CURRENT METHODS IN THE SURGICAL TREATMENT OF STROKES

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SYNOPSIS

The majority of strokes are preventable by the adequate control of hypertension and prompt recognition and treatment of transient ischemic attacks.

The indications and current methods of surgical treatment are reviewed with illustrative cases.

INTRODUCTION

Cerebrovascular disease remains the fourth largest cause of death and disability in Singapore. In 1977 alone, the Departments of Neurosurgery and Neurology admitted 179 cases with various cerebrovascular diseases out of 1613 admissions (or 11.1%) which include head and spinal trauma, infections, neoplasms, degenerative, congenital and demyelinating diseases. The basic pathological lesion is the atherosclerotic plaque which may gradually occlude a blood vessel, send off platelet or cholesterol emboli or erode the wall of an artery or an existing berry aneurysm to cause cerebral or subarachnoid haemorrhage.

The rationale of surgery is to improve cerebral blood flow in occlusive disease by reconstruction of the stenosed blood vessels; making new collaterals; removing the source of emboli where present and removing space occupying blood clots and further sources of haemorrhage where feasible.

THE NATURAL HISTORY OF OCCLUSIVE CEREBROVASCULAR DISEASE

The transient ischemic attack is the harbinger of a major stroke in about 30% of patients within 4 years. In those with a proven stenotic lesion in the carotid territory, 45% will have a major stroke within 3 years. (Ziegler D K et al 1973) Ischemic attacks in the carotid territory usually present with transient hemiparesis, hemiparesthesia, hypoaesthesia; mono-ocular blindness or dysphasia. Vertebral basilar ischemia although less common than carotid ischemia usually present with diplopia, vertigo, ataxia, dysarthria, drop

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attacks or bilateral weakness or sensory deficits. The risk of transient vertebral basilar isohemia developing into infarcts is now believed only to be a little less than that for carotid territory ischemia. Such high risks of a major stroke developing after transient ischemic attacks make it imperative that all such symptoms should be investigated forthwith. Patients should be hospitalised and angiography performed via a transfemoral catheter technique as soon as possible. Medical treatment in the form of "Anti-platelet" agents such as soluble Asprin 300 mgs tid and (Persantin®) 500 — 100 mgs tid are usually given as soon as the diagnosis is made. These are well tolerated except in patients with peptic ulcers and has been shown to reduce thrombosis without the attendant risk of haemorrhage. Standard anti-coagulants such as heparin I.V. should however be considered if the transient ischemic attacks are very frequent, especially if there is a delay between diagnosis, investigation and definitive therapy. In cases of evolving or progressing stroke, prompt heparinization is usually recommended, barring strong contraindications.

REQUIREMENTS OF ANGIOGRAPHY

As nearly 50% of vascular occlusion occur in the extracranial vessels, all the neck vessels should be studied in detail with particular attention to the common carotid artery bifurcations. A full intracranial 4 vessel study is completed at the same time. If both neck and intracranial studies are negative, the aortic origins of the great vessels should then be studied subsequently at a separate occasion to avoid excessive dye administration.

INDICATIONS FOR CAROTID ENDARTERECTOMY

Carotid endarterectomy should be considered in all cases of transient ischemia in which there is a stenosis of over 60% of the vessel diameter or where an obviously ulcerative plaque is seen to be consistent as a source of emboli. Relative contraindications include cases of diffuse and severe atherosclerosis of all the vessels, and those with marked hypertension or serious concomitant illnesses commonly myocardial ischemia and severe diabetes mellitis.

ILLUSTRATIVE CASE

A right-handed 43 year old male presented in May 1976 with persistent attacks of right sided transient hemiparesis and hemianesthesia. He had an associated mild hypertension of 160/100 mm Hg and hypercholesterolemia of 210 mg/100 ml. He was hospitalised, treated with methyldopa 125 mg tid, Clofibrate and persantin 75 mgs tid. His attacks persisted and pan angiography under the cover of I.V. Dextran 40 solution was performed. This revealed an ulcerative plaque at the left common carotid bifurcation (fig. 1a).

Endarterectomy was done in July 1976. Since then he has remained asymptomatic and a repeat study in February 1978 showed satisfactory patency (fig. 1b).

In the acute completed stroke, thrombectomy or endarterectomy has only a limited chance of success if treatment can be given within 6-8 hours of the onset of the stroke. In practical terms, this is usually not feasible. The poor results of such surgery has been ascribed to the conversion of an ischaemic infarct into a hemorrhagic one but Fischer (1977) in his pathological studies believe that deterioration in these cases is usually due to massive embolism caused by surgical manipulation of very soft and friable thrombi during operation.

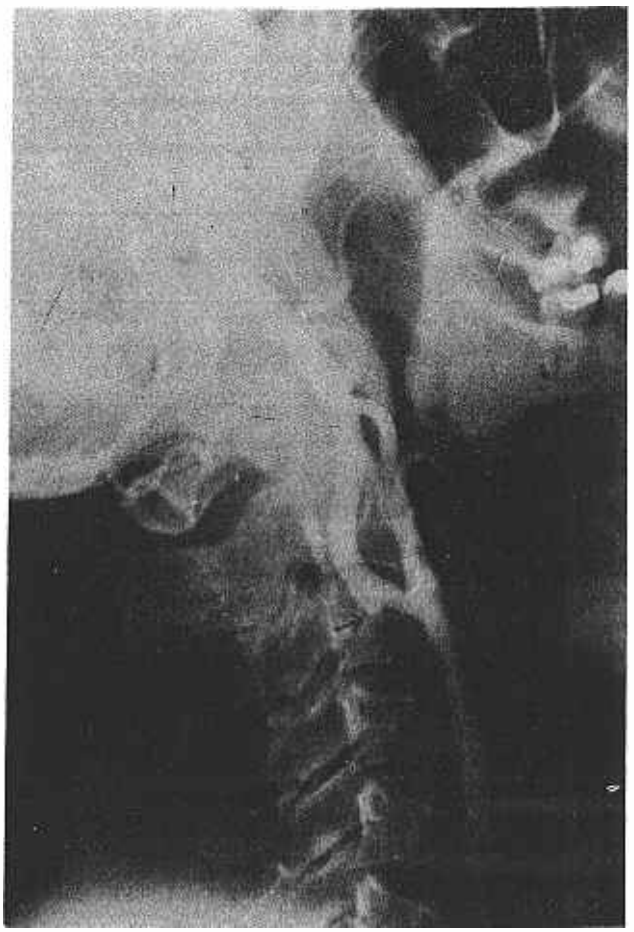


Fig. 1a : Lt. carotid angiogram showing atheromatous plaque at the bifurcation.

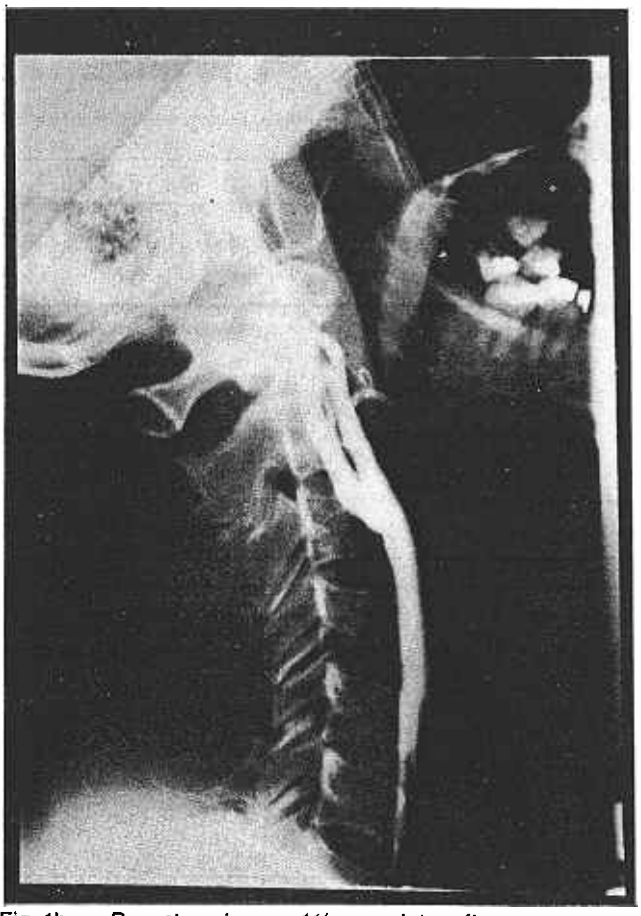


Fig. 1b : Repeat angiogram 1½ years later after endarterectomy.

SURGERY FOR INTRACRANIAL VASCULAR OCCLUSIONS

In intracranial carotid occlusions, the lesions are usually sited at the terminal portion within the carotid canal where access is almost impossible, at the bifurcation of the anterior and middle cerebral arteries or at the trifurcation of the middle cerebral artery. Endarterectomy at the latter site has been performed sporadically but is fraught with significant mortality and morbidity. (Lougheed 1965).

THE EXTRACRANIAL — INTRACRANIAL MICROVASCULAR BYPASS OPERATION

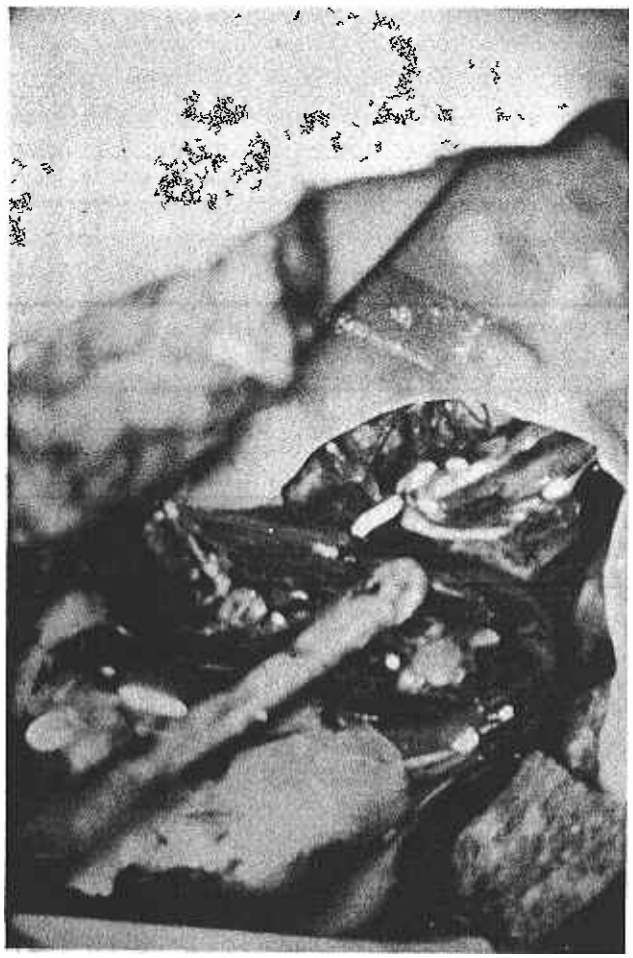
Henschen (1944) attempted to improve revascularisation of the brain in a stroke patient by transplanting a pedicle of temporalis muscle onto the ischemic brain, a procedure perhaps not unlike a Vineberg procedure. By 1966 Donaghy had perfected the microvascular technique experimentally in dogs and in 1967, Yasargil and Donaghy performed the first operation in patients successfully by anastomosing the superficial temporal artery onto a cortical branch of the middle cerebral artery. Over a thousand cases has since been treated by this operation in the west, chiefly in the United States. The consensus of opinion is that it is basically a low risk procedure with extremely encouraging results in well selected cases and when performed by trained microvascular neurosurgeons.

INDICATIONS FOR THE EC-IC BYPASS OPERATION

According to Sundt et al (1976), the definite indications are those patients with intracranial or middle cerebral occlusions with transient ischemic attacks, or in the rarer presentation of a progressive or evolving stroke. Other indications include prophylaxis against iatrogenic stroke as in the trapping or clipping of a complex middle cerebral artery aneurysm (fig. 35), or in the dissection of benign basal tumours that envelope these important blood vessels. Its role in the completed stroke with a deficit is less clear. In younger patients with a completed stroke, whose clinical improvement appear slow and a computer tomographic brain scan may show only a small infarct, angiography may reveal poor collateral vascularisation. In such cases, it seems possible that the microvascular bypass procedure may improve neurological function by furnishing that extra volume of blood required by ischemic but structurally intact neurons around the central infarct (case V). This possibility will require further research and accumulated case studies before strong recommendations are made. Eight patients have had this procedure performed in the Departments of Neurosurgery, Neurology and Radiology at the Tan Tock Seng Hospital since 1975. All patients till date have been asymptomatic. Four cases on re-study show extensive perfusion of the ischemic hemisphere via the anastomoses, three show patency but limited areas of perfusion and one did not consent to check angiography. These cases will be fully documented in a later publication.

ILLUSTRATIVE CASES : I

A male age 38 had a completed right hemiparesis in February 1975. He recovered after 2 months but began to have persistent right-sided TIA's despite medication and was unable to ambulate without an attack. Microvascular bypass was done in June 1975. Fig. 2 illustrate the procedure. Since this time, he has had no other attacks and has been working since. Consent was not obtained for check angiography to record patency, but post-operative serial EEGs has shown improvement (if this could be taken to indicate improved cortical function due to improved blood supply).



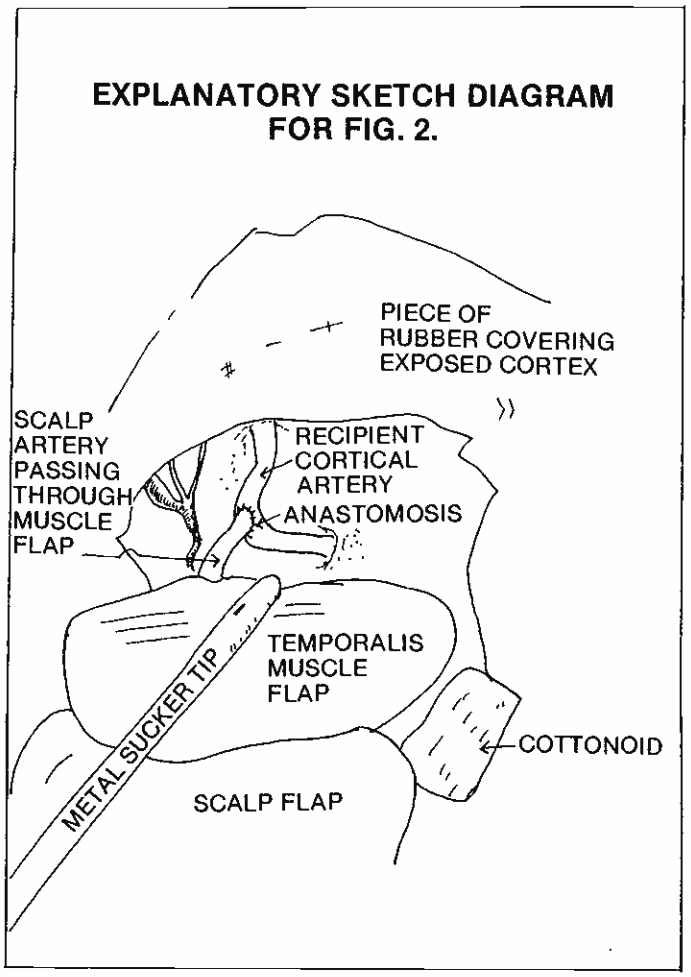


Fig. 2 : This microphotograph show the completed anastomosis between the superficial temporal artery and cortical branch of the middle cerebral. An explanatory diagram clarifies the details.

ILLUSTRATIVE CASE : III

A male aged 28 suffered subarachnoid haemorrhage due to a large left middle cerebral artery aneurysm in January 1978. Angiography showed a complex aneurysm with Rolandic branches arising from a broad neck (Fig 3 a). Before a clip was applied to occlude the aneurysmal sac, microvascular bypass was first performed as a precautionary measure. This was fortunate as although the first author thought he could spare the Rolandic vessels, the clip obviously kinked a major vessel. Figs. 3 b and c show the vascularisation by the bypass graft without which a hemiplegia would definitely have occurred.

ILLUSTRATIVE CASE : IV

A male aged 38 suffered dysphasia and right hemiparesis in October 1974. He recovered but developed frequent attacks of right-sided TIA's and right-sided focal epilepsy which were controllable with anticonvulsants and antiplatelet agents. Subsequently the TIA's became worse and repeated angiography show a worsening collateral blood supply. By March 1978 he had attacks of severe left mono-ocular blindness and daily ischemic attacks. Microvascular EC-IC bypass was done in April 1978 after which he was asymptomatic for 4 weeks. The graft later occluded due to circatrisation of the scalp flap causing a poorly anchored bone flap to become displaced, hence dragging in the bypass graft, kinking this over the bone edge. His symptoms reappeared immediately. A revision of the bypass was performed in June 1978 and again his symptoms disappeared immediately after operation. Figs 4a and b show the pre-operative and post-operative angiograms. This case illustrates the close correlation of the symptoms with the function of the anastomosis indicating that the blood volume delivered though this little scalp vessel is probably significant in relation to the ischemic brain it supplies.

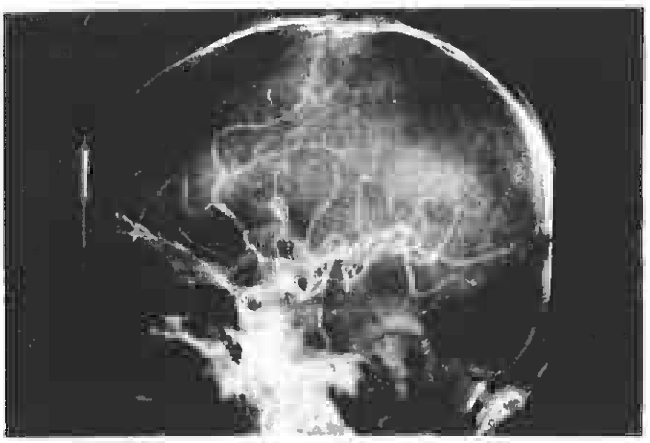


Fig. 3a : Lt carotid angiogram showing complex middle cerebral artery aneurysm with rolandic branches arising from the sac.

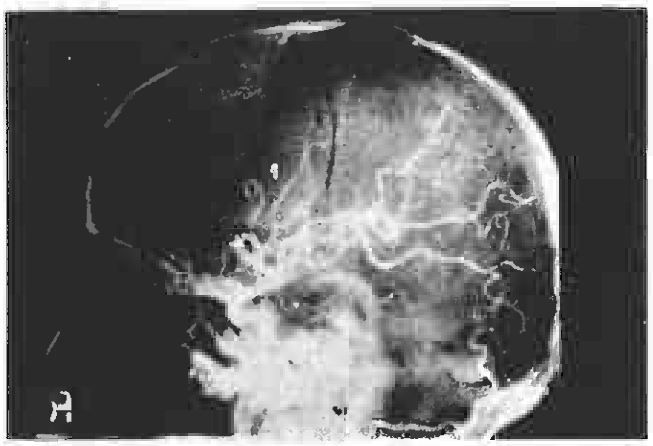


Fig. 3b : Post-operative angiogram showing clip on aneurysm but part of the middle cerebral tree not filling.

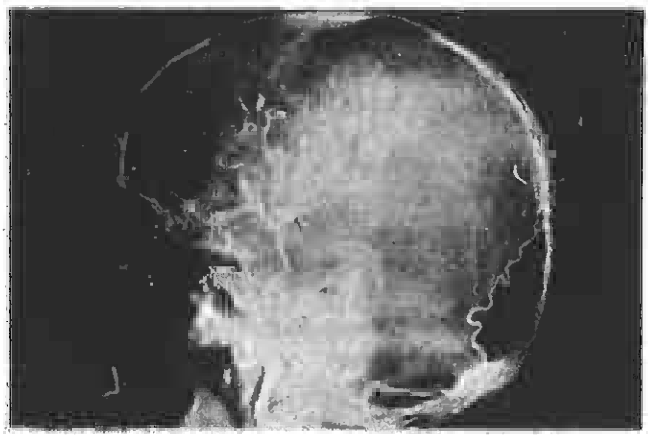


Fig. 3c : External carotid study showing filling of rolandic branches by the EC-IC anastomosis.

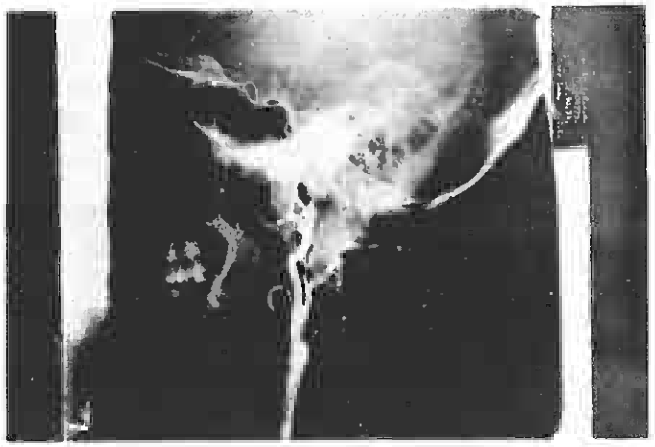
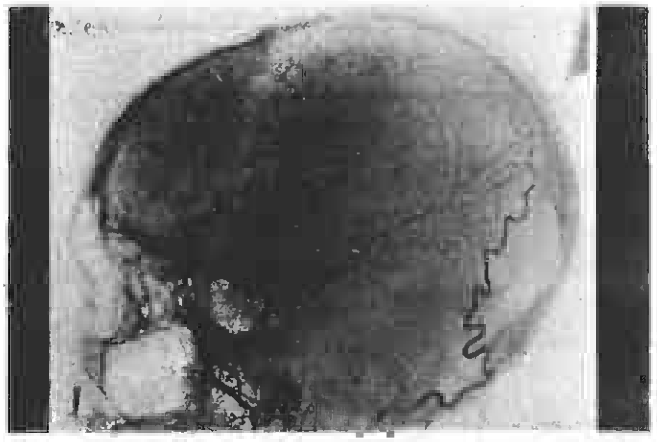
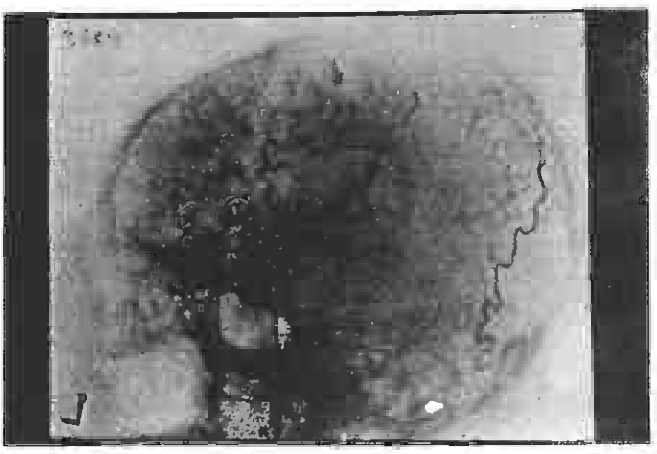


Fig. 4a : Preoperative Lt carotid angiogram showing the total Lt. Internal carotid occlusion in the neck.





Figs. 4b, 4c : Post-operative Lt external carotid study showing extensive perfusion of the left hemisphere by the EC-IC bypass.

ILLUSTRATIVE CASE : V

A male 37 suffered aphasia with right hemiplegia in April 1978. After 3 weeks he improved and was able to walk painfully slowly with a hemiplegic limp but unable to move his right upper limb or utter more than 2-3 coherent words. A CT Scan showed a small infarct in the capsular area but angiography revealed left middle cerebral artery occlusion with poor collaterals from the left anterior cerebral artery, the origin of which was also developing atheromatous occlusion (figs 5a). Because of this danger, bypass was recommended to prevent a further stroke rather than to improve function. This was duly done in early June 1978. 36 hours later, the patient, his family and doctors were pleasantly surprised by a sudden improvement in his speech and the strength of his limbs. He could now speak in sentences howbeit haltingly, and could lift his right upper limb above his head, actions he could not perform previously. Post-operative selective angiography showed extensive perfusion of the left hemisphere by the anastomosis (fig. 5b).

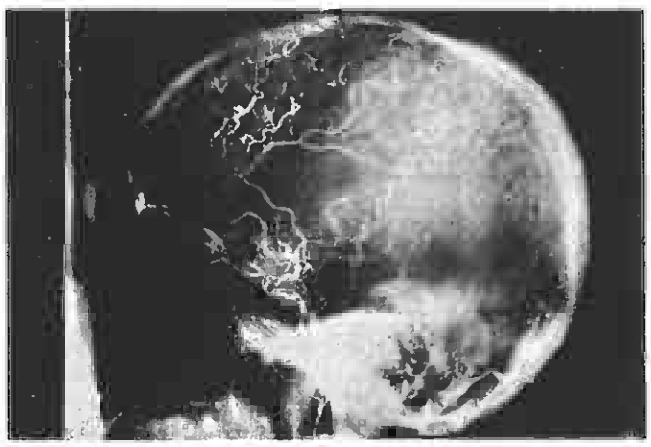
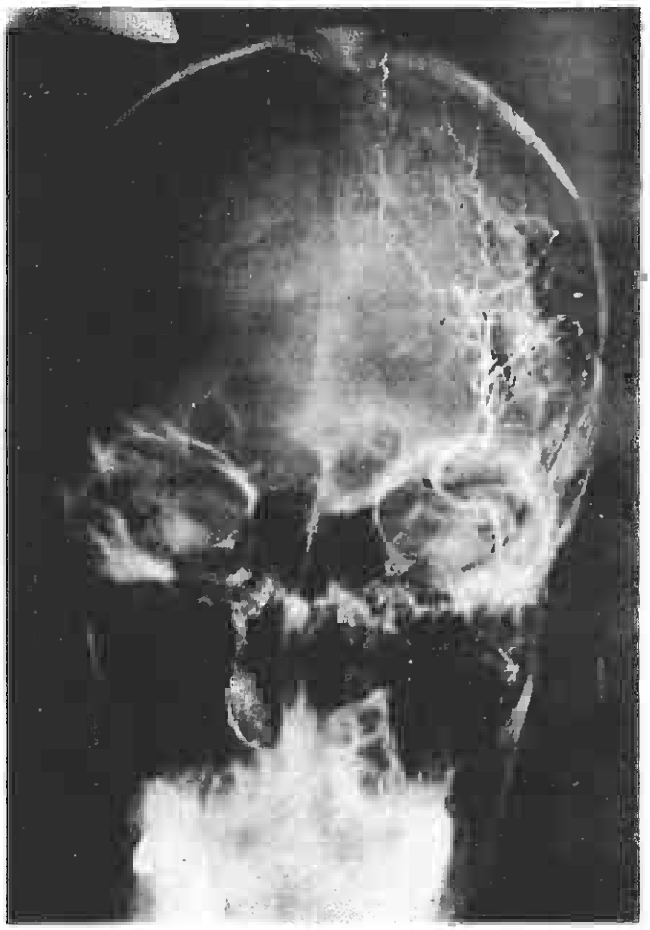


Fig. 5a : Pre-operative Lt. carotid angiogram showing occlusion of the left middle cerebral artery and stenosis of the anterior cerebral artery.



Figs. 5b, 5c : Post-operative Lt. external carotid study showing good perfusion of the left hemisphere via the bypass.

DISCUSSION

This series although small, is consistent with the experience of larger series (Chater 1976, Sundt et al 1976) that the procedure can yield significantly beneficial results with little risks involved. The long term benefits as to whether it can prolong life and prevent stroke over a 10-20 year period will require long term correlative studies as already in progress. (Barnett et al 1977) Austin and his co workers (1976) have provided clear scientific evidence that this operation make a significant contribution to cerebral blood flow and cortical oxidative metabolism as evidenced by the improved redox level of cytochrome a, a3 and cortical PO2 in the human brain during operation when the bypass graft was opened after temporary occlusion (fig 6) Cerebral blood flow studies by the intravenous xenon method has also been shown by Austin (1975) to have significant improvement. Recently, the bypass procedure has been applied to patients with vertebral basilar ischemia by means of an occipital to posterior inferior cerebellar artery anastomosis (Sundt et al 1978) and the results appear encouraging.

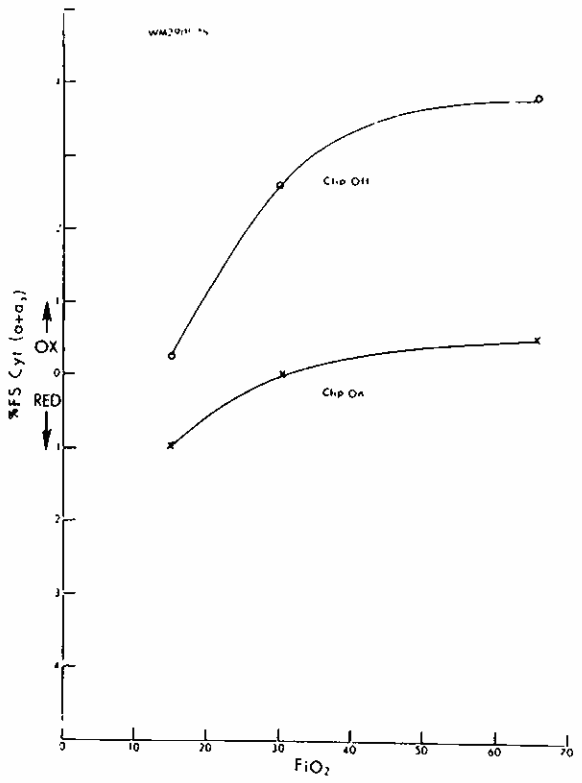


Fig. 6 : Dr Austin's graph showing in brain Red-ox levels of cytochrome a, a3 after bypass in human patients, when a temporary clip is applied to the bypass graft and when this is removed.

SURGERY FOR "HEMORRHAGIC" STROKES

In hypertensive and older patients, spontaneous intracerebral hemorrhage is the second commonest form of stroke. Computerised tomographic brain scanning has made diagnosis easier but clinical management remain essentially the same. Evacuation of the clot will benefit only those patients who are not devastated by the haemorrhage. Patients who are deeply comatose, with decerebrate posturing, stertorous or cheyne-stroke breathing will gain little by surgery. Those who are just drowsy and have a stable or slowly progressing deficit will benefit from surgery. Fig. 7 illustrate the value of CT scanning in a drowsy but stable patient, revealing a striatal haematoma which was duly evacuated.

The rupture of aneurysm and arteriovenous malformation may lead to intracerebral haematomas as well as subarachnoid haemorrhage. They could be considered as forms of stroke. The treatment of these are already well documented. With improved neuroanaesthesia, radiology, microsurgical instrumentation and techniques, most are treatable. An aneurysm is best treated by directly occluding the neck of the aneurysmal sac with a suitable clip, as illustrated in figs 8a and b; or re-inforced by wrapping with a plastic or cotton material when clipping is not possible. Most arteriovenous malformation are excisable save those that ramify the basal ganglia and brain stem.

CONCLUSION

A review of current surgical methods available in the treatment of "strokes" is given with illustrated cases. Prophylaxis remain the mainstay of treatment against major stroke, with medical treatment such as the correction of hypertension, of hyperlipidemia or of a hypercoagulopathic status, a life long process, Surgery is useful only to a minority of patients, the selection of which require a team approach consisting of the referring physician, neurologists, radiologists and neurosurgeons.

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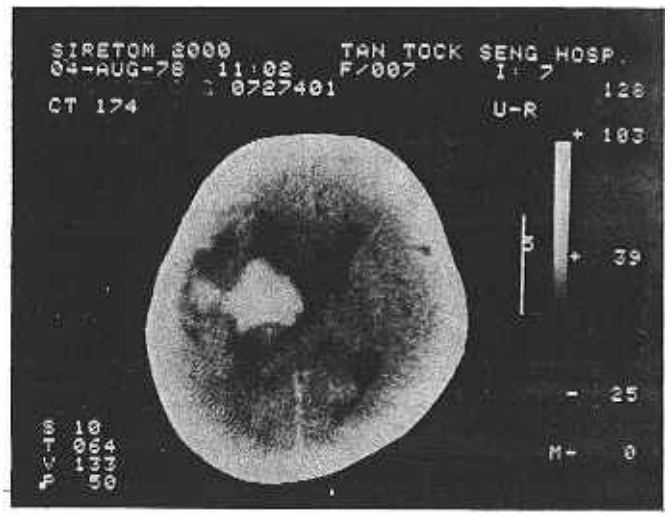

Fig. 7 : CT scan illustrating intracerebral haematoma in the striatal region.

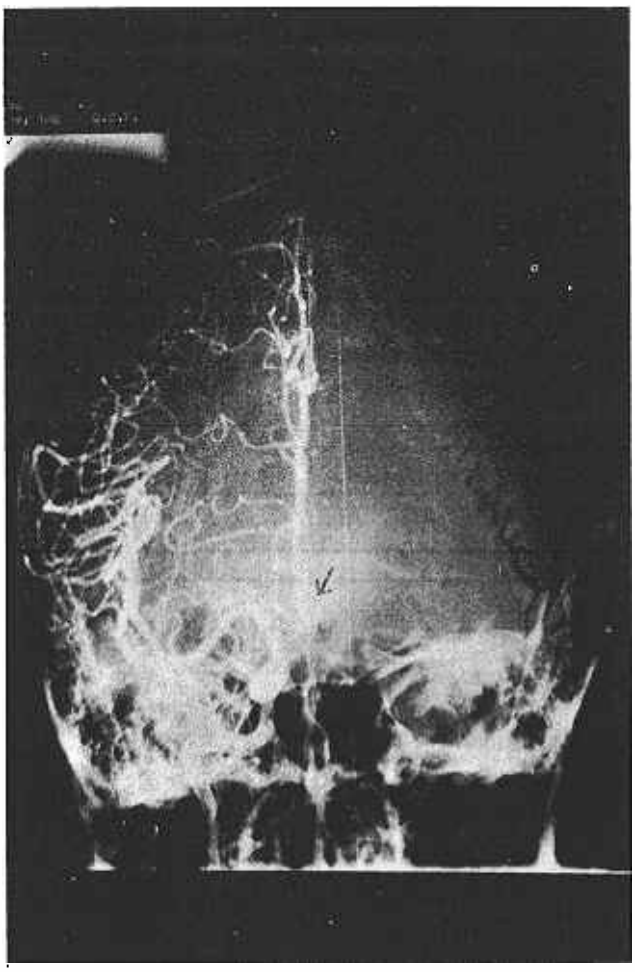

Fig. 8a : Ragged anterior communicating artery aneurysm pointing medially and upwards.

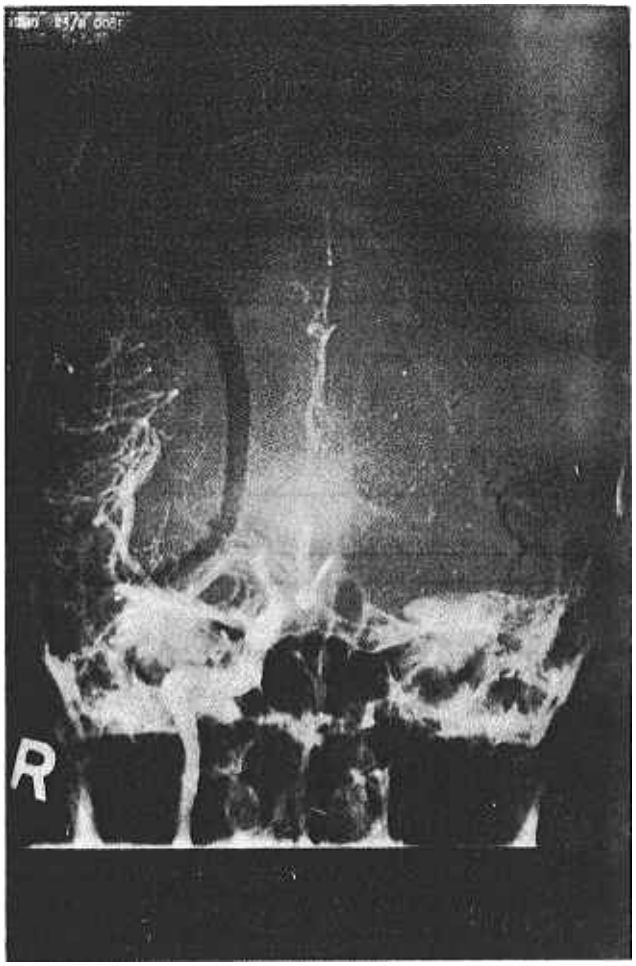

Fig. 8b : Post-operative angiogram after clipping.

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