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Dominant-Hemisphere Arteriovenous Malformations: Therapeutic Embolization with Isobutyl-2-Cyanoacrylate

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Embolization treatment of 16 patients with dominant-hemisphere arteriovenous malformations (AVMs) is described. This group was selected from 50 patients with brain AVMs embolized with isobutyl 2-cyanoacrylate (IBC-2) over a period of 3 years. All 16 AVMs were cortical in location; six involved the rolandic and speech areas, and four had a deep component. Ten AVMs were embolized through a transfemoral approach, an intraoperative approach was used for four cases, and a combined transfemoral/intraoperative approach was used in two cases. Complete obliteration of the AVM was obtained in one case. Partial obliteration and complete surgical resection was obtained in three cases. Obliteration of 70%–95% of the AVM was obtained in six cases and 45%–70% of the AVM was obliterated in six cases. IBC-2 embolization of the venous drainage was observed in three cases. After embolization, eight patients remained neurologically unchanged. Three patients had mild neurologic deficits that resolved completely within 48 hr; one had a deficit that cleared up 1 week later; and one had a deficit that disappeared within 6 months. In three patients a mild permanent neurologic deficit was evident 6 months after embolization.

Cerebral arteriovenous malformations (AVMs) have been considered more benign than aneurysms [1]. Several reports with long follow-ups ([2, 3] and Kjellberg RN, personal communication), however, indicate a more reserved prognosis, concluding that AVMs constitute a major threat to life, many patients dying before the age of 50. Surgical excision has been the treatment of choice of this disease for 20 years. The overall results of surgical excision show a 70% success rate with a 11% morbidity and mortality [1–4]. Recently, methods involving neuroradiologic intravascular embolization techniques [5–9] and focused high-energy radiation [10–12] have been developed. The final technical development, efficiency, morbidity, and mortality of these techniques are not yet fully known.

The development of intracranial, intravascular navigation with microcatheters and calibrated-leak balloons opens an alternative treatment for intracranial AVMs. This technique is particularly useful in high-flow AVMs with large arterial feeders. Intravascular embolization with deposition of the embolizing substance within the nidus of the lesion should aim at complete obliteration of the AVM.

Proximal occlusion of the AVM's feeders by surgical or neuroradiologic techniques is useless as a long-term measure [1]. Furthermore, this approach eliminates the alternative of distal superselective catheterization and embolization.

The site and size of AVMs are the principal concerns in their surgical removal [13, 14]. AVMs located in functionally essential brain regions such as the rolandic or speech areas are traditionally difficult to excise surgically [1–15]. With the development of the latex calibrated-leak balloon [16, 17], it is now possible to repeatedly catheterize and embolize dominant-hemisphere AVMs involving vital brain areas. Even so, close neuroradiologic/neurosurgical teamwork is essential to plan treatment, alternative technical approaches, and to treat complications.

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TABLE 1: **Embolization of Dominant-Hemisphere AVMs: Summary of Cases**

Approach: Case No. (age, gender)	Clinical Status		Cortical Areas of Involvement	Arterial Feeders	No. Embolizations (% Occlusion)
	Preembolization	Postembolization			
Transfemoral:					
1 (20, M)	Seizure	Mild central facial palsy; recovery in 24 hr	Inferior rolandic; speech	MCA	3 (45)
3 (27, F)	SAH; headaches	Normal	Inferior rolandic; speech	MCA	2 (50)
4 (22, M)	Seizures	Mild central facial palsy; full recovery in 24 hr	Frontal; prerolandic; speech*	MCA+++; ACA++; lenticulostriate++	2 (50)
5 (50, F)	SAH; aphasia, R hemiplegic	No changes	Mid posterior frontal; parietooccipital	ACA++; PCA+++	2 (60)
7 (32, F)	Seizures; headaches, R superior quadrant anopia	R homonymous hemianopia; unchanged 6 months later	Parietooccipital	PCA+++; ACA+	1 (80)
9 (20, F)	Headaches	Normal	Rolandic; speech*	MCA+++; lenticulostriate; Heubner	1 (75)
10 (37, F)	SAH; headaches	Normal	Frontal;* prerolandic*	MCA+++; lenticulostriate	1 (95)
12 (21, M)	SAH; headaches; facial palsy	R homonymous hemianopia; unchanged 6 months later	Occipital	PCA+++; MCA+	3 (90)
14 (58, F)	Seizures	Normal	Frontal prerolandic	MCA+++; ACA+	2 (60)
15 (55, F)	Seizures, L leg monoplegia	Unchanged	Supracallosal*	ACA+++; MCA+; posteromedial; choroidal	1 (40)
Intraoperative:					
6 (40, F)	SAH, seizures; headaches	R hand monoplegia; global aphasia; complete recovery 1 week later	Parietal	MCA+++; ACA+	4 (95)
8 (37, M)	SAH; seizures; R hemiparesis	Central facial palsy; full recovery in 48 hr	Rolandic; speech	MCA+++; ACA+	1 (75)
13 (53, M)	Dysphasia	Aphasia; R hemiparesis; full recovery 6 months later	Retrorolandic	MCA	1 (100)
16 (37, F)	Seizures	Aphasia; R hemiparesis; mild dysplasia 6 months later	Inferior rolandic; speech	MCA	1 (95)
Combined:					
2 (30, M)	SAH	Normal	Middle; frontal; interhemispheric	R and L pericallosal	1 (45)
11 (56, F)	SAH; headaches; R homonymous hemianopia	Dysphasia; recovery in 36 hr	Middle parietooccipital; dural	PCA+++; ACA+, MCA+; R and L occipital; R and L middle meningeal	1 (50)

Note.—Complete surgical resection was achieved in cases 2, 8, and 11. Venous embolization occurred in cases 13 (draining vein), 14 (dural sinus), and 16. SAH = subarachnoid hemorrhage; MCA = middle cerebral artery; ACA = anterior cerebral artery; PCA = posterior cerebral artery; R = right; L = left; +++ = dominant blood supply; ++ = moderate blood supply; + = small blood supply.

* These AVMs had a deep component to their cortical locations.

Materials and Methods

We reviewed 16 patients with dominant—hemisphere AVMs treated by isobutyl 2-cyanoacrylate (IBC-2) embolization. They were part of a group of 50 patients with intracranial AVMs treated by this technique between late 1978 and 1981.

The patients were 20–50 years old. The most common clinical presentations were subarachnoid hemorrhage, seizures difficult to control medically, intractable headache, or a combination of these symptoms. Seven of the 16 patients showed neurologic deficits before embolization therapy (table 1). In six patients the AVM was located in the rolandic and/or speech areas. The other AVMs were distributed throughout the left hemisphere.

All the patients were premedicated with 8 mg of Decadron orally every 6 hr, commencing 12–24 hr before embolization to minimize postresection or postembolization edema [18, 19]. Steroid therapy continued for 72 hr after embolization.

Three technical approaches were used: a transfemoral approach (10 cases), an intraoperative approach (four cases), and a combined transfemoral and intraoperative approach (two cases).

Transfemoral Approach

This technique was performed in 10 patients (table 1) under neuroleptic anesthesia. A 6 French Cordis sheath was introduced

into the right femoral artery and the appropriate internal carotid or vertebral arteries were selectively catheterized with a 5.8 thin-walled polyethylene catheter (Elecath, Electro-Catheter Corp., Rahway, NJ). A calibrated-leak balloon microcatheter was then injected through the Elecath catheter and selectively positioned in one of the AVM's main feeders by sequential inflations and deflations of the balloon. In the first four cases we used a Silastic calibrated-leak balloon (Cook Inc., Bloomington, IN). In the other six cases we used a latex calibrated-leak balloon glued to Silastic tubing. The development of this new balloon has led to an apparently safer and more controlled approach. This balloon retains its same size and degree of leak throughout the procedure, which can be as long as 3–4 hr. This physical property minimizes the risk of over-inflation and rupture of the balloon in the arterial feeder [16, 17]. The size of the balloon and its leak can be modified in relation to the size of the feeders to be embolized.

The calibrated-leak balloon was then manipulated as close as possible to the AVM, being careful not to enter the AVM's nidus. A preembolization superselective angiogram through the calibrated-leak balloon was always obtained.

If normal cortical branches were not seen and if no neurologic symptoms occurred during the superselective angiogram, the ionic contrast medium was rinsed from the microcatheter with 5% dextrose in preparation for the IBC-2 (bucrylate, Ethicon, GmbH, Hamburg, W. Germany) embolization. From 0.35 to 1.2 ml of a tantalum-opacified IBC-2/iophendylate mixture was then injected through the microballoon under direct fluoroscopic control. The amount of IBC-2 delivered in each injection was not predetermined, but varied on the basis of the fluoroscopic image of the injected IBC-2 column. When the column stopped progressing and started approaching the balloon, the injection was immediately discontinued, and the balloon was rapidly deflated and removed by pulling the introducer in the groin. Check angiograms always followed the embolization. In some cases a second embolization was done at the same sitting, and in others the second embolization was performed after 2–3 days. This decision was based on the postembolization morphology of the AVM and on the patient's clinical status. The embolization therapy continued if the patient was still able to cooperate fully with the therapist and if postembolization angiograms showed a high-flow AVM with feeders suitable for catheterization. In two patients, three embolization procedures were carried out and in one patient four procedures were carried out.

Intraoperative Approach

This approach was chosen for four cases (table 1) in which it was impossible to catheterize the AVM's arterial feeder with a transfemoral approach (combination of the AVM's peripheral location and long serpiginous arterial feeders). The technique was very similar to the one described by Cromwell et al. [20]. A cortical arterial feeder was surgically exposed as close as possible to the AVM and was cannulated with a 3 French catheter (fig. 1A). A selective angiogram through the cannulated feeder was obtained under direct fluoroscopic control and video recording. This fluoroscopic angiogram verified that the catheter was in an artery and not in a vein, and that the contrast medium was being directed into the AVM. An estimation of the arterial/venous transit time was also made and it was confirmed that there was no reflux of contrast material proximal to the clip or ligature occluding the feeder.

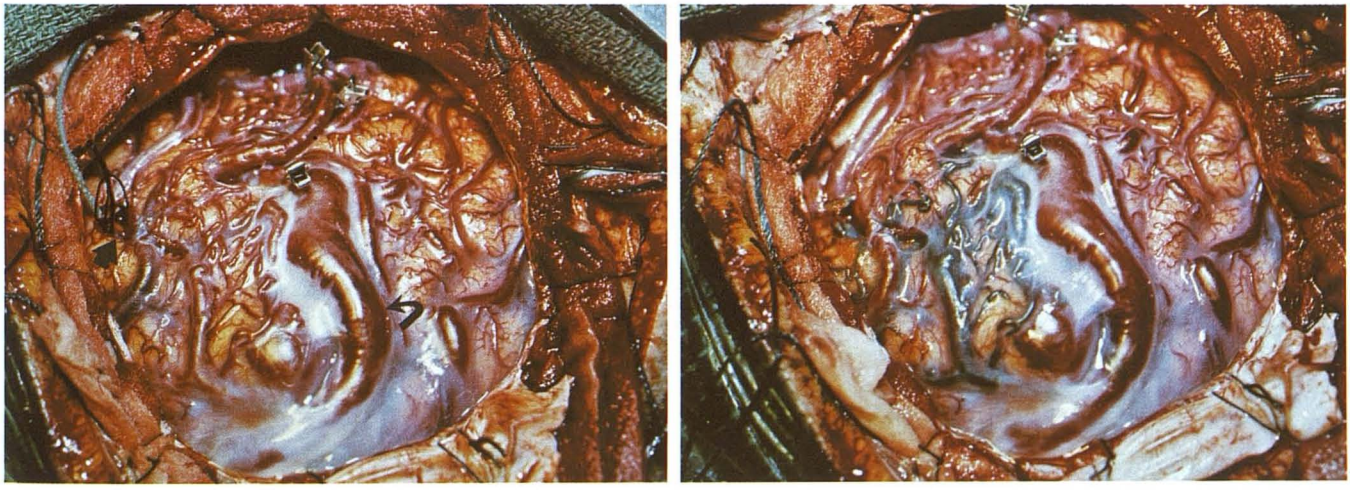
The tantalum-opacified IBC-2/iophendylate mixture was injected through the catheter under direct fluoroscopic control; it was usually followed by dextrose, insuring catheter patency. An immediate postembolization fluoroscopic angiogram was then obtained. At that time, the procedure was either over, or catheterization and embolization of another cortical arterial feeder was performed.

In one patient, the intraoperative AVM embolization was performed with the patient fully awake in order to monitor her neurologic clinical status. With this technique, we were able to embolize four cortical feeders of an AVM located in the left rolandic and immediate postrolandic region. This is probably the ideal, safest approach to intraoperative embolization of AVMs (fig. 1B).

In one of the four patients the AVM was subsequently excised surgically. In the other three patients the AVM was not excised.

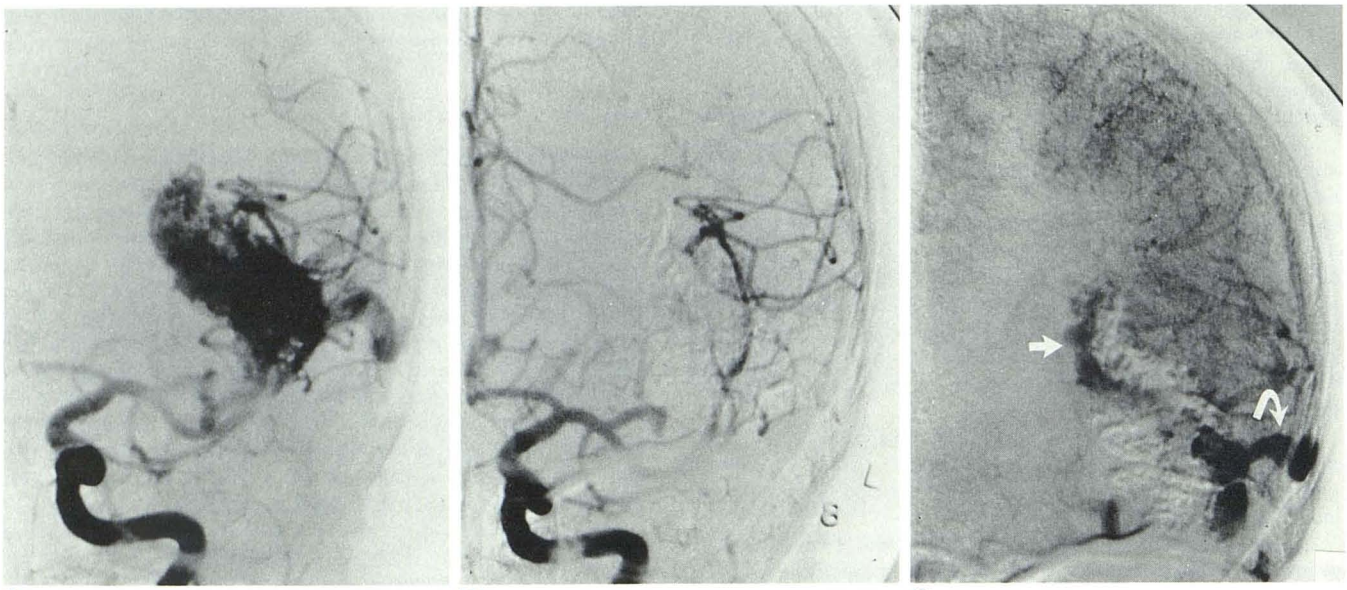
Combined Transfemoral/Intraoperative Approach

This was used in two cases (table 1) in which only palliative transfemoral embolization of the arterial feeders, which were most difficult to expose intraoperatively, was performed. The emboliza-



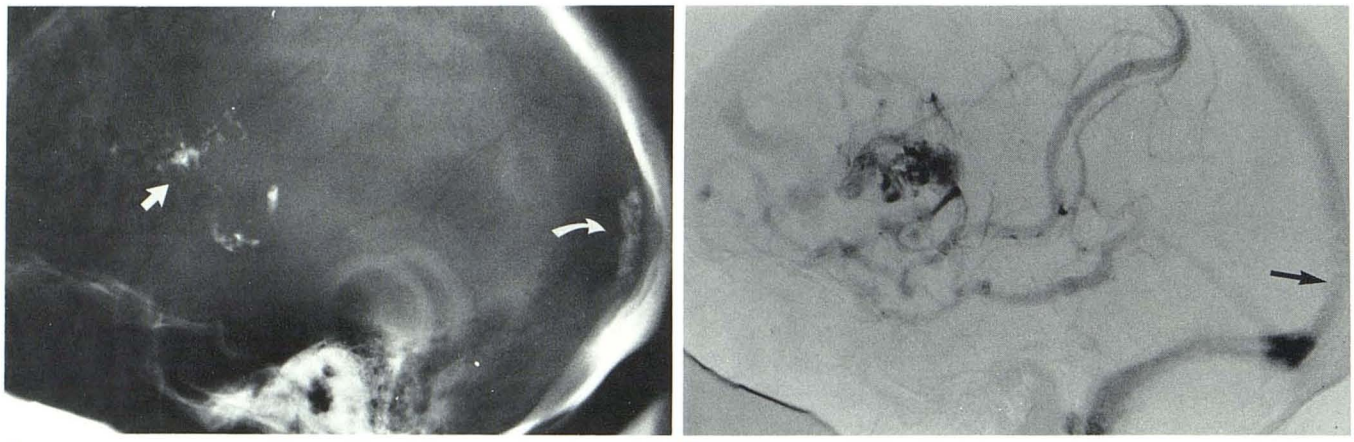
A B

Fig. 1.—Case 6. Surgical exposure of left retrorolandic AVM in patient awake throughout the procedure. **A,** Surgical clips obliterate some cortical feeders. Cannulation of feeder with 3 French catheter (*straight arrow*). Large draining vein in middle of surgical field (*curved arrow*). **B,** Embolization of three cortical feeders. Arterial feeders now filled with cast of IBC-2.



A B C

Fig. 2.—Case 10. Left inferior frontal AVM, deeply situated in sylvian fissure, obliterated by embolization. **A,** Preembolization. Postembolization angiograms in early **(B)** and late **(C)** arterial phases show obliteration of most of AVM's nidus. Its most medial part is still patent (*straight arrow*). Contrast stasis in large cortical draining vein (*curved arrow*).



A B

Fig. 3.—Case 14. Embolization of superior sagittal sinus. **A,** Postembolization skull film. Radiopaque IBC-2 in left frontal AVM nidus (*straight arrow*). Tubular cast of opacified IBC-2 in occipital region (*curved arrow*). **B,** Postembolization angiogram, late venous phase. Tubular cast of IBC-2 observed in **A** is in superior sagittal sinus proximal to torcular Herophili (*arrow*). No complications ensued.

tion was carried out in order that the surgeon would be left with the more easily accessible arterial feeders of the AVM (pericallosal artery embolized in case 2 and, posterior cerebral artery embolized in case 11).

Morphology and Topography of the AVMs

Sixteen AVMs were cortical in location and four had a deep component as well. Six AVMs involved the rolandic and speech areas, three had a frontal prerolandic location, three involved the midfrontoparietal or midparietooccipital regions, and four AVMs involved the lateral parietooccipital regions.

Results

The postembolization clinical and morphologic results of the three different technical approaches are tabulated (table 1).

Transfemoral Approach

About 45%–70% of the AVM was obliterated in six cases, 75%–90% of the AVM in three, and 95% in one (fig. 2). The superior sagittal sinus was accidentally embolized in one

case (case 14, fig. 3). This patient did not have any postembolization clinical deterioration.

Six cases had no postembolization neurologic deficits, two cases (cases 1 and 4) manifested mild neurologic deficits that disappeared completely in 48 hr, and two cases developed complete homonymous hemianopia after embolization of parietooccipital AVMs (cases 7 and 12). Computed tomography (CT) of cases 1, 4, 7, and 12 showed development of persistent low-density areas. CT scanning alone was insufficient to differentiate between postembolization vasogenic brain edema and infarct. The patient's clinical evolution was the final clue to this differentiation.

Intraoperative Embolization

In one case, there was 100% obliteration of the AVM (fig. 4). In another case, after embolization and obliteration of 75% of the AVM, a complete surgical removal was carried out. In two cases, 95% obliteration of the AVM was obtained and surgical resections were not carried out. In case 6, embolization of four arterial feeders of a rolandic AVM was carried out with the patient awake and responding to verbal commands.

The four intraoperative embolizations were followed by postoperative mild neurologic deficits with full recovery in cases 6, 8, and 13 and almost complete recovery in case 16. Case 13 showed complete clinical recovery 6 months later. Again, CT scans in cases 6, 8, and 13 showed new areas of low density produced by brain vasogenic edema and/or infarct. It was difficult to distinguish between surgical and embolization complications. A severe neurologic deficit 12 hr after embolization was seen in case 16. The neurologic deterioration was produced by a postoperative intracerebral hemorrhage and documented by CT (fig. 5); whether this was the result of surgery or embolization cannot be determined. This patient had only a mild expressive aphasia 6 months after embolization.

Combined Transfemoral/Intraoperative Approach

One patient (case 2) had a transfemoral embolization of the left pericallosal artery with complete surgical resection of left midfrontal interhemispheric AVM 24 hr later. The other case (case 11) had 50% obliteration of the AVM through embolization of the left posterior cerebral artery and complete surgical resection of the remaining AVM nidus 48 hr later (fig. 6).

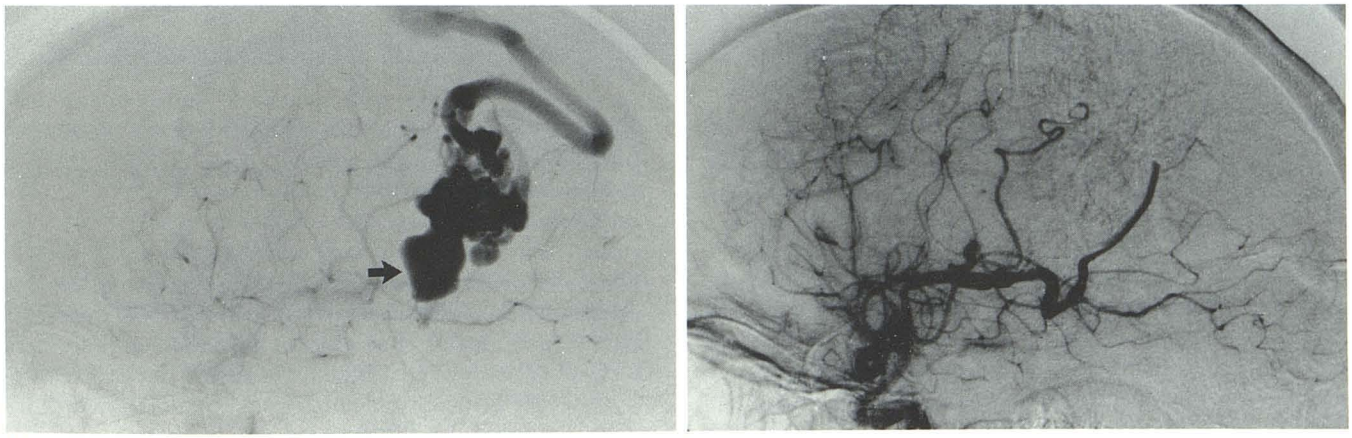
Neither of the two cases had neurologic deterioration after the transfemoral embolization or postembolization surgery.

Discussion

Transfemoral or intraoperative embolization of dominant-hemisphere AVMs, including those involving the rolandic and speech areas, is now possible with the development of safer techniques. The relatively high risk of producing a neurologic deficit when treating these lesions by surgery has forced the development of careful, critical analyses of treatment alternatives. If possible, it is preferable to embolize these lesions with the patient fully awake in order to detect minimal subjective neurologic changes such as headache of sudden onset and contralateral numbness or heaviness, which frequently precede an objective and severe neurologic complication.

Pre- and postembolization steroid therapy is also essential to decrease postembolization edema [20, 21].

It is considered of utmost importance to perform superselective angiography immediately before embolization. This angiogram is obtained to document the location of the balloon, which should be as close as possible to the AVM's nidus, thus reducing the possibility of embolizing undetected normal cortical vessels. The balloon must not, however, enter the AVM's nidus because inflation in that position may produce rupture of abnormal vessels. When inflated it



A B

Fig. 4.—Case 13. Retrorolandic suprasylvian AVM with postembolization edema. **A,** Preembolization angiogram, late arterial phase. AVM nidus with large dilated vein on its most inferior aspect (*arrow*). **B,** Postsurgical embolization angiogram. Complete obliteration of AVM nidus. Regional mass effect manifested by stretching and draping of middle cerebral cortical branches. Postembolization CT showed development of regional "vasogenic edema."

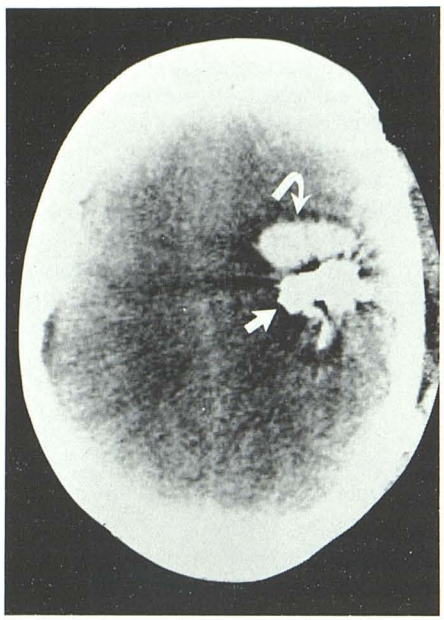
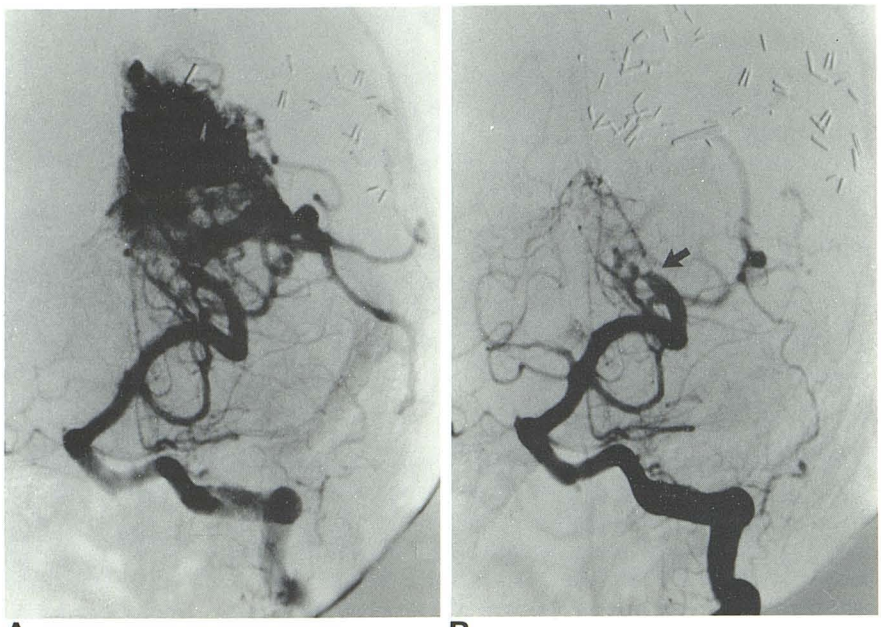
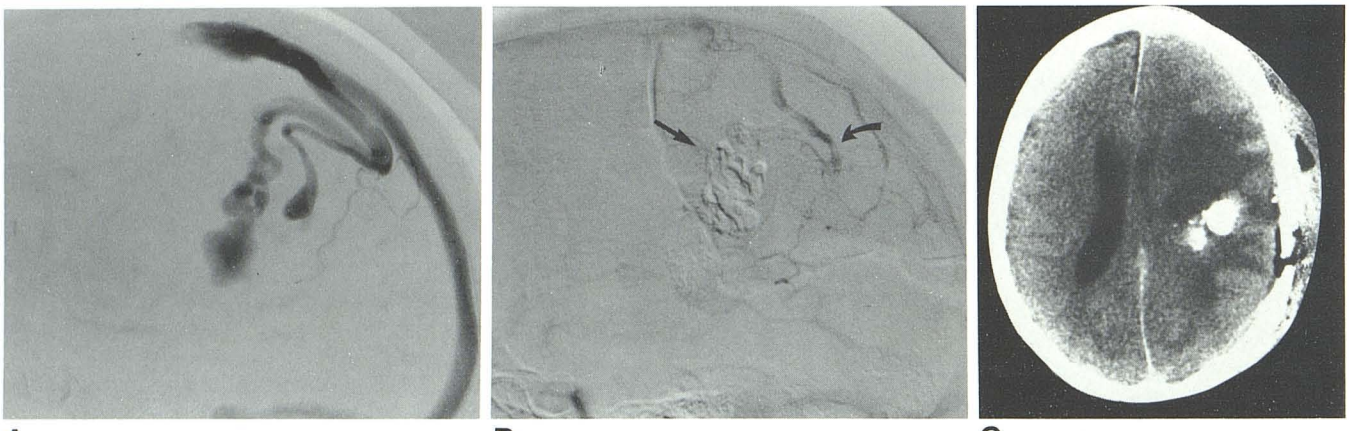


Fig. 5.—Case 16. Postembolization hemorrhage. Postembolization CT scan. Cast of IBC-2 in AVM nidus (*straight arrow*). Intracerebral hematoma is anterior to it (*curved arrow*). Hematoma was not present in preembolization CT scan.



A B

Fig. 6.—Case 11. Occipital AVM treated by embolization and resection. **A,** Preembolization vertebral angiogram. Left occipital AVM supplied by large left posterior cerebral artery. Numerous surgical clips in dural AVM are from previous surgery. **B,** Postembolization angiogram and surgical resection. Complete resection of AVM nidus. IBC-2 occlusion of left posterior cerebral artery (*arrow*).



A B C

Fig. 7.—Case 13. Retrorolandic AVM with postembolization edema. **A,** Venous phase of preembolization angiogram. Left retrorolandic suprasylvian AVM with large dilated veins. **B,** Lateral view of postembolization angiogram. Cast of IBC-2 in AVM nidus (*straight arrow*). Evidence of partial embolization of venous drainage (*curved arrow*). (AP view showed downward displacement of sylvian triangle by suprasylvian mass.) **C,** Postembolization CT scan. Cast of IBC-2 in AVM nidus. Development of low-density areas probably due to edema not seen on preembolization scan.

should completely block the arterial feeder so that the decrease in flow through the nidus of the AVM can be determined and the possibility of embolizing the venous drainage minimized. The proportion of the AVM that can be embolized is estimated from this superselective angiogram. Estimation of the arterial-venous transit time is done from this angiogram as well. The IBC-2 polymerization time can then be modified appropriately [21]. The development of neurologic signs and symptoms can be observed during this superselective angiogram. When contrast medium is injected into a normal cortical branch the patient usually has some neurologic signs. When the calibrated-leak balloon is positioned in an arterial feeder providing no important branches to normal brain cortex, the patient remains asymptomatic. The hemodynamics may change during IBC-2 injection, however, heightening the risk of occluding normal cortical vessels despite their absence on the superselective angiogram.

We strongly believe that the principal goal of AVM embolization therapy is occlusion of the AVM's nidus, not

proximal surgical, particulate, or balloon occlusion of the AVM's arterial feeders.

In this series 100% occlusion of the AVM's nidus was obtained in only one case in which the intraoperative technique was used. However, the AVM's nidus was reached in all the cases, with obliteration of 75%–95% of the nidus in seven cases. When neither the topography nor the size of the AVM precluded surgery, the patients were taken to the operating room and controlled dissection and resection of the remaining nidus of the AVM was performed.

Complications may be divided into two groups. The first group comprises complications produced by occlusion of normal cortical vessels at the time of embolization (cases 7 and 12). The second group comprises delayed complications due to either the probable development of edema (cases 1, 4, 6, 8, 11, 13; Fig. 7) or a postoperative intracerebral hemorrhage (case 16). In the patients in whom immediate postembolization CT scans were obtained, new nonspecific low-density areas in the region of embolization developed. The patient's clinical evolution was the only clue for differentiating between postembolization brain vasogenic edema and infarct. Follow-up CT scans of those patients were not obtained.

Postsurgical or postembolization edema is a well described entity [9, 18]. It is probably produced by a combination of sudden removal of the AVM's sump effect and lack of regional vascular autoregulation of the surrounding normal brain [18]. The production of edema involving the rolandic and speech areas explains the delayed appearance of transient neurologic deficit in some of our patients. All the patients recovered within 48 hr or less.

We observed a catastrophic postoperative hemorrhage in one of our cases 12 hr after operating room embolization. A postoperative CT scan showed bleeding in the area of the AVM's nidus and postoperative angiograms showed obliteration of 95% of the nidus of the AVM, patency of one small arterial feeder, and partial occlusion of the AVM's main draining vein. It is possible that partial obliteration of the venous drainage with preservation of one patent arterial feeder could have produced a sudden increase in pressure within the remaining nidus of the AVM with concomitant rupture of one or several abnormal vessels. Embolization of the venous drainage is not always accompanied by deleterious complications. It may or may not be significant depending on the size and dynamic importance of the embolized vein and on flow changes through the nidus of the AVM. As a rule, it is important to try to avoid embolization of the AVM's venous drainage. This may be accomplished easily in single-feeder AVMs, but it may be difficult in high—flow AVMs with multiple large feeders. An alternative consideration might be to perform an operating room embolization under controlled cardiac arrest, but this complex technique requires intensive laboratory work to develop experience with IBC-2 embolization in a static vascular system.

To conclude, the location of cerebral AVMs in the dominant hemisphere is no longer a contraindication to embolization. Transfemoral, intraoperative, and/or combined techniques of embolization can be used with the goal of obliteration of the nidus of the AVM. Preembolization, superselective angiography through the balloon microcatheter is an essential tool in understanding the dynamics of the AVM and in avoiding unnecessary complications. Intraoperative embolization with the patient awake is ideal for performing a fully controlled AVM nidus obliteration. Postembolization transient neurologic deficit is not an infrequent finding with dominant-hemisphere AVMs and is mostly produced by transient vasogenic edema. The embolization of the venous drainage of the AVM's nidus may be deleterious and should be avoided.

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