

Staged Treatment of Arteriovenous Malformations of the Brain

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Twenty-eight patients treated for arteriovenous malformations (AVMs) of the brain had staged therapy consisting of multiple surgical procedures or endovascular embolization followed by surgical treatment. There were 10 men and 18 women, aged 15 to 60 years (mean, 34 years). The clinical symptoms were those associated with intracranial hemorrhage in 13 patients, progressive neurological deficit not due to hemorrhage in 6, intractable headache in 5, and seizures in 4. Four groups were identified based upon the reason for staging therapy. Thirteen patients with large high flow AVMs (Group A) had staged treatment because of the risk of normal perfusion pressure breakthrough. The initial afferent artery occlusion was accomplished surgically in 9 patients and by endovascular embolization in 4. Postoperatively, no patient in this group had malignant cerebral edema or intracranial hemorrhage suggestive of normal perfusion pressure breakthrough, but 1 patient had an intraventricular hemorrhage after initial embolization. In 9 patients (Group B), the AVM had a complex multiple arterial supply that precluded resection from a single operative exposure. Seven had supratentorial AVMs, and 2 had AVMs of the posterior fossa. In 6 of these cases, the AVM was located in the midline and received bilateral arterial input. Six patients had staged surgical procedures, and 3 had an initial endovascular embolization followed by operation. Two patients had intracerebral hemorrhages, one after an initial surgical procedure and another after initial embolization. In 4 patients (Group C), the AVM had a major dural component that was treated separately from the parenchymal component. In 3 of these patients, embolization through the external carotid artery satisfactorily obliterated the dural component; in the remaining patient, a persistent internal carotid supply necessitated resection of the dural malformation. The parenchymal component was excised surgically in 2 patients. Two patients (Group D) had separate surgical procedures to treat an aneurysm associated with a parenchymal AVM. Overall, 19 of 28 patients had complete excision and 9 had partial obliteration of their AVMs. Late follow-up of 27 patients at a mean of 18.6 months showed that 16 patients were in excellent condition and 8 were in good condition. Three patients were in poor condition with debilitating neurological deficits. One patient had a delayed intracranial hemorrhage 22 months after incomplete obliteration of her AVM. Staged treatment of selected AVMs of the brain may avoid the occurrence of normal perfusion pressure breakthrough. This approach also allows satisfactory obliteration of selected malformations that have multiple complex arterial supplies or a dural component and those associated with an aneurysm. (*Neurosurgery* 21:314-323, 1987)

Key words: Arteriovenous malformation, Embolization, Normal perfusion pressure breakthrough

The treatment of large, complex arteriovenous malformations (AVMs) of the brain continues to challenge the limits of neurosurgical techniques. Certain AVMs are particularly difficult to manage. Those in which a large fistula causes a chronic vascular steal from the adjacent neural parenchyma are at risk of "normal perfusion pressure breakthrough," which may result in edema and hemorrhage of the surrounding brain if excision is attempted in a single stage (4, 6, 21, 22, 27-30, 40, 42). Some AVMs with a multiple complex arterial supply cannot be approached through a single operative exposure. Malformations that receive arterial supply through both parenchymal and dural vessels may be difficult to treat by surgical excision alone. When the AVM is associated with an intracranial aneurysm on an arterial feeding vessel, the lesions may best be treated independently. In all of these cases, the fistula may be satisfactorily obliterated by staged surgical procedures (11, 26, 30, 35-37, 42).

Recent advances in neuroradiological techniques have made possible selective catheterization of intracranial blood vessels and embolization of AVMs by the injection of particles (9, 18, 23, 27, 34, 43, 44) or polymers (3, 7, 12, 18, 38, 41) to occlude the feeding blood vessels or the fistula itself. Attempts have also been made to cannulate such vessels intraoperatively to embolize the AVM before excision (5, 8, 12, 38, 41). Although these maneuvers seldom obliterate the lesion completely (1, 7, 14, 15, 23, 27, 33, 38, 41, 43, 44), they are a valuable adjunct to surgical therapy in selected

cases and at times can take the place of an initial surgical procedure (8, 11, 12, 24, 32-34, 38, 40, 44).

In this report, we review our experience with staged neuroradiological and neurosurgical procedures in 28 patients with complex AVMs and discuss the indications for and the planning of their treatment.

SUMMARY OF CASES

Patient population

Between 1968 and 1985, 319 patients with intracranial AVMs were treated at the University of California, San Francisco, by one of us (C.B.W.). The 28 patients (8.7%) who underwent staged neuroradiological and neurosurgical procedures are the subject of this review. Patients with AVMs involving only the dura mater were excluded, as were those in whom one operation involved only evacuation of an intracranial hematoma and those in whom technical error during the initial operation necessitated reoperation for residual AVM.

There were 10 men and 18 women, aged 15 to 60 years (mean age, 34 years). Their clinical histories at the time of treatment are summarized in Table 1. Symptomatic intracranial hemorrhage was documented in 13 patients, 7 of whom had had multiple previous hemorrhages. Six patients had a history of transient or progressive neurological deficit unas-

sociated with intracranial hemorrhage. Four patients presented with seizures of recent onset, and 5 patients each had a history of intractable migrainous headaches associated with transient visual scotomata.

The location of the AVMs is shown in Table 1. Twelve patients had lesions involving primarily the frontal lobe; 11 AVMs were located primarily in the occipital lobe, and 3 were in the cerebellum. In 6 patients, the AVM was on or near the midline. The patients were divided into four groups according to the primary reason for staging treatment.

Group A: Normal perfusion pressure breakthrough

Thirteen patients had staged procedures to avoid malignant cerebral edema and hemorrhage at the time of excision as a result of normal perfusion pressure breakthrough (4, 6, 21, 22, 27-30, 40, 42). Patients were considered to be at risk for this complication based upon the angiographic criteria of a large AVM with multiple dilated feeding vessels, evidence of a large arteriovenous shunt, and poor filling of the adjacent normal cerebral vasculature (23, 29, 30, 35, 36, 42). Five patients also had a transient or progressive neurological deficit unrelated to previous intracranial hemorrhage; this clinical risk factor was thought to result from cerebral ischemia due to vascular steal through the fistula (29, 30, 35, 36). Four of these 5 patients had progressive hemiparesis and hemisensory loss that were referable to the site of a large hemispheric AVM; the remaining patient had progressive tetraparesis and akinetic mutism thought to be caused by a vascular steal from the basilar artery by a temporooccipital AVM. The AVMs involved the cerebral cortex in all 13 patients.

Staged surgical procedures. Nine patients were treated with staged surgical procedures (Table 1). AVMs chosen for staged surgery had three or more elongated feeding arteries that also supplied regions of functionally important parenchyma proximal or distal to the lesion. In these cases, flow-directed embolization of the AVM was considered dangerous because of the risk of embolic occlusion of vessels irrigating the adjacent brain. The goal of the initial operation was to divide feeding vessels at the edge of the malformation to preserve the parenchymal arterial supply and decrease blood flow through the fistula by at least 50%. To avoid the risk of hemorrhage in a dissection plane, the AVM was not dissected at this stage. Three patients had intraoperative cannulation of a feeding artery: two had embolization with isobutyl-2-cyanoacrylate (IBC) and one with gelatin sponge and tantalum dust. The following case illustrates treatment by multiple staged surgical procedures.

Case 1. A 41-year-old woman had been diagnosed as having an intracranial AVM at 14 years of age after the onset of grand mal seizures. The seizures had been controlled medically, and she had done well until the acute onset of transient facial hypesthesia on the left side 5 weeks before admission. A computed tomographic (CT) scan of the brain at that time showed no evidence of hemorrhage. An intraarterial digital subtraction angiogram (Fig. 1) showed a right frontoparietal AVM supplied by multiple branches of the middle cerebral artery. The right hemispheric parenchymal blood vessels filled poorly from a right internal carotid injection. There was cross filling of the malformation from both the left internal carotid artery and the basilar artery. Contrast material flowed rapidly from the feeding arteries into a large superficial venous varix, draining to the superior sagittal sinus and vein of Labbé. Two weeks before her admission to our institution, the patient had the insidious onset of weakness and hypesthesia, first in the left leg and then in the left arm.

At admission, right carotid and cranial bruits were detected by auscultation. The neurological examination showed a mild pyramidal motor deficit on the left side, which was worse in the leg than in the arm, and hypesthesia over the left hand and forearm. Because the arteries supplying the AVM were tortuous and appeared to supply the adjacent Rolandic cortex (Fig. 1B), occlusion of feeding arteries, rather than embolization, was selected as the first stage of treatment.

The patient was taken to the operating room. Through a frontotemporoparietal craniotomy, three dilated opercular branches of the middle cerebral artery were followed to their entry into the AVM and clipped at that point. Postoperatively, the patient's left hemiparesis worsened slightly over the next 24 hours. A CT scan showed a mild lucency adjacent to the AVM. The patient was treated with steroids and dehydrating agents, and the hemiparesis returned to the preoperative level within a week. A postoperative angiogram showed decreased flow through the AVM and improved filling of normal hemispheric blood vessels from the right internal carotid artery (Fig. 2). Three weeks after the first operation, the malformation was excised through the original exposure without difficulty. The large venous varix was partly thrombosed. Postoperatively, the patient again had a slightly worsened left hemiparesis, which resolved over 10 days. A postoperative angiogram showed complete excision of the AVM and normal filling of hemispheric blood vessels (Fig. 3). One year later, the patient is well except for residual hypesthesia of the left foot.

Embolization followed by operation. Four patients were treated with endovascular embolization before operation (Table 1). These patients had an arterial shunt through one or two markedly enlarged middle cerebral artery branches that directly terminated in the malformation. Parenchymal supply from these vessels proximal to the AVM appeared minimal, and there was no parenchymal supply distal to the malformation. These angiographic characteristics suggested that flow-directed embolization from the internal carotid artery could be performed safely.

Operative results. The AVM was excised completely in 8 of the 13 patients in this group. In 2 patients, massive AVMs involving the basal ganglia, thalamus, and central white matter of the dominant hemisphere were almost completely obliterated after vessel clipping and intraoperative cannulation for the injection of IBC and gelatin sponge; because of the location of these lesions, excision was not attempted. Three had only partial obliteration or excision of their AVM. In one of these patients, intraventricular hemorrhage after initial embolization resulted in a severe neurological deficit, which precluded further therapy. Of the five patients with progressive neurological deficits, 3 each had a reduction of the preoperative pyramidal deficit after initial partial occlusion of the fistula, suggesting reduction of cerebral ischemia. None of the patients in Group A had malignant "breakthrough" cerebral edema or hemorrhage, although 8 had mild transient worsening of their deficits. In 2 patients, CT scans done at the time of the deficit showed mild edema adjacent to the malformation.

Group B: AVM with multiple complex arterial supply

Nine patients had staged procedures because of the complexity of the arterial supply to the AVM. In each case, the AVM was supplied by multiple arterial sources that were distant from each other; at least one major source arose abruptly from the proximal vessels off of the circle of Willis or the basilar artery. The AVM was supplied by two major

TABLE 1
 Clinical Characteristics, Operative Treatment, and Outcome after Staged Treatment for Intracranial AVMs^a

Case	Age	Sex	Presenting History	Preoperative Neurological Deficit	AVM Site	First Procedure
<i>Group A: Normal perfusion pressure breakthrough</i>						
1	41	F	Progressive L hemiparesis	L hemiparesis	R frontoparietal	Feeders clipped
2	28	F	Transient R hemiparesis; aphasia	None	L frontotemporal	Feeders clipped
3	58	F	Previous hemorrhage; seizures	Homonymous hemianopsia	R frontotemporal	Feeders clipped
4	35	F	Headaches	None	R parietooccipital	Feeders clipped
5	15	M	Intracerebral hemorrhage	L hemiparesis	R temporoparietooccipital	Feeders clipped
6	29	F	Progressive R hemiparesis	R hemisensory loss	L basifrontal	Feeders clipped
7	26	M	Seizures	None	R temporoparietal	Feeders clipped
8	19	M	Recurrent intracerebral hemorrhage	None	R frontoparietal	Feeders clipped
9	26	F	Progressive hemiparesis	R hemiparesis	L frontoparietal	Feeders clipped
10	56	F	Progressive akinetic mutism and tetraparesis	Akinetic mutism and tetraparesis	R temporooccipital	Intravascular embolization
11	19	F	Seizures	None	R parietal	Intravascular embolization
12	29	F	Recurrent intracerebral hemorrhages	R hemiparesis	L frontotemporoparietal	Intravascular embolization × 4
13	17	F	Recurrent intracerebral hemorrhages	R hemiparesis	L frontotemporoparietal	Intravascular embolization
<i>Group B: Multiple complex arterial supply</i>						
14	27	F	Subarachnoid hemorrhage	None	Cerebellar vermis/medial hemispheres	SCA feeders clipped
15	30	M	Intraventricular hemorrhage	None	Corpus callosum/fornices	Feeders clipped
16	29	M	Hemorrhage; seizures	None	L parietooccipital	Feeders clipped/partial dissection
17	35	F	Subarachnoid hemorrhage	R hemiparesis; homonymous hemianopsia	L parietooccipital	Feeders clipped
18	23	F	R hemiparesis	R hemiparesis	L frontoparietal/corpus callosum	Lenticulostriate feeders clipped
19	23	F	Headaches	None	Bilateral parietooccipital with vein of Galen aneurysm	Feeders clipped
20	28	M	Subarachnoid hemorrhage	R visual scotoma	Corpus callosum/basifrontal	Selective intravascular embolization × 3
21	31	F	Recurrent intracerebral hemorrhage	None	L occipitoparietal	Feeders clipped
22	16	F	Intraventricular hemorrhage	L hemiparesis/ataxia	Cerebellar vermis/medial hemispheres	Intravascular embolization × 3
<i>Group C: Dural component</i>						
23	60	M	Headaches; transient homonymous hemianopsia	R inferior quadrantanopsia	L occipital	Intravascular embolization of dural-parenchymal AVM × 3
24	60	M	Headaches; progressive R hemiparesis	R hemiparesis	L occipital	Intravascular embolization of dural-parenchymal AVM × 2
25	58	M	Difficulty reading	Dyslexia	L parietooccipital	Parenchymal AVM excised
26	23	F	Headaches; visual scotomata	None	R occipital	Intravascular embolization × 3
<i>Group D: AVM with associated aneurysm</i>						
27	42	M	Intracerebral hemorrhage	None	L basifrontal AVM; L MCA aneurysm	AVM excised
28	57	F	Subarachnoid hemorrhage	L hemiparesis and dysphasia	R cerebellar AVM; R superior cerebellar artery aneurysm	Aneurysm clipped

^a Abbreviations: R = right; L = left; AVM = arteriovenous malformation; MCA = middle cerebral artery; PCA = posterior communicating artery; SCA = superior cerebellar artery.

TABLE 1—continued

Second Procedure	Third Procedure	Operative Result	New Neurological Deficit/ Postop Procedure	Follow-up (mo)	Outcome
<i>Group A: Normal perfusion pressure breakthrough</i>					
AVM excised		Complete excision	Transient hemiparesis	12	Excellent
AVM excised		Complete excision	Transient hemiparesis	42	Excellent
AVM excised		Complete excision	L hemiparesis	15	Poor
AVM excised		Complete excision	L hemiparesis	24	Good
AVM excised		Complete excision	None	6	Excellent
Feeders clipped		Partial obliteration	R hemiparesis	24	Excellent
AVM excised		Partial excision	Transient dysphasia	6	Excellent
Partial excision		Complete excision	Transient hemiparesis	6	Excellent
AVM excised		Complete excision	None	33	Excellent
Feeders clipped		Partial obliteration	Postop hemorrhage	12	Poor
AVM excised		Complete excision	None	12	Excellent
Craniotomy for embolization		Partial obliteration	Homonymous hemianopsia	36	Good
Intravascular embolization	Intraoperative embolization	Incomplete obliteration	Homonymous hemianopsia	33	Excellent
<i>Group B: Multiple complex arterial supply</i>					
AVM excised		Complete excision	None	12	Excellent
Partial excision		Partial excision	Homonymous hemianopsia	12	Good
PCA feeders clipped	AVM excised	Complete excision	Homonymous quadrantanopsia	36	Excellent
AVM excised		Complete excision	None	12	Excellent
Intraoperative embolization	AVM excised	Complete excision	Intraventricular hemorrhage	12	Good
Partial excision		Partial excision	None	Lost	
Partial excision	AVM excised	Complete excision	Abulia; hyperphagia; diabetes insipidus	24	Poor
Intravascular embolization	AVM excised	Complete excision	Diabetes insipidus; R homonymous hemianopsia	20	Good
SCA feeders clipped	AVM excised	Complete excision	None	12	Good
<i>Group C: Dural component</i>					
		Partial obliteration	None	16	Excellent
Feeders clipped		Partial obliteration	Improved hemiparesis	6	Good
Intravascular embolization of dural AVM	Dural AVM excised	Complete excision		48	Excellent
	AVM excision	Complete excision		12	Good
<i>Group D: AVM with associated aneurysm</i>					
Aneurysm clipped		Complete excision: aneurysm clipped	None	12	Excellent
AVM excised		Complete excision: aneurysm clipped	None	12	Excellent

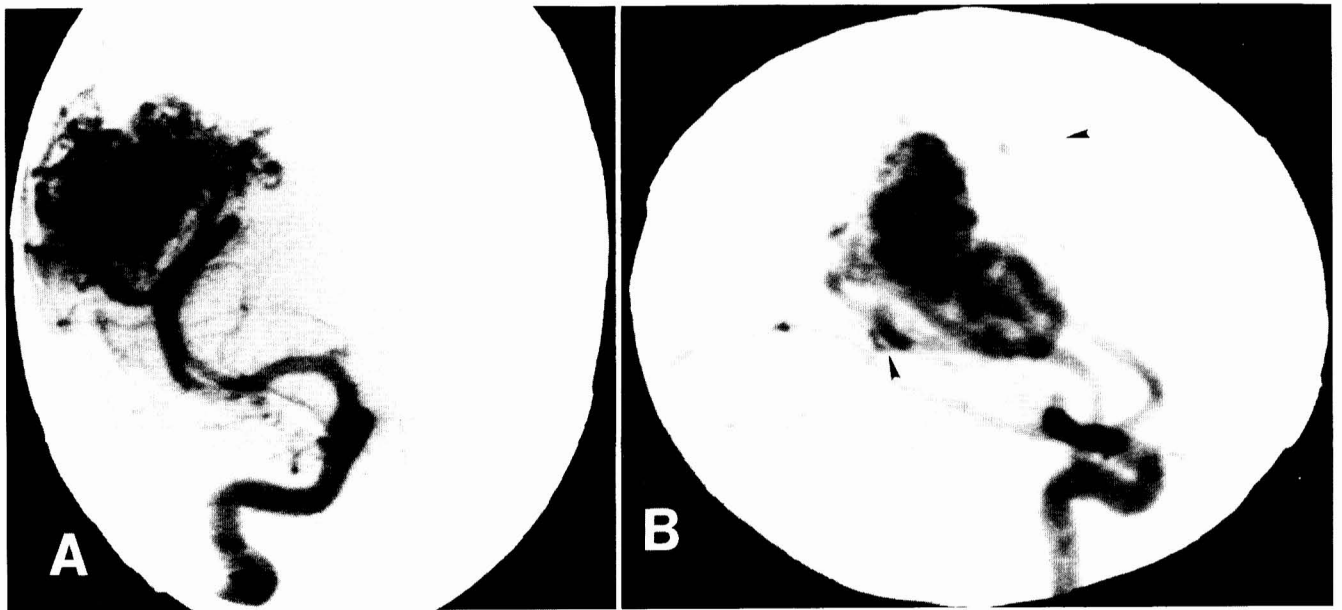


FIG. 1. Case 1. A right internal carotid intraarterial digital subtraction angiogram in anteroposterior (A) and lateral (B) views shows that the AVM is supplied by branches of the middle cerebral artery. Note poor filling of the adjacent cortical blood vessels and the anterior cerebral artery. Branches supplying the AVM also supply adjacent cortex at the periphery of the AVM (arrowheads).

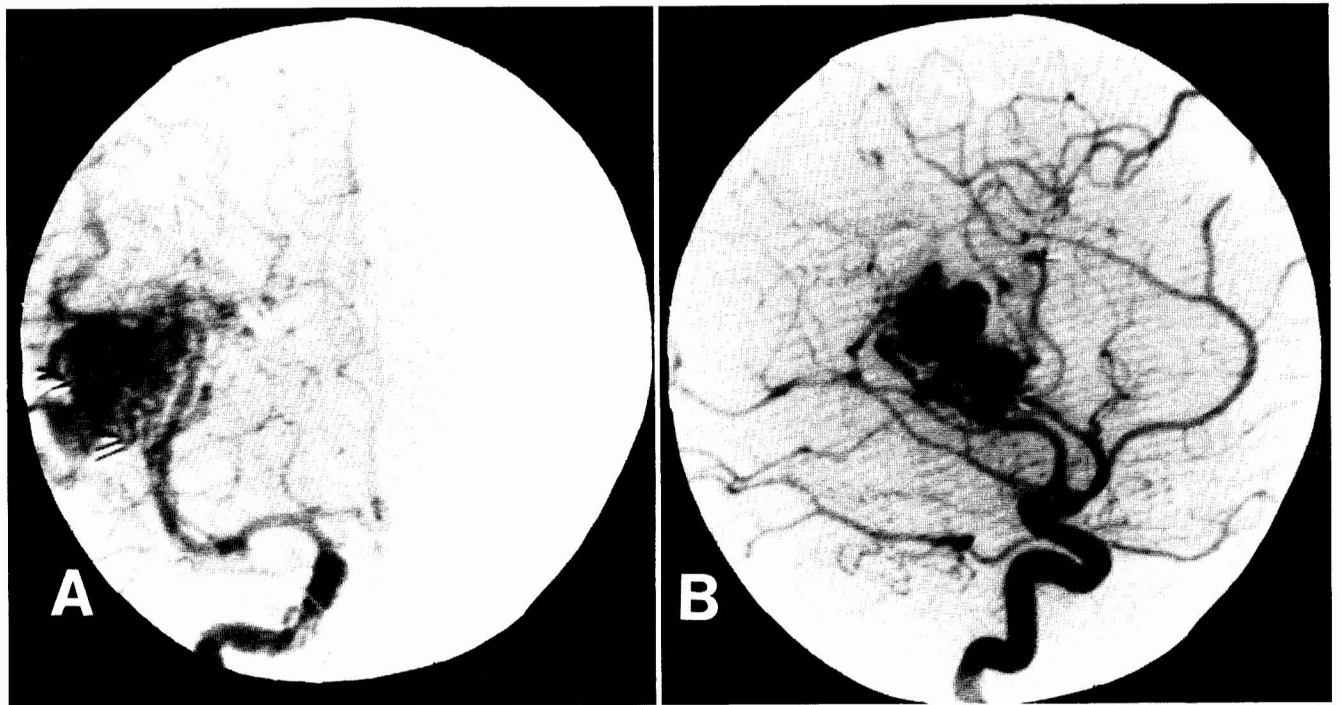


FIG. 2. Case 1. A right internal carotid digital subtraction angiogram in anteroposterior (A) and lateral (B) views after the first operation shows decreased flow through the malformation and improved filling of the adjacent cortical vasculature, including the anterior cerebral artery.

arteries in two patients, by three arteries in five patients, and by four arteries in two patients. In six patients, the lesion was located in or near the midline and received input from arteries from both hemispheres. The treatment for this group is detailed in Table 1. In five patients, the AVM was located in the medial frontoparietal or parietooccipital regions. In these cases, the major arterial supply from arteries of the proximal

circle of Willis was occluded through a subtemporal or pterional approach before excision of the AVM through a paramedian convexity approach. In two patients (Cases 14 and 22), the AVM was located within the superior cerebellum, and major supply from the superior cerebellar artery was occluded through a subtemporal transtentorial approach before excision of the AVM through a suboccipital approach.

Six patients underwent multiple surgical procedures, and three patients had an initial endovascular embolization followed by a surgical procedure. Two patients underwent the intraoperative injection of IBC for partial thrombosis of the malformation before excision.

Staged treatment resulted in complete excision of the AVM in seven patients and partial excision in two. The excision was incomplete in one patient because the AVM involved the roof of the 3rd ventricle and both fornices. In the other patient with a bilateral medial temporooccipital AVM draining into a large vein of Galen aneurysm, the size of the aneurysm limited exposure of the feeding arteries, resulting in persistent

drainage of a single medial posterior choroidal artery into the galenic venous system.

One patient had an intraventricular hemorrhage that resulted in hemiparesis several hours after the AVM had been partially dissected during an initial operation. Another patient had a hemorrhage of a cerebellar AVM 4 days after her second embolization, causing transient worsening of her ataxia and cranial nerve deficits. Six patients had new deficits after final excision of the malformation. In four, the deficits persisted into the late follow-up period.

Group C: AVM with a dural component

In four patients, treatment was staged because the AVM had a significant dural component or a major dural arterial supply. All four patients presented with headaches and visual symptoms (Table 1). The AVM involved the occipital lobe and the dura mater of the occipital convexity or the tentorium in each case (Fig. 4).

In three patients of Group C, the dural component was treated first by multiple endovascular embolization procedures; two patients also had embolization of the parenchymal component of their AVMs. The fourth patient had initial surgical resection of a parenchymal AVM; a dural component involving the tentorium was identified during the operation. Postoperative angiography revealed persistence of the dural AVM supplied by the occipital and middle meningeal arteries. These arteries were embolized; however, persistent supply from the meningohypophyseal artery could not be occluded, as this artery could not be selectively catheterized, and flow-directed embolization of the internal carotid artery would have affected the parenchymal vessels. A temporoparietal craniotomy was performed, and the residual AVM and adjacent tentorium were excised.

In two of the four patients, the dural and cerebral components of the AVM were completely obliterated. Two patients have persistent residual parenchymal AVMs after occlusion of the dural component. Both of these patients were 60 years old and neurologically intact and had only a history of headache without previous hemorrhage. Repeated endovascular embolization procedures for both the dural and parenchymal components of the AVM resulted in complete relief of chronic headaches in both cases. In one patient, a progressive hemiparesis thought to be due to vascular steal was also decreased

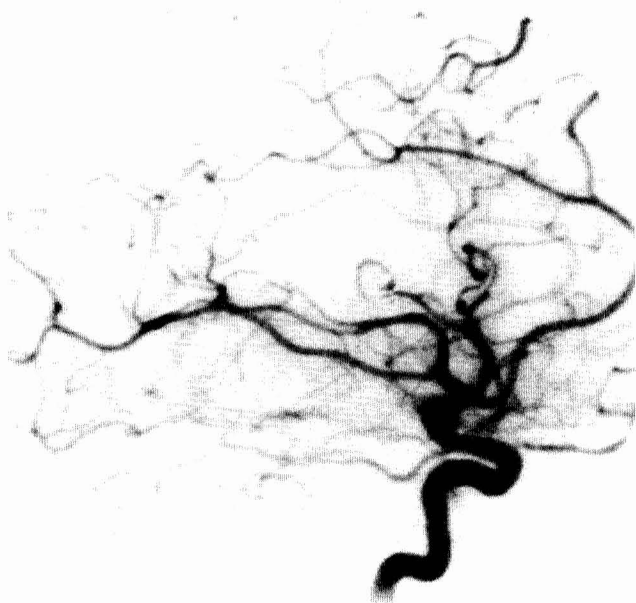


FIG. 3. Case 1. A right internal carotid digital subtraction angiogram, lateral view, shows complete excision of the AVM after the second operation.

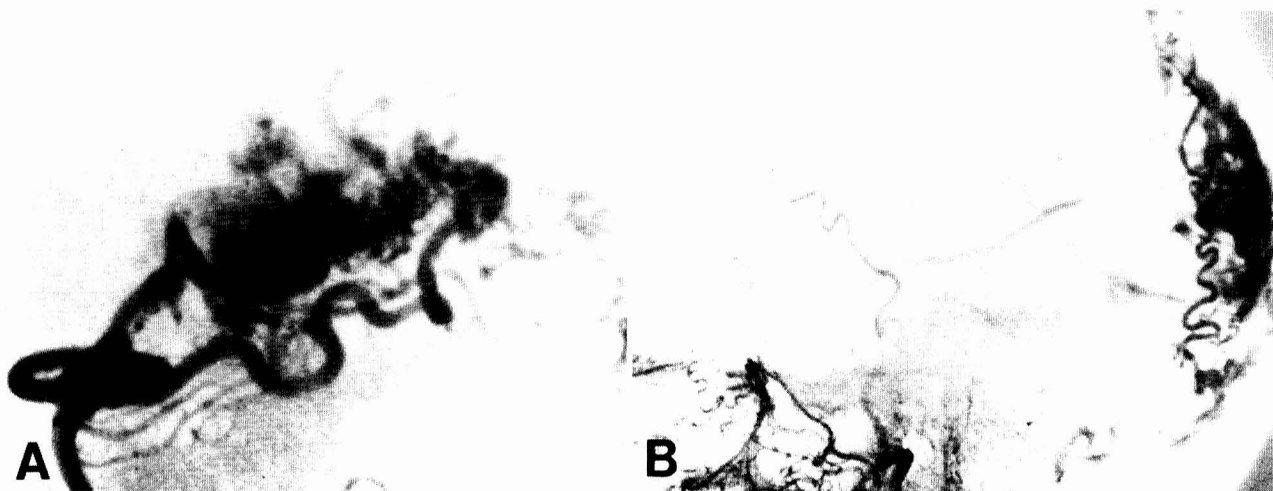


FIG. 4. *A*, left vertebral angiogram in the lateral view shows an arteriovenous malformation supplied by branches of the posterior cerebral artery. *B*, external carotid angiogram in the lateral view shows an occipital malformation with a dural component supplied by the occipital and middle meningeal arteries.

after treatment. No further treatment was pursued because the age of these patients and the absence of a history of bleeding were judged to outweigh the risk of deficit associated with excision of the AVMs from the dominant parietal lobe. One patient, after embolization of the parenchymal component of the AVM, developed a homonymous quadrantanopsia that persisted into the late follow-up period.

Group D: AVM associated with an aneurysm

Two patients had staged procedures because their AVMs were associated with an intracranial aneurysm and it was judged that each lesion required a separate approach for optimal exposure. In one patient, an aneurysm arising from the proximal superior cerebellar artery was approached and treated through a subtemporal transtentorial exposure before a second stage excision of a cerebellar hemispheric AVM. In the other patient, an initial bilateral subfrontal exposure of a medial frontal AVM was performed before a second stage pterional exposure of an aneurysm arising from the ipsilateral middle cerebral artery trifurcation. In each case, follow-up angiography showed complete obliteration of both lesions.

Early morbidity related to staged therapy

Early morbidity directly related to staged treatment in the 28 patients was limited to 3 episodes of intracerebral hemorrhage. Two hemorrhages occurred after initial embolization procedures, and 1 occurred after partial surgical dissection of an AVM with a diffuse arterial supply. None of the patients had infections or complications of wound healing.

Late follow-up

Twenty-seven of the 28 patients were available for late follow-up review at a mean of 18.6 months (range, 6 to 48 months) postoperatively (Table 1). Sixteen patients were in excellent condition and had either no neurological deficit or a minor deficit that did not interfere with normal activity. Eight patients were in good condition and able to live independently, but had neurological deficits. Three patients were in poor condition with severe neurological deficits. One had an intraventricular hemorrhage after an embolization procedure and remained confused and hemiparetic. Another had a residual severe spastic hemiparesis after a two-stage resection of a frontotemporoparietal AVM involving the central white matter. The third patient had an AVM with diffuse arterial supply in the basifrontal and hypothalamic region. After two embolization procedures and three craniotomies, the malformation was finally excised, but the patient is debilitated by dementia, diabetes insipidus, and hyperphagia. One patient who had a large AVM in the basal ganglia and central white matter of the dominant hemisphere had an intracranial hemorrhage 22 months after incomplete obliteration of the lesion. She recovered without incurring an additional neurological deficit and remains stable and in good condition 14 months later.

DISCUSSION

Although cerebral AVMs have been considered more benign than intracranial aneurysms, long term follow-up studies have suggested that AVMs carry a major risk of eventual hemorrhage, especially if they have bled previously (10, 11, 43). Up to 50% of symptomatic patients will die from or be impaired by the AVM during their lifetime (35). Complete surgical excision is the best treatment (8, 11, 32, 36, 42); clipping of feeding arteries or incomplete obliteration of the

fistula does not protect the patient from further episodes of bleeding (8, 12, 35, 36, 42-44). Surgical management may prove difficult if the malformation is large, if it has a diffuse arterial supply or a significant dural component, or if it is associated with an intracranial aneurysm. In such cases, obliteration of the fistula may best be achieved by multiple staged procedures.

Normal perfusion pressure breakthrough

The most common indication for staged treatment of AVMs in this series was the risk of normal perfusion pressure breakthrough. As first described by Spetzler et al. (30), abrupt occlusion of blood flow through a large, high flow AVM may result in malignant cerebral edema or uncontrollable hemorrhage in adjacent brain. This may occur because a vascular steal through the AVM causes ischemia in the adjacent parenchyma, which leads to a chronic loss of autoregulation. Structural changes in these arterioles and a permanent decrease in vessel resistance may also occur (29). Nornes and Grip showed that, when large, high flow feeding arteries are occluded at the malformation, their intraluminal pressure increases acutely (26). Abrupt termination of blood flow through the AVM results in increased perfusion pressure and blood flow through the passively dilated adjacent arterioles. Without the protection of autoregulation, the capillaries in the adjacent vascular bed may be unable to tolerate the suddenly increased perfusion pressure, resulting in edema and hemorrhage (29, 30). Despite frequent reports (4, 6, 22, 29, 30, 35, 42), the incidence of this phenomenon is unknown. Decreasing blood flow through such AVMs in stages provides a graded change in hemodynamics and may allow autoregulatory capacity to recover before the lesion is excised completely (22, 26, 29, 30, 35, 36). Perioperatively induced hypotension (6, 26, 29, 30), proximal internal carotid artery stenosis with an occlusive clamp (4), and pentobarbital-induced coma (6) have been used to avoid the breakthrough phenomenon.

Five of our patients had a history of progressive neurological deficit unrelated to hemorrhage. Cortical ischemia caused by a vascular steal through the malformation has been reported to cause such a deficit (22, 29, 30, 35, 36, 42) as a result of decreased perfusion pressure and ischemia in the adjacent brain (25, 26). Patients with such progressive deficits have been thought to be at particular risk of developing normal perfusion pressure breakthrough (29, 30, 35, 36). The angiographic indications of the potential risk for normal perfusion pressure breakthrough include the presence of a large high flow fistula, poor filling of adjacent normal cortical blood vessels (23, 29, 30, 35, 36, 42), and filling of the malformation from the contralateral hemispheric circulation through the circle of Willis (22, 42).

In our patients with these clinical and angiographic findings, the goal of the first stage was to occlude 50% or more of the arteries supplying the AVM, either surgically or by flow-directed embolization. After the initial procedure, at least 1 week was allowed for the adjacent parenchymal vascular bed to adjust to the increased perfusion pressure before further obliteration or excision of the AVM was attempted. No patient in this group developed malignant edema, although one had an intracranial hemorrhage after embolization. Hemorrhage is a well-recognized complication after embolization of AVMs (8, 9, 12, 16, 19, 23, 38). It may be caused by perfusion pressure breakthrough or by altered hemodynamics in the fistula and increased pressure in feeding arteries (26). Ischemia and infarction of adjacent cortex (1, 7, 12, 23, 38, 41, 43, 44) and cerebral edema (12, 38, 44) have also been reported after embolization of AVMs.

Three of the five patients in this group who had a progressive neurological deficit before treatment improved neurologically after flow through the malformation was decreased. U and Rosenberg also reported the complete resolution of a severe progressive hemiparesis after staged excision of a large AVM in the basal ganglia (36). Neurological improvement in these patients may have resulted from decreasing the vascular steal through the AVM, which improved perfusion of the adjacent cortex.

AVM with a multiple complex arterial supply

Nine patients in our series had staged treatment for AVMs with diffuse arterial supply because control of the major arterial supply to these malformations could not be achieved through a single operative exposure, and a single stage excision was judged unreasonably hazardous. Such cases included large AVMs that extended to the subependymal region or into the ventricular system, where bleeding from uncontrolled arterial supply could prove disastrous (31, 37). In this group of patients, the first stage generally consisted of exposing and occluding the less accessible feeding arteries that arose from the proximal circle of Willis or the basilar artery to prepare for excision of the malformation in a subsequent procedure.

Six patients had AVMs that received bilateral arterial input. In 2 cases, the AVM involved the vermis and both medial cerebellar hemispheres and was supplied by the superior cerebellar, anterior inferior, and posterior inferior cerebellar arteries bilaterally. Because one superior cerebellar artery was dominant, we occluded it through a subtemporal approach before excising the AVM through a suboccipital approach. Samson and Batjer noted that most AVMs involving the cerebellar vermis may have bilateral artery input (28). In their series of 15 patients, excision was generally attempted in a single stage; however, 3 patients required a second operation for complete excision of the malformation (28).

AVM with a dural component

Four patients in our series had AVMs consisting of both dural and parenchymal components. The dural component of a malformation may be supplied by meningeal branches of the external carotid, internal carotid, and vertebral arteries. Venous drainage is usually into the dural sinuses (24, 39, 40).

The dural component was treated separately from the parenchymal component. In three cases, endovascular embolization of the external carotid arterial supply was performed; in one, the dural component was excised because the meningo-hypophyseal arterial supply was considered unsuitable for embolization. AVMs involving the dura mater must be completely occluded or excised because clipping of feeding vessels alone leads to the rapid development of new arterial supply (8, 38). The treatment of dural malformations has been advanced by the development of endovascular embolization techniques. Particulates (20), liquid silicone (21), and IBC (39) have been used successfully to occlude dural AVMs. Vinuela et al. recently performed superselective catheterization of distal branches of the external carotid artery using detachable calibrated-leak balloons to deliver IBC directly into the nidus of the malformation (39).

AVM associated with an aneurysm

Two patients in this series each had staged treatment for an intracranial aneurysm associated with an AVM. Aneurysms may occur on arteries supplying intracerebral AVMs (11, 13, 17) or in sites remote from the AVM (2). Chronically increased flow through the arteries supplying the AVM has been

postulated to account for proximal aneurysm formation (2, 13, 17). The aneurysm must be considered in planning treatment of the malformation. Because pressure within feeding arteries increases after flow to an AVM is occluded (2, 25, 26), an aneurysm located proximally on a feeding artery may rupture if flow into the AVM is occluded first. Often, a proximal aneurysm can be approached and clipped and the malformation can then be excised through the same exposure in a single stage. In our two cases, a different exposure was needed for optimal exposure and treatment of each lesion. Batjer et al., reporting on 22 patients with AVMs and associated aneurysms, described the similar use of an initial procedure to obliterate the aneurysm, followed by elective resection of the AVM through a more optimal surgical exposure (2).

Embolization

In 12 of the 28 patients in this series, the initial procedure consisted of endovascular embolization with polyvinyl alcohol foam particles. In suitable cases, endovascular embolization has replaced open surgical procedures for the occlusion of selected feeding arteries or partial obliteration of the AVM (1, 3, 7, 14, 23, 27, 38, 40, 41, 43-45). Polyvinyl alcohol foam sponge (18), silicone elastomer spheres (7, 9, 11, 14, 23, 33, 43), and fluid silicone polymer (18, 21) have proven to be satisfactory materials for embolization.

Attempts have been made to define angiographic criteria that indicate the effectiveness and safety of embolization. Mullan et al. considered AVMs with a central nidus of small blood vessels and a slow arteriovenous shunt best suited to particulate embolization (23). Hilal suggested that it is safer to embolize AVMs with feeding arteries terminating in the malformation than those with feeding arteries continuing distal to the fistula to supply normal brain because of the risk that emboli will enter the parenchymal vascular bed in the latter case (14). Wolpert and Stein suggested that AVMs originating from a single main arterial trunk and having one or two large, relatively straight, short feeding arteries are best suited for particulate embolization (45). Embolization through the anterior cerebral artery has proved difficult because of the angle at which this vessel branches from the internal carotid artery (1, 43).

A more recent embolization technique is the injection of rapidly polymerizing IBC directly into malformations by flow-directed or superselective catheterization (1, 3, 7, 38, 41). Arteries supplying the AVM have been cannulated directly through a surgical exposure when adequate positioning of a flow-directed microcatheter could not be achieved transfemorally (5, 7, 12, 38, 41). We used this technique to embolize large feeding arteries in 4 cases; embolization was followed in 2 cases by excision of the AVM. Although the firm cast formed by the IBC partially obliterated the fistula, it made subsequent excision more difficult, as reported by others (12, 38). For large unresectable AVMs deep within the dominant hemisphere, Girvin and colleagues have found that direct cannulation of the vessel and embolization with IBC in the awake patient are useful (12). This method allowed subsequent surgical resection in 2 of 14 patients and resulted in at least 80% obliteration of the fistula in 10 of the remaining 12 patients.

Nine of 28 patients in this series had incomplete excision or subtotal obliteration of their AVM as a final result, most often because the nidus of the lesion was located within a critical structure in the dominant hemisphere, where complete excision was judged to carry an excessive risk of unacceptable neurological deficit. In these 9 patients, the goal of therapy was complete obliteration without excision, usually by com-

bined vessel clipping and embolization of the AVM. In 7 of these patients, each without a history of intracranial hemorrhage, the presenting neurological symptoms were reduced postoperatively. Because of the relatively lower risk of subsequent hemorrhage (10), either these patients refused further treatment or it was elected to follow them for further symptoms.

None of the 28 patients in our study had a postoperative infection or other complication directly attributable to multiple staged surgical procedures. Although many patients were discharged from the hospital between operations, the hospital stay was prolonged in most cases because treatment was staged. Nevertheless, we are convinced that this approach decreases the risk of disastrous complications, such as malignant edema or hemorrhage due to normal perfusion pressure breakthrough, and makes the excision of selected AVMs each with a diffuse arterial supply, a dural component, or an associated aneurysm safer and more effective than excision in a single stage in selected cases.

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COMMENT

The authors review 28 cases of complex AVMs in which multiple obliterative procedures were utilized in an attempt to obliterate the malformation completely. In some instances, these procedures include embolization and operation or multiple surgical procedures. The malformations were extremely difficult and probably in most neurosurgeons' estimation might be considered inoperable. Therefore, the results have to be qualified considering the problem being managed. Of the 28 cases, complete obliteration of the AVM or associated aneurysm was accomplished in 19 cases. One would question whether the other patients were afforded any degree of protection from future hemorrhage. Another question is more philosophical, whether these very complex, difficult to reach malformations should be treated or left to their own natural devices. The facts that 19 of 28 were obliterated and that there was approximately 10% severe morbidity from these treatments must be considered. Of course, most physicians who have dealt with large numbers of AVMs feel that the natural history is disaster in almost all cases, although the outcome is

age-related because of the period the patient is exposed during his or her life-span. These questions are very difficult; answers can only be based on broad experience, such as these authors have recorded, and detailed discussions with the patients, pointing out the statistics and what we know of the natural history of these lesions.

We have primarily used silicone elastomer pellet embolization as an adjuvant to surgical resection of complicated arteriovenous malformations, while realizing that even after successful embolization some cases still are not appropriate for operation. Our primary purpose in embolization has been to interrupt deep, somewhat inaccessible feeders to malformations to reduce the overall pressure and flow within the malformation and, on some occasions, to interrupt more accessible feeding arteries to the malformation. In a large series of embolization procedures (over 500), Dr. Sadek Hilal and his group of neuroradiologists have been quite successful with silicone elastomer pellet embolization, in some cases using small tethered balloons to redirect the emboli. Serious complications such as explosion of the malformation during embolization or shortly thereafter have been rare (under 1%). Complications due to stray emboli, which fortunately have been temporary in the vast majority of cases, are rather common. As a surgeon, I find that these maneuvers, when well thought out and planned, are extremely valuable in the subsequent operative resection of the malformation. As regards staging of operative procedures on AVMs, we have done this only rarely. We do find that significant scar and revascularization builds up between surgical endeavors. In one particular case, the entire AVM was revascularized through the closed dural flap, suggesting some angiogenesis factor at work during the interval of 3 weeks since operation. Histological evaluation showed definite revascularization via the dura to the superficial portions of this AVM. In other cases, we have encountered scar difficult to deal with around the previously made margin of the AVM. On the basis of this limited experience, we prefer to stage the treatment of AVMs by first using intravascular techniques followed by a single operation, if at all possible.

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