

Microvascular Anastomosis for Cerebral Ischemia in 19 Patients: A Preliminary Report

P. J. MURRAY

SUMMARY: *The general principles of bypass surgery as they affect the cerebral circulation are reviewed. The preliminary results of an extracranial-intracranial bypass operation performed on a group of 19 patients suffering from cerebral ischemia are presented. The results of the surgery compare favorably with those published in the literature.*

RÉSUMÉ: *Les principes généraux de la chirurgie de dérivation, tels qu'ils influencent la circulation cérébrale, sont passés en revue. Nous présentons également les résultats préliminaires d'une opération de dérivation extra-intracrânienne chez 19 patients souffrant d'ischémie cérébrale. Les résultats de cette approche chirurgicale se comparent favorablement à ceux publiés dans la littérature.*

INTRODUCTION

The purpose of this paper is to present the experience gained from the first 19 patients treated by the author using an extracranial-intracranial bypass operation. Although these observations are uncontrolled, they may serve as a useful interim report until the results of the recently established extracranial-intracranial clinical trial become available. As indications for this procedure are published, it is evident that there are two: — 1. the prophylactic operation performed prior to the surgical correction of certain intracranial aneurysms and — 2. the potentially therapeutic operation for the reduction of transient ischemic attacks and the reduction of the incidence of stroke or death in certain forms of occlusive cerebrovascular disease. It is to the latter group that this paper addresses itself.

Because of the morbidity and mortality of cerebrovascular disease neurosurgeons began to develop techniques to bypass obstructive lesions, just as peripheral vascular surgeons had done successfully for many years. Yasargil, in Donaghy's laboratory, applied Jacobson's techniques and with modern microsurgical methods developed the operation of superficial temporal to middle cerebral artery anastomosis. This operation was first performed in October of 1967 (Donaghy and Yasargil, 1968).

Since then a number of neurosurgeons have reported results of microvascular bypass anastomoses (Chater and Popp, 1977) and it is estimated that over a thousand such procedures have been performed. The basic premise for grafting an artery onto the surface of

the brain is that a significant cell mass in the area of relative ischemia is nonfunctional though viable, and an increase in blood supply might return its function.

Indications for this procedure are being suggested with increasing frequency. The basic assumption is that the expected increase in cerebral blood flow, following the bypass operation, improves the condition of the patient and reduces the incidence of subsequent stroke.

Theoretical Considerations and Selection of Patients

It has been shown that the degree of atherosclerosis is proportional to the blood pressure and the radius of the artery involved (Resch et al., 1970). The sites of greatest disease, therefore, are the larger or more proximal vessels and there is relative sparing of parenchymal vasculature in the brain. Cerebrovascular insufficiency may be the result of microemboli from a proximal source or of deficient collateral circulation. Most transient ischemic attacks occur because of a reduction in global or regional cerebral blood flow due to an occlusive process plus a drop in global or regional perfusion pressure or displaced platelet aggregates arising from an ulcerated lesion in the extracranial vessels or on cardiac valves (Editorial: Platelet Embolism, *British Medical Journal*, 1972; Blackwood et al., 1972). The effect of this is to drop regional cerebral blood flow below the level at which symptoms appear. Cerebral symptoms will appear if regional cerebral blood flow is reduced by 30 to 40 percent (Cushman et al., 1972). A more acute drop in cerebral blood flow to more profound levels will produce neuronal destruction or cerebral infarction.

From the Kingston General Hospital & Queens University.

Reprint Requests to: Dr. Patrick J. Murray, Division of Neurological Surgery, Douglas III, Kingston General Hospital, Kingston, Ontario K7L 2V7, Canada.

It is difficult to know how serious a particular transient ischemic attack may be to the individual patient. Of patients who have transient ischemic attacks, however, 20 to 40 percent will have a completed stroke within three to four years from the time of onset of symptoms (Whisnant et al., 1971). It is safe to say that transient ischemic attacks are the single major risk factor in respect to the occurrence of stroke.

Once stroke has occurred therapeutic endeavor is limited to rehabilitative measures. However, standard medical therapy for cerebrovascular insufficiency includes attention to such risk factors as hypertension, diabetes, left ventricular hypertrophy, cigarette smoking, etc.; and the use of anticoagulants or antithrombotic agents once transient ischemic attack has occurred. This therapy, in the reduction of the incidence of stroke or the reduction of the incidence of transient ischemic attacks, is less than satisfactory.

Surgical treatment in cerebrovascular insufficiency is largely confined to carotid endarterectomy in selected patients whose symptoms are appropriate to the lesion found at angiography. A significant number of patients have lesions which are surgically inaccessible. These include carotid artery occlusion, carotid stenosis high in the neck or in its intracranial portion, or a stenosis extracranially associated with a second or tandem lesion intracranially in the carotid artery. A middle cerebral artery occlusion or a stenosis may also be found. It is these patients which are considered for extracranial-intracranial bypass anastomosis.

The clinical or pathophysiological effect of an occlusive lesion depends to a certain extent on the effectiveness of collateral supply (Feindel et al., 1975). In the brain this occurs from the opposite hemisphere through the anterior communicating artery and from the posterior circulation through the posterior communicating artery or from the external carotid circulation via the ophthalmic artery. It may also arise from the leptomeningeal arteries or epicerebral vessels. In cases of middle cerebral artery stenosis, or as

has been shown by Feindel et al. (1975), in the case of an absent anterior communicating artery, the collateral flow arises mainly from epicerebral vessels. It would appear, therefore, that the basic premise of increasing the collateral blood supply to areas of potentially ischemic brain rests on a sound physiological basis.

Technique

A broad based flap is elevated and the temporalis muscle is divided. A small craniectomy is performed over the temporal region based on the Sylvian fissure. Using the operating microscope, a cortical artery of 1 mm in diameter or larger is selected and dissected free of its arachnoid. A rubber dam is placed under the vessel to protect the brain from subsequent manipulations with sharp instruments. The superficial temporal artery is then dissected free from its bed and the stoma prepared. It is then anastomosed end to side onto the cortical vessel (Fig. 1). The dura is closed leaving a gap for the superficial temporal artery and the temporalis muscle is closed over the craniectomy. The scalp is closed in layers. I prefer interrupted suturing

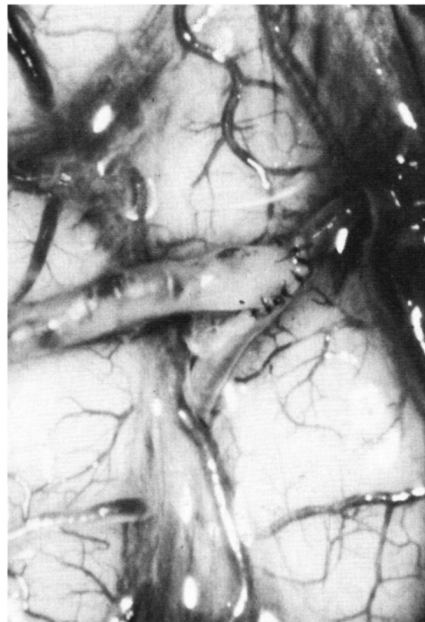


Figure 1—Superficial temporal artery anastomosed end to side to a cortical branch of the middle cerebral artery (Case J.D.).

TABLE 1

Distribution by age and sex, of 19 patients who underwent cerebral bypass surgery

Number of Cases	19
Men	14
Women	5
One Woman had a bilateral procedure	
Average Age	56.5 years
Range of Age	33 - 60 years

with 10-0 monofilament nylon using a BV-8 needle (Ethicon).

The patients are then treated with 500 cc's of low molecular weight dextran given slowly intravenously over a 24 hour period for each of three days.

Clinical Presentation

Twenty extracranial-intracranial bypass operations were performed on 19 patients. One had a bilateral procedure. There were 14 men and 5 women. They varied in age from 33 to 66 years, with a mean age of 56.6 years (Table 1).

The most common clinical presentation (Table 2) was the transient ischemic attack (80 percent). Three patients had a reversible ischemic neurologic deficit (15 percent) and one had a completed stroke of moderate severity.

A wide range of angiographic lesions were found, reflecting the diffuse nature of the disease process (Table 3). Three patients presenting with objective symptoms and signs of cerebrovascular insufficiency showed dementia as well.

Indications for Surgery

Of the 19 patients operated upon, 17 had a preoperative trial period on anticoagulants or platelet inhibitors. Most were being treated with antihypertensive medication. None had serious myocardial or pulmonary disease. All of the patients had a history of transient ischemic attack, or reversible ischemic neurologic deficit, and most of these had occurred while taking platelet inhibiting medication (aspirin or disulfenpyrazone). All patients had an occlusive lesion, angiographically demonstrated, otherwise surgically inaccessible. No patient had significant

TABLE 11

Clinical presentation of 19 patients who underwent cerebral bypass surgery

	Number	Percent
T.I.A.	15	80%
R.I.N.D.	3	15%
Stroke	1	5%

Definitions

- T.I.A. —Neurologic dysfunction lasting less than 24 hours, with complete recovery.
- R.I.N.D. —Reversible ischemic neurologic deficit is a transient cerebral dysfunction lasting longer than 24 hours, but with complete recovery.
- Stroke —A completed stroke is a fixed neurologic deficit which may be mild, moderate or severe, depending on the depth of neurologic impairment.

cerebral small vessel disease, and all had a superficial temporal artery of adequate size.

RESULTS

Of the 19 patients operated on, one died of a myocardial infarction 30

days after surgery; one patient who presented with a potentially reversible neurologic deficit went on to develop a moderate left hemiplegia, and one patient who presented with transient ischemic attacks had three attacks while in hospital in the immediate postoperative period. Of the

other 16 patients all have remained asymptomatic since surgery.

Followup

The period of followup is between two months (the most recent operation) and two years. Table 4 shows that of the 19 patients 12 have had

TABLE 111

Result of 20 angiographic examinations of 19 patients who subsequently had bypass surgery

Bilateral Carotid Occlusion	1
Internal Carotid Occlusion at the bifurcation	5
Internal Carotid Stenosis at the siphon	2
Tandem Stenoses	2
Middle Cerebral Artery Occlusion	1
Middle Cerebral Artery Stenosis	4
Multiple Vessel Occlusion or Stenosis	5

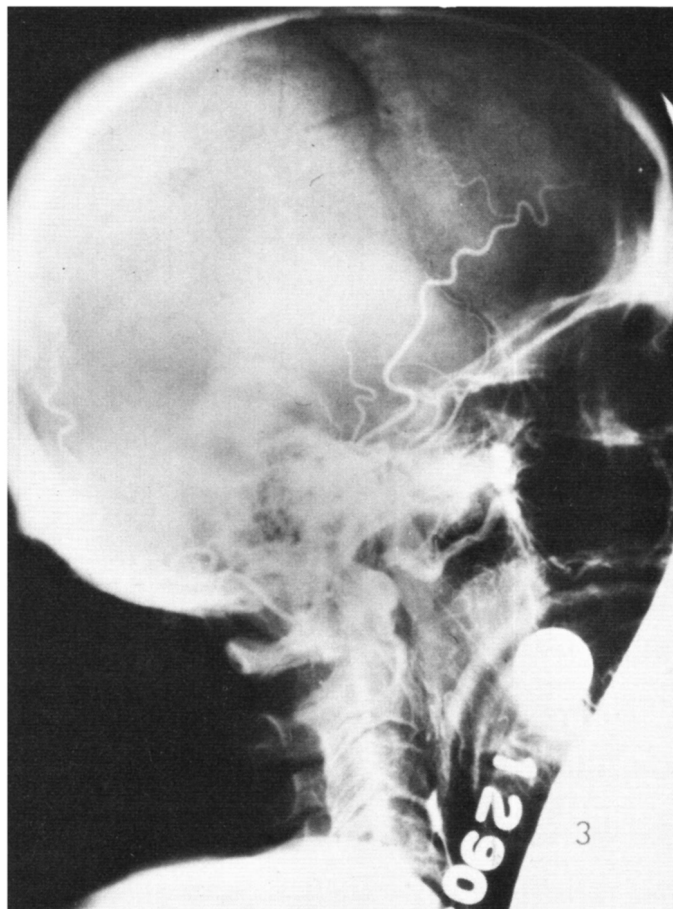


Figure 2—Left internal carotid artery occlusion. Superficial temporal artery is seen (Case J.D.).

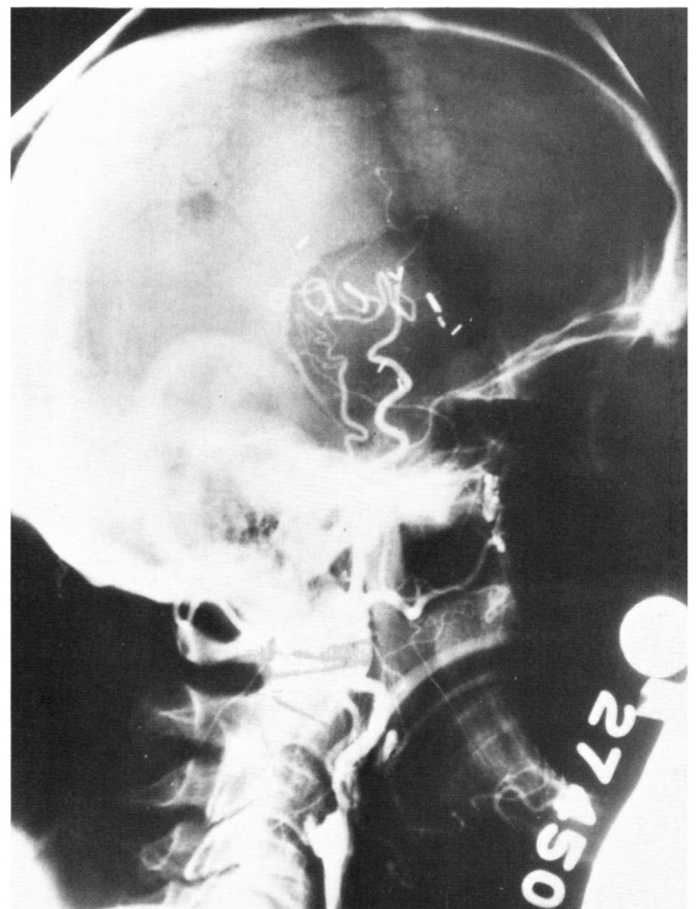


Figure 3—Superficial temporal to middle cerebral artery anastomosis two months postoperatively (Case J.D.).

postoperative angiography and all of these anastomoses are patent. The doppler ultrasound method of examination has shown good flow and an intracerebral direction of flow in the superficial temporal artery.

In Table 5, degrees of relief from transient ischemic attack postoperatively are graded according to Austin

et al. (1976). Of the 19 patients 16 have had no further episodes of transient cerebral ischemia and are classified as excellent. One patient had a series of three transient ischemic attacks while in hospital in the immediate postoperative period and is graded as good. This patient, however, has not had any further

transient ischemic attacks since discharge. There were two poor results, ten percent of the total.

Complications

The complications following this procedure were few and not serious. (Table 6). A permanent increase in neurologic deficit was seen in one

TABLE IV

This table summarizes the clinical presentation and angiographic findings as well as the type of bypass anastomosis performed and condition on followup examination of 19 patients who had bypass anastomoses performed.

PATIENT	SYMPTOM	ANGIOGRAPHY	OPERATION	FOLLOW-UP
S.B. F. 66 yrs	T.I.A.	L car. occl. absent. ant. comm. art	L STA-MCA Sept. 1975	Died
M.P. M. 60 yrs	T.I.A.	R car. occl.	R. STA-MCA Oct. 1975	Asymptomatic
M.G. F. 63 yrs	R.I.N.D.	R int. car. art. stenosis in tandem	R STA-MCA 14 Jun. 1976	Fixed left hemiparesis
M.B. F. 44 yrs	stroke mod.	L MCA occl.	L STA-MCA 1 Jul. 1976	Some improvement
C.R. M. 55 yrs	T.I.A.	R car. occl. L car. stenosis	R STA-MCA 7 May 1976	Asymptomatic
H.G. M. 52 years	T.I.A.	L car. stenosis at siphon	R STA-MCA 17 May 1976	3 TIA's, then asymptomatic
H.S. M. 66 yrs	T.I.A.	R car. occl. L car. stenosis	R STA-MCA 9 Feb. 1976	Asymptomatic
W.M. M 49 yrs	T.I.A.	R car. occl. L car. stenosis	R STA-MCA 8 Mar. 1976	Asymptomatic
S.S. F. 33 yrs	R.I.N.D.	bilat. MCA stenosis	R STA-MCA L STA-MCA 17 Mar 77 13 Apr 77	Asymptomatic
G.C. M 66 yrs	T.I.A.	L car. stenosis in tandem	L STA-MCA 10 Apr. 1977	Asymptomatic
M.S. F. 54 yrs	T.I.A.	L car. stenosis in tandem	L STA-MCA 4 Mar. 1977	Asymptomatic
J.D. M 62 yrs	T.I.A.	L car. occl. R car. stenosis	L STA-MCA 23 Mar. 1977	Asymptomatic
E.H. M. 64 yrs	T.I.A. Dementia	R car. occl. L car. stenosis	R STA-MCA 6 May 1977	Asymptomatic
P.W. F. 63 yrs	T.I.A. Dementia	L car. occl. absnet, ant. comm. art.	R STA-MCA 15 Apr. 1977	Asymptomatic
W.M. M. 51 yrs	T.I.A. Dementia	L car. occl.	L STA-MCA 25 May 1977	Asymptomatic
K.H. M. 53 yrs	T.I.A.	L Car. occl.	L STA-MCA 8 Jun. 1977	Asymptomatic
S.K. M. 45 yrs	T.I.A.	L MCA stenosis	L STA-MCA 8 Jul. 1977	Asymptomatic
P.B. M. 64 yrs	T.I.A.	L MCA stenosis	L STA-MCA 6 Aug. 1977	Asymptomatic

Abbreviations: 1. T.I.A.
R.I.N.D. } as in Table II
Stroke

2. L or R car. occl. = left or right internal carotid artery occlusion

3. L or R STA-MCA = left or right superficial temporal artery to middle cerebral artery branch anastomosis.

TABLE V

The clinical results of 19 patients who had cerebral bypass surgery

Excellent (a total relief from transient ischemic attacks)	16	80%
Good (1 to 8 transient ischemic attacks post-operatively)	1	5%
Fair (greater than 8 transient ischemic attacks but fewer than were occurring preoperatively)	0	0%
Poor (no change or worsening in the neurological state)	2	10%

TABLE VI

The surgical complications encountered in 20 extracranial-intracranial bypass anastomoses performed on 19 patients

Mortality	1
Permanent Increase in Neurological Deficit	1
Marginal Scalp Ischemia	2
Intracranial Infection	1

patient with a fluctuating mild hemisensory loss. He progressed, in the first postoperative week, to a moderate hemiparesis. The marginal ischemia seen was not unexpected as a part of this procedure involves devascularization of the scalp in preparing the donor vessel. Widening the base of the scalp flap to include the posterior auricular artery has, I hope, decreased the incidence of that problem.

CASE HISTORY

J.D.
Mr. J. D., 52 years of age, was treated with Hydrochlorothiazide daily for known hypertension. He was referred because of a history of transient ischemic attacks, characterized by paresthesia and weakness of the right arm and hand. These

occurred three to six times per week during the previous six months. Doppler ultrasound and arteriography showed a complete occlusion of the left internal carotid artery at the bifurcation and a severe stenosis of the right internal carotid artery (Fig. 2).

A left extracranial-intracranial bypass anastomosis was performed. Postoperatively he has been symptom free. He was admitted two months postoperatively for followup angiography. This showed patency of the anastomosis and irrigation of many of the branches of the middle cerebral tree (Fig. 3).

CONCLUSION

These results, through preliminary and in a small group of patients, compare favorably with those of Austin et al. (1976), Gratzl et al., (1976), and Reichman et al., (1976).

More time will be needed to evaluate morbidity and mortality rates. A greater number of patients and a prolonged followup period is required to evaluate the effect this operation might have on the incidence of transient ischemic attacks, on the incidence of subsequent stroke, and on the ability of the patients to return to work or their previous recreations. While encouraged by this uncontrolled experience and by that of others, it is important to study this procedure with a well controlled clinical trial. A large multicenter trial has recently been launched, and it is hoped that this trial will answer many of the questions which remain.

ACKNOWLEDGEMENTS

I wish to express my deep appreciation to W. Feindel of the Montreal Neurological Institute. The first two cases were performed with able assistance of Dr. C. Dila of the Montreal Neurological Institute. I would like to thank Dr. Henry Dinsdale for reviewing this paper, and wish also to thank the other physicians in the neighborhood of Kingston who have allowed me the honour the looking after their patients.

REFERENCES

AUSTIN, G., LAFFIN, D. and HAYWARD, W. (1976). Bypass Anastomosis for Cerebral Ischemia. Current Controversies in Neurosurgery, ed. Morley, W. B. Saunders.

AUSTIN, G., HAYWARD, W., LAFFIN, D. (1976). Modification of Cerebral Ischemia by Microsurgical Intracranial Anastomosis. Ch 22 In: Microsurgical Anastomoses for Cerebral Ischemia. C. C. Thomas, Springfield.

BLACKWOOD, W., HALLPIKE, J. F., KOCEN, R. S., and MAIR, W. G. (1972). Atheromatous Disease of the Carotid Arterial System and Embolism from the Heart in Cerebral Infarction. Brain 92: 897-910.

CHATER, N., and POPP, J. (1977) Microsurgical Vascular Bypass for Occlusive Cerebrovascular Disease Current Status. Advances in Neurology, Vol. 16, ed. Thompson, R. A., Green, J. R. Raven Press.

CUSHMAN, A., ROSTAN, H., MARVIN, S., LAFFIN, D., HAYWARD, W., CIESEL, C., and AUSTIN G., (1972). Abstract. Association of American College of Surgeons, San Francisco.

DONAGHY, P. F., and YASARGIL, M. G. (1968). Extracranial Blood Flow Diversion. A.A.N.S., Chicago.

Editorial. Platelet Embolism (1972). B.M.J. 3: 67.

GRATZL, O., SCHMIEDECK, P., SPETZLAR, R., STEINHOFF, H., and MARGUTH, F. (1976). Clinical Experience with Extracranial-Intracranial Arterial Anastomosis in 65 cases. J.N.S., Vol. 44.

FEINDEL, W., GARRETSON, H., MURRAY, P. J., YAMAMOTO, L., and HODG, C. P. (1975). Collateral Flow in the Cerebral Microcirculation. Recent Evidence to Extend the Views of Thomas Willis. (Abstract) Fifth Congress of European Association of Neurological Surgeons, Oxford.

REICHMAN, O. H. (1976). Arteriographic Flow Pattern Following Superficial Temporal Artery — Cortical Middle Cerebral Artery Anastomosis. Ch 21 In: Microsurgical Anastomosis for Cerebral Ischemia, ed. Austin, G. C. C. Thomas, Springfield.

RESCH, J. A., LOEWENSON, R. B., and BAKER, A. B. (1970). Physical Factors in the Pathogenesis of Atherosclerosis. Stroke 1: 77.

WHISNANT, J. P., MATSUMOTO, N., and ELVEBACK, L. R. (1971). The Effect of Anticoagulant Therapy on the Prognosis of Patients with Transient Cerebral Ischemic Attacks in a Community. Rochester, Minnesota, 1955-1969. Mayo Clinic Proceedings 48: 844-848.