Intracranial Neurosurgical Treatment of Occlusive Cerebrovascular Disease

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SUMMARY. Anastomosis of the extracranial superficial temporal artery to the intracranial middle cerebral artery offers an additional source of blood to the cerebral circulation in patients with transient ischemic episodes. Fourteen cases are reported. Two representative cases with three anastomoses demonstrate the use of this technique in occlusion of the internal carotid artery, occlusion of the middle cerebral artery and stenosis of the middle cerebral artery. Indications and contraindications of the procedure are discussed.

SURGICAL TREATMENT of arteriosclerotic occlusive disease of coronary, aortic, and extremity vessels is well known. Until recently, only a few investigators** have applied microsurgical techniques to deal with severe cerebral arteriosclerotic disease, hitherto considered inaccessible or technically out of reach of usual vascular surgical techniques. In 1967, Donaghy and Yasargil6 proposed a new bypass operation to provide additional blood supply to the ischemic brain in patients with carotid occlusion or middle cerebral artery stenosis or occlusion. This procedure entails an anastomosis of the superficial temporal artery (STA) to a cortical branch of the middle cerebral artery (MCA) by way of a 4-cm craniotomy above the ear (fig. 1). We have carried out such a procedure in 13 cases; in another, the anastomosis was made between the occipital branch of the external carotid artery and the middle cerebral artery (table 1). Another shunting procedure (which we have not yet carried out) was designed by Lougheed7 using a saphenous vein graft to carry blood from the common carotid artery in the neck through a craniotomy into the intracranial portion of the internal carotid artery in cases of internal carotid occlusion in the neck, when the superficial temporal artery is too small to permit an effective anastomosis with the middle cerebral branches. In the two patients presented here, transient ischemic attacks (TIAs) led to angiography which disclosed occlusive disease; one patient had stenosis of the right MCA and occlusion of the left MCA; and the other had occlusion of the right internal carotid artery.

Report of Cases

Case I

A 50-year-old man was admitted to the University of Illinois Hospital because of episodes of transient numbness and weakness of the left face, arm, hand, and leg and associated speech difficulty lasting three to four minutes, occurring five times during the month prior to admission. He was not receiving treatment for arterial hypertension known for about one or two years. Examination showed blood pressure of 160/110 mm Hg; the left nasolabial fold was flattened; and the carotid pulses were somewhat decreased. Chemical tests indicated borderline diabetes, and a Frederickson type IV hyperlipoproteinemia. The EEG and brain scan were normal. Carotid and vertebral angiography showed stenosis of the right MCA (fig. 2) and occlusion of the left MCA, with retrograde flow into the distribution of this artery by way of the left posterior cerebral artery (fig. 3).

The patient had an uneventful recovery from a right STA-
MCA anastomosis. Eleven days after this procedure, a left STA-MCA anastomosis was carried out. A week later, bilateral external carotid angiograms via the femoral artery showed patency of both STA-MCA anastomoses, with filling of the entire MCA, particularly on the formerly occluded side (figs. 4 and 5). Postoperative brain scan and blood flow study with 99mTc pertechnetate showed changes due to surgery, and good flow bilaterally. The patient was stable neurologically at the time of discharge, taking Ascriptin 250 mg b.i.d. and diphenylhydantoin (Dilantin) 100 mg t.i.d. on a prophylactic basis.

**Table 1** Summary of Superficial Temporal MCA Anastomoses

<table>
<thead>
<tr>
<th>Date</th>
<th>Pt., age</th>
<th>Diagnosis</th>
<th>Postoperative angiogram, date</th>
<th>Postoperative results</th>
</tr>
</thead>
<tbody>
<tr>
<td>2/24/74</td>
<td>R.K., 64</td>
<td>Occlusion L MCA, stroke-in-</td>
<td>Occluded anastomosis,</td>
<td>R hemiplegia and aphasia; slightly improved</td>
</tr>
<tr>
<td></td>
<td></td>
<td>evolution</td>
<td>3/8/74</td>
<td>Pt. died due to MI three weeks postoperatively</td>
</tr>
<tr>
<td>2/25/74</td>
<td>H.N., 58</td>
<td>TIAs, occlusion R internal carotid</td>
<td>Not done</td>
<td>Asymptomatic, returned to work</td>
</tr>
<tr>
<td>6/17/74</td>
<td>M.A., 63</td>
<td>R cerebral thrombosis (recovered), stenosis</td>
<td>Patent anastomosis, 6/26/74</td>
<td>Essentially unchanged, no more TIAs</td>
</tr>
<tr>
<td></td>
<td></td>
<td>R MCA (recovered), stenosis R MCA</td>
<td>6/26/74 and 8/13/74</td>
<td></td>
</tr>
<tr>
<td>7/11/74</td>
<td>E.E., 51</td>
<td>TIAs, occlusion L internal carotid, mild</td>
<td>Patent anastomosis, 7/18/74</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>cerebellar dysfunction</td>
<td></td>
<td></td>
</tr>
<tr>
<td>8/12/74</td>
<td>N.C., 70*</td>
<td>TIAs, occlusion R internal carotid</td>
<td>Patent anastomosis, 8/19/74</td>
<td>Asymptomatic</td>
</tr>
<tr>
<td>8/13/74</td>
<td>Z.C., 54</td>
<td>TIAs, 90% occlusion L MCA</td>
<td>Occluded anastomosis, 12/20/74</td>
<td>Pt. unchanged as of 7/26/75</td>
</tr>
<tr>
<td>9/17/74</td>
<td>B.M., 54</td>
<td>TIAs, bilateral internal carotid occlusion</td>
<td>Patent anastomosis</td>
<td>Pt. still has intermittent weakness in L leg,</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>otherwise improved</td>
</tr>
<tr>
<td>9/23/74</td>
<td>N.B., 74</td>
<td>TIAs, occlusion L internal carotid occlusion</td>
<td>Occluded (L occipital MCA</td>
<td>Asymptomatic</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>anastomosis), 9/30/74</td>
<td></td>
</tr>
<tr>
<td>10/18/74</td>
<td>S.A., 50*</td>
<td>TIAs, 90% occlusion R carotid artery, occlusion L MCA</td>
<td>Patent bilateral anastomoses, 11/5/74</td>
<td>Asymptomatic, returned to work</td>
</tr>
<tr>
<td>10/31/74</td>
<td>H.L., 67</td>
<td>TIAs, 60% occlusion L internal carotid</td>
<td>Occluded anastomosis, 11/74</td>
<td>Pt. died due to MI one month postoperatively</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Small L cerebral infarct, mild</td>
</tr>
<tr>
<td>11/1/74</td>
<td>B.W., 74</td>
<td>Small L cerebral infarct (dysphasia recovered)</td>
<td>(Pt. had bilateral</td>
<td>essentially unchanged, no more TIAs</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>anastomoses 11/12/74.)</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>One anastomosis patent</td>
<td></td>
</tr>
<tr>
<td>12/16/74</td>
<td>V.L., 67</td>
<td>TIAs, L internal carotid</td>
<td>Occluded anastomosis, 12/30/74</td>
<td>Small L cerebral infarct, residual confusion</td>
</tr>
<tr>
<td>2/4/75</td>
<td>H.E., 37</td>
<td>Occlusion L internal carotid, residual (minimal)</td>
<td>Patent anastomosis, 2/11/75</td>
<td>essentially unchanged, no more neurological</td>
</tr>
<tr>
<td></td>
<td></td>
<td>L hemiparesis</td>
<td></td>
<td>deficit postoperatively, returned to work</td>
</tr>
</tbody>
</table>

*Cases reported in this paper. L = left, R = right, MCA = middle cerebral artery, MI = myocardial infarction, TIAs = transient ischemic attacks.
Case 2

A 70-year-old man was admitted to the Illinois Masonic Medical Center with a history of TIAs over a two-month period; he claimed more than 50 such episodes, consisting of dizziness and fainting-like spells without definite loss of consciousness. Recently, these were accompanied by weakness of the entire left side of the body, lasting for a few seconds to one minute, improving spontaneously. He had had two myocardial infarcts, and his long-standing hypertension was being treated with hydrodiuril. The admission blood pressure was 220/190 mm Hg. Neurological examination was within normal limits. The brain scan ($^{99}$Tc pertechnetate) was normal but the isotope blood flow study showed decreased flow in the right carotid system and hemisphere, with “flip-flop” phenomenon (decreased density on the right initially, increased density on the right at a time when there was normally decreased flow on the left). The EEG was within normal limits. Angiographical studies showed occlusion of the right internal carotid artery in the neck, but the right hemisphere was irrigated by collateral flow from posterior circulation via the posterior communicating and posterior cerebral arteries (fig. 6). Left carotid angiography showed normal flow in the left cerebrum without cross-flow into the right side. The patient underwent right STA-MCA
anastomosis with excellent postoperative course. Postoperative selective external carotid angiogram showed excellent filling of most of the branches of the MCA by way of the anastomosis (fig. 7). Postoperative brain scan and isotope flow study showed evidence of craniotomy and increased flow to the right hemisphere. The patient was discharged on Ascriptin and prophylactic diphenylhydantoin.

Both patients have been followed (eight and ten months respectively) and both have been asymptomatic with subjective feeling of improvement.

Results

Patent anastomoses have been demonstrated in 8 of 14 instances (7 of 13 patients) by angiography and Tc isotope cerebral blood flow study. Six patients are free of TIAs, feel subjectively improved, and have resumed their usual occupations. These have no neurological deficit demonstrable in follow-up examinations. One patient had mild dysphasia postoperatively, from which he recovered and is doing well.

Occlusion of the anastomosis has been demonstrated by angiography in five patients. One patient (R.K.) had a stroke-in-evolution, resembling TIAs — he was evaluated within 24 hours. Embolectomy of the MCA was attempted but was not completed because of cerebral edema. Superficial temporal artery anastomosis was done but the diameter of the superficial temporal artery was smaller than 1 mm and there was evidence of intracranial arteriosclerosis.

The condition of another patient (Z.C.) was unchanged on July 26, 1974, seven months after the anastomosis was demonstrated to be occluded.

One patient (H.N., the only one who did not have follow-up angiography) died three weeks after the operation from a myocardial infarction.

Discussion

The treatment of cerebrovascular insufficiency is summarized in table 2. The medical treatment of the risk factors appears to be of value, especially treatment of hypertension
which appears to have a statistically valid effect in decreasing stroke incidence.6 The role of the vasodilators is still disputed, with anecdotal reports of improvement, but no statistically demonstrable benefit. Acetylsalicylic acid may well help prevent platelet emboli, but at a risk of provoking bleeding from the gastrointestinal tract; the role of anticoagulants is still disputed, especially by those who have treated patients for coronary artery disease and have had nonhemorrhagic strokes appear during this time.

Baker et al.13 concluded that from the standpoint of mortality, long-term anticoagulant therapy plays no beneficial role in the treatment of thrombotic cerebrovascular disease and may be harmful.

Whisnant et al.10 evaluated anticoagulants in the treatment of TIA's and they felt that the effect of anticoagulant therapy can be expected to occur in the first two months following the onset of TIA's. So the difference between the two groups of patients studied occurred in the first two months. There was little difference in the probability of a stroke occurring after that time between the treated and untreated groups.

Endarterectomy of the carotid vessels for stenosis with, presumably, emboli into the cerebral circulation would appear to have merit, but only when the stenosis is accessible. Treatment by endarterectomy or removal of thrombus has been disappointing when the internal carotid is occluded, unless this is quite acute.11,12 Reports of success in up to 42.8% of cases13 must be balanced by the neurological complications during surgery (e.g., 2.93% mortality and 5.86% morbidity in the 613 cases of Bland et al.14). Sundt et al.5 suggest that in addition to TIA's and lesions demonstrated to be in ACCESS to the middle cerebral trunk but also indirect evidence of lesions such as Delayed or retrograde filling of one or several branches of the middle cerebral artery which fill. Relief of TIAs without subsequent strokes has been reported by Austin et al.24 in all the cases with patent anastomoses. In an 18-month follow-up, Tew4 had five of eight functioning anastomoses performed by Yasargil in 1971 reviewed by Chater were patent25; with experience and better selection of patients, the patency rate should improve. Table 4 gives a summary of the indications for this type of operation. Although numerous tests have been designed to diagnose vascular disease in the cervical and cerebral vessels,26 the key test is angiography, perhaps signaled by cerebral flow studies using 133Xe scanning or actual measurements of flow with 123I. Proper diagnosis of the vascular lesion involves three-vessel or four-vessel angiography, although some authorities in the field of cervical carotid endarterectomy feel that for practical purposes bilateral carotid visualization is sufficient.21 Chater et al.8 include as indication for operation multiple cerebrovascular occlusions associated with low perfusion syndromes such as mental deterioration, memory loss, vertigo, ataxia, etc. Very small vessel disease may be identified with new sophisticated techniques. Bradac et al.27 have shown direct stenotic or occlusive lesions of the middle cerebral trunk but also indirect evidence of lesions such as delayed or retrograde filling of one or several branches of the main artery, slow progression of the contrast material, or lack of filling in the area of infarction. Austin et al.4 suggest that in addition to TIA's and lesions demonstrated to be inaccessible to operation in the neck, another indication for anastomosis may be a reduction of 20% to 25% in the cerebral blood flow on the side of the lesion. Speitzer et al.28 have discussed the importance of flow measurements in revascularization procedures. Postoperative angiograms may show not only the patency of anastomosis but also the number of branches of the middle cerebral artery which fill. Relief of TIA's without subsequent strokes has been reported by Austin et al.24 in all the cases with patent anastomoses. In an 18-month follow-up, Tew4 had five of eight functioning anastomoses, but no patient had recurrence of symptoms or infarction. As with carotid endarterectomy and anticoagulant therapy, so with this new anastomosis firm proof of value, but recent reports are encouraging.18

Revascularization procedures, such as have been reported here, appear to hold great promise in delivering more blood to an ischemic or potentially ischemic brain. The difficulties (table 3) lie in identification of candidates for operation, and finding vessels of adequate diameter for maintaining an open anastomosis. Up to now, the patency of the anastomosis is reported to be between 60% and 70% (e.g., 66% of
itself increases survival rate, but improved quality of survival and reduced numbers of TIAa appear to be benefits to be derived. With TIAa, the incidence of infarction, hemorrhage, or death within two to three years has reached 10% to 40% while disabling stroke may rise to 18% to 60% of cases of TIA within one year. There is insufficient information concerning long-term results in cases of cerebral revascularization procedures, but the short-term follow-up indicates enough promise to warrant cautious extension of the procedure and, perhaps, even extension to insufficiency of the vertebral-basilar system when this appears to steal blood from the carotid system.

References


Correction

An error appeared in the second paragraph of the legend of figure 2 (STROKE 7: 289 [May-June] 1976): hypoxemia should be hyperemia.
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