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# Microvascular Embolization of Arteriovenous Malformations: Predicting Success by Cerebral Angiography

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A retrospective and prospective analysis of cerebral arteriograms in 28 patients with intracerebral arteriovenous malformation (AVM) was undertaken to identify those parameters that predict the efficacy of small silicone particle embolization as a method of treatment. It was noted that when the number and caliber of the arterial feeding vessels were greater than the number and caliber of the draining veins the embolization was successful (15 of 16 patients). On the contrary, when the number and caliber of the draining veins exceeded that of the arteries the embolization failed (seven of seven patients). In four of five patients with nearly equal ratios, the embolization reduced the flow but not the size of the malformation.

For many years, arteriovenous malformation (AVM) of the brain was considered untreatable. Neurosurgical advances now permit management of an increasing number of AVMs by total excision, arterial embolization, or a combination of both. Since surgical removal of moderate or large AVMs is inapplicable in many locations, embolization techniques have been developed to reduce the size and the blood flow to these lesions. Cerebral angiography permits precise anatomic evaluation of this anomaly and is therefore indispensable in the pretreatment work-up. Furthermore, transfemoral catheter embolization of cerebral AVMs under angiographic control is now an important adjunct to successful surgical treatment [1-5].

In a series of 28 consecutive patients who underwent embolization at our institution, we observed that although the same technique was used in all cases, the results varied considerably. In this paper, we report our evaluation of angiographic features that predict our success in embolization treatment of AVMs.

## Materials and Methods

During a 2 year period, 28 consecutive patients were treated for intracerebral arteriovenous malformations (AVM) by transfemoral catheter embolization at University of Chicago Hospitals and Clinics. Most were referred from other institutions with headaches, seizures, hemorrhage, or progressive neurologic deficits.

Diagnostic cerebral angiograms and conventional anteroposterior and lateral views were obtained in every patient. In some patients lateral stereoscopic, oblique, or basal views were also obtained. All the vessels supplying the malformation were studied, including those that originated from the external carotid artery (three cases).

The embolization technique has already been described by Mullan et al. [6]. Briefly, we use small particles of silicone sponge of less than 1 mm to occlude the abnormal vessels

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within the malformation rather than to obliterate the larger feeding arteries [7-9]. Each particle is loaded into an 18 gauge stub-adaptor, connected to the transfemoral arterial catheter and flushed into it with 1-2 ml of saline. Thus the silicone particles are introduced singly, permitting precise monitoring of the embolization. Each patient had one or more embolization treatments separated by days, weeks, or months. As few as seven and as many as 410 emboli were injected during each treatment. Two to six angiograms were made in the course of each embolization session to observe progressive obliteration.

The end point of embolization varied in each patient. For some patients the procedure was terminated after diminution of the AVM or a decrease in the flow of contrast medium through the feeding arteries to the lesion. In others, it was terminated because of the appearance of an abnormal sign or symptom (numb sensation, weakness, flash of light, dizziness, or difficulty with words). These signs or symptoms were always transitory and disappeared within a few seconds or minutes. Occasionally a procedure was terminated because the patient became fatigued or because he had been given the arbitrary upper limit of contrast (100 ml of Conray 60). In none of our patients were there major or long-lasting complications.

Preembolization angiograms were analyzed in each patient. We counted the number of feeding arteries and draining veins. From these an arteriovenous (AV) ratio was calculated. The diameter of the feeding arteries and draining veins were measured with an optically magnified meter. The diameters of the arteries were measured close to their entries to the AVM and the diameter of the veins close to their exits from the AVM. These figures were added to give the total diameters of the arteries and the total diameters of the veins. From these an AV ratio of the total diameters was calculated. Finally we correlated both AV ratios with the outcome of embolization as seen on postembolization angiograms.

In 21 of 28 patients, diminished AVM and favorable hemodynamic changes were found. The latter were manifested by opacification of arteries previously not visualized, improved circulation to the normal brain, decrease in the flow of blood to the AVM, and increase in the circulation time through the malformation.

## Results

From our material we distinguished three groups of patients. In the first group of seven, embolization failed to diminish the AVM or the flow of blood to the lesion (table 1, figs. 1 and 2). In these patients the number and total caliber of the draining veins was greater than the number and total caliber of the feeding arteries. Both AV ratios were less than 1 and the intra-AVM communications were judged to be large. Angiography after completion of the embolization showed patency of all normal vessels, although in most of this group an unusually high number of silicone emboli was used. Thus, most injected emboli probably passed through the AVM and lodged into the pulmonary circulation. Cases 1 and 2 are typical examples of unsuccessful embolization (figs. 1 and 2). In both cases, the number and total caliber of the draining veins exceeded those of the feeding arteries. In case 1, 370, and in case 2, 204 emboli were injected, but in both, pre- and postembolization angiograms were identical.

In the second group (five patients), four had feeding arteries of about equal number and caliber to the draining veins. The fifth had larger AV ratios. The embolizations were judged to be moderately successful. The circulation to the

TABLE 1: AV Ratios Relative to Results of Embolization

Case No.	Age (yrs)	AV Ratio	Caliber AV Ratio	No. Emboli
Embolization failure:				
1	51	1:2.2	1:3.5	370
2	29	1:3.3	1:4.1	204
3	25	1:2	1:2.7	7
4	33	1:2.7	1:3	25
5	59	1:2	1:2.4	410
6	23	1:5	1:3.4	175
7	63	1:2	1:4.1	250
Reduced blood flow:				
8	52	1.6:1	1.1:1	165
9	32	6:1	2.2:1	15
10	20	1.6:1	1:1	60
11	31	1.4:1	1.6:1	100
12	51	1:1.2	1:1.5	76
Diminution of AVM:				
13	21	7:1	1.7:1	207
14	37	5:1	1.6:1	139
15	38	8:1	1.6:1	113
16	26	5:1	1.2:1	89
17	41	2.5:1	1.5:1	32
18	22	3.5:1	1.2:1	180
19	25	2.7:1	1.4:1	73
20	31	3:1	1.5:1	15
21	32	2.3:1	1.6:1	122
22	27	5:1	1.5:1	35
23	21	4.6:1	1.6:1	30
24	24	2.5:1	2:1	63
25	32	4:1	1.7:1	97
26	33	6:1	2:1	50
27	52	5:1	1.8:1	100
28	20	1.1:1	1:1	25

Note.—Case 9 had a favorable AV ratio. The AVM was supplied by both anterior and middle cerebral arteries. Those branches that originated from the middle cerebral artery were successfully occluded. However, the overall size of the malformation was not reduced because of the alternate supply from the nonoccluded branches of the anterior cerebral artery. Case 28 had an equivocal AV ratio. See text.

normal brain was improved, the flow of blood to the AVM was reduced, but the size of the malformation was unchanged. We concluded that in these patients a few emboli were halted within the AVM and many passed through.

In the third group (16 patients), embolization was judged to be successful. In all, both the size of and blood flow to the AVM were reduced. The number and total caliber of the feeding arteries of 15 malformations were greater than the number and total caliber of the draining veins.

One malformation (case 28) showed arteries and veins of comparable number and caliber, but this patient had clipping of several feeding arteries before hospitalization at our institution. In every other case, both AV ratios exceeded 1. The intra-AVM communications were judged to be small and most of the emboli lodged within the lesion. Figure 3 shows a typical example of this group. There are many feeding arteries in this case, and their total caliber is larger than that of the single draining vein.

## Discussion

Several guidelines for successful embolization of arteriovenous malformations have been described. Luessenhop et al. [1] have pointed out that embolization is usually successful if the AVM is supplied by the middle cerebral



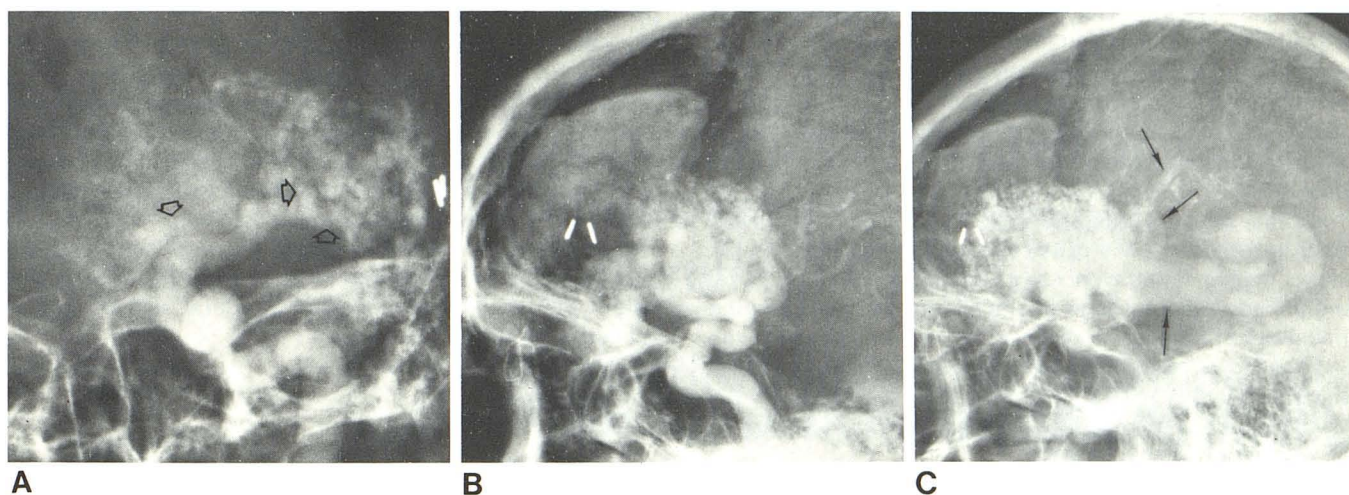


Fig. 1.—Case 1. Large arteriovenous communications; failure of embolization. AVM in frontal lobe with extension into basal ganglia. **A**, Three feeding arteries (arrows). **B** and **C**, Lateral views. Three of nine veins that drained

malformation (arrows). Arrows also indicate site where caliber measurements were made. Predominance of caliber of draining veins.

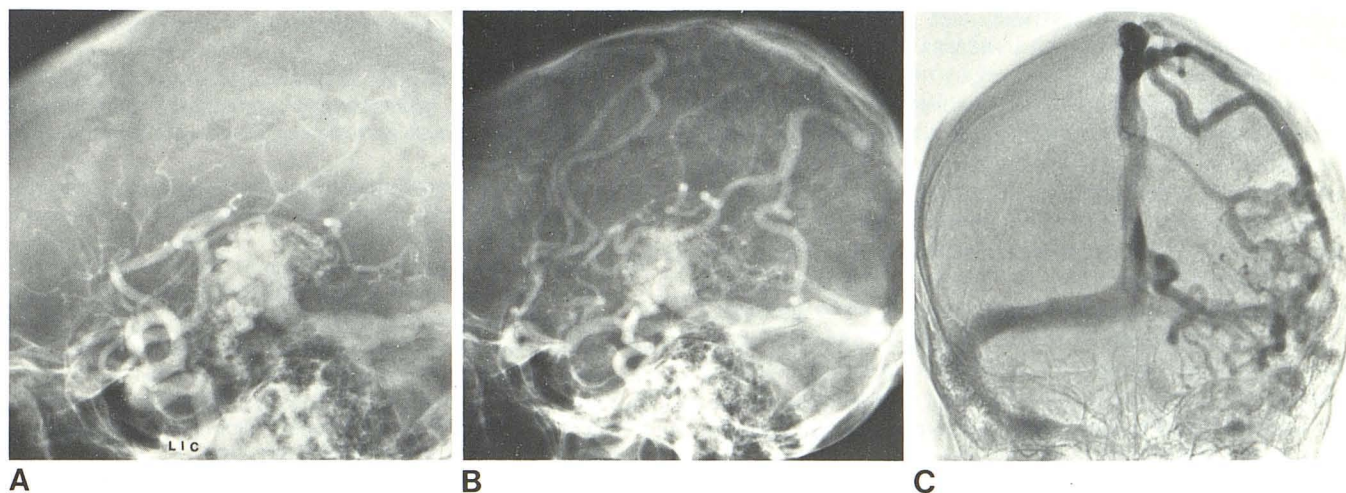


Fig. 2.—Case 2. Large arteriovenous communications; failure of embolization. AVM in temporal lobe. **A**, Two of three branches of middle cerebral

artery that supplied malformation. **B** and **C**, Several draining veins with number and total diameter greater than those of feeding arteries.

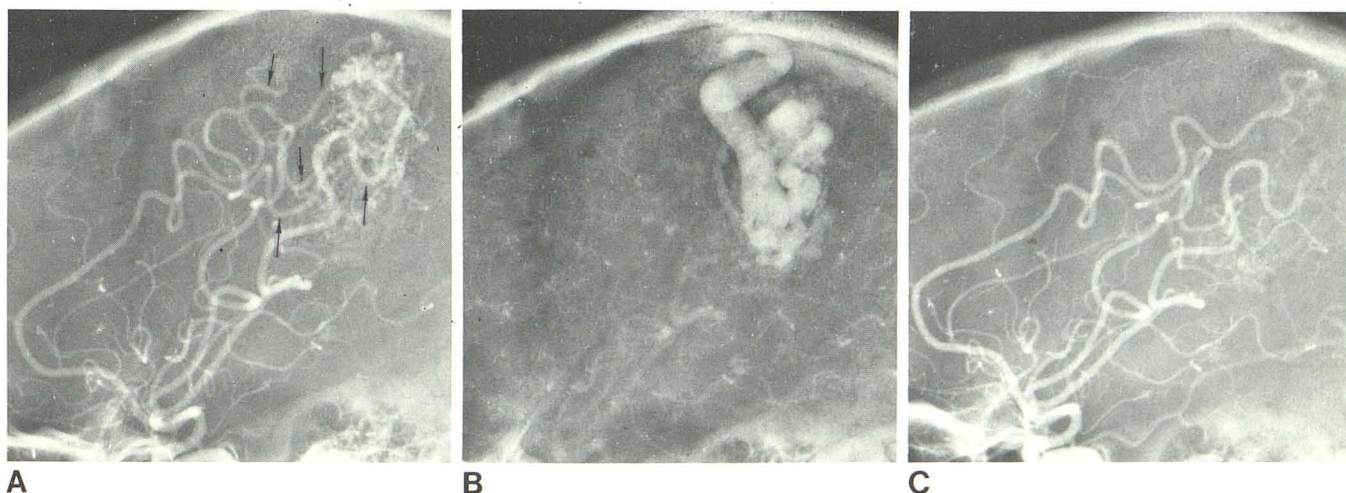


Fig. 3.—Small arteriovenous communications; successful embolization. AVM in parietal lobe. **A**, At least five feeding arteries (arrows), branches of anterior and middle cerebral arteries. **B**, Venous phase. One draining vein.

Both AV ratios are greater than 1. **C**, Postembolization angiogram. Decrease in size and blood flow of AVM indicates most emboli (total of 170) were halted within lesion.



artery and, less frequently, by the posterior cerebral artery. If the AVM is supplied primarily by the anterior cerebral artery, embolization will probably fail. Wolpert and Stein [8] recently emphasized the significance of the ratios of the caliber of the feeding AVM arteries to the caliber of the arteries supplying the normal brain. This ratio should be at least 4.5:1.

Another parameter known to influence embolization treatment is the size of the arteriovenous communications [9]. Large caliber arteriovenous communications (i.e., large shunts) allow the passage of emboli into the veins and the embolization may fail to diminish the lesion. But when the arteriovenous communications are small the emboli are stopped within the AVM and the results of embolization, if the other criteria previously described are also being observed, should be successful.

Factors determining the size of the arteriovenous communications are either congenital or acquired. Embryologic studies [10, 11] have shown that the genesis of AVMs is due to interruption of the normal maturation process of the capillary bed during the first weeks of fetal life. According to Kaplan et al. [12] the arteries may open into large sinusoids or alter their state and become veins without intervening capillary system.

The caliber of the abnormal capillaries may vary from patient to patient and also within the same lesion. An anatomic study by Hamby [13] describes four types of abnormal vessels within a malformation. The first type is a plexus of small, transparent, richly communicating vessels with uniform lumens 0.15–0.5 mm in diameter interposed between the arterioles and veins (fig. 3). The second type is a thin, sparsely branching looped vessel 0.3–0.5 mm in diameter, oriented longitudinally to the axis of the AVM. The third type, 0.4–0.7 mm in diameter, is relatively straight or loosely coiled. The fourth type, 0.3–1.0 mm in diameter, is extremely tortuous and convoluted. This type is delicate, easily broken, and the site of numerous small hemorrhages. It is obvious that when the fourth type of vessel prevails in a malformation, the shunt is large (figs. 1 and 2).

It has also been shown that the fragile walls of the abnormal vessels of the malformation or the dilated veins may rupture [13], which results in larger openings and greater arteriovenous communications. It seems that the caliber of communications increases with advancing age. Indeed, the mean age of patients in the first group was significantly greater ( $p < 0.05$ ) than in the patients of the third group who had small shunts and successful embolization.

Previously, the angiographic feature used to determine the size of the shunt was the initial appearance time of the veins in a serial angiogram. Although this is an important parameter, we believe that it is rarely appreciated, since a very rapid sequence angiogram (six films/sec) is infrequently obtained.

Our observations allowed us to estimate the size of the shunt. The AV ratios described above may be used to

predict the short-term results of embolization and to determine the best way of treating these patients with cerebral AVMs.

We have concluded that the microvascular embolization technique should not be used in any patient with unfavorable AV ratios. In those patients, larger emboli should be chosen. Also the AV ratios should be considered for the selection of patients to be embolized with liquified embolic materials, for it is possible that large solid parts of the injected material may escape the AVM through large openings within the lesion and lodge anywhere from the dural sinuses to the pulmonary arteries.

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#### REFERENCES

1. Luessenhop AJ, Kachman R, Shevlin W, Ferrero AA. Clinical evaluation of arterial embolization in the management of large cerebral arteriovenous malformation. *J Neurosurg* 1965;23:400–417
2. Djindkian R, Cophingnon J, Thelon J, Merland JJ, Houdart R. Embolization by superselective arteriography from femoral route in neuroradiology: review of 60 cases. *Neuroradiology* 1973;6:20–26
3. Robles C, Carrasco-Zanini J. Treatment of cerebral arteriovenous malformation by muscle embolization. *J Neurosurg* 1968;29:603–608
4. Kricheff II, Madayag M, Braunstein P. Transfemoral catheter embolization of cerebral and posterior fossa arteriovenous malformation. *Radiology* 1972;103:107–111
5. Pool JL. Treatment of arteriovenous malformation of the cerebral hemisphere. *J Neurosurg* 1962;19:136–141
6. Mullan S, Kawanaga H, Patronas NJ. Microvascular embolization of cerebral arteriovenous malformations. A technical variation. *J Neurosurg* 1979;51:621–627
7. Boulos R, Kricheff II, Chase NE. Value of cerebral angiography in the embolization treatment of cerebral arteriovenous malformation. *Radiology* 1970;97:65–70
8. Wolpert SM, Stein BM. Factor governing the course of emboli in the therapeutic embolization of cerebral arteriovenous malformations. *Radiology* 1979;131:125–131
9. Wolpert SM, Stein BM. Catheter embolization of intracranial arteriovenous malformations as an aid to surgical excision. *Neuroradiology* 1975;10:73–85
10. Evans HM. *The development of the vascular system: manual of human embryology*, vol 2. Keibel F, Mall FP, eds. Philadelphia: Lippincott, 1912:570–709
11. Arey LB. *Developmental anatomy. A textbook and laboratory manual of embryology*. Philadelphia: Saunders, 1954:106–107
12. Kaplan HA, Aronson SM, Browder EJ. Vascular malformations of the brain. An anatomic study. *J Neurosurg* 1961;18:630–635
13. Hamby WB. The pathology of supratentorial angiomas. *J Neurosurg* 1958;15:65–76