Microsurgical resection of infratentorial arteriovenous malformations

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Successful microsurgical resection of an infratentorial arteriovenous malformation (AVM) requires both surgical skill and intraoperative judgment. Extensive practical experience in treating these complex lesions, which is acquired over many years, is of substantial value during each new operation. The authors present the surgical approaches and techniques used for the treatment of posterior fossa AVMs based largely on the strategies acquired and developed by the senior author (H.H.B.). Emphasis is placed on conceptual principles of AVM excision, as well as principles incorporated for the treatment of each specific type of infratentorial malformation.

KEY WORDS • arteriovenous malformation • cerebellum • posterior fossa • vertebrobasilar region

OVERVIEW

Compact masses of entangled arteries and veins situated within viable neural parenchyma, AVMs of the brain are among the most challenging neurovascular lesions from a pathophysiological as well as a therapeutic standpoint. Infratentorial AVMs, which receive their principal arterial inflow through the vertebrobasilar system, are much less common than their supratentorial counterparts, constituting approximately 10% of all AVMs in modern clinical series. In fact, these malformations were only recognized as a distinct clinical entity in 1906, and it took until 1932 before a report of successful resection by Olivecrona and Rives appeared. Almost despite their relative infrequency, infratentorial AVMs have received a great deal of interest from the neurosurgical community. Significant efforts have been made to understand better both the anatomical and hemodynamic complexities of these lesions, largely in an effort to facilitate more effective treatment strategies.

Despite the advent and subsequent refinements of microsurgical techniques in the latter half of the twentieth century, excision of infratentorial AVMs is a formidable task. In fact, treatment today is often rendered as a combination of the three principal therapeutic modalities: microsurgery, endovascular embolization, and radiosurgery. The difficulty these lesions pose from an operative standpoint relates to the necessity for meticulous preservation of the innumerable critical neurovascular structures housed within and around the brainstem and cerebellum, the cranial nerves, and the deep nuclei.

Beyond just the neuroanatomical complexity entrenched within the posterior fossa, an additional level of technical challenge is conferred because of the inevitably narrow cranial base corridors through which the neurosurgeon must frequently maneuver during resection, particularly in treating lesions in and around the brainstem. Consistently optimal outcomes in infratentorial AVM surgery are only achieved when the neurosurgeon is able effectively to couple skillful surgical technique with sound intraoperative judgment, the latter of which is gradually acquired through extensive practical experience, preferably under the guidance of an accomplished cerebrovascular surgeon during a several-year apprenticeship.

In this paper we review the microsurgical treatment of infratentorial AVMs, primarily based on the extensive neurovascular experience of the senior author (H.H.B.). Preoperative considerations, such as clinical presentation and diagnostic evaluation, are initially discussed because they are crucial in determining not only an appropriate surgical candidate, but also the preferred approach should surgery be the chosen intervention. The nuances of the procedures used to excise these lesions, which have been generated from repeated refinements of operative technique by the senior author over numerous years in neurovascular prac-
Preoperative Considerations

Infratentorial AVMs, like all other AVMs, classically present with some combination of intracranial hemorrhage and neurological deficit, either fixed or progressive. Other less specific symptoms such as headache are often present as well. Neuroimaging evaluation should begin with a noncontrast CT scan to determine whether the AVM has ruptured and, if so, the location of the hemorrhage. The distribution of intracranial blood can be critical in the therapeutic decision-making process, particularly in cases in which the hemorrhage is predominantly subarachnoid and is therefore likely due to the rupture of an associated cerebral aneurysm. Use of MR imaging, and in particular T2-weighted imaging, will reveal regions of hypointense flow voids, representing dilated and tortuous arterialized veins as well as enlarged feeding arteries. All patients must undergo formal six-vessel catheter angiography for accurate characterization of the anatomy and hemodynamics of the AVM. In particular, all feeding arteries and draining veins must be diligently identified preoperatively in preparation for a complete resection. High-resolution magnification studies are required of both VAs, both internal carotid arteries, and both external carotid arteries, because approximately 10% of infratentorial AVMs are fed by one or both external carotid arteries.

Because flow-related aneurysms are not infrequently the origin of hemorrhages in the case of posterior fossa AVMs, angiographic identification of all such lesions is a paramount consideration. Of additional importance to note on angiographic studies is the presence of en passage feeding vessels of the AVM, namely vessels that do not directly communicate with veins through the nidus, but rather travel through the AVM, often yielding branch vessels along the way. The presence of an en passage artery that subsequently suberves eloquent cortex after passing through the AVM immediately renders the AVM "eloquent," even if the lesion does not reside in eloquent tissue per se. Superselective angiography is often required to define this complex vascular anatomy accurately.

Because most posterior fossa AVMs present with intraparenchymal hemorrhage, the initial surgical focus is directed at hematoma removal and treatment of any acute mass effect or hydrocephalus if it is present (Figs. 1–3). When feasible, however, definitive resection of the AVM should be deferred for 4 to 6 weeks. Delayed extirpation in this manner often ensures sufficient time for brain swelling to diminish and the hematoma to liquefy, features which greatly facilitate ultimate AVM excision. Late treatment is also of benefit in that associated cerebral aneurysms, which might initially be obscured by intraparenchymal hemorrhage on the first angiogram, could later be well visualized after a brief period of quiescence. On rare occasions, a patient who needs early hematoma evacuation may require complete AVM resection during the same operation because of intraoperative hemorrhage. In such a situation, every effort is made to preserve all viable cerebellar tissue.
as well as alleviate any undue mass effect on the brainstem. When forced to remove a life-threatening hematoma in the acute phase, however, the goal of surgery is to remove enough of it to decompress the posterior fossa and ventricular system, not to resect the AVM. In the absence of an associated aneurysm that is the source of hemorrhage, the incidence of early rebleeding from a ruptured posterior fossa AVM with intact venous drainage is very low. Clearly, outcomes from resection of the more complex infratentorial AVMs are optimized by allowing several weeks of recovery after the hemorrhage.

General Principles of AVM Resection

The basic tenets of AVM resection are the same for lesions both above and below the tentorium. One must begin with generous bone resection and dural opening to allow for full visualization of the entire cortical extent of the malformation and more. Wide exposure facilitates unobstructed inspection of both the afferent and efferent vasculature, which may be at a distance from the nidus of the AVM. A variety of cranial base techniques, which are beyond the scope of this discussion, may be used to create generous exposure with minimal brain retraction. With regard to resection specifically, we separate the surgical effort into five principal stages of dissection: subarachnoid, pial, parenchymal, periventricular, and extirpation.

Stages of Dissection

Subarachnoid. Superficial feeding arteries are identified, dissected, and followed to the point of arborization as they enter the nidus. They are then coagulated or ligated with clips and divided. Particular attention is paid to en passage vessels to avoid ischemic insult to normal brain parenchyma. While sacrificing the superficial feeding vessels, the cortical margins of the AVM should be identified and dissection initiated deep to the cortically located draining veins in a circumferential fashion. In doing so, meticulous preservation of the venous channels of drainage must be the operative priority; inadvertent sacrifice of even a small venous pedicle draining the AVM can result in distention of the malformation accompanied by easily provoked and difficult to manage bleeding. In contrast, maintenance of the integrity of the draining veins during ligation of arterial feeding vessels results in gradual involution of the AVM, permitting eventual resection. If at any point during the dissection a draining vein must be sacrificed, we advocate placement of a temporary clip across the vessel, with subsequent observation of the malformation for several minutes. If the AVM appears to be under tension during trial occlusion, the clip should be removed and an alternative dissection pattern sought out and initiated. In concert with the obliteration of arterial input to the AVM, associated cerebral aneurysms must be successfully treated during the subarachnoid stage of the operation. Clearly, to avoid aneurysm rupture and potentially severe subarachnoid hemorrhage, each aneurysm must be clipped before the parent artery is sacrificed just above its entry into the AVM nidus.

Pial. After ligation of all superficial feeding vessels, the peripheral margin of the AVM should be dissected along a pial plane, freeing the lesion at its interface with surrounding brain. It is not unusual for the parenchyma around the circumference of the malformation to appear atrophic and gliotic, pathologically representing changes from chronic ischemia.
Parenchymal. After circumferential demarcation of the AVM, dissection proceeds in a spiral fashion from its superficial cortical limit, then into the white matter, and finally to its deepest periependymal extent, maintaining a relatively equal depth of exposure around the periphery throughout the dissection. Minor bleeding, which can be minimized by developing the dissection along the gliotic plane, is usually easily controlled with a combination of bipolar electrocautery and gentle tamponade with cottonoid. Another simple measure used to control hemorrhage consists of moving the dissection 180° from the bleeding site to retract the AVM itself into the hemorrhagic focus. One may also put the retractor directly on the bleeding vessels and tamponade the hemorrhage.

As the deep white matter is traversed, highly friable and hemorrhage-prone vessels of the periependymal region are encountered. Retraction of these vessels into the adjacent white matter can significantly complicate sustaining hemostasis at this stage of the operation. We have found that bleeding from the periependymal vessels is best treated by the application of multiple small microaneurysm clips, even if they are only temporarily applied. Decreasing flow velocity in these fragile vessels permits the bipolar cautery to coagulate the intraluminal blood as well as the vessel wall. It is often desirable to remove the clips after this occurs. This intervention is not only sufficient to abate the bleeding, but also prevents small vessel retraction into the white matter.

Periventricular. After sufficient devascularization of arterial input into the AVM, the dissection can be selectively pushed into the adjacent ventricle to ensure adequate resection of the malformation at its ventricular extent. Delicate vessels of the ependyma and periependyma are meticulously coagulated with bipolar electrocautery.

Exirpation. Once the entire AVM has been dissected free, from its most superficial location at the cortex to its deepest periependymal and ventricular extent, the final venous pedicles that drain the nidus are sequentially removed in a stepwise fashion, ensuring that the malformation is not placed under any undue intravascular hemodynamic tension. Frequently, residual arterial input is still present, and the vessel is carefully ligated with clips in a methodical manner. The AVM is then removed and its resection bed is carefully inspected to ensure that no retained malformation is present and complete hemostasis has been achieved. Mild hypertension can then be pharmacologically induced to ensure the absence of any further bleeding. Attention is then turned toward closure, which is performed in the usual fashion, beginning with a rigorously tight dural closure preceded by copious lavage of both the subarachnoid space and ventricular system.

Postoperative Care

After surgical removal is complete, strict adherence to normotension in the first 24 to 48 hours is critical to protect the fragile, often engorged vascular bed surrounding the resection cavity. On the 1st postoperative day, it has become our practice to obtain a cerebral angiogram for radiographic verification of complete AVM resection. It is important for the neurosurgeon to follow the angiographic injections, particularly in the vertebrobasilar circulation, all the way through the venous phase to ensure the absence of residual malformation or early drainage. In cases in which a complex extirpation was performed, we occasionally obtain an intraoperative angiogram after resection and just before the termination of general anesthesia. If any residual AVM is detected, surgical exploration is of course manda-
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tory because venous drainage has been compromised, setting the stage for early postoperative hemorrhage.

Surgical Technique for Cerebellar Vermian AVMs

The AVMs situated in the cerebellar vermis are some of the most common malformations located in the infratentorial compartment. They frequently receive bilateral arterial supply from both the SCAs and PICAs. The relative contribution of each of these arterial inputs is dependent on the AVM’s superior–inferior relationship with respect to the horizontal cerebellar fissure. In rare circumstances involving very large vermian AVMs, distal branches from the AICA may supply lateral and deep arterial feeding vessels. Venous drainage is usually carried superiorly through pre-central cerebellar veins into the galenic system or, less commonly, through superior vermian veins into the petrosal or straight sinuses. Although vermian AVMs usually contain some degree of ventricular ependymal extension, they very rarely if ever reach the level of the cerebellar peduncles or brainstem. In rare situations, a deep midline AVM can extend both above and below the tentorium (Fig. 4).

Use of preoperative endovascular embolization can be a helpful adjunct in the surgical treatment of vermian AVMs. Endovascular intervention should be targeted at obliterating deep arterial feeding vessels of the malformation, such as distal feeding branches originating from the AICA should they exist, which are not easily accessible at surgery. Also of great assistance is preoperative embolization of SCA feeding vessels distal to the dorsal aspect of the brainstem for AVMs located superiorly to the horizontal fissure. The use of endovascular methods to eliminate this arterial in-

Fig. 4. Axial (A) and sagittal (B) MR images revealing a deep midline AVM involving both the supratentorial and infratentorial compartments, for which radiosurgical management failed. An angiogram (C) demonstrates that the AVM is predominantly supplied by dilated posterior cerebral arteries. A postoperative angiogram (D) was obtained after microsurgical treatment.
flow is particularly valuable because the superiorly directed venous drainage often obscures visualization of the majority of feeding vessels that arise from the SCA. Moreover, aggressive embolization of the SCA in cases of superiorly located vermian AVMs significantly facilitates the dissection effort on the superior aspect of the vermis, which is the site of fragile arterialized venous drainage.

With regard to operative exposure, we approach vermian AVMs through a midline suboccipital craniectomy with the patient in the prone–concorde position. The operating table is placed in an extreme reverse Trendelenburg position. The patient is positioned prone with the knees and head flexed so that the nuchal region is parallel to the floor. The table and patient are oriented according to the handedness of the surgeon. A large midline incision extending from the occipital protuberance to the level of C-2 is fashioned and then a large bone flap is elevated to expose the entire superior extent of the lesion. Wide bone exposure often necessitates drilling of the bone overlying the transverse sinus and torcular herophili to facilitate retraction of the dural edges superiorly. Inferior exposure of the cisterna magna may be beneficial if minor hydrocephalus is present and no external ventricular drainage catheter is used. A wide stellate durotomy is fashioned as a further aid in exposure of the AVM. The initial dissection effort involves subarachnoid dissection and subsequent clip ligation of the PICA feeding vessels just proximal to their entry into the malformation. The operative focus is then turned to the superior aspect of the malformation, where a circumferential cortical margin is fashioned at the cerebellar–AVM interface. Distal interhemispheric AICA feeding vessels, should they exist, are then sacrificed along the lateral margins of the AVM. As dissection is carried deeply in a circumferential manner, feeding vessels arising from the SCA are sacrificed along with deep AICA feeding vessels, if present. Finally, after all arterial input to the nidus is obliterated, the venous drainage is rather easily eliminated in a meticulous stepwise fashion.

**Types of AVMs**

**Cerebellar Hemispheric AVMs.** Because of the large space occupied by the cerebellar hemispheres, AVMs affecting this region of the posterior fossa are comparatively common (Fig. 5). These lesions can vary in size enormously, ranging from small lesions with trivial arterial feeding vessels to profoundly large malformations that are holohemispheric and absorb massive amounts of cerebral blood flow, generating a significant steal phenomenon. Feeding vessels are almost always unilateral and, depending on location, have some combination of input from all three cerebellar arterial trunks. Superiorly located lesions possess a prominent SCA supply, lateral lesions with CPA extension are copiously irrigated through the AICA, whereas the PICA principally supplies inferior lesions. Visualization of AICA involvement on preoperative angiographic studies is important because it signifies almost certain extension into the lateral aspect of the fourth ventricle roof and the adjacent middle cerebellar peduncle. Particularly in cases of large lesions with significant arterial input, flow-related aneurysms are not uncommon. As with vermian AVMs, hemispheric malformations send venous drainage either superiorly into the galenic system through the vermian venous plexus or laterally into the petrosal sinus.

In similar fashion to vermian lesions, preoperative embolization can be a useful adjunct in the treatment of hemispheric AVMs. Aggressive endovascular obliteration of deep AICA and large SCA feeding vessels is a great help for reasons elucidated previously. If the AVM is very large, embolization of flow through the PICA can significantly reduce the global inflow to the lesion, thereby facilitating manual mobilization of the malformation during dissection.

We tend to favor positioning patients in the lateral park-bench configuration during resection. A wide bone exposure is critical, especially in the resection of sizable malformations. Although many different skin incisions have been described, we prefer a paramedian inverted U-shaped incision with inferior reflection of the occipital musculature. This technique allows for adequate surgical exposure from the foramen magnum up through the CPA by reducing the potential interference from the retracted skin, superficial soft tissue, and musculature. The lateral bone exposure runs as far as the cranium overlying the sigmoid sinus and the foramen magnum is then opened inferiorly. Both the PICA and AICA are dissected in the subarachnoid space and followed to their entry point into the malformation, at which point they are clipped and divided. As with vermian AVMs, the SCA feeding vessels to hemispheric malformations are often obscured early in the treatment by the diluted venous drainage running over the superior aspect of the cerebellum into the galenic system. Late bleeding during the resection is invariably due to deep AICA feeding vessels at the periependymal level that are rather easily controlled using bipolar electrocautery with entry into the fourth ventricle. When all arterial inflow to the malformation is eliminated, the AVM can be delivered from the resection bed, tethered only by a final venous outflow pedicle, which can then be transected.

**Cerebellar Tonsillar AVMs.** Primarily because of the limited anatomical confines of the cerebellar tonsils, AVMs restricted to this location are quite rare. When they do occur, however, they are usually fed by a single large, dilated PICA ipsilateral to the involved tonsil. Secondary arterial feeding vessels may arise from distal AICA branches. Venous drainage can be carried laterally into the sigmoid sinus, but more commonly it is transported by superior vermian veins into the galenic system or through the petrosal sinus via a dilated cortical vein that courses laterally into the CPA cistern.

Tonsillar AVMs are rather easily resected by simply performing a limited resection of the affected cerebellar tonsil. It is our preference to position the patient laterally and to fashion the same incision as that used with hemispheric lesions. A wide bone exposure is accomplished by generous resection of the inferior aspect of the foramen magnum and even the posterior arch of C-1. The initial operative effort is directed at subarachnoid dissection of the PICA followed by clip ligation of this artery immediately proximal to its entry into the malformation. Dissection in the cisterna magna, which helps to define one tonsil from the other, carries with it the risk of inadvertent injury to the contralateral PICA at its inferomedial aspect. This may require minimal opening of the inferior vermis in the midline for ventricular access. Once this is accomplished, the tonsil is resected superior to the peripheral margin of the AVM, saving stepwise venous drainage transection until the final stage. Because of the ease with which surgical access to the PICA...
feeding vessels is obtained, preoperative embolization is rarely necessary.

**Brainstem AVMs.** Malformations of the brainstem, which are distinctly different lesions from the cerebellar AVMs described earlier, can be broken down into two broad varieties based on location and operability, that is, pial lesions and parenchymal lesions. The AVMs restricted to the pial surface typically receive arterial input from dilated branches of the SCA or AICA, drain into the prepontine or petrosal sinuses, and rarely if ever have ventricular extension. These lesions can occasionally be resected completely.

In contrast, malformations situated in the parenchyma of the brainstem receive arterial inflow from critical en passage brainstem perforating vessels that arise at right angles to the longitudinal extent of the vertebrobasilar parent vessel from which they originate. This feature alone, in addition to their deep insinuation within vital neural tissue, precludes safe microsurgical extirpation in the overwhelming majority of cases (Fig. 6). Venous drainage of these deeply housed parenchymal AVMs is typically transmitted through normal venous channels of the brainstem substance, which then empty into the petrosal sinus, galenic system, or both. Ventricular extension is common and, for the reasons outlined, it has been our practice to treat these daunting brainstem lesions with either expectant observation or stereotactic radiosurgery.

Because of the critical vessels that originate in the vertebrobasilar system and ultimately perfuse both the brainstem and the AVM, we do not routinely use preoperative endovascular embolization. It has been our experience that

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**Fig. 5.** Axial CT angiogram (A) and MR image (B) revealing a lateral posterior fossa AVM with a large partially thrombosed venous varix discovered in a 6-year-old child. Anteroposterior (C) and oblique (D) projections obtained after left VA injection further define this complex lesion.
with meticulous dissection techniques, arterial feeding vessels are better accessed surgically as they enter the peripheral margin of the nidus, thereby avoiding the potentially severe morbidity of inadvertent brainstem infarction related to endovascular procedures.

Microsurgical resection of pial-based brainstem AVMs begins, as it does with all other posterior fossa malformations, with a wide bone exposure. Most of these lesions are situated on the anterolateral surface of the pons and because of this, we prefer placing the patient in a true lateral position and performing a far-lateral craniotomy to engage the maximal extent of the lesion at the level of the CPA. Brainstem auditory evoked potentials are carefully monitored throughout the resection. Before AVM dissection, the cistern of the CPA should be generously opened, from the site at which the VA penetrates the intradural compartment all the way to the dorsal root entry zone of the trigeminal nerve. As a general rule, the predominant arterial input is derived from the AICA, although SCA feeding vessels are not uncommon. These vessels are ligated with clips at their entry point into the malformation in the usual manner. This must be performed with the utmost care because inadvertent sacrifice of a normal artery will result in irreversible and potentially catastrophic brainstem infarction. After occlusion of the arterial input to the AVM, it is dissected free from the posterior surface of the brainstem and venous pedicles are sequentially transected in the usual manner.

Postoperative Care

The hallmark of postoperative care following AVM resection is strict blood pressure control and the absolute prevention of hypertension for at least the first 3 days. This hemodynamic regimen is performed to prevent excessive perfusion of the chronically ischemic brain tissue surrounding the resection bed, for which normal autoregulatory mechanisms are absent. After this brief period of regimented normotension, parameters may be gradually liberalized as the clinical scenario permits. A formal cerebral angiogram is routinely obtained in the early morning of the day following resection. In patients who demonstrate residual AVMs, either endovascular obliteration of the remaining lesion or, more likely, a return to the operating room is clearly required.

Problems in the early postoperative period—hemorrhage, cerebral edema, hydrocephalus, and normal perfusion pressure breakthrough bleeding—are, in many cases, related to inadequate blood pressure control postoperatively. It is extremely important to recognize and treat each complication optimally even after an apparently complete resection. Clearly, the risk of postoperative hemorrhage is highest when there is significant fragility of vessels in the surrounding microcirculation. Perhaps more importantly, an increased risk of bleeding in the postoperative period occurs when the AVM and the volume of previously shunted blood is largest.

CONCLUSIONS

The resection of infratentorial AVMs, although it is always challenging, can be accomplished with excellent results. One must carefully evaluate all patients before performing a resection. In patients who have sustained a hemorrhage, the initial operative effort is directed at relieving mass effect or hydrocephalus if necessary, with definitive AVM resection being deferred for 4 to 6 weeks. Malformations of the cerebellum are easily categorized according to their anatomical location: vermian, hemispheric, and tonsillar. Lesions in each of these locations, all eminently resectable as described earlier, have their own unique array of arterial input and venous drainage, which varies to some extent based on the size of the lesion and the vertebrobasilar anatomy of the patient.

Brainstem AVMs, which are broadly classified based on operability and location as either pial or parenchymal, are distinctly different malformations from those of the cerebellum. We have achieved generally successful results with resection of pial lesions, whereas AVMs residing within the brainstem parenchyma are typically relegated to radiosurgical therapy. Finally, postoperative care is predicated on the strict maintenance of normotension in the few days imme-

Fig. 6. Axial MR image (A) and lateral VA angiogram (B) revealing a complex AVM involving the brainstem and right cerebellar peduncle.
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diately following the procedure; blood pressure parameters can then be gradually liberalized when the normal autoregulatory capacity of the cerebral circulation is physiologically restored.

Reference


Manuscript received June 27, 2005. Accepted in final form July 18, 2005. Address reprint requests to: H. Hunt Batjer, M.D., Department of Neurological Surgery, The Feinberg School of Medicine, Northwestern University, 676 North St. Clair Street, Suite 2210, Chicago, Illinois 60611. email: hbatjer@nmff.org.