Cerebral Arteriovenous Malformations

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INTRODUCTION

Arteriovenous malformations (AVMs) are vascular abnormalities consisting of fistulous connections of arteries and veins without a normal intervening capillary bed. In the cerebral hemispheres, they frequently occur as cone-shaped lesions with the apex of the cone reaching toward the ventricles. Nearly all AVMs are thought to be congenital. Supratentorial location is the most common (90%). The most common presentation of an AVM is intracerebral hemorrhage (ICH). After ICH, seizure is the second most common presentation. Other presentations of AVMs include headache and focal neurological deficits, which may be related to steal phenomena or other alteration in perfusion in the tissue adjacent to the AVM, such as venous hypertension from arterialization of normal draining veins.

In managing unruptured AVMs, it is important to understand the natural history of these vascular malformations. The decision for no treatment or for a single modality or multimodality treatment paradigm also involves being familiar with the outcomes and risks of each treatment modality—microvascular resection, endovascular embolization, and stereotactic radiosurgery. Finally, the patient-related factors, such as age, general medical condition, neurological condition, occupation, and lifestyle must also be taken into consideration before reaching a conclusion. The treatment of AVMs is highly individualized. There is no universal algorithm or protocol to be followed when dealing with these unique problems.

The currently used treatments for AVMs include microsurgical resection only, preoperative endovascular embolization followed by microsurgical resection, stereotactic radiosurgery only, preprocedural endovascular embolization followed by radiosurgical treatment, endovascular embolization only, and observation only. The ultimate goal for all of these modalities is cure for the patient; however, the only way to achieve cure is with complete obliteration of the AVM. Microsurgical resection, whenever it can be perfromed safely is the "gold standard" treatment for brain AVMs, and other methods of treatment must be measured against it. There is

certainly a well-established role for adjunctive endovascular embolization of some AVMs. Clearly, there are specific situations, such as small deep AVMs in eloquent brain structures, in which microsurgery should not be used as the primary treatment modality; stereotactic radiosurgery and occasionally embolization (if there is reasonable expectation of complete obliteration by embolization) are the preferred treatment options in these cases. We also make a case for observation in patients with large AVMs in or near critical areas of the brain that are not ideal for surgical resection or radiosurgery. Here, the pursuit of treatment may actually be more harmful to the patient than the natural history of the AVM.

EMBRYOLOGY, ETIOLOGY, AND GENETICS

AVMs of the brain are congenital lesions developing during the late somite stages between the 4th and 8th weeks of life. The lesion consists of persisting direct connections between the arterial inflow and venous outflow without an intervening capillary bed.[29]

The primordial vascular plexus first differentiates into afferent, efferent, and capillary components over the rostral portions of the embryonic brain. The more superficial portion of the plexus forms larger vascular channels, evolving into arteries and veins, whereas the deeper portion of the plexus forms the capillary component more closely attached to the brain surface. Beginning circulation to the brain appears around the end of the 4th week. AVMs arise from persistent direct connections between the embryonic arterial and venous sides of the primitive vascular plexus, with failure to develop an interposed capillary network.[80,100,115]

Genetic variation may influence pathogenesis and the clinical course of brain AVMs.[102] Identification of genetic polymorphisms associated with clinical course would help in stratifying risk and understanding the underlying biology. Molecular studies of brain AVMs have revealed an altered expression profile compared with normal tissue, including upregulated expression of genes involved in angiogenesis and inflammation.[38] Brain AVM patients homozygous for the interleukin (IL)-6−174G allele had a greater risk of ICH at presentation than IL6−174C carriers; a polymorphism in the inflammatory cytokine IL6 was associated with ICH presen-

tation of brain AVM.[95] Local IL6 release by endothelial cells within the brain AVM nidus may, therefore, contribute to vascular wall instability by stimulating release and activation of matrix metalloproteases.[12,20]

NATURAL HISTORY

A number of series have evaluated the natural history of AVMs with regard to the risk of hemorrhage. In a series of 168 patients without a history of previous hemorrhage, 18% of patients had subsequent hemorrhage over a mean follow-up of 8.2 years.[8] Annualized hemorrhage rate was 2.2%. In a study reported by Graf et al.,[31] hemorrhage risk at 1 year was 2%, at 5 years was 14%, and at 10 years was 31%. A retrospective study of 217 patients with AVMs followed for an average of 10.4 years yielded an annual hemorrhage rate of 3.4%.[11]

An important study by Ondra et al.[88] outlined the natural history of AVMs among 160 patients who presented with symptomatic AVMs and were followed for a mean follow-up of 23.7 years. The mean age at presentation was 33 years. The re-hemorrhage rate was 4% per year with an average of 7.7 years for the next hemorrhage to occur. The yearly morbidity rate was 1.7%, and the mortality rate was 1%. Interestingly, the yearly rate of hemorrhage for those AVMs that had never bled in patients who presented with seizures or vague symptoms was very similar (4.3% and 3.9% per year, respectively). This study demonstrated the high morbidity and mortality associated with AVMs regardless of initial mode of presentation, including hemorrhage, headache, or seizure.

The only prospective study of the natural history of AVMs resulted in an annual hemorrhage rate of 2.2%. However, follow-up of this group of 139 patients was short, at an average of only 1 year.[73]

Patients who present with ICH experience a 6 to 7% risk of hemorrhage during the 6 months subsequent to the initial hemorrhage; however, after that, the risk of hemorrhage is the same as that of patients that have never bled (3–4% per year).[31,32,88]

Table 13.1 summarizes the previously published studies on the natural history of AVMs.

DECISION MAKING

AVMs of the brain present a formidable challenge to the clinician in terms of what to recommend to the patient presenting with an AVM. At the start, we confess to some of the senior author's (RC Heros) biases. We think that a neurosurgeon is in the best position to recommend a plan of treatment or no treatment to a patient with an AVM. Furthermore, it would be preferable for that neurosurgeon to be experienced with AVM surgery because an essential part of the decision-making process concerns the risk of the treatment being considered. As indicated, we think that there is no formula or specific protocol in which a patient can be fitted into the decision scheme. Each patient is different, and each AVM is different, and, furthermore, the risk of each particular treatment differs depending on who will perform the treatment. Another important bias that the senior author has emphasized in writings and discussions on AVMs concerns the responsibility of the neurosurgeon to give the patient clear and unambiguous recommendations regarding what is the best form of treatment (or no treatment) for that particular patient. In the great majority of cases, there will one "best option" and that option ought to be recommended to that particular patient without ambiguity. Occasionally, there are equally satisfactory options and, in those cases, it is fair to present those options for the patient to choose, but we feel strongly that it is unethical to present options that are not equal in terms of the best interest to the patient as equally acceptable options. If it is difficult for the knowledgeable and experienced neurosurgeon to engage in the decision-making process for a particular patient with a particular AVM, one

TABLE 13.1. Natural history studies for arteriovenous malformations

Series (ref. no.)	Type of study	No. of patients	Average follow-up (yr)	Annual hemorrhage rate
Graf et al., 1983 (31)	Retrospective	164	4.8	2–3% in patients without hemorrhage; 6% at 1st year after hemorrhage, then 2% in patients with hemorrhage
Crawford et al., 1986 (11)	Retrospective	217	10.4	2%; 36% cumulative risk at 10-year in patients with hemorrhage; 17% in patients without hemorrhage
Brown et al., 1988 (7)	Retrospective	168 (all unruptured)	8.2	2.2%
Ondra et al., 1990 (88)	Retrospective	160	23.7	4% overall; 3.9% in patients with hemorrhage; 4.3% with seizure; 3.9% with other symptoms
Mast et al., 1997 (73)	Prospective	281	1.0	2.2% in patients without hemorrhage; 17.8% in patients with hemorrhage
Halim et al., 2004 (32)	Retrospective	790	4.0	7% for first year, then 3%

can only imagine how difficult it would be for the patient with no knowledge whatsoever of the topic to be presented with a number of statistics and perhaps references to the literature and be told that he or she needs to make the decision with no specific recommendation forthcoming from the neurosurgeon. Most patients confronted with this situation would either choose to see another neurosurgeon or, more commonly, to ask the neurosurgeon, "What would you do if it were you or one of your loved ones in my situation?" To continue to refuse to give an unambiguous answer under these circumstances is to forego our duty as physicians. Of course, it may be that the neurosurgeon truly does not know the answer and, in that case, it may be best to refer that patient to another colleague with more experience in this particular area. With the above preface, we next discuss some of the multiplicity of factors that the neurosurgeon must consider in making an appropriate recommendation to a patient with an AVM.

Diagnostic Evaluation

A computed tomography (CT) scan may be used as an initial screening tool for patients presenting with neurological sequelae related to unruptured or ruptured AVMs. This study can be used quickly to determine location of the lesion, acute hemorrhage, hydrocephalus, or areas of encephalomalacia from previous surgery or rupture. A non-enhanced CT scan may show irregular hyperdense areas frequently associated with calcifications in unruptured AVMs and acute hemorrhage on plain CT scan with ruptured AVMs. With the addition of intravenous contrast material, a CT scan can demonstrate the nidus and feeding vessels or dilated draining veins.

Magnetic resonance imaging (MRI) is superior to CT scan in delineating details of the macroarchitecture of the AVM, except in the case of acute hemorrhage. These architectural features include exact anatomic relationships of the nidus, feeding arteries, and draining veins as well as topographic relationships between AVM and adjacent brain.[65] MRI is sensitive in revealing subacute hemorrhage.[107] The AVM appears as a sponge-like structure with patchy signal loss or flow voids, associated with feeding arteries or draining veins on T1-weighted sequences. MRI and angiography in combination provide complementary information that facilitates understanding the three-dimensional structure of the nidus, feeding arteries, and draining veins. Magnetic resonance angiography (MRA) currently cannot replace conventional cerebral angiography. In the case of acute hemorrhage, the hematoma obscures all details of the AVM, making MRA virtually useless. This calls for direct use of cerebral angiography if the characteristics of the hematoma suggest AVM as an etiology.

Complete cerebral angiography with multiple projections is a mandatory step in the preoperative evaluation of a patient with an AVM. Cerebral angiography can localize the nidus, the feeding arteries, and draining veins. Angiography can be invaluable in supplementing MRI information in terms of operability of the lesion; for example, an AVM located in the ventricular surface of the thalamus may be "operable" if fed only by choroidal arterial branches, whereas the presence of deep thalamic perforating arterial feeders may render it "inoperable." Angiography will also assess the flow dynamics within the nidus of the AVM. The search for associated aneurysms is part of the preoperative evaluation. External carotid injections to determine the presence of an external supply are necessary in cases of large convexity AVMs. It is important that the angiogram be performed close to the time of surgery because AVMs can change in size and configuration over time. Vessels that were not observed secondary to compression from a hemorrhage may appear on a follow-up angiogram, weeks later.

Many techniques are available for studying the functionality of cortical structures surrounding the AVM. These include the use of positron emission tomography, functional MRI, magnetoencephalography, and direct provocative testing of cortical function. Judicious use of these techniques will enhance safety of AVM therapy. Such information may allow the surgeon to tailor treatment modalities to increase the margin of safety during treatment and decrease periprocedural flow-related hemorrhagic or ischemic complications.[2,98]

AVM-related Factors

Clearly, one of the most important considerations in terms of decision making is the AVM itself. Location, size, and configuration (compact versus diffuse) of the nidus; the pattern and location of the feeding and draining vessels; and the association of abnormalities, including aneurysms, direct arteriovenous fistulae, stenosis, or occlusion of the venous draining system are all factors that must be taken into consideration to estimate not only the risk of surgical excision of a particular AVM but also the risk of no treatment. To help the neurosurgeon estimate the surgical risk, a number of classifications have been developed, beginning with the classification proposed by Luessenhop and Gennarelli.[68] Although other classifications have been proposed,[68,90] the one most commonly used today is that proposed by Spetzler and Martin.[110] This classification simplifies the estimation of the surgical risk by considering the size and location of the AVM as well as whether it has deep drainage, which is an objective indicator of the fact that the AVM is located in or extends to the deep portions of the brain. Although the Spetzler and Martin grading has been used widely and has been confirmed by many experienced surgeons to be very useful, we should keep in mind the important factors that are not included in this classification. Such factors include the pattern of arterial supply (superficial versus deep perforating), abnormalities of the venous drainage (the arterialized venous drainage, for

example, may make it difficult and dangerous to gain access to the AVM), configuration of the nidus (compact versus diffuse), the presence of aneurysm in feeding pedicles, and, very importantly, the experience of the neurosurgeon who will be performing the procedure. In brief, a classification of surgical risks is extremely helpful in terms of analysis of an individual surgeon's series of cases and comparison with other series, and it also serves as a beginning for the decision-making process, but, as emphasized, the number of factors that the neurosurgeon must consider is such that it defies precise placement of a single patient on a particular risk category. We next discuss a few of the factors that the neurosurgeon must consider when studying a particular AVM and assessing the risk of treating that AVM by surgical excision as opposed to leaving it untreated or treating it by other modalities.

Location of AVM

Cerebellar and pial brainstem AVMs should be given strong consideration for surgical resection because it seems that AVMs in these locations carry a higher risk of bleeding as compared with supratentorial AVMs; however, such locations may make the surgery more difficult.[118] A case may also be made for surgical excision of basal ganglia and thalamic AVMs, because they carry an annual bleed rate of 9.8%,[21] considerably higher than the average bleeding rate when AVMs in all locations are considered. In addition, morbidity rate associated with these hemorrhages is significant, with 85.5% of patients suffering hemiparesis or hemiplegia. Here again, the risk of operating in these locations, especially in cases in which the lesion is fed by deep perforators, may outweigh the risk of observation or treatment with another modality, such as radiosurgery.

Size of AVM

In a series of 168 patients followed after presentation without a previous hemorrhage, the size of the AVM was not found to be predictive of future hemorrhage, using a multivariate statistical analysis.[69] However, other studies have found AVMs of small size to be at higher risk of hemorrhage. Spetzler et al.[109] compared the feeding artery pressures in small and large AVMs. They found higher feeding artery pressures in the small AVMs and suggested that small AVMs bleed more often than large ones. A more recent study from the same institution calculated hemorrhage risk at 1.5% per year for grades IV and V AVMs, which are, by definition, large AVMs.[34] However, the question of whether small AVMs bleed more often than large ones continues to be controversial at present; clearly, small AVMs present more often with hemorrhage, but this may be related to the fact that because their small size they are less likely to produce seizures, headaches, or a focal neurological deficit unless they bleed.

Draining Veins

Deep drainage has been thought to be an important risk factor for hemorrhage from an AVM. Nataf et al.[83] reported a strong correlation between frequency of hemorrhages and presence of deep drainage in AVMs. AVMs with a single draining vein were found to have a higher risk in some studies.[1,82] This can be explained by the fact that impaired drainage through a single vein leads to a high risk of hemodynamic overload and eventual rupture. Impairment in venous drainage caused by stenosis or kinking may also increase the risk of bleeding.[76]

AVMs and Aneurysms

Prevalence of the association of AVMs with aneurysms varies from 2.7 to 22.7%. This association seems to be correlated with a higher risk of hemorrhage.[59] Brown et al.[6] studied 91 patients with unruptured AVMs. Among these, 16 patients had 26 saccular intracranial aneurysms. They found the risk of ICH in patients with coexisting AVM and aneurysm to be 7% at 1 year, compared with 3% among those with AVM alone. At 5 years, the risk persisted at 7% per year, whereas it decreased to 1.7% per year in patients with an AVM unassociated with aneurysms. Ninety-six percent of 26 aneurysms were located on an AVM arterial feeder. The significance of intranidal arterial or venous aneurysms, which are quite common in large complex AVMs, is unknown, although it has been suggested that this finding may be associated with an increase risk of hemorrhage.[120]

Other Factors

As emphasized above, there is no magic formula to dictate to the neurosurgeon how to proceed in managing a patient with a cerebral AVM. For example, deep venous drainage may actually be an advantage intraoperatively because the draining veins are hidden away from the surgeon until the last moments of AVM removal. Patients with AVMs that present with major hemorrhage, progressive neurological deterioration, inadequately controlled seizures, intractable headache, or venous restrictive disease should be strongly considered for surgical resection, even when the risk of surgical excision may be relatively high.[118]

AVM resection should be strongly considered in patients with intractable seizures or, in rare cases, intractable headaches, because these symptoms are likely a hinderance on activities of daily living. The chance of relieving the symptoms of these patients and giving them a normal life back may outweigh the risks of surgery. Patients with venous restrictive disease may present another strong argument for surgical excision. With the occlusion of venous outflow from the nidus of the AVM, the intranidal hemodynamics begin to change: acutely, pressure begins to rise in different compartments of the AVM, and chronically, new, fragile venous draining pathways are recruited. These changes are likely caused by the increase in the risk of AVM hemorrhage.

The above comments regarding indications are only to suggest that, in these cases, stronger consideration should be given to treatment as opposed to observation. However, we emphasize that all AVMs, whether they have bled or not, causing symptoms or not, should be considered for treatment. The basis for this statement is the well-known fact that, as we stated above, after the first few months of a hemorrhage, the risk of hemorrhage is the same for AVMs that have bled than for those that have not bled.[48,88] As always, the ultimate recommendation should rest on the balance between the presumed risk of treatment and the risk of future hemorrhage or progressive disability, taking into account the multiple factors discussed, and, very specifically, the likely number of years at risk if the AVM is left untreated, which obviously is directly related to the age and general health of the patient.[48]

Patient-related Factors

As emphasized above, decision-making in determining the best management pathway for patients harboring AVMs must include consideration of the patient's age, general health and clinical condition, occupation, and lifestyle.[47,48] The patient's age is most important in determining the cumulative risk of AVM rupture during the remainder of the patient's life expectancy. Assuming an annual hemorrhage rate of 2 to 4% and an average life expectancy of 70 years, the cumulative risk (in percentage) of AVM rupture may be estimated by the following formula: 105 minus the patient's age in years.[5,61] Hence, one may justify a more aggressive approach for surgical treatment in younger patients because their cumulative risk of hemorrhage is so high. In addition, neurological deficit caused at a young age is generally better tolerated and has a greater chance of recovery. The general health of the patient is important, because a patient with severe comorbid conditions may preclude surgery as a reasonable treatment option. The clinical presentation and neurological condition of a patient will often dictate timing of surgery; for example, as indicated above, the patient may need emergent evacuation of a hematoma caused by a ruptured AVM, or it may be best to wait until the patient has improved to a neurological plateau when AVM resection can be approached electively. The occupation and lifestyle of a patient are important considerations as the neurosurgeon begins to weigh the risks and benefits of treatment of an AVM in a critical area of the brain. For example, a patient who is a pilot and is dependent on perfect vision presenting with an occipital AVM may think differently about surgical resection with a greater than 50% chance of causing a postoperative hemianopsia than a patient with an AVM in the same location who is a homemaker.

Surgeon-Related Factors

For obvious reasons, the surgeon's experience with AVMs is an important factor to be considered. Ethical considerations related to surgeon's experience come into play at a point at which the surgeon determines whether an AVM is operable or inoperable. Most competent neurosurgeons can remove safely a small AVM located in non-eloquent brain. However, with lesions that are more complex, the decision should preferably be made by an experienced cerebrovascular neurosurgeon at a referral center who specializes in AVM surgery. The surgeon should be familiar with the literature as well as their own personal experience and should be able to explain to the patient all treatment options with their associated risks and benefits. Importantly, as emphasized above, the surgeon should inform the patient clearly and unambiguously of what, in their opinion, is the best treatment option, which, in certain cases, may be no treatment at all.

SURGICAL RESECTION

In general, AVM surgery is elective. As discussed above, we recommend operating on ruptured AVMs that lead to intracranial hemorrhage and significant neurological deficits in a delayed fashion. Even if the AVM has resulted in a large hemorrhage that must be evacuated to relieve life-threatening mass effect, we generally prefer to evacuate the clot "gently" without interfering with the AVM and defer definitive treatment until later. Exception is with small superficial AVMs that can easily be removed at the time of evacuation of the clot. We have observed many "good" results reported after excision of large AVMs of the thalamus and basal ganglia operated on early after a hemorrhage that rendered the patient hemiplegic. The thinking is that the hemorrhage has already destroyed critical areas of the brain that lead to devastating neurological deficits, and, therefore, surgery cannot do further harm to the patient. "Good" results in these instances frequently mean that the patient's neurological condition was the same as before surgery. However, it is possible that the patient's preoperative condition would have changed for the better with time to recover from the ictus. Frequently, the hemorrhage does not destroy functional parts of the brain; instead, the mass from the hemorrhage splays apart gray and white matter, producing a deficit from pressure rather than destruction of critical brain. As the hematoma begins to resolve, these areas of the brain may recover to variable degrees. After a reasonable delay to allow such potential recovery to occur, the surgeon will be in a better situation to judge whether, given the degree of the recovery, it may not be preferable to treat the patient with an alternative treatment modality (i.e., radiosurgery) or to recommend conservative therapy. This "delay" in treatment is justified because, as opposed to rebleeding from an aneurysm, the risk of early rebleeding from an AVM is relatively low (approximately 6% during the first 12 months after a hemorrhage).

Outcomes

Microsurgical resection of Spetzler-Martin grades I, II, and most, although not all, grade III AVMs by experienced surgeons carries high cure rates and low complication rates with immediate elimination of risk of hemorrhage.[3,36,46] Angiographic cure rate with microsurgery ranges from 94 to 100%. Microsurgery can achieve 100% angiographic obliteration for unruptured convexity AVMs less than 3 cm, with superficial venous drainage.[118] The combined surgical morbidity and mortality for AVMs grade I, II, and III is reported to be less than 10% in several large series.[33,36,46,51,54,85,91,92,99,106,119]

In a series of 110 patients harboring grade I to III AVMs taken to the operating room for microsurgical resection, 99% had angiographically confirmed obliteration of the AVM. Two patients (1.8%) required reoperation for residual AVM. The risk of neurological deterioration in the immediate postoperative period was 10.9%, but many of these patients improved and the serious morbidity by 6 months was only 2.7%.[18] In another series of small AVMs,[112] 67 patients underwent microsurgical resection with a surgical outcome of 1.5% morbidity and 0% mortality. Pikus et al.[92] reported a series of 19 patients with small AVMs, grade I to III, yielding a 0% rate of morbidity and mortality.

In the senior author's personal series[47] of 311 patients who underwent microsurgical resection alone before 1993 for AVM, 89.9% of grade I to III patients had a good outcome, 9.5% had significant disability, and 0.5% died during the early postoperative period. However, grade IV and V patients had only a 60.7% good outcome, 37.5% significant disability, and 1.8% mortality. In a follow-up study of 153 consecutive patients with AVMs of all grades with a mean follow-up period of 3.8 years, we looked at the immediate morbidity and mortality rate and compared it with the late morbidity and mortality rate.[46] The overall immediate postoperative rate of serious morbidity was 24.2%; the serious morbidity at follow-up was 7.8%. The mortality rate at follow-up was 1.3%. There was no history of intracranial hemorrhage in any patient during the follow-up period. At follow-up, 97.8% of patients with grade I to III AVMs were in good or excellent condition, 1.1% experienced a poor outcome, and 1.1% died. In the group of patients with grade IV and V AVMs, 79.0% had good outcome, 17.7% had poor outcome, and 3.2% died. A recent large series of 220 patients with grade I or II AVMs reported an overall morbidity of 0.9% and an overall mortality of 0.5%, but also found a risk of adverse outcome for AVMs in eloquent cortex to be 9.5% as opposed to 0.6% for AVMs in non-eloquent regions[78]

Grade III AVMs are a heterogenous group, each type possessing different surgical risks. Grade III AVMs that are smaller than 3 cm in size, with superficial venous drainage in eloquent brain region have low risk that is similar to that of grade I or II AVMs. Grade III AVMs that are 3 to 6 cm in size, with superficial drainage in eloquent brain regions have a surgical risk similar to that of grade IV or V AVMs. Grade III AVMs 3 to 6 cm in size, with deep drainage in non-eloquent brain regions have intermediate risks.[64]

Tables 13.2 and *13.3* summarize the microsurgical outcomes from some of the larger published case series for Spetzler-Martin grades I to III and grades IV and V AVMs, respectively.

Completely obliterated AVMs lead to the best outcome in terms of seizure control. After surgical excision, 81% of patients with a history of seizures were seizure-free, whereas seizure-free outcome after radiosurgery and embolization was at 43% and 50%, respectively.[50] Heros et al.[46] reported a seizure-free survival in patients with AVM that had preoperative seizures of 43.6%.

TABLE 13.2. Postoperative outcomes for Grade I-III arteriovenous malformations

Series (ref. no.)	No. of patients	Morbidity and mortality rates
Pik et al., 2000 (91)	110	10.9% early morbidity; 2.7% late morbidity
Sisti et al., 1993 (106)	67 (small AVMs)	1.5% combined
Pikus et al., 1998 (92)	19	0%
Heros et al., 1990 (46)	91	1.1% late morbidity; 1.1% late mortality
Tokunga et al., 2000 (119)	12	0% for grades I and II; 75% early morbidity, 50% late morbidity, and 0% mortality in grade III
Irie et al., 2000 (54)	27	0%
Hongo et al., 2000 (51)	20	4% mortality
Russell et al., 2002 (99)	35	8.6% morbidity; 0% mortality
Hartmann et al., 2000 (36)	95	5.3% morbidity; 0% mortality
Spetzler and Martin, 1986 (110)	100	5% early morbidity
Lawton, 2003 (64)	76 (grade III AVMs only)	3.9% morbidity; 3.9% mortality
Morgan et al., 2004 (78)	220 (grades I and II AVMs)	0.9% morbidity; 0.5% mortality

TABLE 13.3. Postoperative outcomes for Grade IV and V arteriovenous malformations

Series (ref. no.)	No. of patients	Morbidity and mortality rates
Heros et al., 1990 (46)	62	17.7% late morbidity; 3.2% late mortality
Tokunga et al., 2000 (119)	4	25% morbidity; 0% mortality
Irie et al., 2000 (54)	4	25% morbidity; 0% mortality
Hashimoto et al., 2000 (37)	3	75% morbidity; 0% mortality
Russell et al., 2002 (99)	9	22.2% morbidity; 11.1% mortality
Hartmann et al., 2000 (36)	29	6.9% morbidity; 0% mortality
Hamilton and Spetzler, 1994 (33)	44	21.9% combined for grade IV; 16.7% combined for grade V
Hessler and Hejaza, 1998 (49)	62	20.5% combined for grade IV; 30.4% combined for grade V
Nozaki et al., 2000 (84)	32	9% morbidity; 0% mortality
Jizang et al., 2000 (56)	50	26% early morbidity; 12% late morbidity; 0% mortality

RADIOSURGERY

Indications

Stereotactic radiosurgery can be accomplished with a cobalt x-ray source (gamma knife), with the linear accelerator, or by taking advantage of the Bragg peak effect of heavy radioactive particles produced by a cyclotron.[45] Stereotactic radiosurgery is ideal for small (<3 cm) AVMs located in critical areas of the brain, in which the morbidity of surgical excision would be considered to be unacceptable.[44,45,91,101,118] It is also a good treatment choice for patients whose age or comorbidities make the risk of general anesthesia unacceptable.

Outcomes

The results of radiosurgery in terms of obliteration rate are hard to evaluate and compare because some series report obliteration rates based only on patients who had late angiography. Other series include obliteration whether observed by angiography or by MRI. A long-term follow-up study[95] of 118 patients who underwent first-time radiosurgery only for AVM showed an obliteration rate of 78.0%, good outcome in 75.4%, poor outcome in 11.9%, and mortality in 4.2%. Friedman[24] achieved a 79% obliteration rate in AVMs of less than 10 cm^3. For AVMs greater than 10 cm^3, the obliteration rate dropped to 47%. Pollock et al.[93] reported an overall obliteration rate for 222 patients of 61%; the obliteration rate increased to 83% if only AVMs of less than 4 cm^3 in volume were considered. Steinberg et al.[114] published an obliteration rate of 100% for AVMs less than 4 cm^3 and a 70% obliteration rate for AVMs greater than 3.7 cm in diameter. Pollock et al.[94] reported less than 50% of patients with deeply located AVMs were cured of future risk of hemorrhage without new neurological deficits, emphasizing the difficulty in treating patients with deeply located AVMs.

Table 13.4 summarizes the obliteration and complication rates of some large series of stereotactic radiosurgical treatment of AVMs.

Disadvantages

The main disadvantages of radiosurgery are the lack of certainty of obliteration and the delay in complete obliteration in those patients whose AVM is eventually obliterated. During these periods of delay, which may range between 1 year and several years, the patient remains at risk for hemorrhage and the risk is almost the same as if no treatment had occurred (3–4% per year), although one recent report claims a low latency hemorrhage rate (1.7%).[72] In addition, there is a small but significant risk of neurological injury from radiation damage (3–10%, depending on location).

Radiosurgery is not therapeutically effective for all lesions. Increasing AVM size reciprocally affects radiosurgical obliteration rate.[25] Radiosurgery is not effective in 10 to 15% of even small AVMs.[3] Repeated radiosurgery for previously incompletely obliterated AVMs carries a worse rate for subsequent obliteration than primary radiosurgery.[23] As stated above, there is no definitive evidence to suggest a reduction in the rate of hemorrhage in patients whose lesion is not completely obliterated.

A possibility exists that AVMs may reappear after having been totally occluded after radiosurgery, especially in the pediatric population.[67] A summary of factors associated with radiosurgical treatment failure has been compiled and consists of: changes in nidus morphology after radiosurgery because of resolution of hematoma, recanalization of a previously embolized portion of the AVM, technical errors in treatment planning, large nidus size (10 cm^3), and increasing Spetzler-Martin grade.[22,26]

Shin et al.[105] reported a series of 236 patients with complete follow-up data who underwent gamma knife radiosurgery for their AVMs. All had complete angiographic obliteration with median 77 months follow up. Of these 236 patients, 4 patients experienced a hemorrhage in the area of the previously demonstrated AVM. The calculated risk of hemorrhage was 0.3% annually. Persistent enhancement after

TABLE 13.4. Post-radiosurgical outcomes for arteriovenous malformations

Series (ref. no.)	Obliteration rates	Morbidity and mortality rates
Pollock et al. 2003 (95)	78.0% by angiography	11.9% morbidity; 4.2% mortality
Nozaki et al., 2000 (84)	N/A	36% morbidity from rehemorrhage; 0% mortality
Friedman, 1997 (24)[a]	79% (<10 cm^3); 47% (>10 cm^3)	N/A
Pollock et al., 1998 (93)	61% overall; 83% (<4 cm^3) by angiography	N/A
Steinberg et al., 1991 (114)	100% (<4 cm^3); 70% (>3.7 cm in diameter) by angiography	0.9% permanent morbidity; 11% hemorrhage rate
Stieg et al., 2000 (112)[a]	76% at 3 years	N/A
Inoue and Ohye 2002 (53)	81.3% by angiography	N/A
Shin et al., 2004 ()	87.1% by angiography	1.9% annual hemorrhage rate; 1.5% permanent morbidity
Pollock et al., 2004 (96)[b]	75.4% by angiography; 22.8% by MRI	12% morbidity from hemorrhage; 17.6% morbidity from radiation; 9% mortality
Maruyama et al., 2004 (72)	66% by angiography	latency-interval hemorrhage rate 1.7%/year for 1st 3 years, then 0%

[a]It is not clear that obliteration rates in these articles were confirmed by angiography or MRI and angiography.

[b]A series of grades IIIB and IV AVMs in the basal ganglia, thalamus, and brainstem. Grade IIIB is defined as small Spetzler-Martin grade III AVM located in areas where surgical resection is either too difficult or prohibitive (14).

complete angiographic obliteration was the only significant factor correlating with the risk of hemorrhage.

AVMs in or adjacent to functional brain tissue have a higher risk of radiation injury.[27] There have been rare reports of secondary tumors from radiation exposure from radiosurgery.[112] Other complications include death (<0.2%), cranial nerve injury (1%), new or worsened seizures (0.8%), increased risk of re-hemorrhage after radiosurgery in large AVMs and older patients,[22,66] and occlusive hyperemia.[10]

Radiosurgery has been praised in terms of its cost-effectiveness when used as an alternative to surgical excision of potentially operable AVMs and that is, of course, true if only the initial cost of treatment is considered. However, we found that in this setting (small AVMs that could be treated either with surgical excision or radiosurgery), microsurgical excision proved to be much more cost-effective as compared with radiosurgery when one considers, in a decision-making analysis model, the cost of future hemorrhages on patients treated with radiosurgery.[85]

EMBOLIZATION

Indications

Endovascular embolization can be used to eliminate proximal aneurysms before microsurgery or radiosurgery, particularly if the aneurysms could have been the source of hemorrhage. Embolization can also be used to obliterate deep arterial pedicles that are inaccessible during the early surgical exposure. It can also be used to obliterate the supply from lenticulostriate and thalamoperforating arteries before microsurgical resection. These small fragile feeding arteries are difficult to access and coagulate with electrocautery because they retract into the surrounding parenchyma when sectioned;

however, it is also true that embolization of these small deep arteries is more difficult and probably more dangerous. Embolization of superficial feeding arteries is usually not necessary because they can be easily controlled intraoperatively. Progressive, staged reduction in arteriovenous shunting by endovascular embolization may result in gradual restoration of normal cerebral perfusion and vascular reactivity, reducing potential for perioperative complications of brain swelling and hemorrhage with very large high-flow AVMs.[71] Frequently, we use embolization simply to reduce the flow and make surgery safer in AVMs that are either in or adjacent to critical areas of the brain. With these AVMs, we defer a final decision to excise the AVM until after completion of "safe" embolization; if embolization has decreased the flow very significantly, we proceed with excision; otherwise, we consider radiosurgery. Another situation in which we attempt embolization before committing to excisional surgery is with AVMs of deep structures, such as the thalamus, where there is both surgically accessible arterial supply (choroidal or circumferential) and surgically inaccessible perforator supply. If embolization can eliminate the perforator supply, we recommend surgery (*Fig. 13.1*), otherwise we advise radiosurgery.

Outcomes

Even in experienced hands, embolization of AVMs carries significant morbidity and mortality. Deruty et al.[18] reported 25% overall morbidity and 8% mortality rates. In the study by Wickholm et al.,[125] embolization resulted in severe complications in 6.6%, moderate complications in 15.3%, and mild complications in 17.3% of 150 patients. A meta-analysis of 1246 patients in 32 series during a 35-year period
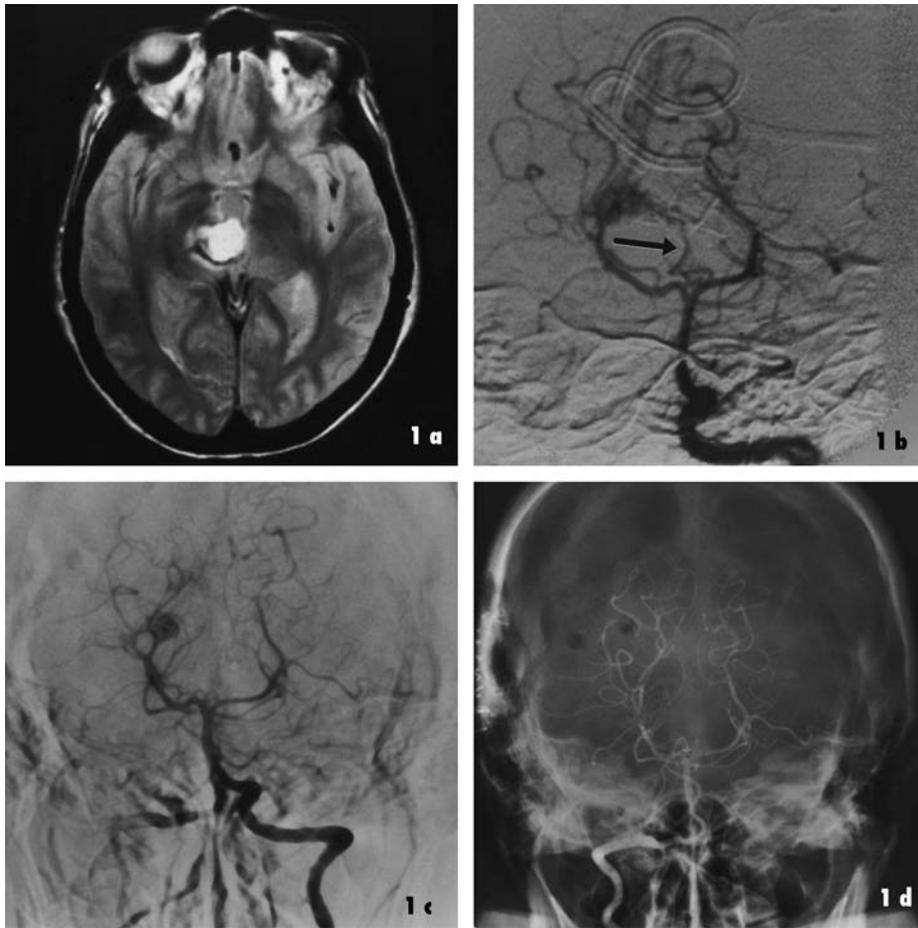
FIGURE 13.1. *A*, T2-weighted MRI of a patient who presented with subarachnoid and intraventricular hemorrhage shows a lesion consistent with an AVM in the left thalamus. *B*, anteroposterior (AP) view of angiogram shows a thalamic AVM fed by the branches of the posterior cerebral artery and the posterior thalamoperforator (*arrow*) from the P1 segment. *C*, this patient underwent preoperative embolization to eliminate the perforator supply, which made this lesion surgically safely removable. *D*, postoperative AP angiogram confirms complete obliteration.

showed a permanent morbidity rate of 9% before 1990 and 8% after, and mortality rate of 2% before 1990 and 1% after.[28] In a series[84] of 36 patients who underwent transarterial embolization of grade IV and V AVMs, 8% morbidity and 8% mortality was observed. Taylor et al.[117] reported that preoperative embolization was associated with a 1.2% mortality rate and 6.5% permanent neurological deficit rate per procedure in 339 procedures performed in 210 patients. Their overall mortality rate was 2% and permanent neurological deficit rate was 9% per patient. In a recent review of our data at the University of Miami, preoperative embolization of AVMs in 142 patients was associated with a 9% rate of major complications and a 1.2% rate of mortality per procedure (unpublished data).

Kwon et al.[63] followed 27 patients with greater than 4 cm diameter AVMs in eloquent cortex (≥ Spetzler-Martin grade III), that were thought to be inoperable. Eleven patients underwent embolization; 27.3% deteriorated after embolization, and 45.5% experienced hemorrhage after embolization. These results were compared with 16 patients who underwent medical treatment only; 31% of patients under medical treat-

ment deteriorated, and 25% experienced hemorrhage. Palliative embolization of AVMs does not seem to improve clinical results when compared with conservative treatment of AVMs though to be inoperable; therefore, it seems unjustified to put these patients through the significant risks of embolization, unless embolization has a reasonable chance of resulting in complete obliteration of the AVM.

Complete obliteration rates after embolization alone have been reported to be between 5% and 18% in the majority of series.[28] Valavanis and Yaşargil[122] and Yu[128] have recently reported the highest rates of complete obliteration (40.8% in 387 patients and 60% in 10 patients, respectively) for AVMs selected for embolization; the overall cure rate for Yu's series was 22%, however. It is important to know that this high rate of obliteration probably results from careful selection for embolization of those AVMs in which it was thought that there was a reasonable chance of complete obliteration by embolization. Vinuela et al.[123] reported an obliteration rate of 9.9% in 405 patients with AVMs. Gobin et al.[30] reported an obliteration rate of 11.2%. In the previously mentioned studies of Deruty et al.[18] and Wickholm et al.,[125] obliteration rates

of 13% and 13.3% were reported, respectively. Debrun et al.[16] and Hurst et al.[52] published obliteration rates of 5.5% and 15%, respectively.

In 32 patients undergoing preoperative silk suture embolization for AVM, 100% embolization was never obtained. More than 50% obliteration was accomplished in 10 patients, and less than 50% obliteration was achieved in 22 patients.[17]

Table 13.5 summarizes the obliteration and morbidity and mortality rates found in some reports of embolization as a treatment for AVMs.

Disadvantages

Despite some preconceived notions, endovascular procedures are not truly noninvasive, innocuous, or risk-free, as emphasized above. The procedures are frequently long and uncomfortable and may require general anesthesia and all of its associated risks.[71]

Endovascular embolization alone is rarely curative because of a high degree of delayed recanalization. Delayed refilling or recanalization is caused by almost completely embolized AVMs rapidly recruiting collateral supply.[71]

Embolization should not be performed for most grade I and II convexity AVMs because they can be surgically resected with minimal blood loss and low morbidity.[46,78] Medium-size grade III AVMs adjacent to or in critical cortical regions can be carefully embolized to reduce surgical risk, but cortical lesions or lesions in non-eloquent brain regions generally can be safely excised without embolization. For some grade III AVMs located in eloquent brain regions,

radiosurgery is an alternative. The risks of embolization include cerebral infarcts from inadvertent obliteration of penetrating arteries or vessels *en passage*,[71] AVM rupture from occlusion of draining veins before feeding arteries,[117] and hemorrhage from perforation of proximal arteries by either the guidewire (usually well tolerated) or the microcatheters (generally more serious). Hemorrhage can frequently occur during the first hours and days after embolization. Unfortunately, we observed several instances of this major problem. Each of these hemorrhages occurred after aggressive attempts at embolization with "almost complete obliteration" of the lesion. We think that altered hemodynamics were responsible for these hemorrhages, some of which occurred in patients who had not bled before.

The majority of complex AVMs have additional sources of arterial supply—small branches of perforating arteries, such as lenticulostriate and anterior choroidal arteries, small leptomeningeal collateral vessels, and tiny deep transmedullary arteries—that are not amenable to embolization.[71] Partially embolized AVMs rapidly and aggressively recruit new sources of arterial supply; indiscriminate embolization may result in occlusion of large cortical vessels, which could have been easily controlled surgically, only to be replaced by collateral flow from many tiny subcortical arteries—thin-walled, fragile, and distended—which are more difficult to control surgically.[71]

Another frequent use of embolization is to reduce the size of large inoperable AVMs and make them amenable to

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TABLE 13.5. Post-embolization outcomes for arteriovenous malformation

Series (ref. no.)	Obliteration rates	Morbidity and mortality rates
Deruty et al., 1996 (18)	13%	25% morbidity; 8% mortality
Wickholm et al., 1996 (125)	13.3%	6.6% severe morbidity; 15.3% moderate morbidity; 17.3% mild morbidity
Frizzel and Fisher, 1995 (28)	N/A	5% major morbidity; 15% minor morbidity; 2% mortality
Nozaki et al., 2000 (84)	N/A	8% morbidity; 8% mortality
Kwon et al., 2000 (63)	N/A	27.3% morbidity (45.5% post-emboliztion hemorrhage rate)
Hartmann et al., 2002 (35)	N/A	14% early morbidity; 2% permanent morbidity; 1% mortality
Jahan et al., 2001 (55)	N/A	4% morbidity; 0% mortality
Valavanis and Yasargil, 1998 (122)	40.8%	1.3% severe morbidity; 1.3% mortality
Vinuela et al., 1995 (123)	9.9%	N/A
Gobin et al., 1996 (30)	11.2%	N/A
Debrun et al., 1997 (16)	5.5%	N/A
Hurst et al., 1995 (52)	15%	N/A
Yu et al., 2004 (128)	60% curative intent; 22% overall	N/A
Taylor et al., 2004 (117)	N/A	6.5% morbidity; 1.2% mortality per procedure
University of Miami, 2005 ()	N/A	9% major complication; 1.2% mortality per procedure

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radiosurgery. However, in many of these cases, radiosurgery may fail to obliterate the AVM nidus because of delayed recanalization and reappearance of the portion of the nidus that was not included in the radiosurgical target planning.[96] Previous embolization is actually a negative predictor of successful AVM radiosurgery, and some considered it to be contraindicated before radiosurgery.[93] We can find no good evidence that embolization before radiosurgery reduces the risk of hemorrhage during the "latent" period before complete obliteration.

OBSERVATION

Observation alone seems to be the best option for most patients with deep thalamic, brainstem, and basal ganglia AVMs that are too large for radiosurgery. In addition, it has gradually become apparent to experienced surgeons that almost all grade V and many grade IV AVMs should be left alone unless the patient has a serious progressive deficit or has suffered multiple hemorrhages.[34,46,47,118] In one series, a hemorrhage risk of 1.5% per year for grade IV and V AVMs was lower than that reported for grades I through III.[34] As stated above, there is no evidence that partial treatment with embolization reduces the patient's risk of hemorrhage and, by definition, grade IV and V AVMs are too large for optimal radiosurgical treatment, again lending support to the option of conservative treatment for these lesions.[34]

GENERAL SURGICAL TECHNIQUE

We, as well as many other experienced surgeons, have written extensively regarding this topic and, therefore, we will be brief here. As indicated above, we generally think of AVM surgery as elective surgery. The occasional patient that requires immediate surgery because of an intracerebral hematoma is approached "conservatively," with gentle evacuation of the clot without interfering with the AVM; only in the case of small surface AVMs that can easily be removed with the clot do we attempt to excise the AVM on an emergency basis, as we stated above. Because we wait sometimes 3 to 6 weeks, depending on the size of the hematoma and other factors, such as the accessibility of the AVM, age of the patient, etc., we prefer to repeat the arteriogram the day before surgery to make sure that angiographic features of the AVM have not changed, because the earlier arteriogram may have been performed on an emergency basis after the bleeding. We have found that, in these cases, sometimes a portion of the AVM that was not well visualized in the immediate post-hemorrhage arteriogram can be clearly observed on a later arteriogram and, of course, it is critical to know this before surgery. For deep lesions, of course, the craniotomy depends on the approach, as we will discuss below. For lesions with cortical representation, we prefer a large craniotomy for several reasons. The most important reason is to be able to examine the cortical vascular anatomy and to compare

the location of the feeding arteries and the draining veins with the arteriogram. Sometimes the feeding arteries can be observed on the surface at some distance from the AVM before they dip into sulcus to reach the AVM. With a small craniotomy, these feeding arteries may not be apparent. We like to observe the gradual change in color of the veins that drain the AVM as their arterial supply is controlled. Sometimes the color of these veins begins to change only a few centimeters away from the AVM where they begin to receive some normal cortical drainage. Additionally, it is sometimes difficult to control a parenchymal hemorrhage away from the AVM if the surgeon is working through a small craniotomy. Clearly, with frameless stereotactic guidance, the craniotomy can be more accurately placed, but still we prefer a craniotomy with a margin of a few centimeters around the AVM.

We make it a point to identify as many of the feeders as possible on the surface and then follow them to the AVM through a sulcus where they usually "hide" before they reach the AVM. Sometimes it is not easy, particularly with less experience, to be sure whether a particular vessel is a feeding artery or a draining arterialized vein. With experience, it is clear that the veins are usually larger, more delicate, and have thinner walls; however, when in doubt, it is useful to place a temporary clip that will make a draining vein less turgid and frequently bluer distal to the clip, whereas an artery will, of course, continue to vigorously pulsate against the clip. After we have taken all of the visible feeders, always correlating the operative anatomy with the arteriogram, we proceed to systematically open every sulcus around the AVM to try and identify other, usually smaller, feeders. We then proceed with a circumferential cortical incision immediately adjacent to the AVM to a depth of about 2.5 to 3 centimeters. From experience, we have found that with a circumferential corticectomy to this depth, all of the superficial feeders are controlled. This corticectomy can comfortably be made a few millimeters from the AVM in a non-eloquent region of the brain and this, of course, tremendously facilitates the dissection. Clearly, when working in eloquent areas or immediately adjacent to them, the surgeon must work right on the loops of the AVM. This is why, in these cases, we frequently use preoperative embolization to decrease the flow, which makes it safer to work right at the periphery of the AVM, frequently stroking gently the loops of the lesion with bipolar coagulation to shrink it away from the brain; this, of course, cannot be performed safely when the AVM is turgid from high flow.

After the initial circumferential incision, we proceed with further circumferential dissection of the AVM in a spiraling fashion. During this stage, the surgeon frequently encounters bleeding from the AVM, and the important point here is to not attempt to use coagulation when the AVM is still turgid with high flow. Of course, this can be performed with relative safety once most of the arterial supply to the AVM has been controlled, or in cases in which there has been

extensive preoperative embolization. Most frequently, the surgeon can control the bleeding by just placing a small cottonoid against the AVM and sometimes placing a self-retaining retractor over the cottonoid for a few minutes. When bleeding occurs, it is frequently best to control it, as just stated, and then move to another area of the AVM. It is critical to never "pack" bleeding away from the AVM on the brain's side. In the senior author's early experience, this was the cause of a few serious intraoperative hemorrhages. When bleeding occurs from a vessel coming from the brain to the AVM, or from a piece of AVM that has been disconnected from the rest of the AVM, that bleeding must be controlled and the surgeon must be doggedly persistent regarding this.

The most difficult stage of AVM surgery, particularly with large cortical AVMs, is bleeding from the deepest aspect of the AVM near or within the ventricular ependyma once the circumferential dissection has been almost completed. Every neurosurgeon knows of the difficulty in controlling these small fine vessels at the bottom of the AVM. We will be eternally grateful to the late Dr. Thoralf Sundt for having designed tiny microclips precisely for this purpose. In the past, we used large aneurysm clips and it is remarkable to see how these tiny fragile vessels continue to bleed right through those clips. Bipolar coagulation sometimes is completely ineffective. The usual solution nowadays is the precise use of a Sundt microclip as the point of bleeding is isolated and held in a fine suction tip. The moment of relief for the surgeon is usually when the ventricle is reached, because this is where the last few remaining tiny deep feeders come from and can be controlled with more ease.

In general, intraoperative angiography should be used for all cases of AVM surgery and we make it a point of doing so whenever possible, however, there are times when the position of the patient makes intraoperative angiography very difficult and, in these cases, if it is not practical to place a femoral sheath before positioning, we have to depend on immediate postoperative angiography. There are a few cases of small surface AVMs in which an experienced surgeon does not really need intraoperative angiography to know that they have performed the job, but still, even in these cases, it is better to do at least a postoperative angiogram.

SURGICAL APPROACHES TO DEEP LESIONS

In this section, the senior author discusses his preferred approaches to deep AVMs. Obviously hemispheric AVMs with cortical representation are approached directly, frequently with the use of frameless stereotaxis for accurate placement of the craniotomy.

Sylvian AVMs

The more anteriorly located of these lesions are approached by opening the sylvian fissure, working from lateral to medial through a pterional craniotomy.[41] The lesions located in the middle and posterior portions of the sylvian fissure are approached directly by opening the fissure laterally through a temporal craniotomy[41] (*Fig. 13.2*). The most important point to remember with these lesions is that they tend to be fed by vessels *en passage* that supply the AVM through small side branches and then go on to supply important areas distally. The difficulty here is in carefully skeletonizing these vessels as they pass through the AVM, coagulating or clipping and dividing all of the side branches to the lesion but preserving the main trunk. Occasionally, a temporary clip is used on the arterial branch as it comes into the area of the AVM to facilitate its dissection; however, if this is performed, one must be particularly careful with hemostasis, keeping in mind that the side branches to the AVM were controlled under markedly reduced pressure with a temporary clip in place. For fear of insecure hemostasis, we prefer to use this technique of temporary clipping only when excessive bleeding makes it necessary.

Medial Temporal AVMs

The more anteriorly located lesions, in the region of the amygdala, uncus, and the anterior hippocampal complex, are approached through the medial aspect of the sylvian fissure using a pterional craniotomy.[41] As the sylvian fissure is opened and the temporal lobe is retracted laterally, all of the feeding vessels to the AVM are stretched slightly and can be easily identified. These feeders are, from superficial to deep, anterior temporal branches of the middle cerebral artery (MCA), the anterior choroidal branches, branches from the posterior communicating artery, and early temporal branches of the posterior cerebral artery (*Fig. 13.3*). As these feeders are controlled, one must be particularly careful to preserve the venous drainage, which is usually to anterior medial sylvian veins and, more posteriorly, to the basal vein of Rosenthal. Early temporal branches should be followed from their origin to feeder branching because they may occasionally give rise some of lentoculostriate arteries right after their origin from the MCA.[116] Once the feeders are controlled, the lesion can be removed, generally with ease, by working from a medial to lateral direction.

The more posteriorly located lesions of the medial temporal lobe involve the hippocampal and parahippocampal region and the fusiform gyrus and can extend to the trigone. The lesions are approached through a temporal craniotomy, working either subtemporally or through the inferior temporal gyrus, as described previously by the senior author.[40] The transtemporal approach has the advantage of preserving the vein of Labbe, which may be arterialized, and minimizing retraction of the temporal lobe, which sometimes is considerable when using the subtemporal approach. The direction is toward the temporal horn, which, once identified, serves as a good anatomic landmark for orientation. The anterior choroidal artery feeders, which invariably supply these lesions, can
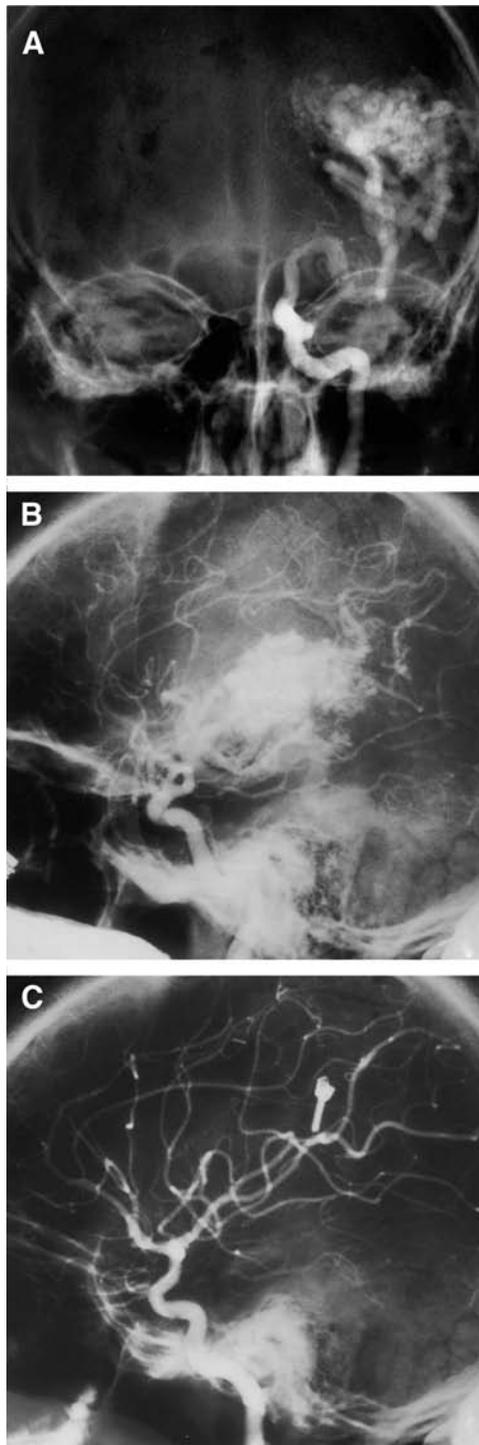
FIGURE 13.2. Sylvian AVM. The AP (*A*) and lateral (*B*) views of angiogram show an AVM located in the left sylvian fissure. Main feeders originate from "vessels *en passage*," which are M2 branches of the MCA. These vessels were skeletonized after widely splitting sylvian fissure and feeders from small side branches were coagulated and divided. Postoperative lateral angiogram (*C*) shows complete obliteration of this AVM.
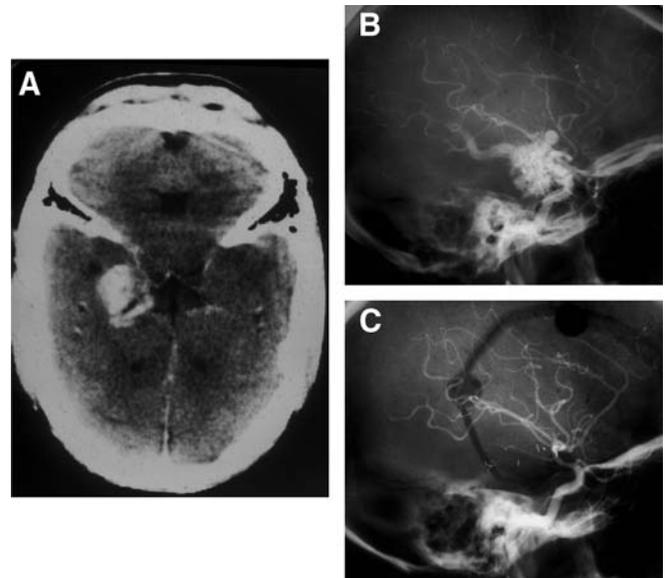

FIGURE 13.3. Anteromedial temporal AVM. *A*, CT scan shows a lesion consistent with an AVM located in the anteromedial temporal lobe. *B*, lateral angiogram confirms the diagnosis of AVM with feeders from the posterior communicating artery, anterior choroidal artery, and the early branches of the posterior cerebral artery. *C*, postoperative angiogram shows complete obliteration.

usually be controlled through the choroidal fissure on the medial aspect of the temporal horn. The posterior cerebral feeders are controlled either subtemporally or directly transtemporally as they enter the lesion (*Fig. 13.4*). A superior quadrantanopsia is commonly observed after removal of these lesions, whether the subtemporal or the transtemporal approach is used, and the patients need to be informed of this complication.

A recent article highlighted the orbitozygomatic approach as an alternative to maximize the exposure of the tangential approach to medial temporal lobe AVMs and has advantages over traditional lateral approaches. It provides early access to critical feeding arteries from the anterior choroidal artery, posterior cerebral artery, and posterior communicating artery; it minimizes temporal lobe retraction and risk to the vein of Labbe; and it avoids transcortical incisions or lobectomy that might impact language and memory function. For these reasons, it may be the optimal approach for small- and medium-sized compact AVMs in the dominant medial temporal lobe.[19]

Medial-Temporal Insular AVMs

Purely insular AVMs are approached through the sylvian fissure with skeletonization of the MCA sylvian branches to control the medially directed feeders to the AVM that sits just deep to the web of MCA branches. Substantial lenticulostriate perforator supply means deep extension of the
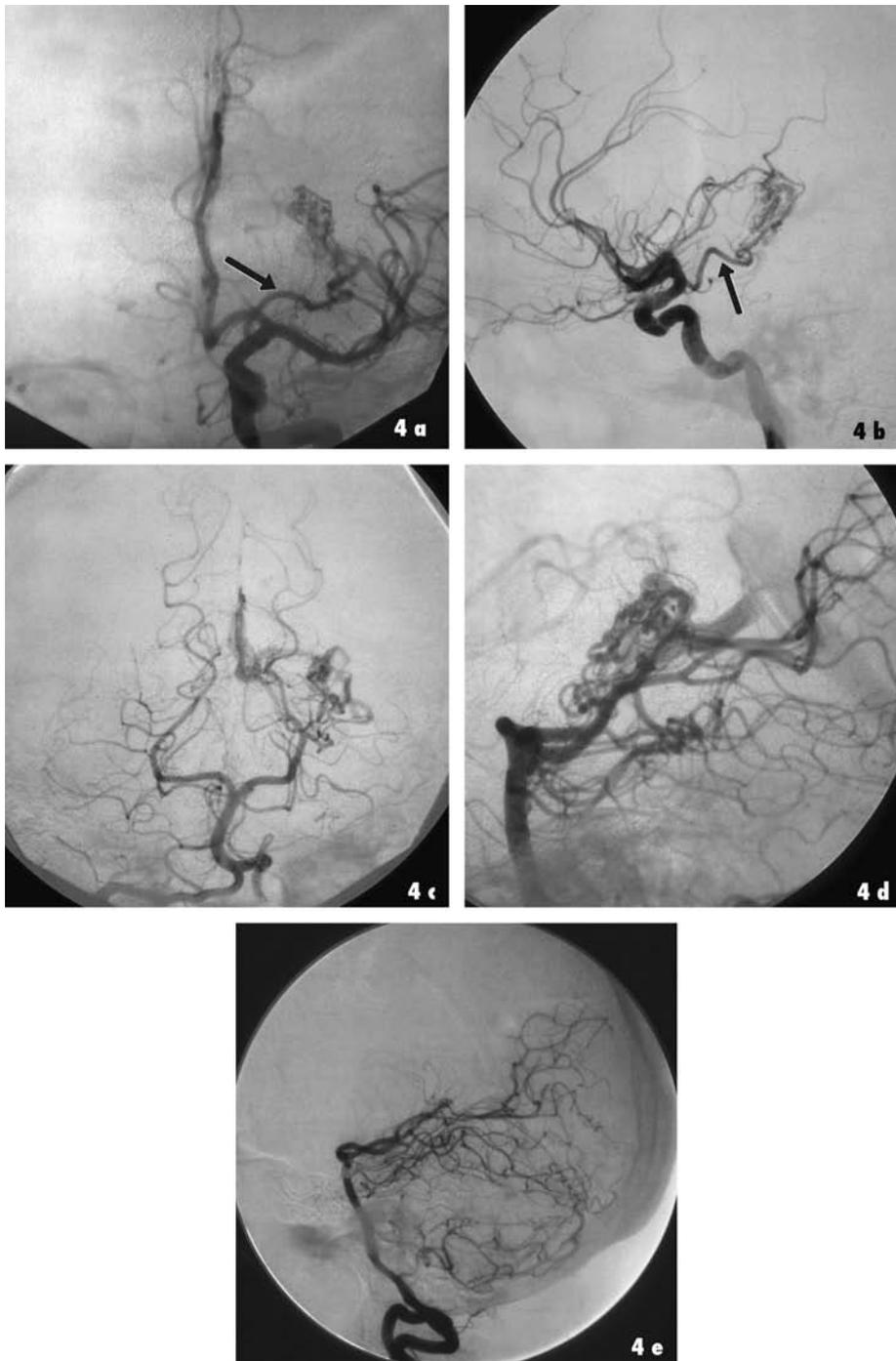
FIGURE 13.4. Mesial temporal AVM. AP (*A*) and lateral (*B*) carotid angiograms show a feeder from the anterior choroidal artery (*arrow*) and AP (*C*) and lateral (*D*) vertebral angiograms show feeders from the P2–P3 junction. Postoperative lateral vertebral angiogram (*E*) shows complete obliteration.

AVM, which usually make it inoperable. Some of these AVMs can be more extensive and involve the temporal lobe stem, the medial temporal lobe, and reach the atrium (*Fig. 13.5*).

Trigonal AVMs

In terms of surgical approach, we like to divide these AVMs into two groups. We tailor the surgical approach depending on where the bulk of the AVM is located. The first group is lesions that are more inferiorly located and involve the floor and the lateral wall of the trigone. We approach these lesions transtemporally, through the inferior temporal gyrus on the dominant side to avoid speech problems, and either through the inferior temporal gyrus or middle temporal gyrus on the nondominant side. Again, a superior quandran-
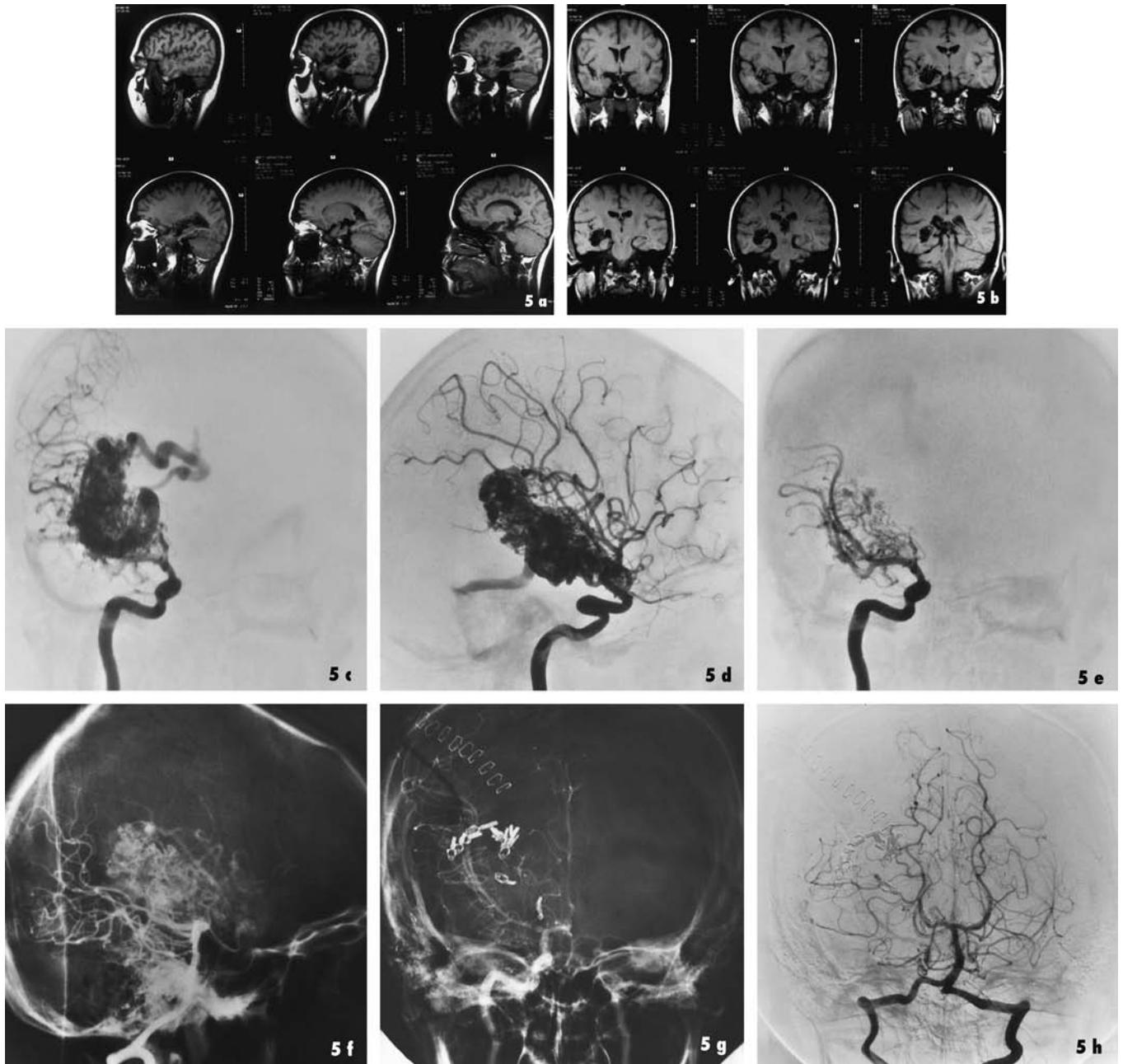
FIGURE 13.5. Insular temporal AVM. Sagittal (*A*) and coronal (*B*) T1-weighted MRI scans show an AVM located in the right temporal lobe and insula. AP (*C*) and lateral (*D*) carotid angiograms show a large AVM extending into the medial temporal region and the insula. Note lenticulostriate feeders on early arterial phase (*E*). A lateral vertebral angiogram (*F*) shows PCA feeders. Postoperative AP carotid (*G*) and vertebral angiograms (*H*) show complete excision.

tanopsia is very common after this approach, but frequently these patients have a visual field cut already and generally it is not a disabling deficit. Sometimes, we choose a subtemporal approach to obtain control of the feeders from the posterior cerebral artery, depending on how inferior the lesion is (*Fig. 13.6*).

The second group of lesions involves the medial aspect of the trigone, which has been called the parasplenial region,[127] as well as the roof of the trigone and sometimes the dorsal surface of the pulvinar of the thalamus (*Fig. 13.7*). We prefer to use a parieto-occipital approach to these lesions.[41] This transcerebral approach sometimes takes advantage of a
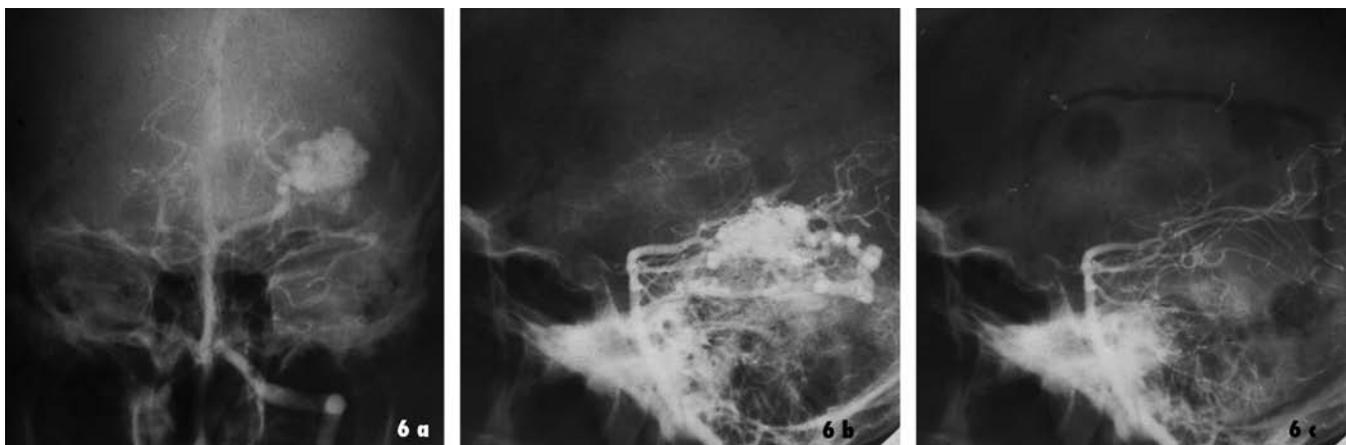
FIGURE 13.6. Inferolateral trigonal AVM. AP (*A*) and lateral (*B*) vertebral angiograms show an AVM in the inferolateral aspect of the left trigone. Arterial supply was via posterior choroidal branches of the PCA. Postoperative angiogram (*C*) confirms complete obliteration achieved via inferior temporal gyrus approach.

sulcus that can get the surgeon 1.5 to 2 cm closer to the ventricle before he has to transgress white matter. The approach is between the parietal sensory association fibers and the occipital visual association fibers; we have used this approach routinely without resulting sensory or visual deficits, particularly if the AVM is small. With large AVMs, sometimes it is impossible to prevent some visual field loss from damage to the visual radiations along the tapetum. We use the semi-sitting or the prone position for this approach. The incision in the brain is made approximately 7 cm up from the occipital tip, which corresponds to approximately 9 cm above the inion as an external landmark. The incision is centered approximately 3 cm off of the midline and the direction of approach is toward the trigone, which can usually be identified with ultrasound or frameless stereotactic guidance. We usually extend the bone flap to the midline to allow an initial parasagittal approach to identify the splenium for better orientation, because the trigone is exactly at the same axial plane of the splenium but approximately 3 cm laterally. This transcerebral approach has been more satisfactory than the parasagittal approach through the precuneus, which others have recommended.[127] The parasagittal approach requires considerable brain retraction because the trigone is approximately 3 cm lateral to the midline. In addition, the line of vision is tangential when one uses the parasagittal route, as opposed to the more direct transcerebral route.

Splenial-Posterior Third Ventricular Region AVMs

These AVMs differ from trigonal AVMs in that the arterial supply is more complicated because they reach the midline. The more lateral trigonal AVMs are supplied primarily by branches of the posterolateral choroidal artery as well as by direct branches of the posterior cerebral artery. The more medial splenial AVMs, although they can extend laterally to the atrium and obtain posterolateral choroidal supply, are primarily supplied medially by the posteromedial choroidal artery, direct branches of the posterior cerebral artery, and pericallosal branches from the anterior and posterior cerebral complexes. Because of this, the parasagittal approach to control the medial blood supply is imperative. We have used the same positioning as for trigonal AVMs and also a similar bone flap, although, more recently, we have used the lateral position, with the ipsilateral side down to allow the occipital lobe to fall away by gravity. The difficulty comes at the end of the procedure because the AVMs need to be followed laterally in a tangential direction toward the trigone, where they almost invariably receive significant blood supply. Another difficulty is that the AVM may be intimately related to the deep venous system and one must be careful not to injure any of the important deep cerebral veins. Others have recommended a contralateral parafalcine approach for callosal AVMs, which provides a less tangential view and less need for brain retraction.

We have dealt with some AVMs of the area of the roof of the third ventricle in the velum interpositum through the same exposure. A small callosal incision just anterior to the splenium gets the surgeon into the region of the velum interpositum.

Deep Parasagittal AVMs

Parasagittal AVMs are approached through a unilateral frontal craniotomy that crosses the midline. The position depends on the afferent blood supply. If the lesion has MCA supply over the convexity, as well as the usual anterior cerebral artery (ACA) supply, we use the neutral supine position with the head flexed and a craniotomy that extends more laterally for ease of control of the MCA feeders. If the
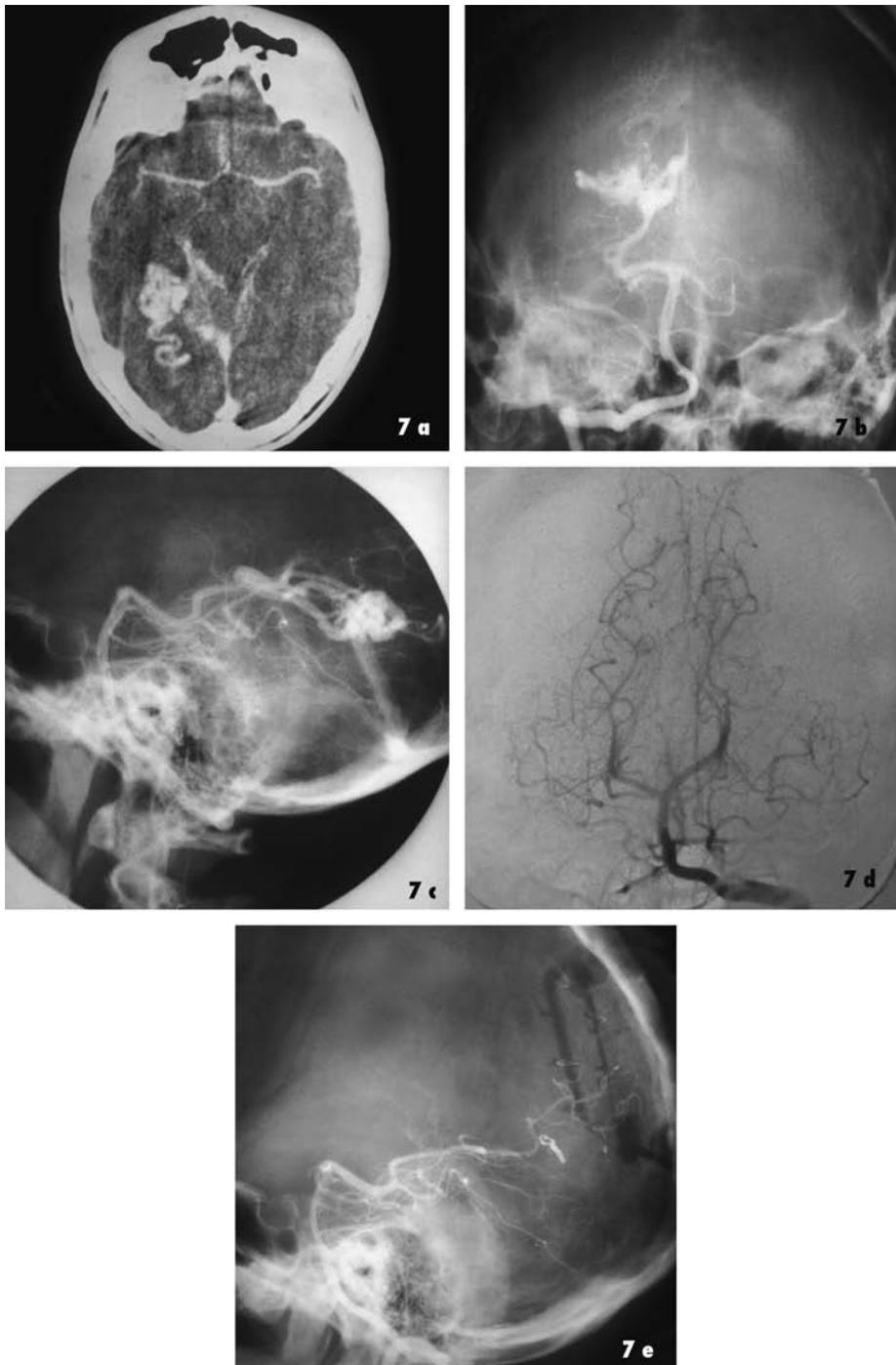
FIGURE 13.7. Medial trigonal (parasplenial) AVM. CT scan (*A*) shows an AVM located in the medial aspect of the right trigone. AP (*B*) and lateral (*C*) vertebral angiograms show an AVM fed by the posterior lateral choroidal branches of the PCA. Postoperative AP (*D*) and lateral (*E*) angiograms confirm complete excision via superior parietal lobule approach.

supply is only from the ACA, the approach is strictly inter-hemispheric and, in these cases, we prefer the lateral position with the ipsilateral side down. We also prefer this latter position for parietal and occipital parasagittal AVMs. It is important to use this position with the ipsilateral side down only when the lesion does not reach the convexity, because, when this position is used, the dura must be opened only with

a very narrow flap medially that allows the brain to fall under the dura. If a wider dural flap is opened, the brain tends to fall against the edge of the dura and there can be substantial damage. The contralateral parafalcine approach has also been used for these lesions.

Larger parasagittal lesions, particularly in the frontopa-rietal region involving the motor-sensory strip, present a

serious surgical problem. These patients can be expected to have some degree of weakness of the contralateral lower extremity and possibly also of the shoulder and proximal arm when the lesion is large and, therefore, a conservative approach may be preferable in these cases, depending on the circumstances. The technical problem with these larger lesions is that they are supplied both by pericallosal branches and by MCA branches and the position required to access the pericallosal branches is very different than the position that facilitates dealing with the MCA branches along the convexity.[41] To deal with the pericallosal branches, a parasagittal approach is preferred, for which either the semi-sitting position with the head neutral, or the lateral position with the ipsilateral side down is preferred, so that the brain can fall away without undue retraction. To deal with the convexity portion of the AVM, one would prefer a lateral position with the ipsilateral side up, which makes it very difficult to work parasagittally. A way around this is to have the pericallosal branches occluded by preoperative embolization, so that the surgeon needs to deal only with the branches of the MCA along the convexity; this can best be performed in the lateral position with the head elevated to have the convexity uppermost in the field.

A potential pitfall with these larger parasagittal-convexity lesions is injury to the arterialized draining veins, which can occur easily as a result of retraction for the parasagittal approach. Sometimes the entire AVM is densely stuck through many small draining veins to the falx and the sinus and it is virtually impossible to work parasagittally in the area of the AVM. In these instances, the surgeon would be well advised to have a much broader-based bone flap so that the surgeon can develop the parasagittal approach either anterior or posterior to the lesion or both. Again, preoperative embolization, when successful in obliterating essentially all of the pericallosal supply, obviates need for an early parasagittal approach.

Anterior Callosal AVMs

These lesions are usually fed by ACA branches and drain to the sagittal sinus superiorly and to the septal vein intraventricularly. The lesions can be very complex and extend laterally into the basal ganglia, particularly into the head of the caudate nucleus. They can also extend inferiorly below the genu to involve the basal frontal region and anterior aspect of the hypothalamus (*Fig. 13.8*). When the lesions extend more laterally, they acquire perforator supply frequently from the recurrent artery of Heubner. When the lesions are supplied by lateral lenticulostriate branches, they usually become inoperable, because this is an indication that these lesions involve the internal capsule. The simpler lesions are approached with the patient supine, through a unilateral frontal craniotomy and an interhemispheric parasagittal approach; the arterial supply is secured by skeletonizing the

pericallosal and callosomarginal arteries as they pass through the lesion.[41] It is important to preserve the main arterial trunks, which are usually vessels *en passage*, and take only the side branches that go to the AVM. Incidentally, this tendency to be fed by small side branches of vessels *en passage* makes embolization of these lesions difficult and hazardous.

For the more extensive lesions, one can perform a more extensive frontal or bifrontal flap that reaches to the floor of the frontal fossa. The head is kept neutral for the subfrontal phase of the exposure, where the feeders from the anterior communicating complex and the early portions of the pericallosal arteries are controlled. Once this is accomplished, the head is flexed and elevated for the higher interhemispheric approach to control the more distal pericallosal and callosomarginal branches, and also for the final intraventricular aspect of the dissection in those lesions that extend to the head of the caudate. The main problem with these lesions comes from medial lenticulostriate perforating supply that comes through the medial aspect of the basal ganglia and can lead to deep bleeding during the final stages of the resection.

Intraventricular AVMs

Although many AVMs have some ependymal representation, purely intraventricular AVMs make up only 4 to 13% of large series of AVMs.[130] In the opinion of the senior author, AVMs in this location have a higher tendency to bleed than other AVMs surrounded circumferentially by brain parenchyma, but this is only an unsubstantiated observation. Operability is determined by the relative importance of choroidal as opposed to perforator supply. That is, the lesions that we consider operable are supplied mostly by choroidal arteries, which can be readily controlled at the different ependymal surfaces of the ventricles. Some lesions involve all of the tela choroidea of the lateral ventricle and they can be approached transcallosally with a subchoroidal approach to the tela choroidea on the roof of the third ventricle (*Fig. 13.9*). When the predominant supply is perforating vessels and through the basal ganglia or through the thalamus, operability becomes very questionable because the chance of morbidity from deep bleeding becomes too high. When these lesions with considerable perforator supply are small enough, radiosurgery becomes an excellent alternative.

The lesions located in the head of the caudate region can usually be excised with safety. They are fed by choroidal branches but also deeply by the recurrent artery of Heubner and by medial lenticulostriate arteries. They are approached through an anterior transcallosal route or, if there is ventriculomegaly, by a transfrontal route. Again, deep bleeding from perforators is a problem, but parenchymal damage in this region (anteromedial to the internal capsule) is usually well tolerated.
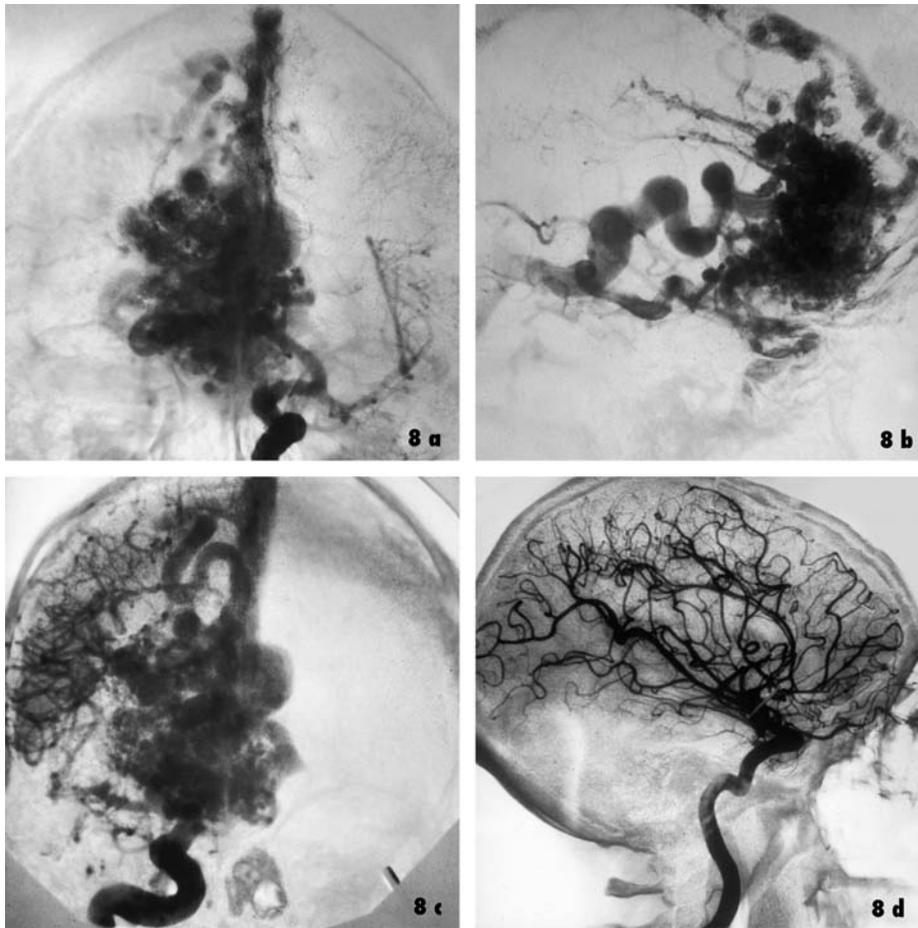
FIGURE 13.8. Anterior callosal AVM. AP (*A*), lateral (*B*), and oblique (*C*) views of carotid angiograms show extensive anterior callosal AVM reaching the basal frontoorbital regions and anterior aspect of the hypothalamus. Postoperative lateral angiogram (*D*) shows complete obliteration.

We alluded above to the AVMs of the trigonal region and dorsal thalamus, which are usually supplied by posterolateral choroidal arteries. As stated above, the more medial lesions involving the posterior third ventricle and the area of the velum interpositum are approached parasagittally through a callosal incision just anterior to the splenium, and, here, the predominant blood supply is from the posteromedial choroidal arteries.

Striato-Capsulo-Thalamic Region AVMs

When these lesions are large and have predominantly perforator supply, we prefer either to leave them alone or to treat them with radiosurgery. Clearly, the lesions lateral to the internal capsule, involving the lateral basal ganglia and insula, can be operated on with acceptable morbidity. The lesions of the head of the caudate, as indicated above, are also quite operable, as are the lesions involving the dorsal posterior aspect of the pulvinar of the thalamus. The lesions involving the posterolateral inferior aspect of the thalamus invariably involve the lateral geniculate body and can be removed by a temporal approach if the patient already has a complete hemianopsia; otherwise, radiosur-gery should be considered. Other surgeons have been more aggressive with these thalamic and basal ganglia parenchymal lesions, and good results have been reported frequently.[70,103,108,121,124]

Cerebellar AVMs

We approach cerebellar AVMs differently depending on where they are located. Superior vermian AVMs are invariably supplied by branches of the superior cerebellar arteries, most frequently bilaterally. To reach these branches safely, we prefer a supracerebellar infratentorial approach, for which we favor the sitting position so that the cerebellum can fall away from the tentorium. The surgeon must be particularly careful with the arterialized veins that drain the lesion from the cerebellum to the tentorium and sometimes working "around" these veins presents a problem.

Posterior and inferior vermian AVMs are approached suboccipitally in the prone or the three quarters "park bench" position, which allows the surgeon to sit, which is important during these frequently long operations. These lesions are supplied predominantly by posterior inferior cerebellar arteries, but frequently get some deep supply
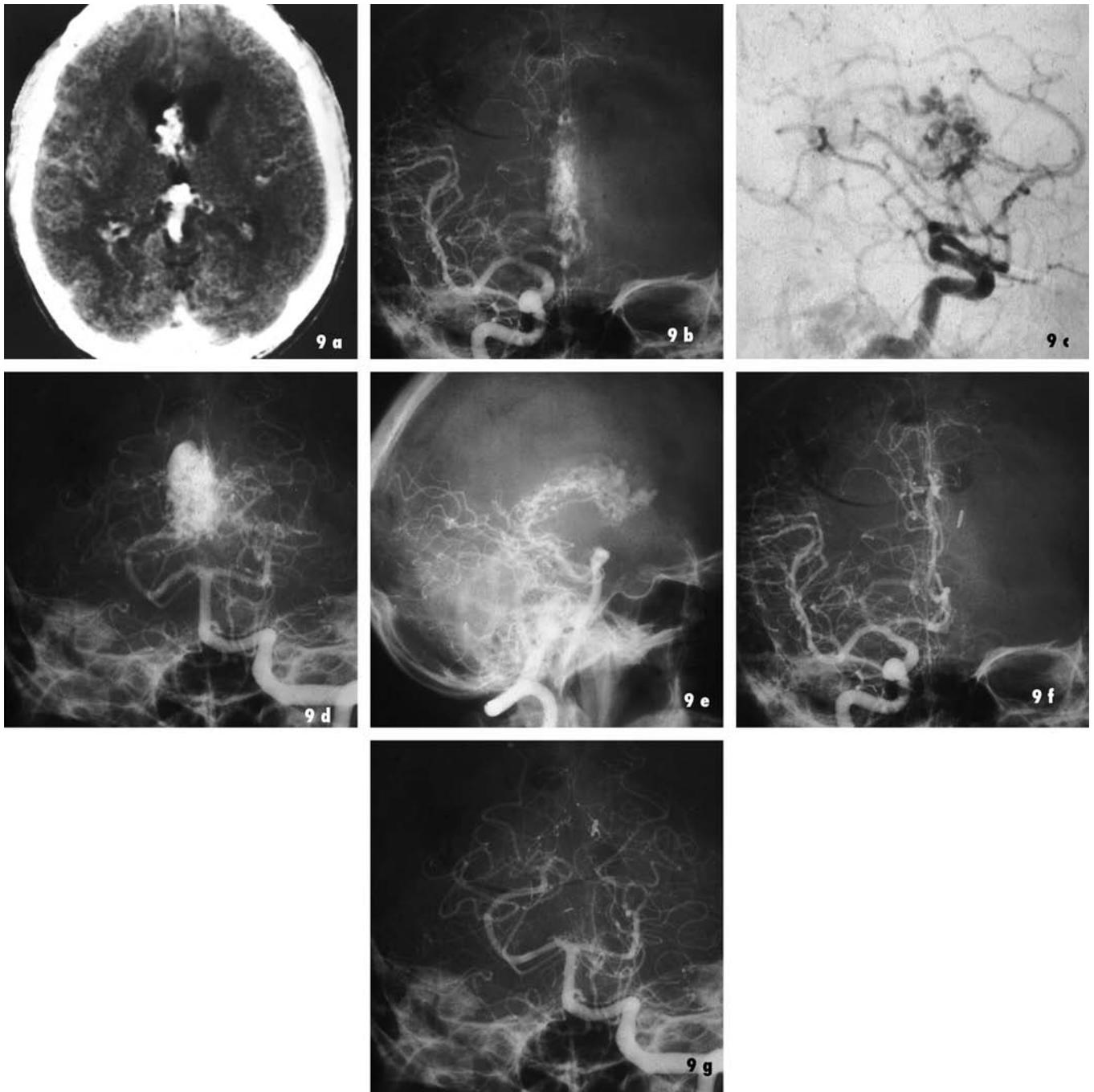
FIGURE 13.9. Intraventricular AVM. CT scan (*A*) shows an intraventricular lesion consistent with an AVM. Note dilated veins in the septal region, tela choroidea, and in the body of the lateral ventricle. AP (*B*) and lateral (*C*) carotid; and AP (*D*) and lateral (*E*) vertebral angiograms confirm the diagnosis of AVM fed by the ependymal branches of the distal ACA and the medial posterior choroidal branches of the PCA. This AVM was excised via a transcallosal/subchoroidal approach. Postoperative AP carotid (*F*) and vertebral (*G*) angiograms show complete obliteration.

from superior cerebellar branches. When they reach the fourth ventricle, they invariably get deep transependymal feeders.

The more lateral hemispheric cerebellar AVMs, which frequently can be quite large, are generally approached on the lateral position through a retromastoid craniectomy, which

can be extended to the midline and/or to a far lateral suboccipital approach, depending on the location of the AVM. With a combined generous suboccipital retromastoid craniotomy extending into the midline inferiorly and far laterally on the ipsilateral side, the surgeon can approach the superior cerebellar supply by working above the cerebellum, the anterior inferior cerebellar supply by identifying those branches at the cerebellopontine angle, and the posterior inferior cerebellar supply by following the posteroinferior cerebellar artery branches to the lesion. The senior author has been surprised at the relative mildness of the neurological deficit resulting from excising very large lesions that involve practically all of the cerebellar hemisphere (*Fig. 13.10*).

Brainstem AVMs

We generally do not operate on AVMs of the brainstem. Most of the time these lesions are fed by perforating branches that come across the critical parenchyma of the brainstem and intolerable damage can result from an attempt to remove these lesions. The only lesions we have operated that involve the brainstem are subpial lesions, generally in the area of the cerebellopontine angle, that are supplied purely by circumferential branches that can be controlled before they reach the lesion. However, we recall one occasion when even after placing temporary clips in all of the branches that we thought fed one of these lesions, the draining vein was still red. We presume that although we did not see it angiographically, this lesion had deeper perforator supply and, therefore, we left it alone and referred that patient for radiosurgery.

We have also operated on a couple of lesions that involved the tectum of the mid brain in patients who already had some degree of Parinaud's syndrome. Again, these lesions were fed by circumferential branches of the superior cerebellar and posterior cerebral arteries and did not have deep perforating blood supply (*Fig. 13.11*).

COMPLICATIONS

The management of cerebral AVMs is one of the most important challenges to neurosurgeons, which requires careful preoperative and intraoperative judgment.
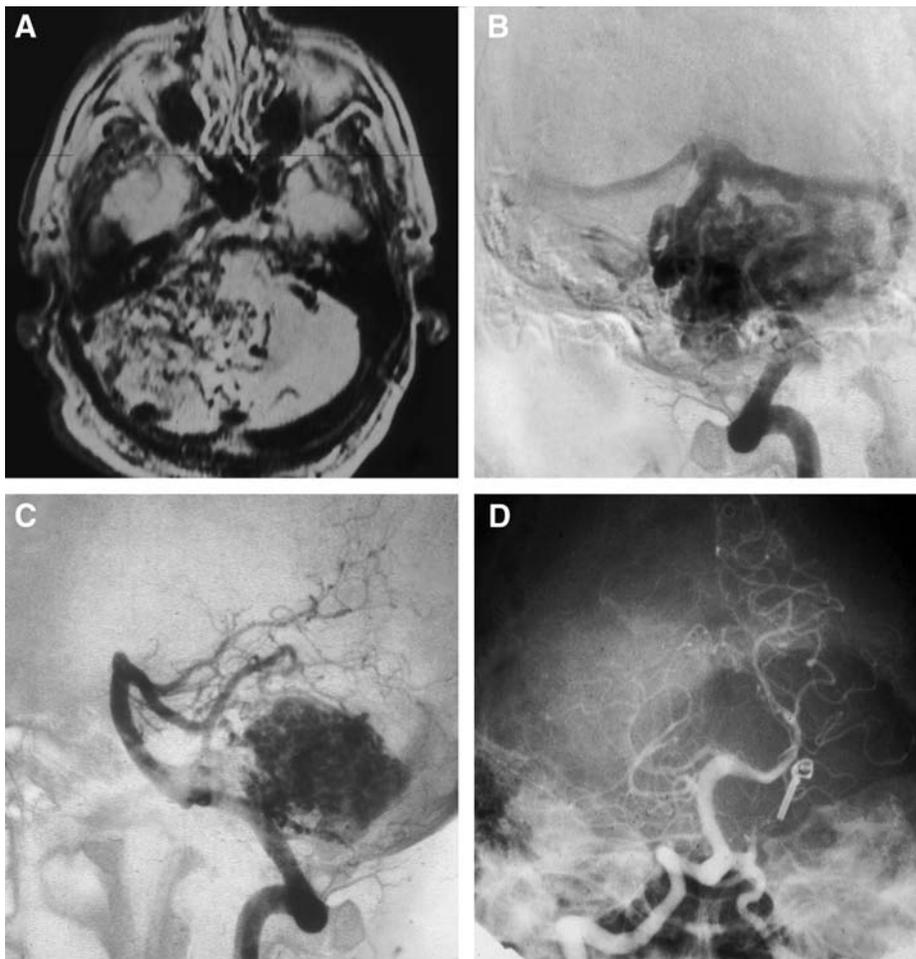

FIGURE 13.10. Lateral cerebellar AVM. T1-weighted axial MRI scan (*A*) shows a large AVM involving almost the entire right cerebellar hemisphere. AP (*B*), lateral (*C*), and late phase oblique (*D*) vertebral angiograms show this AVM fed by the posterior inferior cerebellar, anterior inferior cerebellar, and superior cerebellar arteries. Surgical approach was via large lateral suboccipital craniotomy extending into the midline, retrosigmoid region and far laterally on the ipsilateral side. Postoperative AP vertebral angiogram (*E*) shows complete obliteration.
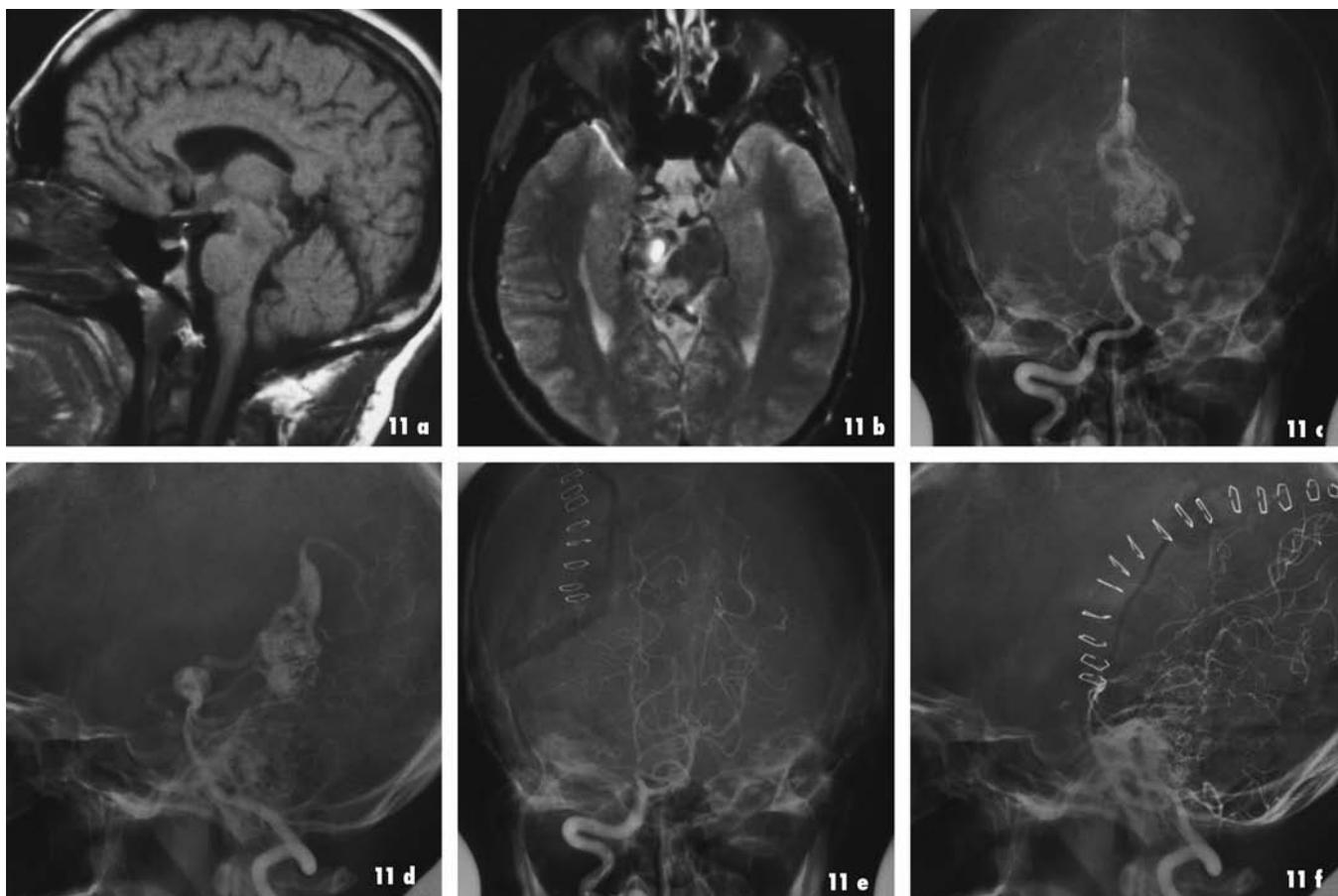
FIGURE 13.11. Tectal AVM. Sagittal (*A*) T1- and axial (*B*) T2-weighted MRI scans show a lesion consistent with an AVM in the pial surface of the tectal region. AP (*C*) and lateral (*D*) vertebral angiograms show an AVM fed by the circumferential branches of the superior cerebellar and posterior cerebral arteries. Postoperative AP (*E*) and lateral (*F*) vertebral angiograms show complete excision achieved via large posterior temporal occipital transtentorial approach.

Preoperative Judgment Problems

Selection of Treatment Modality

There are lengthy discussions in the literature regarding the importance of proper selection of patients with AVMs for conservative treatment, surgical resection with or without embolization, palliative embolization, or radiosurgery. In previous communications,[45,85] we have made the point that relatively young patients in good health with Spetzler-Martin grade I, II, and III AVMs should generally be treated by microsurgical resection because surgery results in immediate and permanent elimination of risk of hemorrhage, is very safe and is also extremely cost-effective in the long run when compared with conservative management or radiosurgery. As presented in *Tables 13.7* and *13.8*, results of the senior author's surgical series indicate that surgery for grade I and II AVMs is extremely safe (no mortality and no poor results).[43] Grade III AVMs can generally be resected with acceptable morbidity although we have gradually understood that some

grade III AVMs cannot be excised safely and we now regret not having treated some of these AVMs in critical regions with radiosurgery. However, as our own results demonstrated, early morbidity is very high for grade IV AVMs and forbidding for grade V AVMs. This leads to the inevitable conclusion that the majority of patients with grade V AVMs should be treated conservatively. It is clear that patients with grade V AVMs (larger than 6 cm, involving or immediately adjacent to eloquent brain regions and extending deeply to the point of acquiring deep venous drainage) who underwent microsurgical resection comprise the largest number of our complications. We have not operated electively on a single grade V AVM since 1999.

Faulty Spatial Conceptualization

It is important to fully understand the topography of the lesion when planning surgical resection of AVMs. Compared with CT and angiography, high-resolution MRI provides an excellent definition of the exact location of the AVM and its

extension into the eloquent brain regions. One of our most common errors has been the assumption that a large AVM was adjacent to rather than intrinsic to the sensory-motor or speech cortex, to the internal capsule, or to vital brainstem structures. Most of these complications occurred before MRI was available, when we depended to a large degree on the arterial supply to estimate where the AVM was located.

Improper Assessment of Medical Condition

Underestimation of the surgical risk attributable to medical comorbidities is another judgment problem encountered during management of AVMs. For example, our single mortality among patients with grade III AVMs came from our faulty judgment. This particular patient had an elevated prothrombin time caused by hepatic cirrhosis. Because he was relatively young, he underwent surgery for resection of his AVM with preoperative administration of vitamin K and under intraoperative coverage with fresh-frozen plasma, as recommended by the consulting hematologist to prevent bleeding. We did not, in fact, have any hemorrhagic problems during surgery, but the patient never woke up from hepatic coma because of massive liver necrosis induced by anesthesia. The error here was an improper weighing of the risk of surgery as contrasted with the natural history of the disease. Additionally, we paid too much attention to the patient's young age, when, in fact, we should have realized that his longevity was limited because of his serious comorbidity. This patient would have most likely died of his liver disease before bleeding from his AVM if we had left the AVM alone.

Intraoperative Complications

Parenchymal damage can occur in various ways. Injury occurs when the surgeon uses *too wide a margin of resection* around the AVM. A plane of resection more than a few millimeters away from the lesion reduces bleeding but also damages surrounding eloquent brain. A bloodless, relatively avascular surgical plane usually indicates that dissection has been carried into normal brain. Another important point is that the so-called "gliotic plane" around the AVM may not be found in every case, and, therefore, cannot always be used as guidance. In the senior author's experience, such a plane exists practically only when there has been previous hemorrhage and, even then, it is almost never around the entire circumference of the lesion.

Another source of parenchymal damage is to *take feeders too far away from the AVM*. With some exception (frontal and temporal polar lesions, some lesions of the cerebellar hemisphere, and occipital lesions in patients with full hemianopsia) feeders should be taken only at the point where they enter the nidus. This is important to prevent damage to arterial branches that contribute to the perfusion of the normal surrounding brain. During exposure of the AVM, if feeders are not visible on the cortical surface, they should be searched meticulously in a systematic fashion in the depth of the sulci surrounding the nidus. For this purpose, the angiogram should be used as guidance. These feeders should be identified, followed to the nidus, and secured and divided only when there is no doubt that they are feeding the AVM. "Vessels *en passage*" are most frequently found with AVMs in the sylvian fissure and callosal region where the distal MCA and pericallosal branches, respectively, should be skeletonized, dividing only the side branches that clearly feed only the lesion and preserving the main trunks, which are then likely to go on to supply normal brain.

Deep perforators are fragile vessels that are difficult to coagulate and that tend to retract into the normal brain tissue. Parenchymal damage can occur when they have to be followed into normal parenchyma to stop bleeding. This is the reason that the senior author does not generally recommend surgery for AVMs intrinsic to the basal ganglia, thalamus, and brainstem. The exceptions are dorsal and lateral thalamic AVMs that have an ependymal or cisternal representations and, therefore, a choroidal or circumferential (as opposed to perforating) supply; lateral basal ganglia AVMs, whose lenticulostriate supply is lateral to the internal capsule; and small superficial brainstem AVMs supplied entirely by circumferential or choroidal vessels. When deep bleeding occurs, it is important not to "pack" the hemorrhage because packing can result in deep parenchymal or intraventricular hemorrhage, which may not be recognized immediately and can result in significant damage. Micro-AVM clips are found to be very useful to stop bleeding from these deep perforators, which frequently do not respond to bipolar coagulation.

Excessive retraction is another source of parenchymal injury. Brain edema adjacent to the AVM caused by excessive retraction may be responsible for a large number of temporary deficits. Kattah et al. considered that spreading depression of cortical activity caused by surgical manipulation is an important factor in these transient deficits.[57] Yaşargil coined the term "temporary blocked syndrome" for these deficits observed after surgery, and suggests that there is a similarity between this syndrome and the transient postictal deficits observed after seizures.[126] Retraction injury can be prevented with adequate positioning and a wide-enough craniotomy, including a cranial base approach if necessary to gain access to deeper lesions or to deep arterial supply with a minimum of brain retraction. The senior author long advocated that resection of a small portion of non-eloquent brain is preferable to excessive retraction to gain deep access, which can not only damage brain parenchyma directly but also through injury to bridging veins.[42] Early surgery after hemorrhage from an AVM should be avoided in all but small superficial AVMs because AVM surgery is usually elective surgery that should be performed under optimal conditions. Because the risk of early rebleeding is relatively low (approximately 6% during the first 6 months), there is little reason for

early resection of an AVM after hemorrhage when the brain may be swollen and the plains are not clear. As discussed above, when the patient requires evacuation of a life-threatening hematoma, we prefer a "conservative" evacuation, avoiding getting into the AVM unless the AVM is small and simple.

Damage to normal major bridging veins may also result in significant parenchymal injury. This is another reason we always recommend wide-base craniotomy flaps that allow the surgeon to find alternative routes around or between major veins, particularly with parasagittal and subtemporal approaches. Preoperative embolization often eliminates the need for early parasagittal or subtemporal retraction, thus, allowing the surgeon to approach the AVM by a more direct transcortical approach without putting these bridging veins in danger. Here again, resection of a small amount of non-eloquent brain, as in the inferior temporal gyrus for example, may reduce the risk of tearing or thrombosis from stretching of an important vein, such as the vein of Labbe.[42]

The visual radiations can be damaged during the resection of AVMs in the temporal and occipital lobes. We analyzed the effect of surgery on the visual fields in 156 patients with supratentorial AVMs.[62] Of these, 18 patients developed a new visual field deficit or worsening of a preoperative deficit as a result of surgery. Most of the deficits were expected by the surgeon and explained preoperatively to the patient, and 72% of the deficits occurred in patients with deep temporal or occipital AVMs. The three-dimensional course of the geniculocalcarine fibers and the Meyer's loop around the temporal horn of the lateral ventricles should be kept in mind in planning an approach to AVMs located in these areas. We prefer the transsylvian approach for anteromedial temporal AVMs. Although the subtemporal approach can be used for resection of small AVMs of the deep mid- and posterior temporal lobe, for more posterior and medial AVMs, we use an inferior temporal gyrus approach, staying underneath the optic radiation and avoiding excessive temporal lobe retraction. AVMs of the roof and medial wall of the atrium are usually approached by a transcortical posterior parietal lobule route, as previously described by the senior author.[42] Yaşargil advocates the posterior interhemispheric-transprecuneus approach to medial trigonal (parasplenial) AVMs.[127] The senior author has tried this approach but, not being endowed by Yaşargil's surgical finesse, has found it extremely difficult and will not recommend it except for very small AVMs.

Intraoperative hemorrhage resulting in major morbidity and mortality results frequently from bleeding from the AVM or from premature occlusion of venous drainage. Excessive and difficult to control bleeding from the AVM usually results from the faulty judgement of the surgeon who starts dissection of the AVM before a reduction in afferent arterial supply by early surgical control of feeders or by

preoperative embolization. It is also essential to preserve venous drainage from the AVM until the arterial supply to the lesion has been completely occluded. However, in some instances, it is difficult to work around the superficial venous drainage, but only if there is major deep drainage should superficial draining veins be sacrificed in the early phase of dissection. Differentiating draining veins from feeding arteries might be difficult in some cases because the latter can be quite thin and abnormal as they approach the AVM. The feeding arteries can be identified by their relatively thicker wall and more vigorous pulsations under high microscopic magnification. In case of doubt, as discussed above, temporary clipping of the vessel may enable the surgeon to tell whether the vessel is relatively collapsed distally, as the vein would be expected to be, or whether it continues to pulsate vigorously as a feeding artery would.

Bleeding from the AVM can be stopped by gentle packing against the AVM, but packing of bleeding from the brain side or in the depth of the dissection should be avoided at all costs, as mentioned above.

Postoperative Complications

Hemorrhage

The most serious complication after AVM surgery is hemorrhage from residual fragments of an AVM or from insecure hemostasis. An unrecognized small piece of residual AVM is most frequently the source of bleeding because the necessity to excise an AVM on a plane very close to its margin creates the potential for leaving behind small remnants of AVM which represent a significant risk of hemorrhage because they are still arterialized and frequently disconnected from their venous drainage. Additionally, at the end of the resection, in the deeper portion of the AVM, the surgeon frequently has difficulty differentiating true AVM from fragile deep feeding and draining vessels. Intraoperative angiography is very useful in detecting residual portions of AVM. However, certain operative positions may pose some difficulties in obtaining proper images. In these instances, the patient should undergo immediate postoperative angiography before awakening from anesthesia and, if any residual AVM is found, the patient should be then taken back to surgery for resection of the remaining AVM. The exception to the need for intraoperative or immediate postoperative angiography is with simple, small superficial AVMs, when the experienced surgeon can be relatively sure that the AVM has been completely removed.

To avoid the second error or "*insecure hemostasis*," we perform the entire procedure under normotensive blood pressure. The use of hypotension could reduce the amount of bleeding during surgical dissection but may increase risk of postoperative hemorrhage from insecure hemostasis. After resection of the nidus, we routinely elevate the blood pressure by approximately 20 to 30 mmHg over the pressure through-

out the operation. With this maneuver, we have encountered spontaneous bleeding within a few minutes in several patients. Meticulous control of the blood pressure to within a level below the level at which hemostasis was achieved is essential during the immediate postoperative period because blood pressure control has been shown to be the single most important factor in preventing delayed hemorrhages in patients with complete removal of their AVMs.[79] The overall incidence of delayed hemorrhage (within a week time) has been reported as approximately 2%.[79] Risk factors include AVMs with a grade higher than Spetzler-Martin grade II, size larger than 3 cm, and AVMs fed by the lenticulostriate arteries.[79]

Normal Perfusion Pressure Breakthrough

Reduction or elimination of blood flow through a high-flow AVM normalizes and redistributes blood to the adjacent normal brain tissue, which may have impaired autoregulation caused by chronic ischemia from chronic hypoperfusion (steal) by the AVM. The autoregulatory control, located possibly at the arteriolar level, having been chronically dilated, cannot sufficiently increase the resistance to the new perfusion pressure to protect the capillaries, which leads to breakthrough with resultant edema and hemorrhage.[111] Although we do not observe this problem frequently because of the selective use of preoperative embolization in large, high-flow AVMs, we are convinced of the validity of this theory. Angiographic features that may be associated with normal perfusion pressure breakthrough are long and tortuous, high-flow, large-caliber feeding arteries, diminished perfusion of adjacent brain, rapid early venous drainage, and diffuse margins of the nidus. The problem of normal perfusion pressure breakthrough can be avoided by a staged reduction in flow to the malformation through preoperative endovascular embolization.

In an analysis of our first 200 patients, we identified only 2 patients who developed a serious clinical problem that we attributed to this complication; we attribute this low rate to the liberal use of preoperative embolization in patients thought to be at risk for this complication.[9] In the large series of Yaşargil,[127] no patient developed this complication. Day et al.[13] recommended aggressive treatment with anti-edema therapy, barbiturate coma, and sometimes delayed evacuation of the hematoma. They reported three patients who developed this complication and, with aggressive therapy, as outlined above, two made an early good recovery and one showed slow improvement.

Venous Thrombosis

Although there is a theoretical risk of inducing stasis in long segments of veins where high flow is suddenly interrupted after resection of the AVM, retrograde thrombosis or postoperative venous infarction in the draining veins is a rare complication of AVM surgery. Miyasaka et al.[77] published the first case report of a well-documented postoperative venous thrombosis and hemorrhagic infarction after resection of AVM. In their case, clinical deficits developed on the third postoperative day. We also had a well-documented case in which there was a high-flow AVM of the cerebellar vermis that was excised without any problem. At the end of surgery, we noticed that the markedly enlarged internal cerebral veins, basal veins, and vein of Galen were relatively collapsed. The patient did not wake up from surgery and postoperative plain CT scan showed a clean resection bed with evidence of thrombosis of both basal veins of Rosenthal. Postoperative angiogram showed no filling of the deep venous system. The patient remained in coma and then gradually began to improve to the point that he had incomplete, but remarkable, recovery (*Fig. 13.12*).

Common features of the previously reported cases and the one presented above are high flow to the AVM, extensive retrograde venous drainage and frequent occlusion or stenosis of the antegrade venous drainage.[77] According to a study reported in 33 patients in whom flow velocities and CO_2 reactivities were measured, the flow velocity in the draining vessels was close to zero after removal of the AVM nidus.[39] This may lead to venous thrombosis in the draining vessels because pathological changes in the draining veins have already taken place because a normal vein with normal endothelium is not likely to thrombose.

When faced with a high-flow AVM in which the venous drainage is retrograde into normal veins that ordinarily drain parenchyma, it is desirable to reduce the flow gradually in the malformation with staged embolization or staged ligation of feeding arteries. Profound neurological deficits caused by venous thrombosis can have a much better prognosis for eventual recovery than similar neurological deficits caused by arterial occlusive disease, as in our case. An important clue that this type of complication would result in a good recovery is that the CT scans may not show significant, irreversible brain damage despite the profound neurological deficit during the initial postoperative period. Finally, these patients should be kept well hydrated during the intraoperative and postoperative periods to avoid further collapse of veins.

Vasospasm

In a series of patients who were surgically treated by the senior author, there was no postoperative complication that could be related directly to development of vasospasm. However, out of 414 patients who were operated by Yasargil, two patients postoperatively and one preoperatively developed this complication.[127] These patients had extensive dissection and exposure of the A1 and M1 segments of the ACAs and MCAs, respectively. This is considered as a rare complication, because, with AVMs, it is unlikely that large subarachnoid clots in the basal cisterns are observed, which
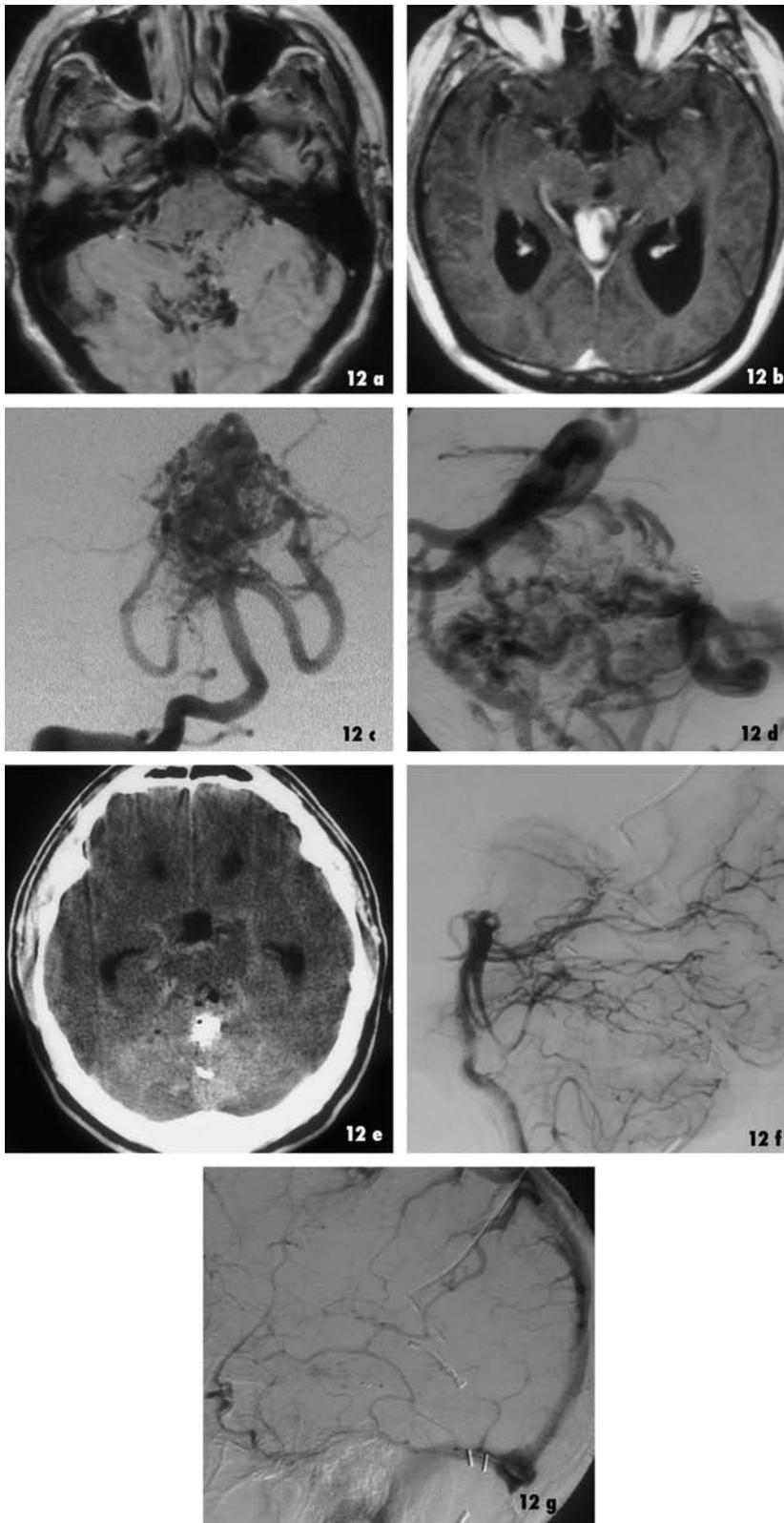
FIGURE 13.12. *A*, preoperative T1-weighted MRI scan shows a large but compact AVM located in the anterior superior aspect of the vermis. Note the much-distended vein of Galen on postcontrast T1-weighted MRI (*B*). AP vertebral angiogram (*C*) shows a large AVM mainly fed by the branches of the superior cerebellar artery. Note the later arterial phase (*D*) showing dilated arterialized vein of Galen draining toward into the deep venous system without any drainage into the straight sinus, which was presumably occluded."? CT scan (*E*) obtained immediately postoperatively shows the high-density area suggesting thrombosis in the basal veins of Rosenthal. Postoperative lateral (*F*) arterial phase vertebral angiograms show complete obliteration of the AVM. Lateral venous phase (*G*) shows no filling of the deep venous system.

are prerequisite for vasospasm after subarachnoid hemorrhage.

Retrograde Feeding Artery Thrombosis

Retrograde thrombosis of former feeding arteries has been described as a rare complication after resection of AVMs. Miyasaka et al.[75] reported five patients with this complication in a series of 76 patients. Out of five, three patients developed clinical symptomatology caused by hypoperfusion and ischemia. Old age, larger AVM size, and marked dilation and elongation of feeding arteries have been identified as potential risk factors in their study.

Stagnant arterial flow can frequently be observed immediately after surgical obliteration of AVMs and it may last up to 1 month. In a series of 52 patients, 61% had a prolonged transit time for contrast material in former feeding arteries and, in 29%, the washout occurred in the late venous phase in the angiogram, which is, by definition, stagnant arterial flow.[74] However, the question of whether this stagnant flow leads to hypoperfusion or hyperperfusion remains to be answered at present.

Seizures

In our earlier series of patients with preoperative seizures, seizure frequency improved in approximately 55%, remained unchanged in 33%, and worsened in approximately 12%. On the other hand, we have encountered new onset postoperative seizures in approximately 15% of patients who had no history of seizures preoperatively. This incidence ranges from 6.5 to 22% in the pertinent literature.[81] We found that immediate postoperative seizures carry a good prognosis and should be considered separately. We recommend antiseizure prophylaxis for at least 6 months in all patients after surgical excision of a supratentorial parenchymal AVM.

FINAL COMMENTS AND CONCLUSIONS

We have attempted to briefly discuss the various individual treatment modalities and multimodality paradigms for the management of AVMs. We have suggested indications for the major accepted modes of treatment: microsurgery, radiosurgery, embolization, and observation.

With cerebral AVMs, it is important to keep in mind that the treatment is most frequently aimed at preventing hemorrhage in the future. Rarely, we treat a cerebral AVM to improve symptomatology, such as intractable seizures or a progressive neurological deficit from "steal," venous hypertension, etc. In this context, it must be kept in mind that the risk of hemorrhage is essentially the same in a patient with an AVM that has never bled than that presented by a patient with an AVM that has bled in the past (longer than 6 months).[48] In other words, in general, the risk of hemorrhage of an AVM, whether it has bled or not, is approximately 3 to 4% per year. After hemorrhage, the risk is approximately 6% during the first 6 months, but then it settles down to approximately the same 3 to 4% per year risk of hemorrhage of AVMs that have never bled. This risk of hemorrhage from an unruptured AVM is considerably greater than that from an incidental aneurysm, a fact that is not widely recognized. Granted, the morbidity of aneurysmal subarachnoid hemorrhage is significantly higher than the morbidity of hemorrhage from an AVM, but, still, the latter is significant (approximately a 10% risk of death and approximately a 30% risk of serious neurological morbidity from each hemorrhage from an AVM). With these considerations in mind, we consider treatment of patients harboring cerebral AVMs whenever possible with acceptable risks whether they have bled or not in the past.[48]

As emphasized previously, each patient with an AVM must be approached individually, considering a multitude of factors, including the size, configuration, and location of the AVM, the age, health, and occupation of the patient, and the skill and experience of the treating team.

As stated above, the ideal treatment for cerebral AVMs is microsurgical excision, which immediately eliminates the risk of future hemorrhage. Therefore, in general, we recommend surgical excision of the AVM in patients who are relatively young and in good health, provided that the treatment can be accomplished with relatively low risk. As stated above, this is the case with practically all patients with Spetzler-Martin grades I and II AVMs and with most patients with grade III AVMs. As a result of an analysis of our own series,[46] as well as others from the literature, we have concluded that the treatment of grade V AVMs carries an unacceptable risk and, therefore, we rarely recommend treatment of these lesions. The same is the case with the majority of grade IV AVMs, although, by careful selection, we have kept the risk of operating on patients with grade IV AVMs to an acceptable level (serious morbidity and mortality of 12.2%).[46] When we use embolization, we use it specifically for the purpose of making the overall treatment plan of preoperative embolization and surgical excision safer; that is, when we consider the risk of embolization, which is not small, as discussed before, we must be convinced that the combined risk of preoperative embolization and surgery is smaller than the risk would be if we undertook surgical excision without embolization. Additionally, we very carefully discuss the aim of preoperative embolization with our endovascular colleagues, which is simply to make the surgery safer. Generally, this entails occluding deep feeding pedicles that are inaccessible during the early surgical stages. There is no point in taking the risk of occluding endovascularly feeding pedicles to which the surgeon has immediate access after exposure of the AVM, for example, cortical middle cerebral branches in a superficial AVM. Another aim of preoperative embolization is to significantly decrease flow in AVMs that are adjacent to critical areas of the brain, which then allows the surgeon to work at the very margin of the AVM with a considerably reduced risk of hemorrhage and

damage to critical brain. We do not think that embolization simply for the sake of reducing flow and making the surgery quicker and easier is justified under most circumstances, given the risk of embolization, except in the rare high-flow AVM in which there is a risk of "perfusion breakthrough" from sudden occlusion of the shunt by removal of the AVM.[111]

We think that there are few indications for primary treatment of an AVM by embolization without subsequent surgical excision. As discussed above, simply reducing the flow to an AVM without obliterating it completely by embolization seems not only to not reduce the risk of future hemorrhage, but it is likely that it increases it. Clearly, there are AVMs that can be occluded completely with embolization, but, as discussed above, these are usually AVMs that can be readily excised with minimal risk and, in general, we prefer the latter tactic because the risk is generally lower than the risk of embolization with these AVMs. Of course, there are patients who, because of their age or comorbidities, may be best treated by embolization to complete occlude their AVM, if this is possible and the AVM is too large for radiosurgery. There are other indications for embolization simply to reduce the flow of the AVM, such as in patients who present with progressive deficit from a steal, with intractable headaches caused by dilated dural feeders, or in patients who have particular features, such as aneurysms or direct fistulae that make us presume a higher risk of hemorrhage if left untreated. We have also used "palliative" embolization in patients with large, unresectable AVMs that present with accumulating deficits from multiple hemorrhages, but we are not sure that we alter favorably the natural history in these patients.

Radiosurgery is a most welcome addition to our armamentarium for treating cerebral AVMs. We recommend radiosurgery to patients who have a relatively small AVM (generally less than 3 cm, although we may stretch that size limit to 3.5 or 4 cm in some circumstances) and have AVMs located in critical areas of the brain where the surgical morbidity would be unacceptable. For patients with small AVMs in accessible areas of the brain, we recommend microsurgical excision, given its very low risk and immediate elimination of the risk of hemorrhage, as discussed above. However, in elderly patients or in patients with significant comorbidities, radiosurgery may be considered under these circumstances. As discussed before, one treatment paradigm that we have not been enthusiastic about is that of using preoperative embolization to "reduce the size of the AVM" so that it then becomes amenable to radiosurgery. We remain unconvinced that those parts of the AVM that seem to be completely obliterated by the embolization in the immediate postembolization angiogram remain, in fact, occluded and without risk of hemorrhage and, therefore, when we recommend radiosurgery, we recommend including all of the AVM,

whether embolized or not, in the field. That limits our indications for radiosurgery to relatively small AVMs and, in these cases, there seems to be no reason to use embolization given the fact that, as discussed above, embolization does not reduce the risk of future hemorrhage unless it results in complete obliteration of the malformation and, clearly, there is a substantial risk to embolization, as discussed above.

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