GIANT INTRACRANIALANEURYSMS

Giant intracranial aneurysms, or intracranial aneurysms greater than or equal to 2.5 cm in diameter, account for approximately 5% of all intracranial aneurysms. Their natural history, clinical presentation, location, and structural features are unique. They often present formidable technical and conceptual challenges to the neurosur-geon. In addition to special techniques such as tandem clipping, adjuncts such as brain resection, temporary arterial occlusion, cerebral ischemic protection, and hypothermic circulatory arrest are frequently required for successful surgical treatment.

Pathophysiology. Giant aneurysms may be broadly classified as traumatic, infectious, or idiopathic, with a saccular or fusiform structure. Fusiform or serpentine giant aneurysms often have vertebrobasilar or MCA location and have been associated with connective tissue disorders and luetic arteritis (syphilis). Idiopathic saccular aneurysms are the most common giant aneurysm type and will be the focus of this section.

Natural History. Although their natural history is not fully understood, giant aneurysms carry a rupture risk similar to or greater than the 3% per year risk of smaller aneurysms. Approximately half of those that rupture are fatal. Giant aneurysms typically present at age 40 to 60 years with pro gressive symptoms resulting from mass effect or ischemia. Like smaller berry aneurysms, they occur at arterial bifurcations and are more common in women. Unlike smaller aneurysms, which usually (90%) present with subarachnoid hemorrhage, giant aneurysms often present with severe headaches, progressive neurologic deficits, transient ischemic attacks, or stroke. Only 30% of giant aneurysms present with hemorrhage. The location of giant aneurysms differs from that of the smaller ones. Giant aneurysms arise with relatively greater frequency at the paraclinoid and cavernous internal carotid artery and in the posterior circulation **(Table 1)** (Anson J.A., 1995). They are also more frequently associated with connective tissue disease and comprise a greater percentage (20%) of childhood intracranial aneurysms. Most patients with untreated symptomatic giant aneurysms die within a few years of hemorrhage, ischemia, or mass effect.

TABLE 1. Comparison of giant and berry intracranial aneurysm location distributions

Intracranial location	Internal carotid	Anterior cerebral artery	Middle cerebral artery	Posterior cerebral artery	Vertebrobasilar	Distal or mino arteries
Berry aneurysms (%)	33 39-55	3810-13	17 12-25	23	12 7-27	2
Giant aneurysms (%)						4

Management. Options include expectant treatment, endovascular treatment, and direct microsurgical treatment. The goals of treatment are to improve or maintain the patient's neurologic status, exclude the aneurysm from the circulation, and reduce or eliminate aneurysmal mass effect. Surgical treatment options include bypass grafts, proximal (hunterian) ligation, simple trapping, and direct clipping with vessel reconstruction. Direct clipping is the treatment gold standard but is often difficult to achieve. Structural features of giant aneurysms that present uniquely difficult problems in clip application and aneurysm occlusion include the following:

1. Their large size, makes dissection treacherous and increases the risk of damaging adjacent structures.
2. Thin-walled areas are common and increase the risk of intraoperative rupture.

3. Wide-based necks, often incorporating parent or perforating arteries, are difficult to occlude without compromising arterial lumens; permanent clips may be pushed into the residual arterial lumen by neck distention.

4. Intraluminal thrombus and other debris increase the danger of distal embolization into the parent vessel during surgery.

5. Frequent calcification and tough atheroma in the neck prevent clip blade closure, cause clip slipping onto the parent artery, and may tear the intima during clipping.

6. Partly extradural necks in carotid ophthalmic and paraclinoid artery aneurysms increase the likelihood of parent vessel sacrifice.

Proximal control allowing temporary arterial occlusion and cerebral ischemic protection are fundamental to giant aneurysm surgical treatment. Special clip application techniques, such as tandem or stacked clips, are also frequently useful. In many cases, temporary aneurysm trapping is required, followed by wide opening and evacuation prior to definitive clip reconstruction. Endovascular treatment is a seductive option that has yielded excellent early angiographic results. However, because endovascular occlusion fails to exclude the aneurysm mechanically from the circulation, the hemody-namic stresses that led to initial aneurysm development often produce recurrence. Preliminary studies indicate a high recanalization rate. **Endovascular occlusion is best reserved for the following situations: (a)** after subarachnoid hemorrhage in the poor grade patient, (b) recurrent posterior circulation giant aneurysms, and (c) patients whose medical condition precludes general anesthesia or surgery.

Surgical Principles. The following section briefly discusses surgical principles -proximal ligation, revascularization, and hypothermic circulatory arrest - uniquely helpful in treating giant intracranial aneurysms. Previously discussed principles that are helpful in giant aneurysm treatment include temporary arterial occlusion and booster, tandem, and stacked clips.

Proximal Arterial Ligation. Although proximal arterial occlusion obliterates or reduces giant aneurysm size, the associated increased risk of cerebral ischemia has limited its practice. Currently, proximal arterial ligation is employed for aneurysms involving the basal carotid and intradural vertebral arteries. To help predict which patients can withstand permanent carotid occlusion, numerous techniques using endovascular balloons have been devised. The most commonly used methods include the trial balloon occlusion test and regional cerebral blood flow measurements. In patients who demonstrated adequate collateral circulation with these methods, a reduction in but not complete elimination of ischemic complications was found. Miller (1977) found that if *25%* reduction in cerebral blood flow was used as a critical level for tolerance, patients with less than this percentage had little or no risk for ischemic complication after carotid artery occlusion. Gradual or graded occlusion to permit collateral blood flow development is advocated by some, but results from previous studies show no significant benefit. A long-term follow-up report after carotid arterial occlusion demonstrated an 8% rebleed rate and a 16% delayed stroke rate. In addition, hypertension and new intracranial aneurysm development contralateral to the ligation have been reported. Despite these risks, one study noted that 86% of aneurysms treated by carotid ligation showed a reduction in size or obliteration (Roski R.A., Spetzler R.F., 1985). Trial occlusions of the basilar artery, in the author's experience, have been unreliable and accompanied by numerous neurologic complications. Occlusion of perforating vessels in the «dead» arterial segment may produce false positive studies.

Bypass Procedures. When a giant aneurysm cannot be definitively clipped and the patient does not tolerate trial occlusion while anticoagulated, revascularization procedures should be considered. Giant aneurysms of the basal carotid artery (cavernous sinus, petrosal, and paraclinoid segments) and giant MCA aneurysms are often treated with bypass. Depending on the tolerance of a trial occlusion, a large-volume conduit or a low-flow anastomosis may be selected as a revascularization method. In our experience, low-flow anastomoses carry a lower morbidity than high-flow anastomoses. In the large-volume conduit, a long saphenous vein graft is interposed between the external carotid artery and an M2 branch. For the low-flow revascularization, the superficial temporal artery is directly anastomosed to an M3 branch or extended with a short vein interposition graft (Samson D.S., 1990). For intracavernous giant aneurysms, revascularization through a short venous graft

from the petrous to the supraclinoid carotid artery has been described (Spetzler R.F, Et al., 1990). During the revascularization period, the patient should be in a drug-induced EEG burst-suppressed and normotensive state.

Hypothermic Circulatory Arrest. Hypothermic depression of cerebral metabolism is well known. Whereas temporary occlusion of less than 15 minutes can be managed with pharmacologic metabolic suppression alone, mild hypothermia with the body temperature decreased to approximately 32°C should be considered for more complex cases. Profound or deep hypothermia to 16°C and complete circulatory arrest should be used when a greater than 20-minute occlusion time is anticipated. To accomplish hypothermic circulatory arrest, a multispecialty team including a neurosurgeon, a cardiothoracic surgeon, a bypass pump team, and an anesthesiologist with cardiothoracic and neurologic experience is required. Numerous groups over the past few decades have reported improved treatment results for giant intracranial aneurysms with hypothermic circulatory arrest (Giannotta et al., 1991; Spetzler R.F. et al., 1988; Williams M.D. et al., 1991). In addition to increased ischemic tolerance of up to 50 minutes, the drainage of blood into the bypass pump reservoir allows aneurysm collapse and facilitates neck dissection and aneurysm clipping.

INTRACRANIAL VASCULAR MALFORMATIONS

Over the past decade, the advent of magnetic resonance imaging (MRI) and refinements in endovascular emboli-zation, microsurgery, and stereotactic radiation have provided more options for treatment and better outcomes in patients with intracranial vascular malformations. For complex vascular malformations, a multidisciplinary team approach that combines neuroradiology, radiation oncology, and neurosurgery has been the best management strategy (Lawton M.T., Hamilton M.G., Spetzler R.F., 1995). A thorough understanding of the natural history, pathophysiology, benefits and risks of each procedure, and outcome are required to counsel patients. In this chapter the classification and pathology of the four basic intracranial vascular malformations are presented: arteriovenous malformations (AVMs), cavernous malformations, venous malformations, and capillary telangiectasias. The natural history, clinical presentation, radiologic features, treatment options, and perioperative management of intracranial vascular malformations are discussed.

CLASSIFICATION AND PATHOLOGY

Our understanding of the anatomy and pathology of intracranial vascular malformations has evolved during the past three decades, largely driven by improvements in radiographic imaging. Cerebral angiography come into widespread use in the 1960s, computed tomography (CT) in the 1970s, and MRI in the 1980s. The most widely used classification SCHEME relies on MeCormick's large autopsy series published in 1966. Based on that study, intracranial vascular malformations are divided into four major categories.

Arteriovenous Malformations. AVMs are congenital lesions with primitive arteries that shunt directly into veins or venule with no intervening capillary network (Fig. 2).

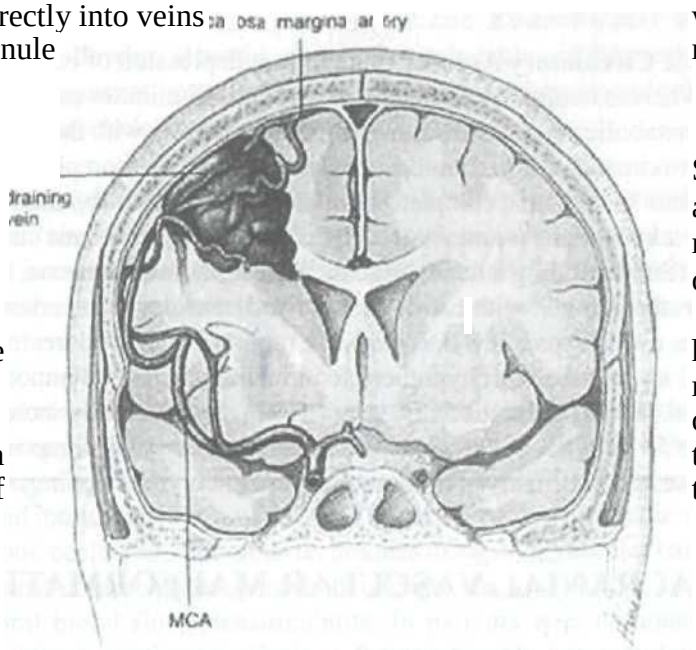


Fig. 2 Schematic drawing showing an arteriovenous malformation with multiple feeding arteries and a primary draining vein.

They are high-pressure and high-flow vascular malformations that cause significant damage to the surrounding brain when they rupture. AVMs on the surface of the brain are often cone

shaped and extend to the ventricular system. The arteries vary in width (transitional) and often have duplication or absence of the internal elastic lamina and tunica media. The veins become dilated, tortuous, and elongated in response to the high arterial pressures. The vein walls often become thickened and stenotic, or thin and aneurysmal (Fig. 3). There may be intervening, nonfunctional gliotic brain tissue. Rapid arterial shunting leads to poor perfusion and ischemia in the surrounding brain. Capillaries adjacent to the AVM become enlarged in response to the ischemic tissue. Alternatively, the capillaries can be dilated and irregular and may represent a transition zone from capillary telangiectasia to AVM.

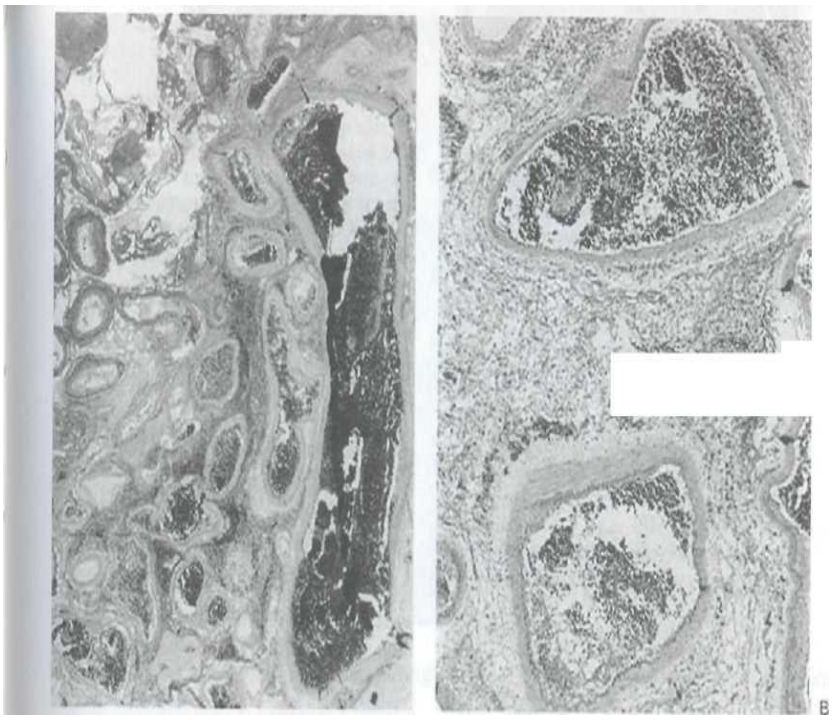


Fig. 3. (A, B) Hematoxylin and eosin stained micrograph of the surgically removed arteriovenous malformation. The vessels are often dilated, and the vessel walls vary in thickness. [18x (A) and 58x (B) magnification.]

Cavernous Malformations. Cavernous malformations are dilated sinusoidal spaces lined by a single layer of endothelium, usually without intervening brain tissue. Macroscopically, they are lobulated and resemble a purple mulberry. These are low-flow lesions that tend to grow by internal thrombosis and hemorrhage within the sinusoidal spaces. The hemorrhage is usually not catastrophic and rarely extends into the subarachnoid space. The surrounding brain forms a well-defined plane that is hemosiderin stained and gliotic. The walls of the malformation lack smooth muscle and an elastic lamina; therefore, there are no true arteries or veins. Occasionally, cavernous malformations have intervening brain tissue (racemose type). There is a 15% association with venous malformations (Robinson J.R. et al., 1993).

Venous Malformations. Venous malformations are an abnormal collection of histologically normal veins that converge in a radial fashion into a single large draining vein. This pattern has been described as caput medusae or spoked wheel. In contrast to cavernous malformations, there is normal intervening brain tissue. There is no arterial input. Venous malformations are located in the white matter, frequently near an ependymal surface. If a hemorrhage or seizure occurs, then an associated cavernous malformation or AVM should be suspected. In fact, venous

malformations drain normal brain tissue and are left in situ when associated cavernous malformations or AVMs are removed (Sasaki O. et al., 1991).

Capillary Teiangiectasias. Capillary teiangiectasias are an abnormal collection of dilated capillaries of variable size with intervening brain tissue **(Fig. 4**). They are clinically silent and are discov ered most frequently in the pons. They rarely exceed 1 cm in size and are detected only on contrast-enhanced MRI scans. Capillary teiangiectasias may represent a precursor or transitional form of cavernous malformations (Awad l.A. etal., 1993).

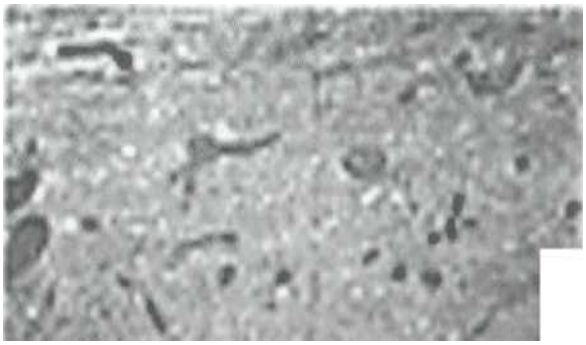


Fig. 4. Capillary teiangiectasias with multiple dilated irregular capillaries.

NATURAL HISTORY

The natural history of intracranial vascular malformations is poorly understood. Clinical evaluations have been neither prospective nor repetitive, and a large percentage of patients have been lost to follow-up. Many studies performed before the advent of CT and MRI probably underestimated the rate of hemorrhage. It is with these limitations that the natural history of intracranial vascular malformations is described below.

Arteriovenous Malformations. AVMs are the best studied vascular malformations in the brain. Several studies show that the yearly risk of hemorrhage is approximately 4% for symptomatic AVMs and 2% for asymptomatic AVMs. Whether the patient has suffered a hemorrhage or a seizure, the risk of rebleeding remains the same. The average interval between the initial symptoms and the rehemorrhage is 8 years. In a cooperative study of 453 patients who had AVMs, there was a 20% risk of a major neurologic deficit and a 10% risk of death from each hemorrhagic episode. Several long-term studies show an average mortality rate of 1% per year (Ondra S.L., Troup H., George E.D., Schwab K., 1990).

Cavernous Malformations. Knowledge of the natural history of cavernous malformations is closely tied to the advent of MRI because they are angiographically occult; CT does not provide enough resolution to document subacute, chronic, and silent hemorrhages (Robinson J.R., Awad I.A, Little J.R., 1991). Symptomatic cavernous malformations have a 4.5% yearly hemorrhage rate, and asymptomatic or incidental cavernous malformations have a 1% yearly hemorrhage rate (Robinson J.R., Awad LA, Little J.R., 1991). These hemorrhage rates are calculated from birth. If the hemorrhage rate is calculated from the onset of symptoms, frequently the recurrent hemorrhage rate is 35% to 55% per year. The rate of repeat hemorrhage varies greatly for patients with cavernous malformations; some patients have an interval of 25 years between hemorrhagic episodes, whereas others suffer three or more hemorrhages in less than a year. Patients at higher risk for hemorrhage may include those who have the familial

form, are pregnant, have received whole-brain irradiation, have an associated venous malformation, or have a cavernous malformation in a deep location (Robinson J.R. et al, 1993).

Venous Malformations and Capillary Telangiectasias. Venous malformations and capillary telangiectasias are clinically silent, and the natural history is considered benign (Garner T.B., 1991). A hemorrhage may be mistakenly ascribed to the venous malformation when there is an associated cavernous malformation. Capillary telangiectasias are usually incidental lesions found at autopsy. Of 10 patients with pontine capillary telangiectasias followed by the authors prospectively (for 1 to 5 years with a mean 3.2 years) using MRI studies, none have suffered a hemorrhage.

CLINICAL PRESENTATION

Hemorrhage is the most frequent presentation for patients with intracranial vascular malformations. AVMs have higher arterial pressures than cavernous malformations and cause more damage to the surrounding brain. Unlike cerebral aneurysms, early rebleeding (within 2 weeks) and cerebral vasospasm is rare in AVMs because the hemorrhage is located WITHIN THE BRAIN PARENCHYMA RATHER THAN THE SUBARACHNOID SPACE. Many patients will show neurologic improvement as the clot shrinks and is reabsorbed. Most operations are performed 1 to 4 weeks after the hemorrhage when the patient has reached a plateau in terms of neurologic recovery, the clot is organized, and the brain is less friable (Lewis A.I. et al., 1995).

Arteriovenous Malformations. Most AVMs are discovered in patients during their third and fourth decades. The presenting symptom is a spontaneous hemorrhage. Children are seven times more likely to suffer a hemorrhage than a seizure as the initial presentation. In neonates, vein of Galen malformations may cause high-output congestive heart failure. Hemorrhage is the initial presentation in 90% of patients with AVMs located in the basal ganglia, thai amus, and posterior fossa (Tew J.M., Lewis A.I., ReichcrtK.W., 1995). Pregnant women are more likely to suffer a rehemorrhage than nonpregnant women with AVMs. However, the increased risk of rebleeding does not parallel increases in blood volume during pregnancy. The peak incidence of hemorrhage occurs between the 15th and 20th week of pregnancy.

In comparison with ruptured cerebral aneurysms, hemorrhage from AVMs is usually less severe. Intraven-tricular hemorrhage is seen in only 5% to 10% of cases. Several studies have shown an increased risk for hemorrhage in AVMs that are small in size. Higher perfusion pressure in smaller AVMs may be the cause of the increased incidence of hemorrhage. In addition, AVMs that have pedicular or intranidal aneurysms, a single draining vein, or venous ectasia are at higher risk for hemorrhage.

Seizures are the second most common presentation in patients with AVMs, occurring in 11% to 33% of cases. Patients with large convexity AVMs are more likely to suffer a seizure than hemorrhage. The seizures may be caused by gliosis in the surrounding brain that occurs from hemosiderin deposition and perinidal inflammation. The onset of a new headache prior to hemorrhage or seizure ranges from 5% to 35%. The headaches are usually ipsilateral to the AVM. Patients with occipital AVMs frequently have visual scotomas and severe mi-grainous headaches. The headaches are thought to arise from stretching of the dura, elevated venous pressures in the dural sinuses, or hydrocephalus.

A small percentage of patients develop a progressive neurologic deficit or cognitive decline from arterial steal. These AVMs are usually very large and frequently occupy more than half of a cerebral hemisphere. The rapid shunting of blood leads to decreased perfusion and ischemia in the surrounding brain. Alternatively, the progressive neurologic deficit may be caused by venous hypertension. Stenosis and occlusion of the arterialized draining veins leads to high pressures within the nidus that creates edema and reduced blood flow in the surrounding brain.

Cavernous Malformations. Seizures are the most common presentation for supratentorial cavernous malformations. Hemosiderin and gliosis surrounding the cavernous malformation is the presumed cause of seizures. Hemorrhage is the second most common presentation and headaches are third. Frequently, cavernous malformations are found incidentally on MRI scans. Epidemiologic studies show that hemorrhage and progressive neurologic deficits occur more frequently in women and seizures occur more often in men (Maraire J.N., Awad I. A., 1995). Fifteen percent of patients with cavernous

malformations have a familial inheritance pattern that is autosomal dominant. Seventy per cent of patients with the familial type have more than one cavernous malformation. By contrast, only 10% of patients with the sporadic form have multiple cavernous malformations.

Similar to patients with deep-seated AVMs, 90% of patients with cavernous malformations in the subcortical nuclei and brainstem present with hemorrhage and progressive neurologic deficits (Tew J.M., Lewis A.I., Reichcrt K.W., 1995). The hemorrhage is intraparenchymal and rarely extends into the ventricles or subarachnoid space. The most common location is the pons, and neurologic deficits include cranial nerve palsies (particularly abducens and facial), hemiparesis, hemianesthesia, and cerebellar ataxia.

Venous Malformations. Previously, investigators have reported that venous malformations can cause seizures, progressive neurologic deficits, and headaches (Rigamonti D., Spetzler R.F., Medina M. et al., 1990). Recent studies, however, suggest that they are clinically silent (Garner T.B., 1991). The hemorrhage and seizures ascribed to venous malformations most likely represent coexisting cavernous malformations.

DIAGNOSTIC IMAGING

During the past decade, MRI and magnetic resonance angiography have been the most important technical advances in neuroimaging. In comparison with CT, MRI provides better resolution and multiplanar images. Additionally, MRI can show the presence of clinically silent hemorrhages, can chronicle multiple hemorrhages, and can document the status of the regional parenchyma in terms of mass effect and edema formation. MRI serves as an excellent tool for planning an operative approach or calculating dosimetry for stereotactic radiosurgery. Imaging in three planes demonstrates the relationship of the nidus to deep brain structures. After stereotactic radiosurgery, MRI demonstrates thrombosis within an AVM and serves as a screening test for AVM obliteration.

Arteriovenous Malformations. Cerebral angiography is the gold standard for AVM evaluation. A complete study includes both internal carotid arteries and both vertebral arteries with sequential evaluation of all phases including arterial, capillary, and venous. The sequence gives a clear understanding of the vascular dynamics of the AVM such as rate of blood flow, differential in regional flow (arterial steal or venous hypertension), and patterns of collateral flow. Additional information includes nidus size and configuration as well as the position, number, and size of the feeding arteries and draining veins. Angiographic images demonstrate pedicular and intranidal aneurysms that may be the actual source of hemorrhage.

Cavernous Malformations. MRI is the best imaging study to evaluate cavernous malformations because they are angiographically occult. Cavernous malformations have a characteristic morphologic appearance on MRI: a central focus of mixed signal intensity representing hemorrhages of various ages. The nidus is surrounded by a hypointense rim of hemosiderin from chronic microhemorrhages **(Fig. 5**.). Enhancement with intravenous contrast is usually faint. In the case of a rare dural-based cavernous malformation, the MRI signal characteristics resemble meningiomas. The cavernous malformation is isointense on T weighted imaging and enhances strongly and homogeneously with gadolinium (Lewis **A.I.etal.,** 1994).

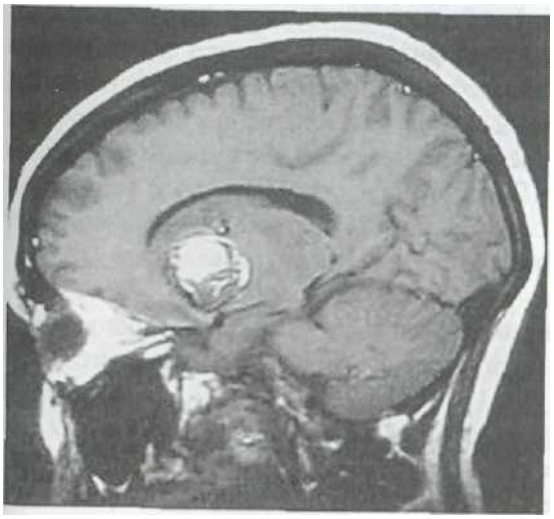


Fig. 5. MRI of a caudatothalamic cavernous malformation. Note the variegated appearance of the core caused by multiple hemorrhages of different ages and the hypointense rim caused by deposition of hemosiderin.

Venous Malformations and Capillary Telangiectasias. Venous malformations are best seen with angiography in the venous phase. They have the characteristic appearance of a caput medusae. On contrast CT and MRI scans, venous malformations appear as a linear enhancement, frequently located near an ependymal surface. Capillary telangiectasias are not visualized by cerebral angiography. Although the lesions have not been confirmed pathologically, we have identified ten patients in whom MRI scans showed a I-cm area of hypenntensity (usually in the pons), which is visualized only with gadolinium enhancement **(Fig. 6**). We suspect that this lesion represents a capillary telangiectasia.

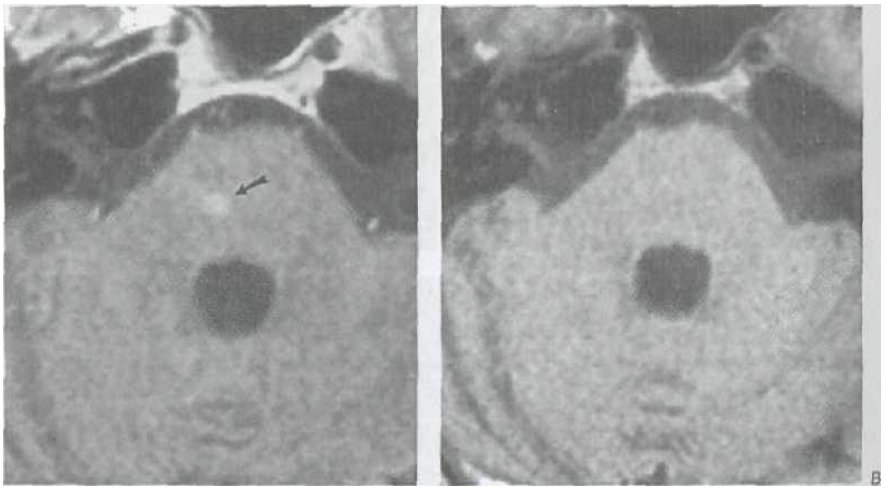


Fig. 6. T_2-weighted MRI scans of a pontine capillary telangiectasia with (A) and without (B) gadolinium.

TREATMENT

The therapeutic options for AVMs include embolization, microsurgery, and stereotactic radiosurgery. The treat ment options for cavernous malformations include microsurgery. Stereotactic radiosurgery is associated with a high rate of radiation injury and its efficacy has not been demonstrated for cavernous malformations. Venous malformations and capillary telangiectasias are benign lesions that do not require therapeutic intervention. Microsurgery and stereotactic radiosurgery are complementary treatment modalities that are used to treat patients with different AVM characteristics. The risk of surgically removing or radiating intracranial vascular malformations must be balanced against the lifetime risk of hemorrhage. Microsurgery is the most definitive treatment to eliminate the risk of hemorrhage. Stereotactic radiosurgery has a 2-year average latency period to obliteration and an 80% rate of cure (Fig. 41) (Lunsford L.D. et al.[5]1991). The yearly risk of hemorrhage remains unchanged until the vascular malformation is completely eliminated.

Arteriovenous Malformations. Embolization is primarily an adjunct to microsurgery or stereotactic radiosurgery to reduce size of the arterial shunt (Fig. 42). In a small percentage of cases, intranidal embolization of small AVMs with acrylic glue may lead to cure. In most cases, embolization of the arterial supply is performed in stages to allow the surrounding brain to adjust to the changes in circulation after the malformation is removed. Recanalization or formation of collateral arterial supply is likely after embolization, so AVMs are removed within 1 week of the embolization. When embolization is considered in the treatment of AVMs, an important question to answer is whether the benefit of embolization in combination with microsurgery sufficiently reduce operative morbidity compared with microsurgery alone, and whether embolization and Stereotactic radiosurgery improve the cure rate compared with Stereotactic radiosurgery alone. The answers are unknown.

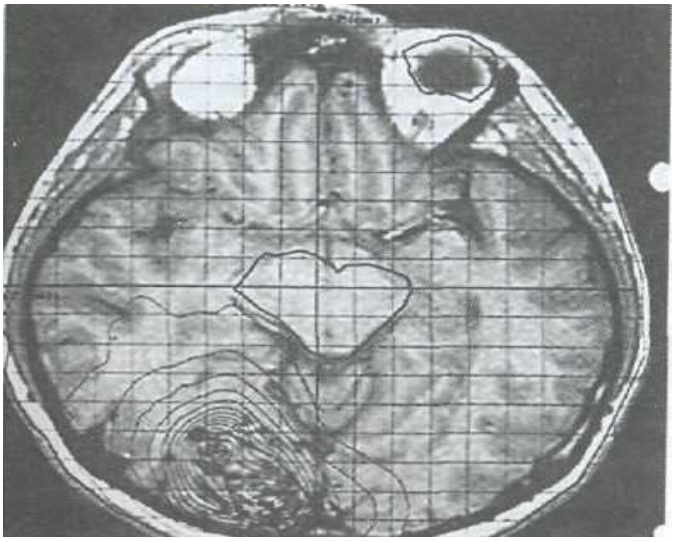


Fig. 7. Stereotactic radiation may be used to obliterate small, unruptured arteriovenous malformations (AVMs) in eloquent brain. $T_r$weighted MRI scan shows isodose radiation curves for treatment of an AVM.

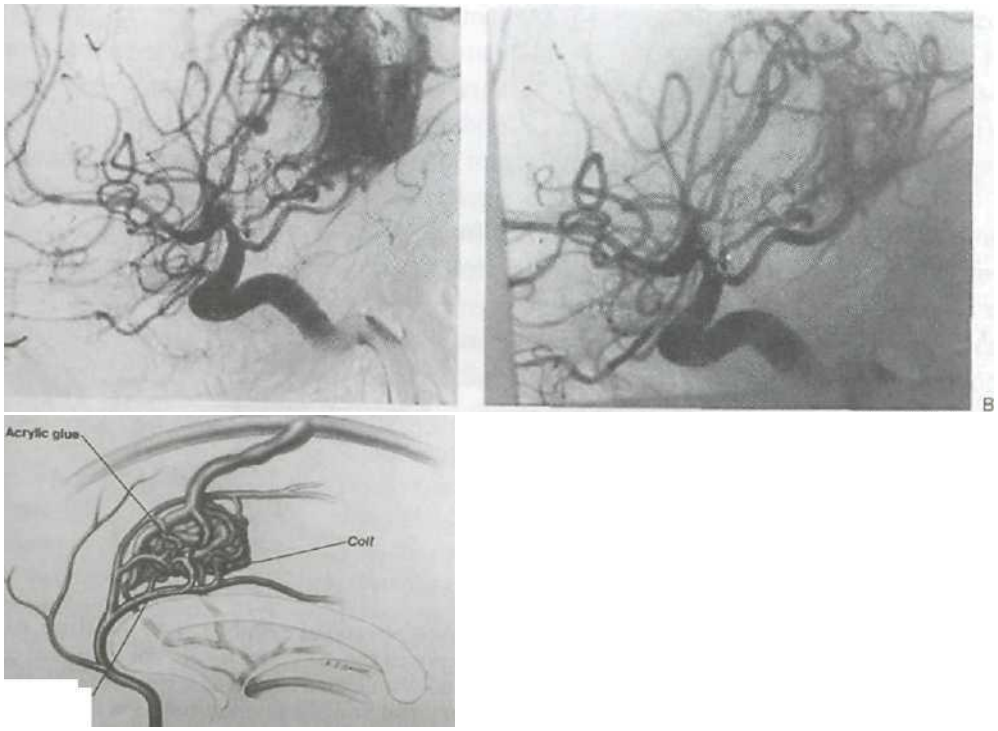


Fig. 8. An arteriovenous malformation is shown preembolization (A) and postembolization (B). (C) Delivery of the embolic materials (e.g., platinum coils, pellets, silk threads, acrylic glue, polyvinyl alcohol sponge) via the microcatheter to the interstitices of the malformation.

In general, the risk of neurologic deficit from microsurgery increases with increasing AV JVI in eloquent brain tissue, and deep venous drainage (Hamilton M .G., Spetzler ients 20 to 40 years of age who have a small AVM located in noneloquent superficial venous drainage, microsurgery is the best option. Patients with ible epilepsy and severe headaches are also candidates for microsurgery, nore effective than stereotactic radiosurgery for eliminating seizures and pas D.G., Sundt T.M., Ragoowani A.T., Stevens L., 1993). Stereotactic served for patients who have small (less than 3 cm), unruptured AVMs in sue with deep venous drainage (Steiner L., Linquist C, Adler J.R., 1992). AVMs are best treated with staged embolization and microsurgery. As the is 3 cm, the cure rate with Stereotactic radiosurgery drops significantly, and xerosis to the surrounding brain rises. Stereotactic radiosurgery may be used ith microsurgery when there is a small postoperative AVM remnant located The operative approach is based on the shortest distance with a perpendicular not traverse functional brain tissue. Patients are positioned with the head above the craniotomy as the highest point.

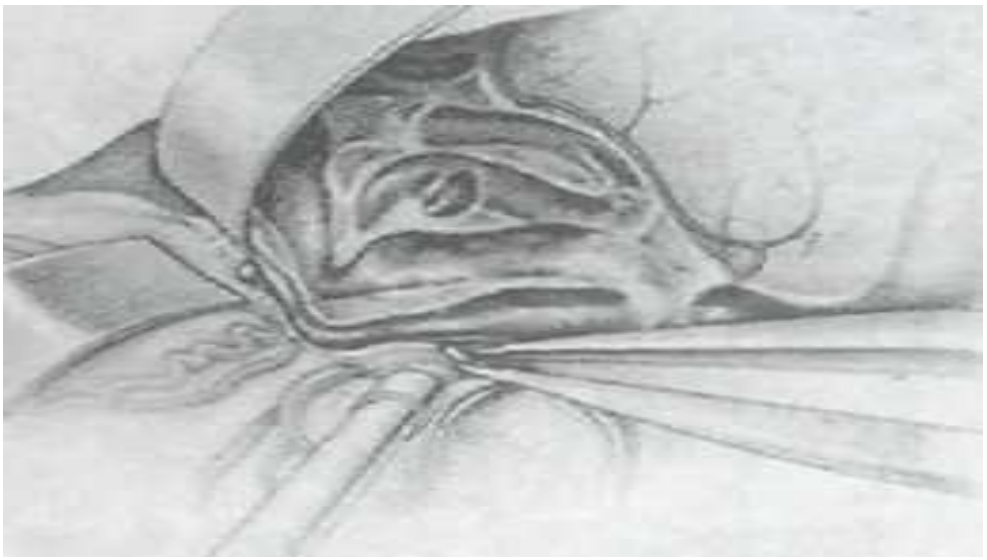


Fig. 9. Circumferential dissection is performed with a cleavage plane developed between the surrounding brain.

The craniotomy must be larger than the AVM to obtain a circumferential dissection. A large exposure also provides easier orientation to the feeding arteries, draining veins, and cortical landmarks. Mannitol, hyperventilation, cerebrospinal fluid drainage, and cerebral protection with barbiturates are used to obtain brain relaxation. Great care must be exercised when opening the dura to avoid tearing adherent draining veins. The arterialized draining veins are usually superficial and lead back to the feeding arteries and the nidus. As an AVM maintains pial planes, a circumferential plane of dissection is developed around it using a bipolar electrocautery and a microscope. Feeding arteries are coagulated and cut as close to the nidus as possible to avoid occluding normal vessels that supply adjacent brain tissue. The hematoma from the hemorrhage often provides a useful plane of dissection and additional room to maneuver, especially in the removal of deep-seated AVMs (Lawton M.T., Hamilton M.G.,

Spetzler R.F., 1995; Tew J.M., Lewis A.I., Reichcrt K.W., 1995). During the dissection, retractors are placed on the AVM to avoid damage to the surrounding brain. Draining veins should be preserved until the arterial supply has been eliminated. Some minor draining veins may be sacrificed to mobilize the AVM prior to removal of the arterial supply. The major draining vein is preserved until all feeding arteries are eliminated. A temporary clip is used to determine if a vein can be sacrificed safely. Induced hypotension with nitroprusside (mean arterial pressure 50 to 60 mm Hg) is used to control bleeding during removal of the AVM. In general, AVMs should be removed in one stage to avoid postoperative hemorrhage. Intraoperative angiography is used to confirm complete obliteration.

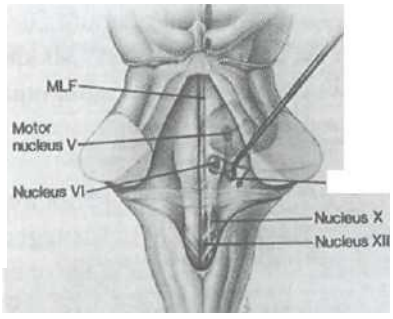
Cavernous Malformations. Asymptomatic cavernous malformations are not treated because the risk of hemorrhage is approximately 1 % or less per year. **Microsurgery** is indicated for supratentorial cavernous malformations that present with hemorrhage or cause medically intractable epilepsy or severe headaches (Maraire J.N., Awad LA., 1995). Brainstem cavernous malformations are removed when there is repeat hemorrhage, superficial location, and progressive neurologic deficit (Lewis A.I. et al., 1995).

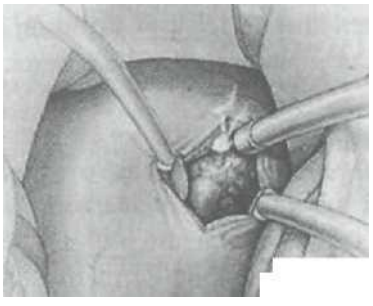
Stereotactic-guided approaches are useful for deep-seated supratentorial cavernous malformations to minimize disruption of cortex. Intraoperative ultrasound is also useful for localization and to confirm complete removal. When cavernous malformations are located in the brainstem, electrophysiological mapping of the fourth ventricle floor avoids permanent injury to the cranial nerve nuclei. Surgical resection of a cavernous malformation is similar to removing a metastatic tumor. A circumferential dissection is performed using bipolar electrocautery or laser between the cavernous malformation and the gliotic plane (Fig. 44). Small feeding arterioles are coagualated and cut as they enter the malformation. Microretractors are used to minimize manipulation of the surrounding brain.

Stereotactic radiosurgery remains an experimental treatment option for cavernous malformations. It is a valid option for cavernous malformations in the hypo-thalamic capsular region, tectum, and deep brain stem, particularly in patients without a hematoma or neurologic deficit. One drawback to radiating cavernous malformations is that no current imaging modality confirms obliteration. A recent study of Kondziolka et al., 1995) however, showed that after Stereotactic radiation of deep-seated cavernous malformations, the yearly hemorrhage rate was reduced compared with the hemorrhage rate before radiosurgery.

POSTOPERATIVE MANAGEMENT

After surgical removal of the AVM, systemic hypotension is maintained for 48 to 72 hours after surgery to avoid postoperative hemorrhage and brain swelling. A postoperative angiogram is performed to confirm AVM obliteration. Postoperative hemorrhage from incomplete hemostasis, uncontrolled blood pressure, or residual malformation are the most devastating consequences of AVM surgery. A postoperative hemorrhage in the resection bed requires urgent return to the operating room to evacuate the hematoma. Reperfusion phenomena from altered hemodynamics or excessive retraction may cause ischemia and swelling of the surrounding brain. Anticonvulsant levels are monitored daily until high therapeutic levels at steady state are reached. More than one anticonvulsant may be required in the perioperative period to control seizures. Obstructive hydrocephalus from intraventricular blood or brain swelling usually resolves after a ventriculostomy. Parenchymal infarction is usually caused by unplanned occlusion of normal arteries or veins. Vasospasm is a rare cause of cerebral ischemia after surgical resection of AVMs.





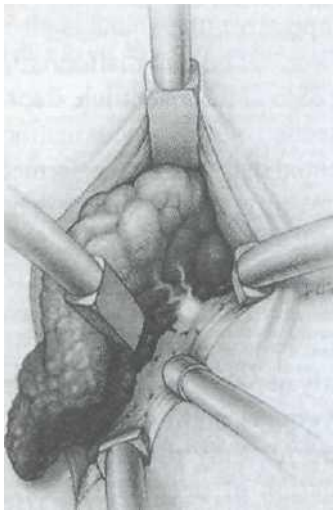


Fig. 10. Removal of a cavernous malformation: (A) mapping of the floor of the fourth ventricle is performed with monopolar stimulation of cranial nerve nuclei before removal of the cavernous malformation. (B) A zone of safe entry is defined after mapping the cranial nerve nuclei. Laser and micro-retractors reduce manipulation and injury to the brain stem. (C) A plane is developed between the cavernous malformation and the surrounding brain.

ARTERIOVENOUS MALFORMATIONS. With the caveat that combining a wide range of heterogeneous series into a single group is not representative of any series, a recent review of several reports on the embolization of AVMs showed a cure rate of 5% in 708 patients embolized since 1990. Temporary morbidity was 10%, permanent morbidity 8%, and mortality rate 1%. There was no long-term morbidity from the use of neurotoxic embolization materials (Frizzel R.T., Fisher W.S., 1995). Because of variability in endo vascular techniques, experience of the operators, different AVM types, and different embolic materials, these percentages provide only a general estimate of outcome after embolization. For giant inoperable AVMs, embolization may palliate the symptoms of arterial steal or venous hypertension. Embolization may also be used to occlude pedicular aneurysms that are the source of hemorrhage. AVMs in the brainstem with perforator supply from the basilar artery cannot be resected completely.

Among a wide range of radiosurgical series, the rate of AVM obliteration ranges from 60 to 90%. The smaller the AVM, the higher the rate of obliteration. In general, the expected obliteration rate for an AVM less than 3 cm in size is 80% after 2 years (Lunsford L.D. et al., 1991; Steiner L., Linquist C, Adler J.R., 1992). The rate of permanent radiation injury to the surrounding brain ranges from 3 to 9%. The latency interval to obliteration ranges from 1 to 3 years (mean 2 years). There is no significant change in the hemorrhage rate of AVMs during the latency interval compared with the expected hemorrhage rate based on the natural history. Also, there was no protective benefit from radiosurgery in patients who have had partial obliteration of the AVM nidus. Finally, pedicular aneurysms should be obliterated prior to radiosurgery to avoid hemorrhage during the interval to obliteration.

Cavernous Malformations. By Adam I. Lewis, Sumeer Sathi, and John M. Tew, Jr (1999), less than half the patients with deep-seated cavernous malformations met the criteria for surgical removal. Among 33 operated patients with cavernous malformations of the basal ganglia, thalamus, and brainstem, 65% suffered a transient neurologic deficit, 20% suffered permanent neurologic deficits, and 6% were dead at 1 year follow-up. Three (9%) of 33 patients required reoperation for residual brainstem cavernous malformations. Overall, 90% of the operated patients made a good or excellent recovery from preoperative deficits. The benefits of stereotactic radiosurgery for cavernous malformations are unknown. In the past, radiosurgical series have shown a higher rate of radiation injury and neurologic deficits in patients with cavernous malformations compared with AVMs. A recent study of 47 patients with cavernous malformations in critical brain regions showed an 8.8% hemorrhage rate in the first year. Although that rate dropped to 1.1% per year in the subsequent 5 years, the morbidity remained high. Ten (21 %) patients had neurologic worsening and two (4%) died after radiosurgery (KondziolkaD. et al., 1995). Despite a lack of imaging studies to document obliteration, radiosurgery may be a valid treatment option for deep-seated cavernous malformations not

adjacent to a pial or ependymal surface. Microsurgery is reserved for symptomatic cavernous malformations near an ependymal surface, and observation is warranted for asymptomatic or minimally SYMPTOMATIC LESOINS IN DEEP LOCATIONS.