Intracranial Neurosurgical Treatment of Occlusive Cerebrovascular Disease

JOSE L. SALAZAR, M.D., F.A.C.S., ABDUL R. C. AMINE, M.D., AND OSCAR SUGAR, M.D., F.A.C.S.

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 Yasargil 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 Lougheed using a saphenous vein graft applied microsurgical techniques to deal with severe cerebral arteriosclerotic disease, hitherto considered inaccessible or technically out of reach of usual vascular surgical techniques. In 1973, it was designed by Lougheed using a saphenous vein graft.
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-evolution</td>
<td>Occluded anastomosis, 8/19/74</td>
<td>R hemiplegia and aphasia; slightly improved</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>Pt. died due to MI three weeks postoperatively</td>
</tr>
<tr>
<td>6/17/74</td>
<td>M.A., 63</td>
<td>R cerebral thrombosis (recovered), stenosis R MCA</td>
<td>Patent anastomosis, 7/18/74</td>
<td>Asymptomatic, returned to work</td>
</tr>
<tr>
<td>7/11/74</td>
<td>E.E., 51</td>
<td>TIAs, occlusion L internal carotid, mild cerebellar dysfunction</td>
<td>Patent anastomosis, 7/15/74</td>
<td>Essentially unchanged, no more TIAs</td>
</tr>
<tr>
<td>8/12/74</td>
<td>N.C., 70*</td>
<td>TIAs, occlusion R internal carotid</td>
<td>Patent anastomosis, 8/10/74</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, otherwise improved</td>
</tr>
<tr>
<td>9/23/74</td>
<td>N.B., 74</td>
<td>TIAs, occlusion L internal carotid</td>
<td>Occluded (L occipital MCA anastomosis), 9/30/74</td>
<td>Asymptomatic</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, 11/4/74</td>
<td>Pt. died due to MI one month postoperatively</td>
</tr>
<tr>
<td>11/1/74</td>
<td>B.W., 74</td>
<td>Small L cerebral infarct (dysphasia recovered), 90% occlusion L MCA, 60% occlusion L anterior cerebral artery</td>
<td>Occluded anastomosis, (Pt. had bilateral anastomoses 11/12/74.)</td>
<td>Small L cerebral infarct, mild dysphasia — improved</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 one month postoperatively</td>
</tr>
<tr>
<td>2/ 4/75</td>
<td>H.E., 37</td>
<td>Oclusion L internal carotid, residual (minimal) L hemiparesis</td>
<td>Patent anastomosis, 2/11/75</td>
<td>Pt. stable, no further neurological 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 TIA’s 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 (\(^{99m}\) 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 $^{99m}$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 atherosclerosis.

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 (N.B.) is essentially asymptomatic. One (H.L.) died due to myocardial infarction, one month postoperatively. Another (V.L.) is alive, complaining of poor memory. Signs and symptoms of residual infarction have disappeared postoperatively.

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. 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.11 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 TIAs and they felt that the effect of anticoagulant therapy can be expected to occur in the first two months following the onset of TIAs. 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.15 have shown direct stenotic or occlusive lesions of the middle cerebral trunk but also indirect evidence of lesions such as loss, vertigo, ataxia, etc. Very small vessel disease may be identified with new sophisticated techniques. Bradac et al.16 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 TIAs and lesions demonstrated to be 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.16 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. 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itself increases survival rate, but improved quality of survival and reduced numbers of TIAs appear to be benefits to be derived. With TIAs, the incidence of infarction, hemorrhage, or death within two to three years has reached 10% to 40% \(^{9-11}\) 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 verteobasilar 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.
Intracranial neurosurgical treatment of occlusive cerebrovascular disease.
J L Salazar, A R Amine and O Sugar

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