GLUE EMBOLISATION OF INTRACRANIAL AND EXTRACRANIAL ARTERIOVENOUS MALFORMATIONS

Francis Hui MBBS FRCR FAMS[1]
Kheng-Hin Lee MBBS FRCS (E) FRCS (G) FAMS[2]
Peck-Leong Ong MBBS FRACS[3]
Samuel Ng MBBS FRCR FAMS[1]
Hong-Khim Boey MBBS FRCR FAMS[1]

ABSTRACT

Anteriovenous malformations of the head are formidable to treat. Embolisation with N Butyl Cyanoacrylate glue was carried out in 22 patients; 16 had cerebral AVMs, 4 had facial AVMs and 2 patients had dural AVMs. Complete obliteration of the shunt by the endovascular route was achieved in 7 patients. Altogether, 15 patients were cured by embolisation alone or in combination with surgery. Embolisation enhanced subsequent surgical or radiosurgical management by reducing nidus size, occluding intra nidal aneurysms and decreasing flow through the shunt. 3 more patients have not completed surgery or are awaiting follow up after radiosurgery. 2 permanent complications occurred, one deficit was mild and the other moderate in severity. Glue embolisation was found to be very effective in the treatment of patients with intra and extracranial AVMs.

INTRODUCTION

Arteriovenous malformations (AVMs) are always formidable lesions to treat, particularly if they occur in the head. AVMs occurring in the brain are particularly dangerous because of their propensity to cause intracerebral haemorrhage. Surgery has previously been the only modality available in the treatment of these lesions. Recently, endovascular therapy has emerged as a viable alternative in the treatment of these conditions either as a pre operative procedure or as the sole form of treatment.[1] Many types of embolic agents are available, including PVA and gelfoam particles, liquid glue, coils and balloons. This study involves the treatment of such AVMs with liquid glue in the form of N-Butyl-Cyanoacrylate (NBCA).

MATERIALS & METHODS

22 consecutive patients with intracranial and facial AVMs treated by glue embolisation were studied. All cases were done in the neuroangiographic suite at Tan Tock Seng Hospital, Singapore. The study period was from March 1994 to April 1995. Follow up period ranged from 1 to 12 months (mean = 3.5 months). 16 patients had cerebral AVMs, 2 patients had dural AVMs and 4 patients had facial AVMs. All patients were evaluated by digital subtraction

[1] Section of Neuroradiology, Department of Diagnostic Imaging, Tan Tock Seng Hospital, Moulmein Road, Singapore 1130.
[2] Consultant Neurosurgeon, Gleneagles Hospital, 6A Napier Road, Singapore 1025.
[3] Department of Neurosurgery, Tan Tock Seng Hospital, Moulmein Road, Singapore 1130.

Correspondences and reprints to: Dr. Francis Hui, Consultant In Charge, Section of Neuroradiology, Department of Diagnostic Imaging, Tan Tock Seng Hospital, Moulmein Road, Singapore 1130, Republic of Singapore.

angiography (DSA) using a General Electric Advantx® single plane fluoroscopic unit. In each case, super-selective catheterisation of the arterial pedicle was obtained with a microcatheter (Magic® Std by Balt, France or Minitorquer® , France). NBCA glue mixed with Lipiodol® and occasionally tungsten powder was injected under DSA.. An angiogram is then obtained to confirm the result of the embolisation. Glue was the sole embolic agent in 16 patients and was combined with polyvinyl alcohol and gelfoam particles in the other 6 patients. The procedures were carried out under neuroleptic analgesia or general anaesthesia. Systemic heparinisation was initiated if the guiding catheter was placed in the internal carotid or vertebral arteries. Amytal testing was carried out if embolisation was carried out at or near eloquent regions. Corticosteroids were given routinely after embolisation of a cerebral AVM. The effect of embolisation on the AVM and any complication that occurred were recorded. Patient outcome was studied immediately post embolisation and at follow up.

RESULTS

Altogether, 35 vessels were glued in 29 sessions, averaging 1.6 vessels and 1.3 sessions per patient. 11 patients had embolisation done as the sole therapeutic procedure and 9 had surgery cum embolisation and 1 patient had radiosurgery after embolisation. 1 patient is awaiting radiosurgery for a small nidus remaining after surgery and pre operative embolisation.

Among the patients who have embolisation of their AVMs as the sole therapeutic procedure, total occlusion of the AVM was achieved in 7 (32%) out of the 22 patients. Surgery after embolisation successfully eliminated the AVM completely in another 8 (36%) patients. In one case, embolisation accomplished complete obliteration of the residual nidus remaining after surgery and pre operative embolisation. Of the remaining 7 patients, 2 (8%) patients have not completed surgical treatment and 2 more are awaiting the result of radiosurgery. 2 (8%) patients had between 40 to 90% occlusion of their AVM after completing therapy. 1 (4%) patient had a large facial AVM which regrew to its original size each time after repeated episodes of embolisation.

Among the patients with cerebral AVMs, complete obliteration of the AVM was achieved in 3 (19%) of 16 patients. Embolisation combind with surgery resulted in complete occlusion of the nidus in another 8 (50%) patients. Complete obliteration of the arterio-venous shunt was achieved in 2 (50%) of the patients with facial AVMs and both (100%) of patients with dural AVMs.

One patient had a temporary complication. This was a 15 year old boy with a corpus callosal AVM. He was noted to have alexia after the embolisation but recovered fully from it within 3 days. Two patients had permanent complications. One patient with a dural AVM had a mild residual left hemiparesis at follow up but was able to lead a normal lifestyle. The other patient had an inoperable AVM in the right basal ganglia with enormous cerebral venous congestion because of venous outlet stenosis. Embolisation of the lenticulostriate feeders resulted in decrease in the flow through the AVM but the patient had worsening of the existing left hemiparesis.

At follow up, 6 (27%) patients had improvement of their clinical deficits. 9 (41%) patients who were neurologically intact remained normal. 6 (27%) patients with pre existing deficits had unchanged clinical status. 1 (5%) patient had worsening of her clinical status compared to her pre embolisation state. One patient had a rebleed 4 months after the embolisation while waiting for radiosurgery. His AVM was subsequently excised.

DISCUSSION

Management of arteriovenous malformations of the head and neck is always a challenging one. Vascular architecture of the lesion can be very complex. Previously, the only mode of treatment is surgery. However, surgery itself can be difficult particularly in lesions associated with a large nidus or a fast shunt. Proximal ligation of feeders is ineffective.[2,3] Obliteration of the nidus must remain the main aim of any form of therapy. In recent years endovascular treatment of these lesions has become an important mode of management.[1]

The need for intervention has been particularly well studied in patients with cerebral AVMs. As with any other disease, any form of therapy must be compared with the natural history of the disease. The risk of haemorrhage for an average lesion is about 2-4% a year.[4,5] The risk is increased if the haemorrhage is the presenting clinical feature. The presence of arterial ectasias, venous stenoses or intranidal aneurysms also increases the risk of haemorrhage.[6,7] The primary goal of any form of treatment of a cerebral AVM must be the prevention of subsequent haemorrhage, the most devastating complication. The risk of haemorrhage will still be present as long as the AVM is still patent. Therefore, the ultimate aim of therapy has to be the

complete elimination of any residual arteriovenous shunting. In the treatment of an extracerebral AVM, the aims are similar although the risk of cerebral haemorrhage may be less or not present.

The advent of variable stiffness microcatheter has enabled superselective catheterisation of AVM vessels. There is also a choice of different embolic agents. Initially, large particle embolisation using gelfoam or PVA through 5F catheters lodged in the internal carotid or vertebral artery was carried out. The technique and materials used in embolisation have changed over the years and presently most interventionists introduce the embolic agent through a microcatheter positioned with its tip close to the AVM nidus.

The selection of an optimal embolic agent depends on the familiarity of the operator. The 3 most common embolic agents utilised in AVM embolisation are glue such as N-butyl cyanoacrylate (NBCA), thrombogenic coils and particles like polyvinyl alcohol (PVA).

NBCA glue is a vinyl monomer which polymerises to a solid compound on contact with an ionic solution such as blood or saline.[10] When injected into any blood vessels, it will then form a cast of the vascular structure. It has the advantage of being suitable for use through any microcatheter. Being liquid initially, it has the capability of penetrating into the nidus of the AVM. In high concentrations, glue is suitable for occlusion of arteriovenous fistulae. It has also been shown to be permanent and AVMs occluded with glue do not recanalise.[11] However, it also requires considerable operator experience to master the technique so as to avoid a high rate of complications. Pulmonary complications can also occur with reflux into the venous side of the shunt.[12] Particles, on the other hand are more controllable and easier to handle. But they can be difficult to inject through a microcatheter, occasionally resulting in clogging of the catheter itself.[13] Recanalisation of occluded vessels can also occur.[14] In cases of large arteriovenous fistulae, particles may also be ineffective and will pass directly into the pulmonary circulation. Thrombogenic coils, when employed are usually delivered in the feeding artery to reduce blood flow or close the whole vessel itself. However, it tends to produce proximal occlusion and collateral circulation to the nidus results.

In a certain percentage of patients, penetration of the nidus by the glue is complete, obviating the need for any other form of treatment. In this series, this was achieved in 19% of cerebral AVMs. Unlike surgery, the size of the AVM vessels may not be an important consideration as illustrated by a 62 year old Malay man who had a left frontal AVM which had bled and resulted in subarachnoid and intraventricular haemorrhage (Fig 1). He was admitted to hospital in a coma. Embolisation of the AV shunt resulted in complete occlusion and the patient had no neurological deficit. He was discharged fully ambulant 3 days after the procedure.

Occlusion of the actual shunt and not just the arterial feeders is essential to the success of the endovascular procedure. Collateral feeders from other sources close up completely if the shunt is obliterated. An example is a 34 year old Chinese man who had a right facial AVM. After occlusion of the shunt via the right facial artery, feeders from the internal maxillary artery disappeared (Fig. 2).

To patients who had undergone complete occlusion of their AVM by endovascular techniques alone, embolisation confers obvious benefits. The patient is spared a craniotomy or facial incision, experiences less pain and scarring and eventually pays less costs for a safer procedure. Apart from this benefit to the patient, there are also advantages to the health service in the form of reduced hospital stay and less intensive care management.

Embolisation can also be used as a complementary mode of therapy in combination with surgery and radiosurgery. Spetzler[15] indicated in his classification of cerebral AVMs that the larger sized AVMs are associated with a higher operative morbidity rate. Embolisation prior to surgery can help by reducing the size of the AVM besides decreasing the flow. In this way, it can help in lessening the surgical morbidity.[16] Many of the patients in this series benefited from reduction in nidus size. In one case (Fig. 3), it allowed awake surgery, a specialised operative technique meant to decrease surgical morbidity in operations near eloquent regions of the brain. Pre operative embolisation can help in other ways. For example, it is well known that certain angiographic features such as aneurysms[6,7] predisposes to bleeding. There are points of weakness within the AVM which have a high potential for haemorrhage. Bleeding from aneurysm rupture can also complicate AVM surgery. In one patient (Fig. 4), glue filling of the aneurysmal sac helped obviate this possibility. Patients with deep periventricular AVMs can also benefit from a combination of embolisation and surgery.[17]

Embolisation followed by surgery is an effective way of treating large AVMs.[18] One patient in this series had a large inoperable right cerebellar AVM. She was treated with staged embolisation until the nidus size shrank by about 80%. This meant that the patient can then undergo surgery with potential complete excision of her AVM.

Embolisation can complement radiosurgery by reducing the size of the nidus prior to radiation.[19] It can also be used effectively to treat lesions that have not responded to initial radiosurgical treatment.[20]

Complications of glue embolisation can occur as a result of glue occluding normal arterial branches or occlusion of an important venous outlet. In one patient with a dural AVM which bled, embolisation of the feeding artery was carried out with a microcatheter positioned a short distance from its origin from the left vertebral artery (Fig. 5). Unfortunately, some reflux of glue into the main artery was noted. The fistula was closed but the patient had a left hemiplegia and hemianopia. He, however, recovered fully from his hemianopia and at follow-up, was found to have only a mild hemiparesis (Grade 4+/5 motor power) and was able to lead a normal lifestyle.

In conclusion, glue embolisation is an effective mode of therapy in the management of intra and extracranial AVMs. Careful embolisation of the AVM nidus produces efficient results and helps keep procedural complications to a minimum.

Figs. 1 (a)-(c) 62 year old man who was admitted to hospital in a coma. Enhanced CT scan (a) shows subarachnoid and intraventricular haemorrhage with large dilated vessels in left frontal lobe (arrowheads). Frontal view of left internal carotid angiogram (b) revealed the AV shunt to be supplied by the left middle cerebral artery. Post embolisation angiogram (c) showed no residual shunt and good filling of the left anterior and middle cerebral arteries (arrows). The patient had no deficit and was discharged 3 days post embolisation ambulant and neurologically intact.

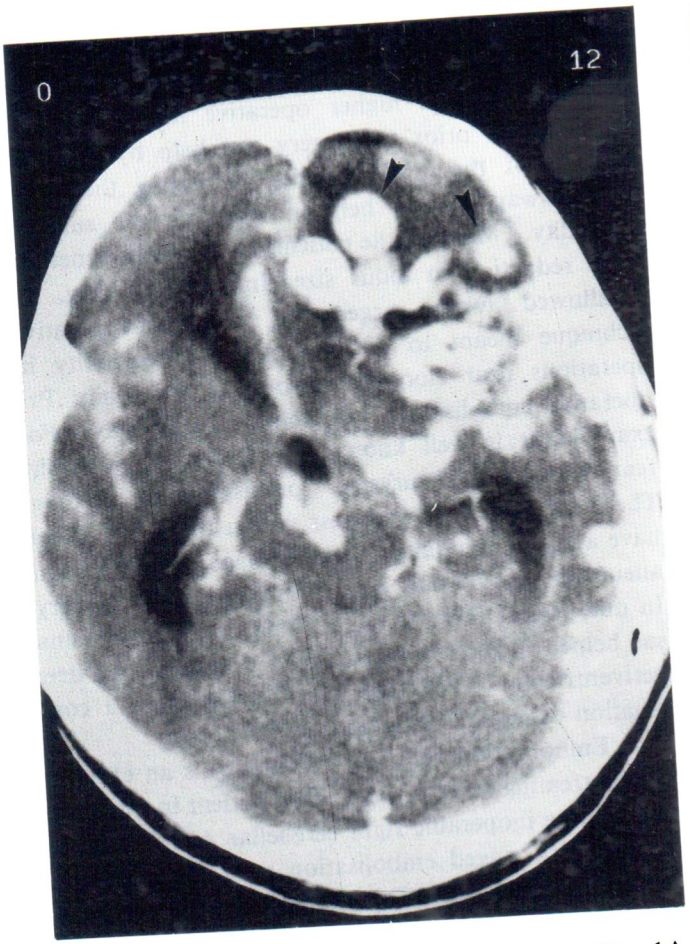

Fig. 1A.

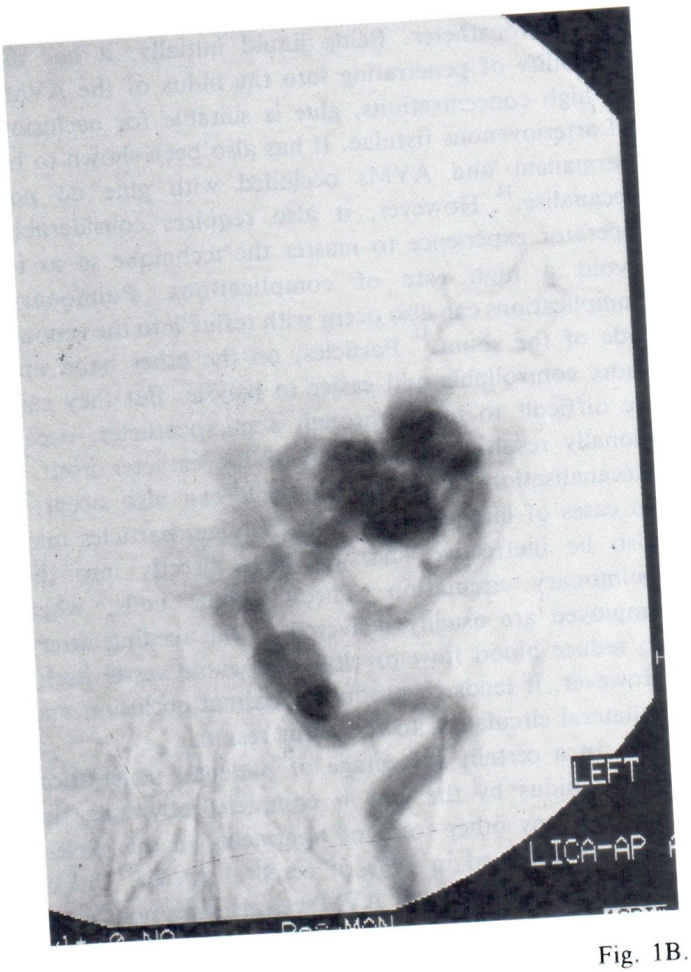

Fig. 1B.

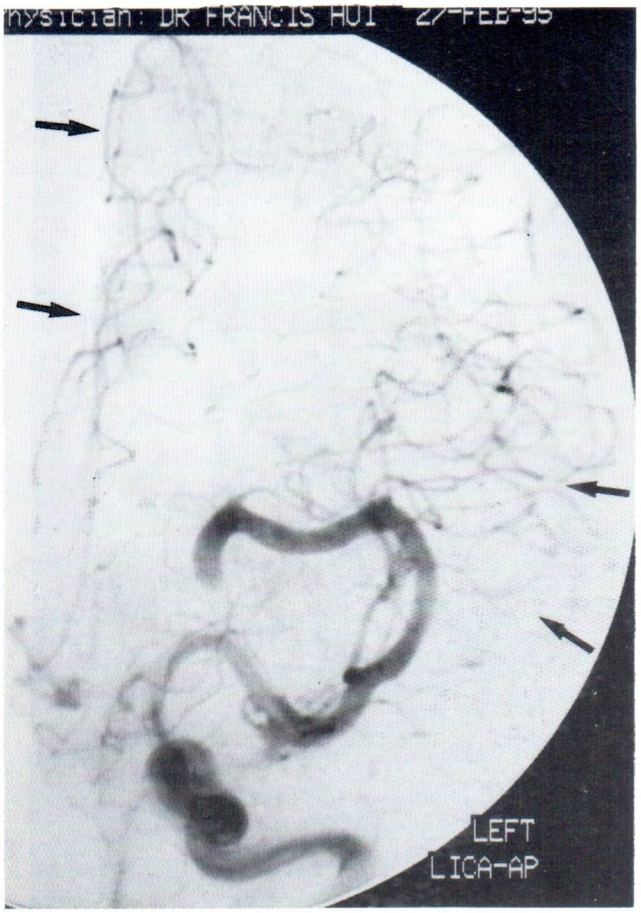

Fig. 1C.

Figs. 2 (a)-(f) 34 year old man with a right facial AVM. Diagnostic angiogram (a) showed an AV fistula in the right cheek. The angiographic catheter is in the right facial artery (arrows). Right internal maxillary angiogram (b) showed opacification of the AV shunt via collateral feeders (arrowheads). Film done after embolisation of the right facial artery feeder (c) demonstrated glue (fat arrow) in the feeding artery, fistula and in the draining vein. Post embolisation right facial angiogram (d) revealed complete occlusion of the AV fistula. The collateral feeders from the right internal maxillary artery have also disappeared even though this artery was not embolised (e). 6 month follow up angiogram (f) showed persistent obliteration of the AV shunt.

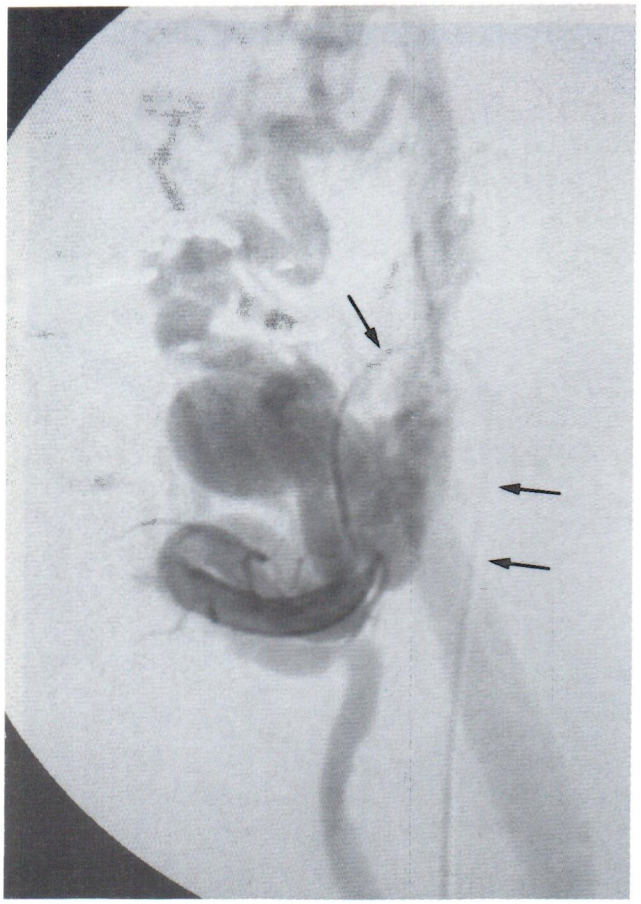

Fig.2A.

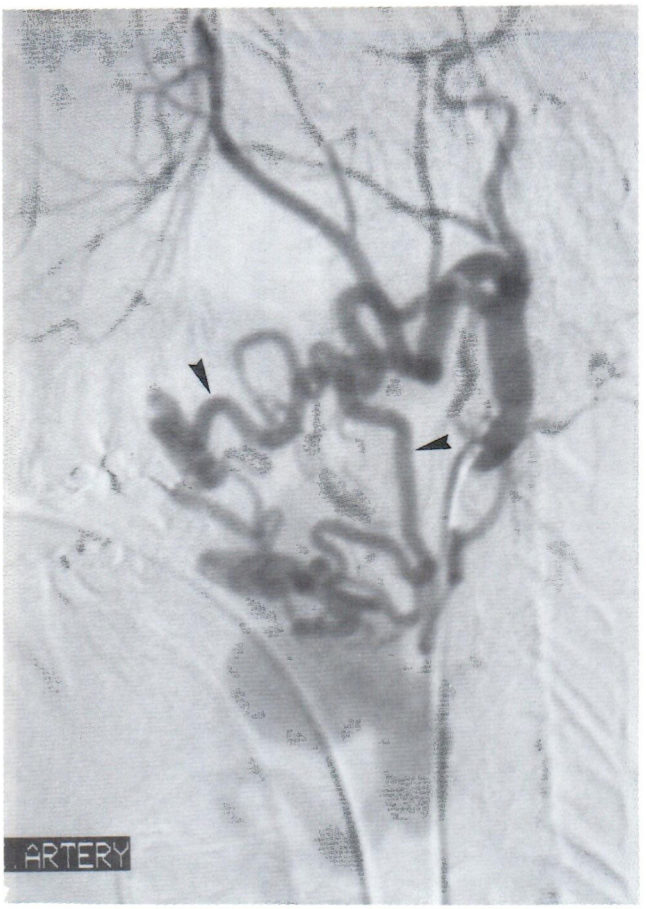

Fig.2B.

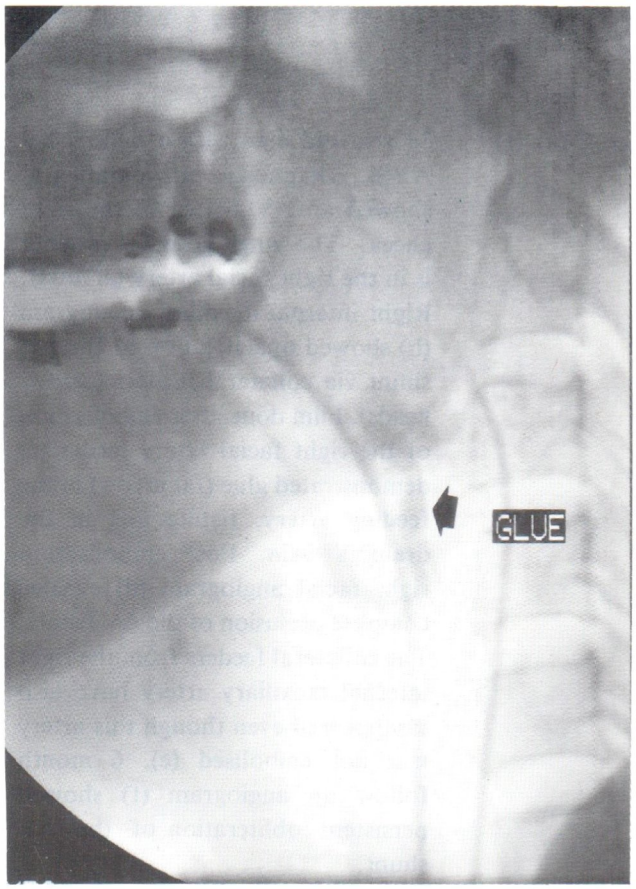

Fig.2C.

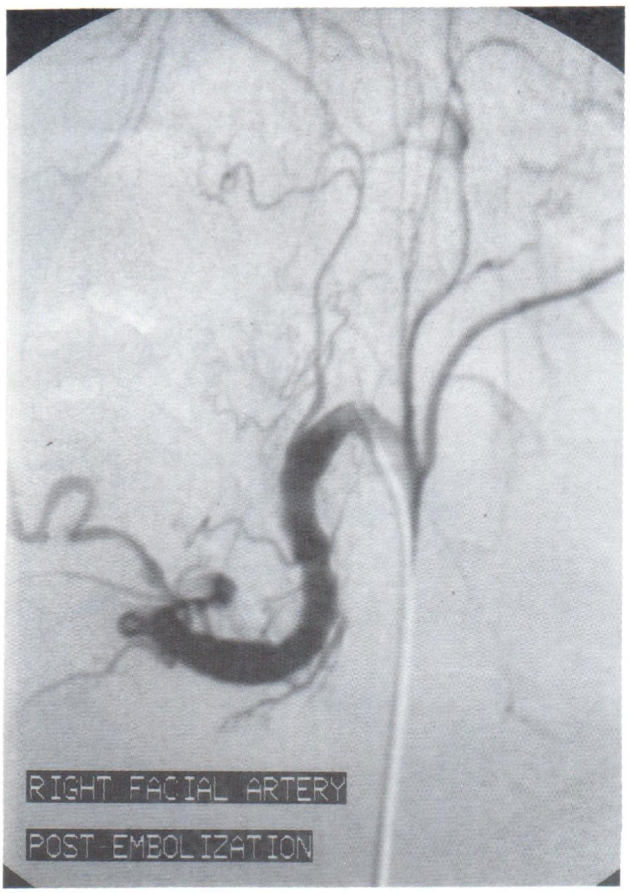

Fig.2D.

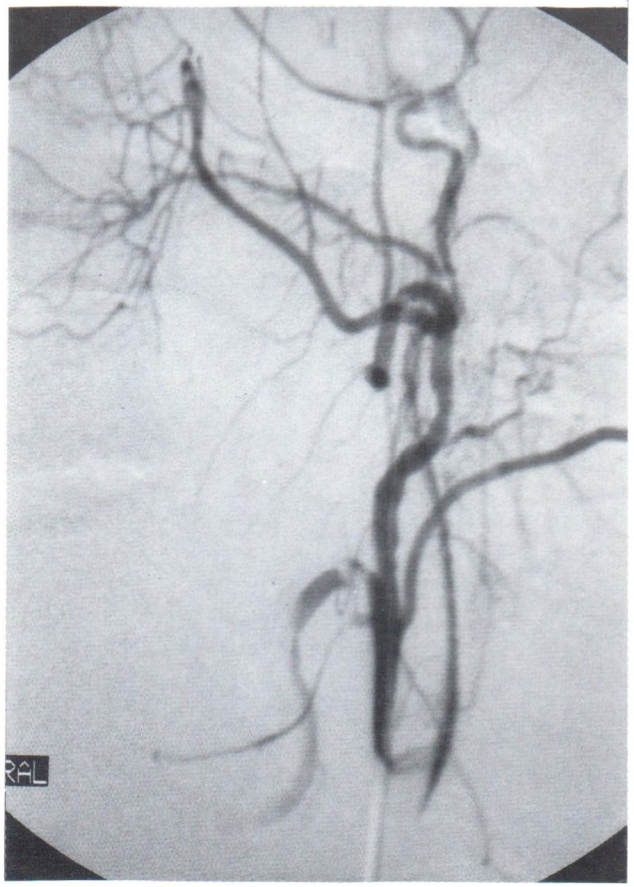

Fig. 2 E.

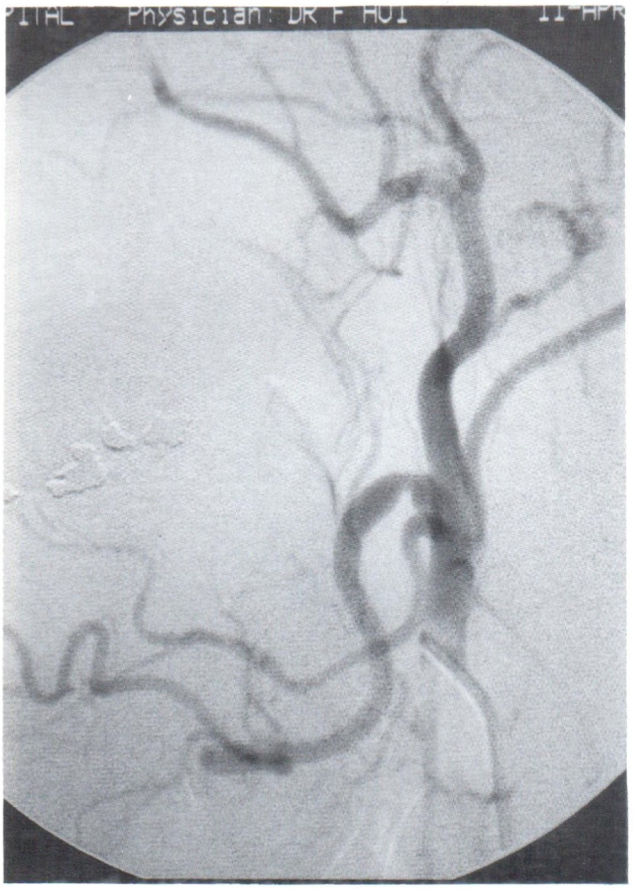

Fig. 2 F.

Figs. 3 (a)-(d) 15 year old girl with left temporal lobe AVM (fat arrow) shown on MRI (a). Pre embolisation angiogram (b) shows the AVM nidus (open arrow-head) with feeders arising from the left MCA. After 2 injections, the nidus is almost completely filled with glue (arrows in c). Post embolisation angio-gram (d) indicated good occlusion of nidus. The patient remained well and underwent awake surgery at a later date with complete obliteration of the AVM.

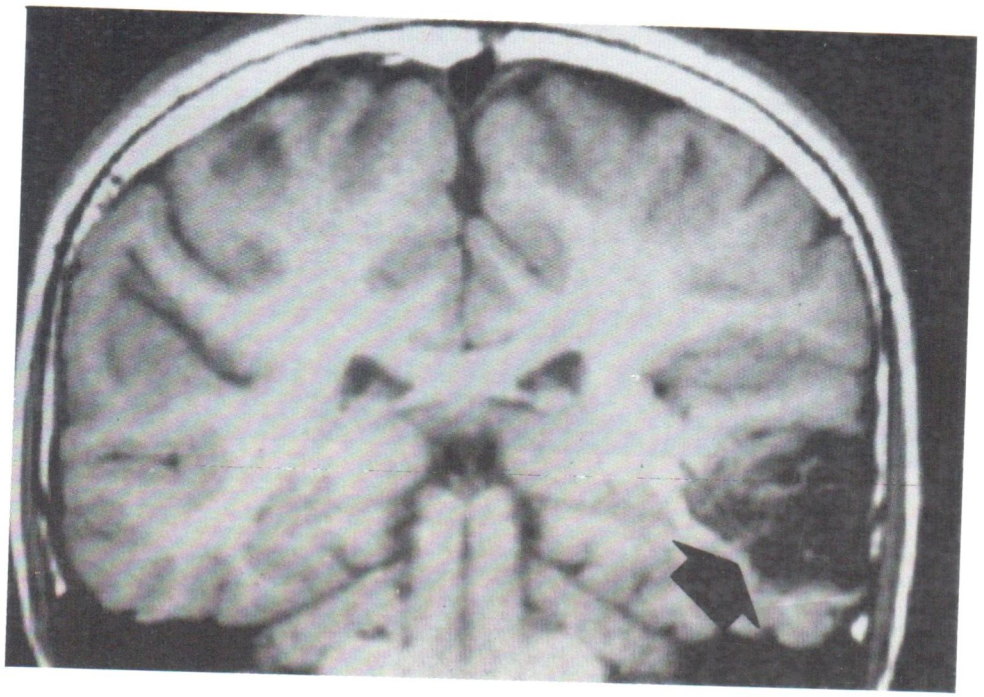

Fig.3A.

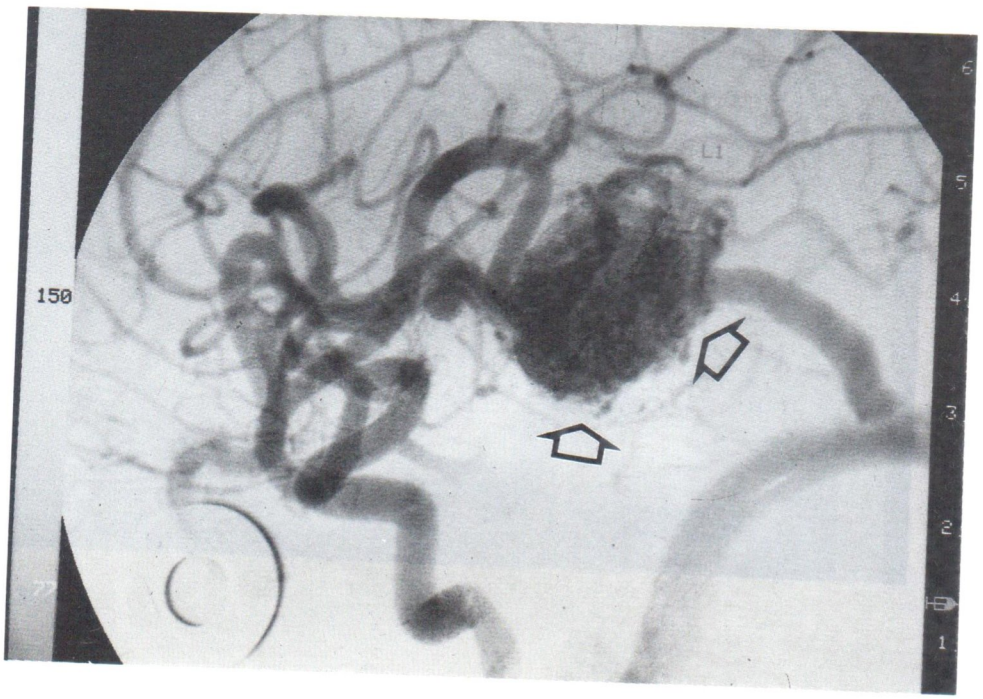

Fig. 3B.

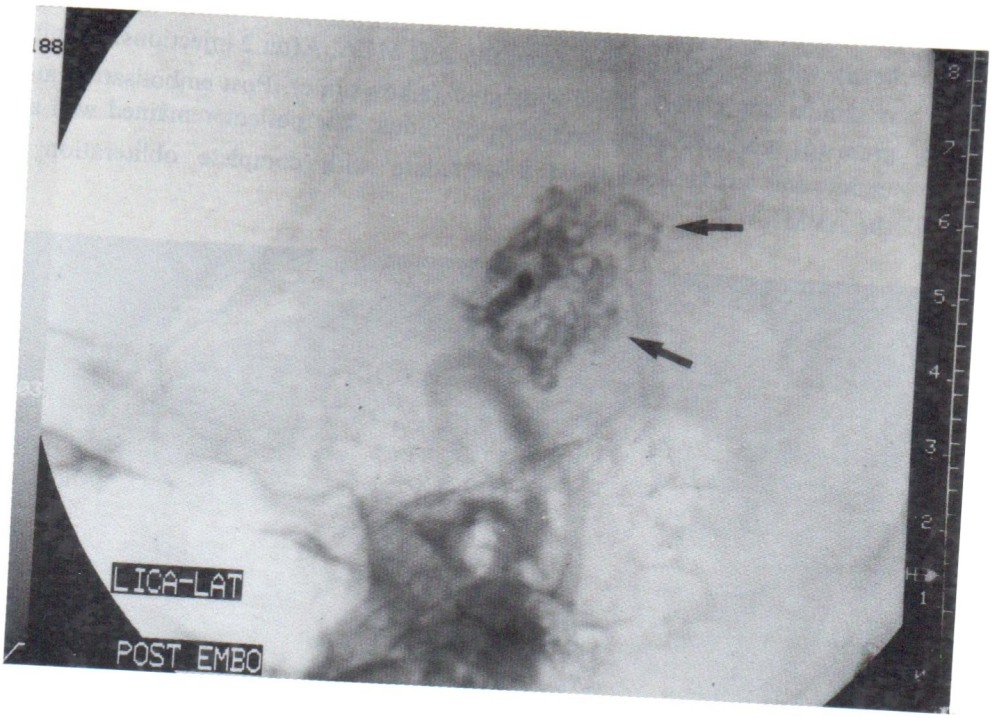

Fig. 3C.

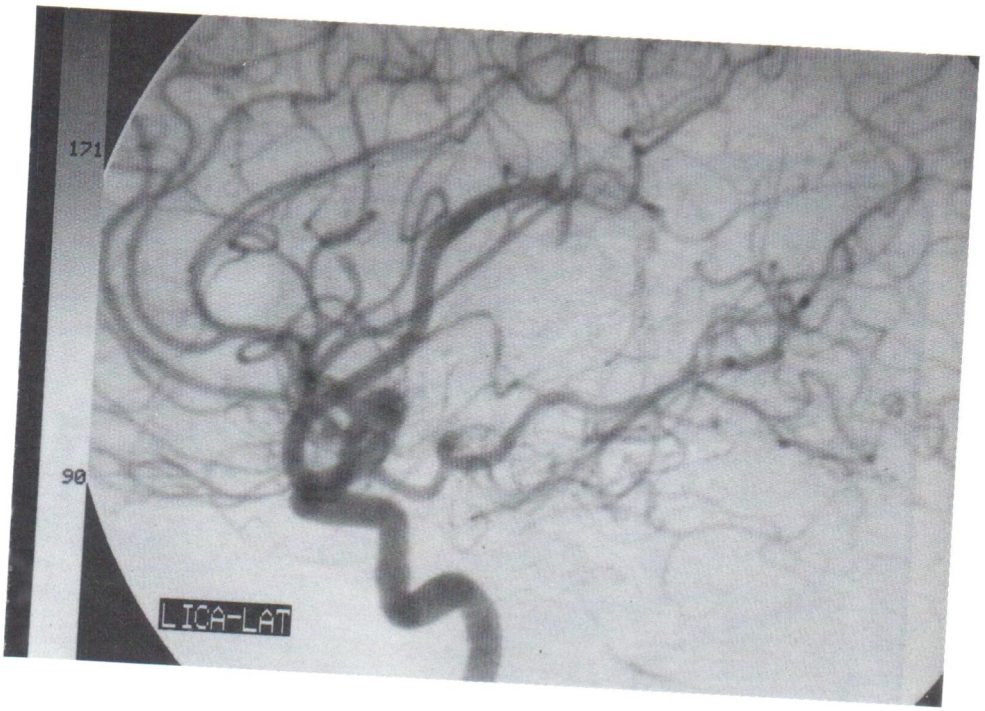

Fig. 3D.

Figs. 4 (a)-(d) 40 year old woman with right frontal AVM which bled and resulted in deep coma on presentation. The diagnostic angiogram (a) indicated the presence of an aneurysm (arrows) in a feeding pedicle from the right anterior cerebral artery. A microcatheter was manoeuvred into this pedicle and glue was then injected into it (b). Post embolisation film (c) showed glue filling the whole aneurysm (fat arrow). The angiogram thereafter (d) showed obliteration of the aneurysm and part of the nidus. The surgeon was then able to resect the whole AVM without any complication.

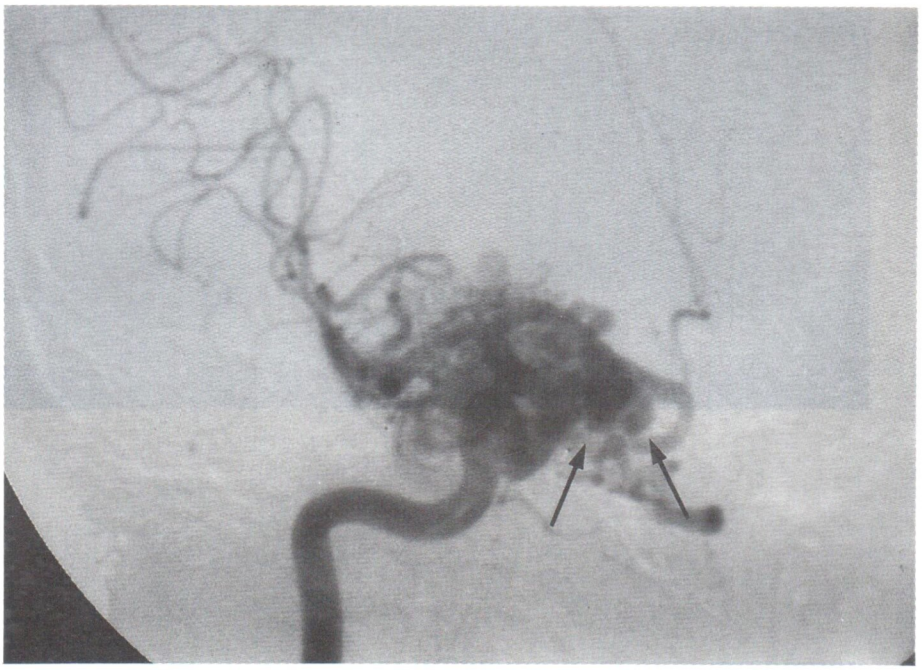

Fig. 4A.

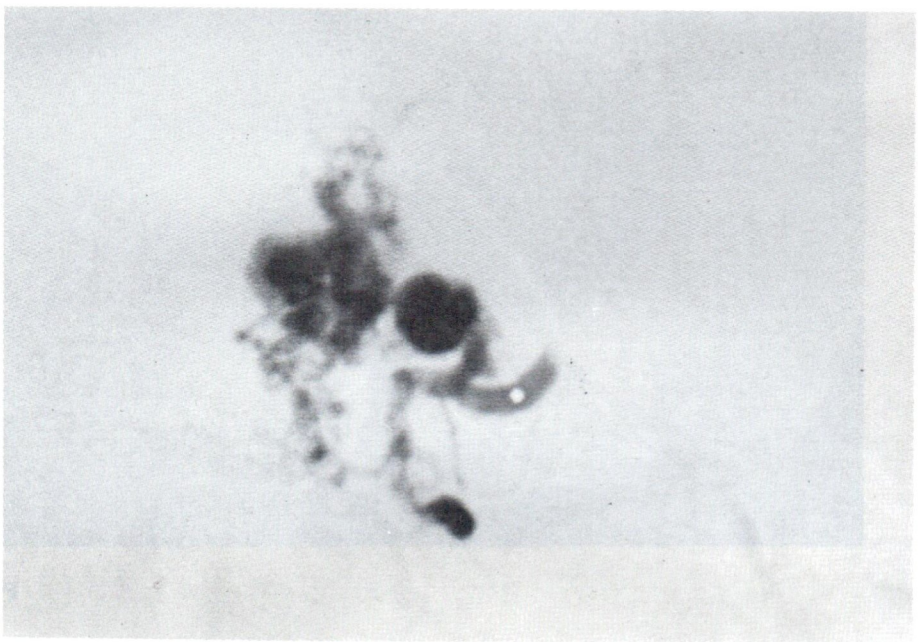

Fig.4B.

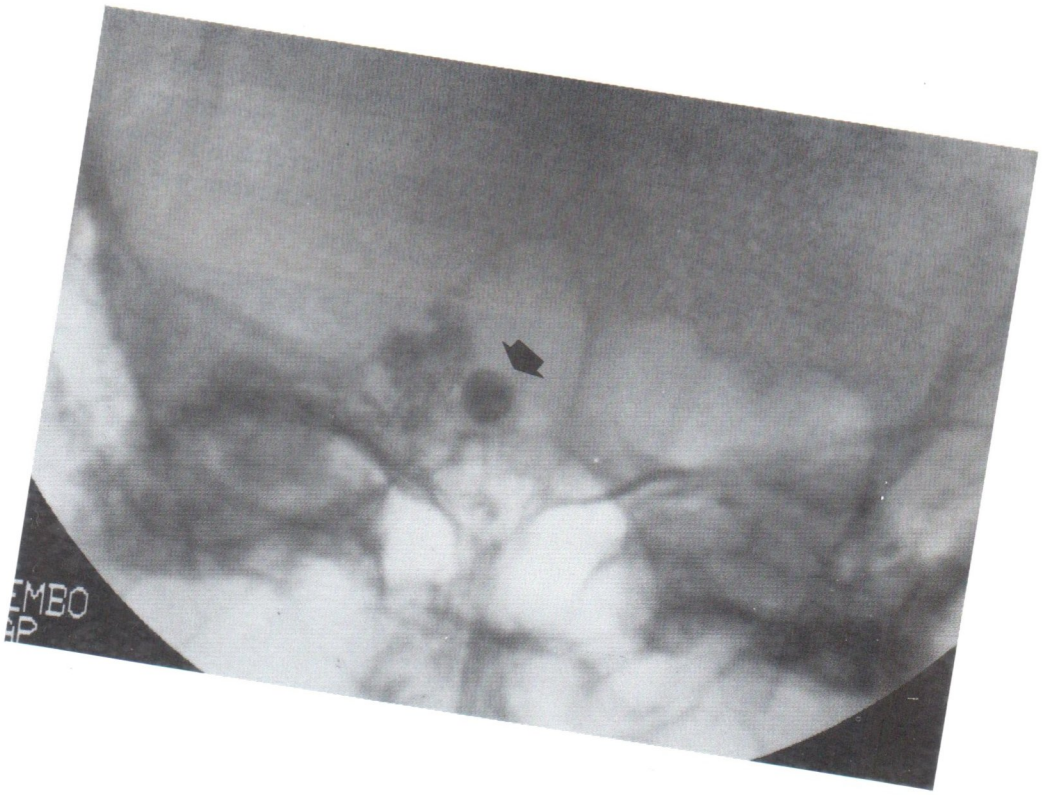

Fig.4C.

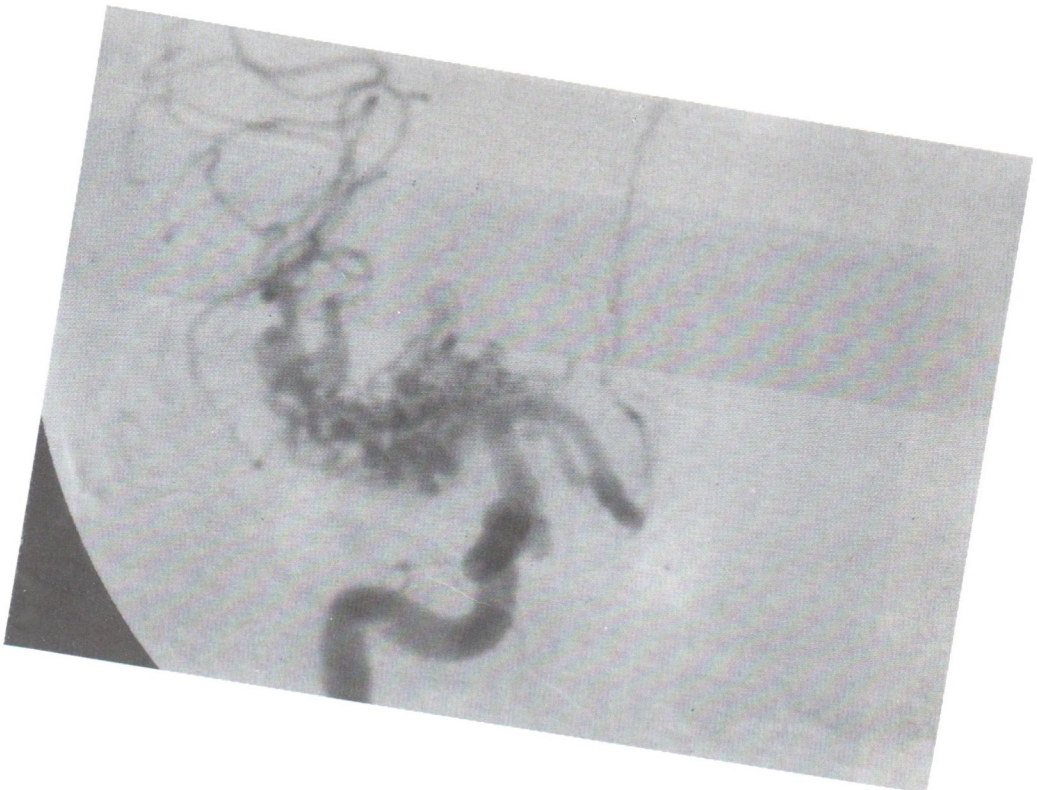

Fig 4 D.

Figs. 5 (a)-(e) 50 year old man who presented in a comatose state. Unenhanced CT scan (a) showed subarachnoid haemorrhage (arrows). Left vertebral angiogram (b) showed considerable spasm in the basilar and left vertebral artery (arrowheads) and a dural fistula (arrows) at the skull base. Superselective angiogram of meningeal feeder (c) with a microcatheter in place showed the AV shunt (arrows) and the arterial aneurysm (open arrowhead). Post embolisation angiogram (d) showed occlusion of the AV shunt but also narrowing of the right posterior cerebral artery due to reflux of glue. He was noted to have a left hemianopia and moderately severe left hemiparesis. Follow up angiogram 2 months post embolisation (e) revealed relief of the spasm in the basilar and left vertebral arteries (arrows) with no filling of the AV fistula. The proximal right posterior cerebral artery remains narrowed (open arrowhead). The patient recovered well and suffered no further haemorrhage. At 6 month post embolisation follow up, he had no visual field loss and only mild left hemiparesis (Grade 4+/5 motor power) and was able to lead a normal lifestyle.

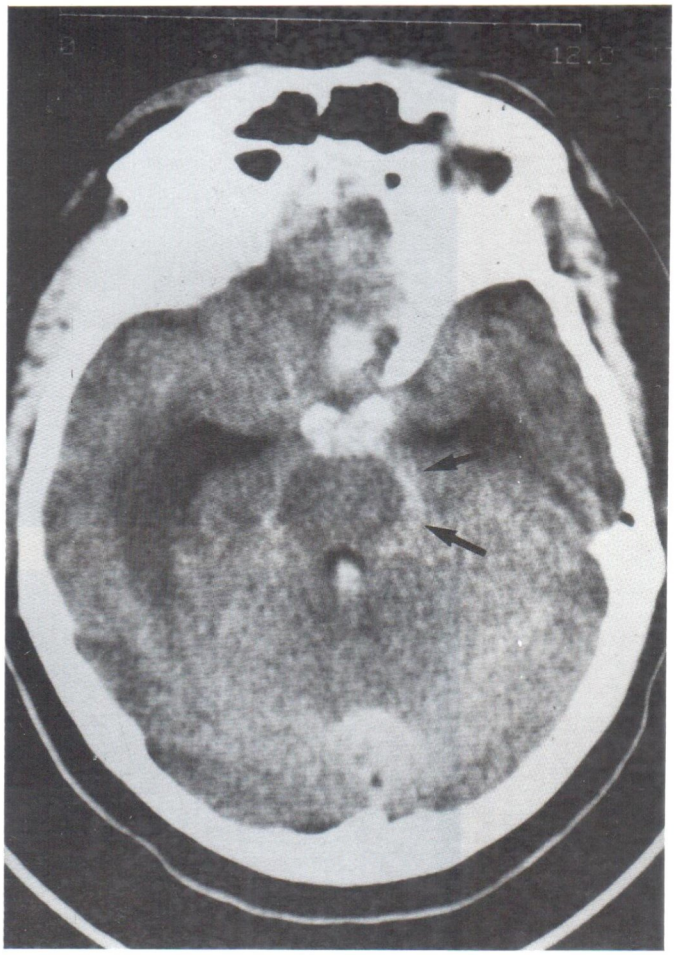

Fig. 5 A.

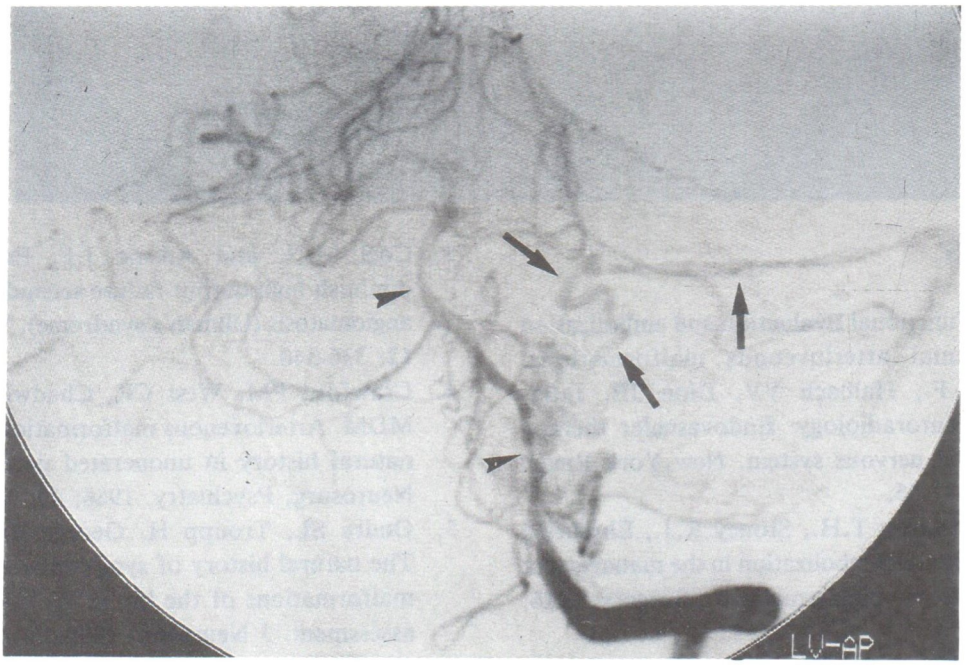

Fig. 5 B.

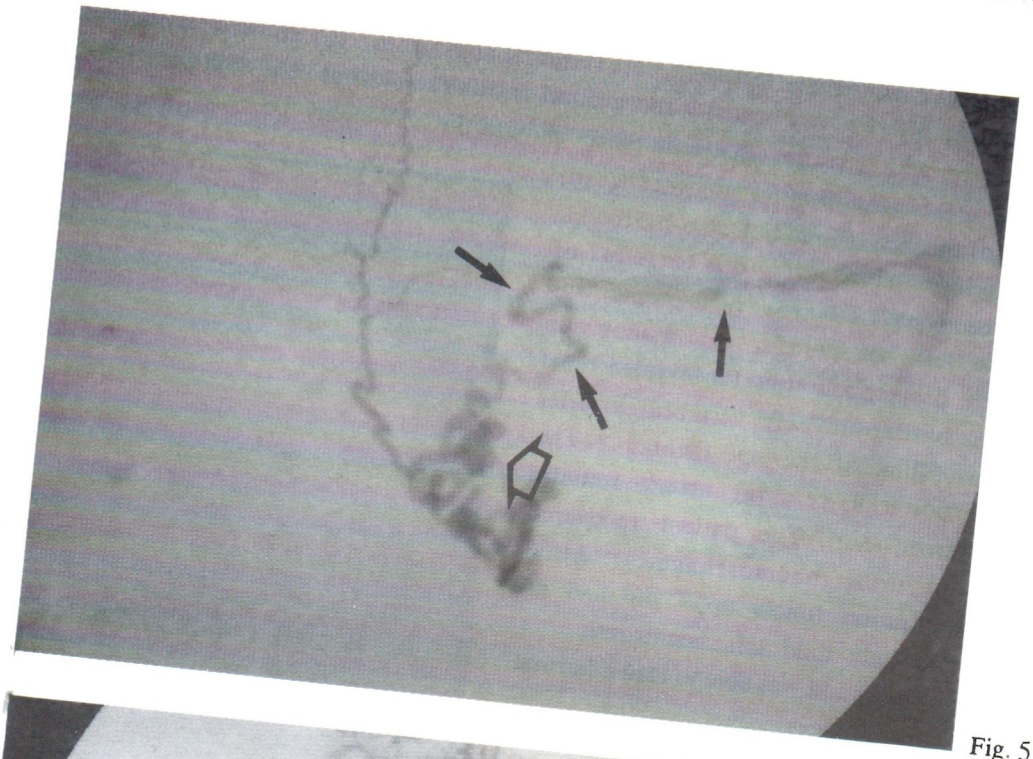

Fig. 5 C.

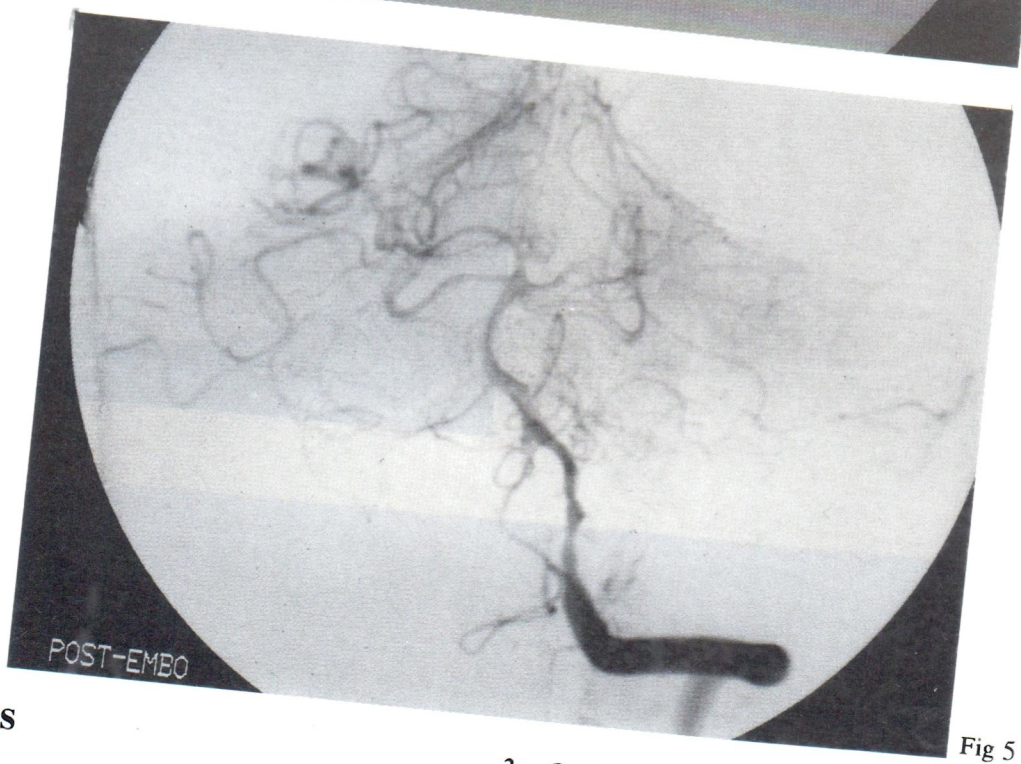

Fig 5 D.

REFERENCES

1. Vinuela F. Functional Evaluation and embolization of intracranial arteriovenous malformations. In Vinuela F., Halbach VV, Dion JE. Interventional Neuroradiology: Endovascular therapy of the central nervous system. New York Raven Press 1992; 77-86.
2. Olcott C., Newton T.H., Stoney R.J., Ehrenfeld W.K. Intra arterial embolization in the management of arteriovenous malformations. Surgery 1976; 79: 3-12.
3. Coel M.N. and Alksne J.F. Embolization to diminish high output failure secondary to systemic angiomatosis (Ullman's syndrome). Vasc Surg 1978; 12: 336-340.
4. Crawford PM, West CR, Chadwick DW, Shaw MDM. Arteriovenous malformations of the brain: natural history in unoperated patients. J Neurol Neurosurg, Psychiatry, 1986; 49: 1-10.
5. Ondra SL, Troupp H, George ED, Schwab K. The natural history of symptomatic arteriovenous malformations of the brain: A 24 year follow-up assessment. J Neurosurg 1990: 73: 387-91.

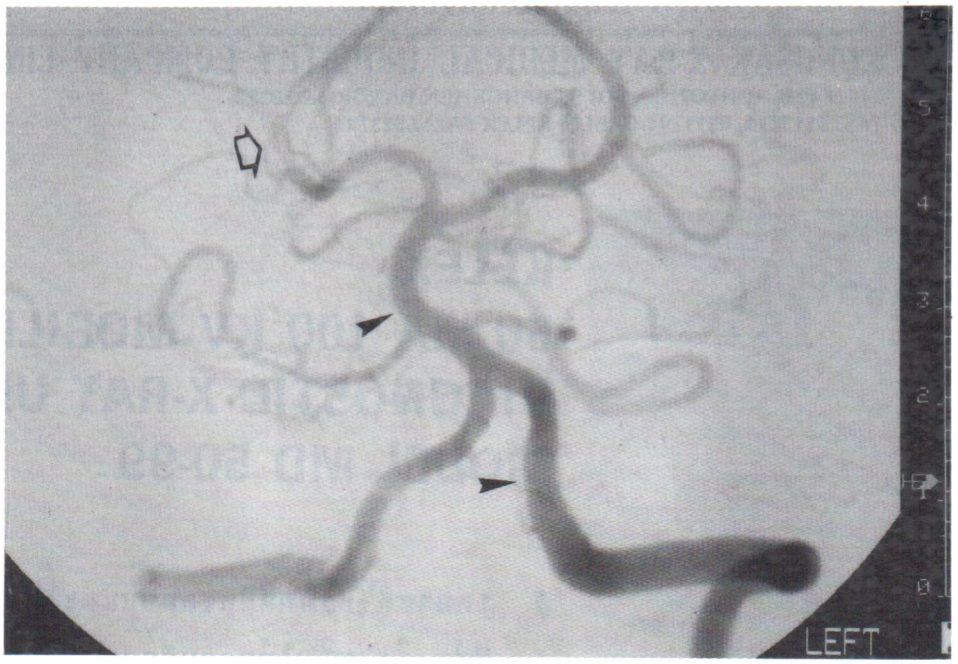

Fig. 5 E.

6. Willinsky R, Lasjaunias P, Terbrugge K, Pruvost P. Brain arteriovenous malformations: analysis of the angioarchitecture in relationship to hemorrhage (based on 152 patients explored and/or treated at the Hospital de Bicetre between 1981 and 1986) J Neuroradiol 1988; 15: 225-37.
7. Marks M, Lane B, Steinberg G, Chang P. Hemorrhage in Intracranial Arteriovenous Malformations: Angiographic determinants. Radiology 1990; 176: 807-13.
8. Wolpert SM, Stein BB. Factors governing the course of emboli in the therapeutic embolization of cerebral arteriovenous malformation. Radiology 1979; 131: 125-131).
9. Mullan S, Kawanaga H et al. Microvascular embolisation of cerebral arteriovenous malformations. J Neurosurg 1979; 51: 621-627.
10. Lasjaunias P., Berenstein A. Surgical Neuroangiography Vol 2. (1987) Springer Verlag. Heidelberg, New York.
11. Wikholm G Occlusion of cerebral arteriovenous malformations with N-Butyl Cyanoacrylate is permanent. AJNR 1995; 16: 479-83.
12. Pelz DM, Lownie SP, Fox AJ, Hutton LC. Symptomatic pulmonary complications from liquid acrylate embolisation of brain arteriovenous malformations. AJNR 1995; 16: 19- 26.
13. Derdeyn CP, Moran CJ, Cross DT, Dietrich HH, Dacey RG. Polyvinyl alcohol particle size and suspension characteristics.AJNR 1995; 1335-43.
14. Germano IM, Davis RL, Wilson CB, Hieshima GB. Histo pathological follow up study of 66 cerebral arteriovenous malformations after therapeutic embolisation with polyvinyl alcohol. J. Neurosurg 1992; 76: 607-14.
15. Spetzler RF, Martin NA. A proposed grading system for arteriovenous malformations J Neurosurg. 1986; 65: 476-83.
16. Fox AJ, Pelz DM, Lee DH. Arteriovenous malformations of the brain: Recent results of endovascular therapy. Radiology 1990; 177: 51-7.
17. U HS, Kerber CW, Todd MM. Multi modality treatment of deep periventricular cerebral AVMs. Surg Neurol 1992; 38: 192-203.
18. Spetzler RF, Martin NA, Carter P, Flom RA, Raudzens PA, Wilkinson E. Surgical management of large AVMs by staged embolisation and operative excision. J Neurosurg 1987: 67: 17-28.
19. Dawson III RC, Tarr RW, Hecht ST, Jungreis CA, Lunsford LD, Coffey R, Horton JA. Treatment of arteriovenous malformations of the brain with combined embolization and stereotactic redio-surgery: Results after 1 and 2 years AJNR 1990; 11: 857-864.
20. Marks MP, Lane B, Steinberg GK, Fabrikant JI, Levy RP, Frankel KA, Phillips MH. Endovascular treatment of cerebral arteriovenous malformations following radiosurgery. AJNR 1993; 14: 297-303.