Arteriovenous malformations of the posterior fossa

Clinical presentation, diagnostic evaluation, and surgical treatment

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Arteriovenous malformations (AVM's) supplied from the vertebrobasilar system and located beneath the tentorium have traditionally been considered relatively rare, representing only 5% to 7% of all AVM's described in the major clinical series reported from 1960 to 1980.3,8,14,19 The improved sophistication of neuroradiological techniques and the growing appreciation of the varied symptomatology referable to these lesions have increased the number of posterior fossa malformations diagnosed over the past several years. Several recent publications have dealt with the clinical and radiographic features of AVM's of the vertebrobasilar system, and have reported the preliminary results of various types of surgical therapy.3,4,7,8,10,11,21 To expand this still small clinical experience, we have reviewed the modes of clinical presentation, results of diagnostic evaluation, and the surgical considerations and outcomes in 30 such patients who were treated surgically during an 8-year time span at one institution.

Clinical Material and Methods

Between July 1, 1977, and July 1, 1985, 32 patients with angiographically documented intracranial intradural AVM's of the brain stem or cerebellum were evaluated at our institution. Thirty of these patients underwent surgical treatment and now have a follow-up period of 6 weeks to 7 years 9 months. All survivors were either examined or interviewed within a 6-month period of time (February 1 to July 1, 1985). Of the two nonsurgical patients, one has been lost to follow-up review and the other is currently being reevaluated for surgical therapy. In all patients, referral data including admission histories, physical examinations, hospital summaries, and radiographic studies have been correlated with our own diagnostic findings to provide information regarding the symptomatology and pertinent anatomical features of these AVM's. Long-term therapeutic results have been assessed by routine clinic visits and by annual follow-up telephone interviews with...
patients living at a great distance from our medical center. These 32 patients represent 14% of the total number of 171 AVM cases evaluated during this time period; the 30 patients undergoing surgery comprise 18% of our total surgical experience with AVM's.

Summary of Cases

Clinical Presentation

The signs and symptoms of sudden intracranial hemorrhage characterized the initial clinical presentation of 23 (72%) of our patients; these patients suffered documented subarachnoid, intraventricular, or intraparenchymal hemorrhages, most of which were either intraventricular or subarachnoid in location. Only eight patients presented with brain-stem or cerebellar hemorrhages identifiable on computerized tomography (CT) scanning. Recurrent hemorrhage appears to have occurred in a disproportionately large number of patients (11 of 23, or 34%), separated by intrahemorrhage intervals of 3 months to over 6 years. The diagnosis of recurrent bleeding was, of necessity, retrospective in three of these 11 patients, and was documented by lumbar puncture in five and CT in two. One patient had undergone a ventriculoperitoneal shunting procedure for hydrocephalus following her initial bleed 3 years before our evaluation.

Nine patients (28%) were initially investigated for symptoms of brain-stem and/or cerebellar dysfunction believed at first to be due to either a neoplasm or a demyelinating process; they were found instead to harbor AVM's of the posterior fossa. At the time of their first evaluation, none of these patients had experienced symptoms suggestive of definitive intracranial hemorrhage, although one patient did suffer a documented subarachnoid hemorrhage (SAH) in the interval between initial neurological evaluation and diagnosis. Patients with initial complaints referable to brain-stem or cerebellar deficits were generally slightly older than those presenting with intracranial hemorrhage (mean age 42 years vs 29 years).

Clinical Symptomatology

In an attempt to reconstruct the total clinical symptomatic picture prior to presentation in these patients, we questioned the patients and their families and also searched referral evaluations and hospital admission records to identify any symptoms predating the symptom that resulted in definitive diagnosis or that occurred between diagnosis and therapy. Somewhat surprisingly, we found a relatively pure symptom complex with only a small degree of commonality between the two major presenting complaints: intracranial hemorrhage, and progressive neurological deficits presumed due to an ischemic or "steal" phenomenon. Of the 23 patients presenting with intracranial hemorrhage, only three had significant prior complaints possibly related to brain-stem or cerebellar dysfunction, although seven did have a significant history of headache (Table 1). One patient had had a prior hemorrhage from an intracranial aneurysm. Similarly, only two of the nine patients with supposed vertebrobasilar ischemia related to AVM steal developed a clinically apparent intracranial bleed (one from a ruptured aneurysm and one from the AVM), although four patients did have a history of chronic headache. We are uncertain if these findings represent a real difference in natural history between posterior fossa malformations that bleed and those that produce ischemia, or whether they are simply manifestations of the small sample size and/or selected patient population. On further examination of these two subgroups of AVM as regards such factors as location, size, angiographic anatomy, and surgical outcome, we could not identify any other substantial differences. In both groups, 11 patients had complaints of a chronic refractory headache, one of whom was demonstrated to have active hydrocephalus following two prior intraventricular hemorrhages.

Radiographic Evaluation

CT Scanning. All patients in this series underwent at least two preoperative CT scans, both with and without contrast enhancement. Although several of the plain scans, especially in patients with small malformations located deep within the posterior fossa, were unremarkable, none of the contrast-enhanced scans could be interpreted as normal. Occasionally, these studies did not specifically identify the nidus of a malformation, but they were very sensitive to the invariable presence of associated abnormalities such as dilated draining veins, subarachnoid or intraventricular hemorrhage, or hematoma in the brain stem or cerebellum. Computerized tomography scans within the narrow confines of the posterior fossa may lack the specificity of studies above the tentorium; however, in this series CT performed both before and after administration of contrast material was an excellent technique to screen for the presence of an AVM.

Cerebral Angiography. Cerebral angiography is the critical radiographic technique in patients with a suspected intracranial AVM. Because many of these lesions are so complex and the extent and quality of arteriographic procedures so variable, all patients in this

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**TABLE 1**

<table>
<thead>
<tr>
<th>Type of Symptom</th>
<th>No. of Cases</th>
<th>Type of Symptom</th>
<th>No. of Cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>intracranial hemorrhage</td>
<td>23</td>
<td>headache</td>
<td>7</td>
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<tr>
<td>progressive ischemic deficit</td>
<td>9</td>
<td>headache</td>
<td>4</td>
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<tr>
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<td>1</td>
<td>AVM hemorrhage</td>
<td>1</td>
</tr>
<tr>
<td>aneurysm hemorrhage</td>
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<td>headache</td>
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*AVM = arteriovenous malformation.*
AVM's of the posterior fossa

series (including the two nonoperative cases) underwent angiography at least twice, and all surgical patients had postoperative studies. The average number of angiograms per patient was 2.8; the maximum number of procedures any one patient had was six. In studying the angiograms in these cases, the surgeon is looking for several AVM characteristics: location, size, arterial feeding, and venous drainage. The location of the AVM's in this series is shown in Fig. 1. In addition, these studies offer important information regarding the presence of other important vascular anomalies which may have an impact on both the natural history of the disease and the complexity of any anticipated surgical procedure.

The majority of malformations in this series were located totally or in part in the cerebellar vermis in close proximity to the roof of the fourth ventricle (Table 2). These lesions ranged from 1 to 7 cm in diameter and were typically between 3 and 5 cm in size. As distinct from the other AVM's in this series, they were routinely supplied bilaterally by branches of the superior cerebellar (SCA) and the anterior inferior cerebellar arteries (AICA's), and less commonly from the posterior inferior cerebellar arteries (PICA's). All of the vermian AVM's drained superiorly, directly into the galenic system via the vermis and midline cerebellar veins. Two of these lesions had important transcerebellar supply from major external carotid artery branches, but none of the malformations in this series demonstrated any significant internal carotid artery input.

Seven of the malformations were isolated in one cerebellar hemisphere, and only two of these were in direct continuity with the lateral aspect of the fourth ventricle (Table 2). These hemispheric malformations ranged in size from 2 to 6 cm, with a mean diameter of 3 cm. These laterally placed lesions were always supplied at least in part by branches of the SCA, and in four cases had additional input from either the AICA or the PICA. Venous drainage of these malformations was variable; some drained directly into the galenic system through cortical veins or the vein of Rosenthal, and others drained laterally into the superior petrosal vein and subsequently into the transverse sinus.

Four malformations of varying size were located exclusively within the brain stem, all in the floor of the fourth ventricle and presenting through the ependymal surface of the ventricular wall. The largest of these malformations was 3 cm in diameter and the smallest 5 mm. Blood supply was from branches of the AICA (in two) and/or the PICA (in two), and drainage was

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**TABLE 2**

<table>
<thead>
<tr>
<th>Feature of AVM</th>
<th>Vermis</th>
<th>Hemisphere</th>
<th>Brain Stem</th>
<th>Tonsil</th>
<th>CPA</th>
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<tbody>
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<td>4</td>
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<tr>
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<td>2-6</td>
<td>0.5-3</td>
<td>2-3</td>
<td>3-5</td>
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<tr>
<td>mean</td>
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<td>3</td>
<td>1.5</td>
<td>2.5</td>
<td>4</td>
</tr>
<tr>
<td>arterial supply</td>
<td>SCA</td>
<td>14</td>
<td>0</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>AICA</td>
<td>10</td>
<td>2</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>PICA</td>
<td>9</td>
<td>2</td>
<td>2</td>
<td>1</td>
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<td></td>
<td>ECA</td>
<td>2</td>
<td>0</td>
<td>0</td>
<td>0</td>
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<tr>
<td></td>
<td>bilat</td>
<td>16</td>
<td>0</td>
<td>2</td>
<td>0</td>
</tr>
<tr>
<td>venous drainage</td>
<td>galenic</td>
<td>17</td>
<td>3</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>petrosal</td>
<td>0</td>
<td>5</td>
<td>2</td>
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</table>

* AVM = arteriovenous malformation; SCA = superior cerebellar artery; AICA = anterior inferior cerebellar artery; PICA = posterior inferior cerebellar artery; ECA = external carotid artery; CPA = cerebellopontine angle.

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**Fig. 1.** Artist's drawings illustrating the location and frequency of posterior fossa arteriovenous malformations in this series. Seven lesions were in a single cerebellar hemisphere, two in a cerebellar tonsil, 17 in the vermis, two in the cerebellopontine angle, and four in the brain stem.
via ependymal veins posteriorly and superiorly into either the galenic system (in three) or the superior petrosal sinus (in two). Each of these malformations lay in direct continuity with the fourth ventricle.

Two malformations were located entirely within a single cerebellar tonsil. One AVM was fed exclusively by a large PICA, and the other by a combined AICA-PICA vessel. Both of these lesions filled the entire tonsil, protruding to some extent into the fourth ventricle, and were between 2 and 3 cm in greatest diameter. The venous drainage of these tonsillar malformations was via enlarged vermal veins into the galenic system.

Finally, two of the AVM's in this series occupied the subarachnoid space, the pia arachnoid, and varying amounts of cerebellar cortex and superficial pons in the cerebellopontine angle. These unusual lesions, previously described at length by Drake, have disproportionately large subarachnoid components, with exuberant feeding branches principally from the AICA. They frequently lie in continuity with or actually extend into the lateral recess of the fourth ventricle via the foramen of Luschka. The two AVM's in this location in our series both received small additional feeding arteries emanating bilaterally from the SCA's and PICA's and both drained rostrally through cortical channels into the vein of Galen and laterally through the petrosal vein into the superior petrosal sinus.

Associated Arterial Anomalies

Six (19%) of the 32 patients in this series were demonstrated to have one or more arterial aneurysms located on major branches of the circle of Willis or in close approximation to the malformation in the posterior fossa. A total of 10 such aneurysms were identified, eight of which were located on branches of the vertebrobasilar system. Of these eight aneurysms, four were in anomalous locations situated distally along feeding arterial branches of the AVM. Two of these aneurysms presented with documented subarachnoid hemorrhage: one in a patient with symptoms of cerebellar ischemia who had a large cerebellar hemispheric malformation, and the second over 2 years before a subsequent hemorrhage from a midline vermal AVM.

Patient Management

Thirty of the 32 patients in this series underwent AVM resection; however, this number perhaps portrays a distorted picture of our overall approach to these difficult surgical problems. We have, in general, recommended a surgical procedure in all patients having recurrent episodes of intracranial hemorrhage, in patients with a single intracranial bleed secondary to a small or moderate-sized vermal, hemispheric, or tonsillar malformation, and in all patients with documentation of a progressive disabling brain-stem or cerebellar deficit (Table 3). At the time of their initial evaluation, seven patients were thought to harbor malformations the extirpation of which carried risks greater than those anticipated with the presumed (and admittedly unclear) natural history of those lesions. As mentioned previously, two of these patients did not undergo surgery, although one 51-year-old woman who has had a significant increase in her cerebellar deficit and disabling headache since her initial evaluation 4 years before is currently being reevaluated for surgical treatment. The remaining five patients have been operated on at an interval ranging from 6 months to 5 \( \frac{1}{2} \) years following their first evaluation. Three of these patients presented with SAH and subsequently had a repeat hemorrhage: one was admitted with complaints relating to brain-stem ischemia and later suffered a cerebellar hemorrhage; and one had steady progression of a brain-stem cerebellar deficit over a 2-year-period.

Surgical Timing

When managing patients who have suffered a recent SAH, our policy in general has been to defer definitive surgery until the patient has clinically recovered from the effects of the initial hemorrhage, the CT scan demonstrates resolution of all subarachnoid or intraparenchymal clot, and any questions regarding the development of posthemorrhage hydrocephalus have been resolved. Obviously in the face of a life-threatening intraparenchymal mass, such a delay is inadvisable. We have operated on three patients early to evacuate the hematoma only, believing that microsurgical removal of posterior fossa AVM's with acceptably low morbidity and mortality is not possible immediately after hemorrhage. An additional four patients have required procedures to divert the cerebrospinal fluid (CSF) in the first several days to weeks following their initial hemorrhage, and implantation of a permanent shunting system has been necessary in two patients. These permanent CSF diversionary procedures were performed several weeks prior to definitive surgery (Table 4).

Patients whose presentation has suggested brain-stem or cerebellar ischemia have routinely undergone a relatively protracted radiographic evaluation, including sequential CT scanning, four-vessel angiography, and sequential xenon-inhalation studies of regional cerebral blood flow, both at rest and under pharmacological manipulations. We have found that AVM's with this presentation are commonly but not exclusively large lesions, with multiple feeding arteries, and are

**Table 3**

<table>
<thead>
<tr>
<th>Surgical Indication</th>
<th>No.</th>
<th>Percent</th>
</tr>
</thead>
<tbody>
<tr>
<td>recurrent hemorrhage</td>
<td>11</td>
<td>37</td>
</tr>
<tr>
<td>single hemorrhage</td>
<td>10</td>
<td>33</td>
</tr>
<tr>
<td>progressive neurological deficit</td>
<td>7</td>
<td>23</td>
</tr>
<tr>
<td>progressive neurological deficit plus</td>
<td>2</td>
<td>7</td>
</tr>
<tr>
<td>single hemorrhage</td>
<td></td>
<td></td>
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</tbody>
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Surgical Procedure

The goal of operative intervention in all AVM's, including those of the posterior fossa, is complete removal of the malformation as documented by postoperative angiography. Only total AVM excision can avoid the risk of subsequent rehemorrhage or progression of ischemic neurological deficits. Each patient in this series was operated on with this express object, although the individual size, location, and extent of the malformation under attack obviously influenced the planning and conduct of the surgical intervention. Seven patients underwent staged procedures, five were subjected to intraoperative embolization followed by resection, and three had hematoma evacuation followed later by definitive AVM extirpation. A total of 44 operative procedures directed at AVM removal were performed on these 30 patients (Table 4).

In general, we preferred to approach all of these AVM's through the posterior fossa via a suboccipital craniectomy. Whereas some authors favor exposure of lesions of the superior vermis and lateral hemisphere via a subtemporal or suboccipital transtentorial approach, we have found that these malformations are exposed and operated on with equal facility from beneath the tentorium with the patient in the sitting position. We approached all other malformations of the brain stem and cerebellum with the patient in either the park-bench or true prone position. A large suboccipital craniectomy was used to facilitate exposure of the entire posterior fossa and to aid in the initial identification of feeding arteries, even at some distance from the malformation, if necessary. Our experience suggests that early clip-ligation of all possible feeding arteries at the AVM margin is a key first step in resection, and should be followed by meticulous microsurgical dissection of the entire AVM periphery, sparing all possible venous drainage. We prefer to use small aneurysm clips on all entering arteries; we also believe clipping with these is the best method presently available to deal with fragile enlarged periependymal arteries which frequently prove refractory to any type of bipolar cauterization. The intimate relationship of most of these malformations to the ventricular system has mandated intraventricular exploration in almost all cases to ensure complete AVM removal. Twenty-three of these 30 patients required significant dissection within the body or the lateral recess of the fourth ventricle for mobilization of the malformation, and only those small lesions confined to the lateral cerebellar hemisphere could be resected routinely without intraventricular exposure.

The microsurgical technique of AVM resection confines the surgeon's dissection to the often uncertain margin of the AVM. Here, working within the boundary between abnormal gliotic brain and functional parenchyma, use of bipolar cautery, microscissors, aneurysm clips, and an ever-present suction tip will minimize the chance of iatrogenic injury to normal surrounding brain tissue and important adjacent vasculature. Unfortunately, this type of dissection carries with it the risk of inadvertent isolation of small tufts of malformation from the larger mass of the lesion, a complication that may result in serious intraoperative or postoperative hemorrhage and resultant potentially devastating neurological consequences. We believe that many of the unwelcome sequelae reported in the first few days after AVM surgery that have been attributed to reperfusion phenomena or cerebral edema in fact represent single or multiple foci of hemorrhage from occult retained malformation. For that reason, all patients in our AVM protocol undergo immediate angiography while still under general anesthesia following presumed complete AVM resection. The presence of residual malformation is an indication to return the patient immediately to the operating room to complete the surgical procedure. We believe that this routine has been instrumental in minimizing the incidence of serious postoperative complications. In the current series of malformations, five of 30 patients believed to have undergone complete resection were demonstrated at angiography immediately after surgery to harbor significant residual malformation, and all were returned to the operating room for further AVM resection.

Complete removal of the AVM in each of the 30 patients at a total of 44 operative procedures was documented by postoperative angiography. Two patients died in the perioperative period: one failed to emerge from anesthesia following resection of a large brainstem AVM, and the other who was undergoing a staged removal of a vermian AVM suffered a catastrophic intraparenchymal hemorrhage on the evening following
his initial procedure. He was returned immediately to the operating room where both AVM and clot were completely removed, but he failed to recover to his normal level of consciousness and died of intercurrent infection several months later.

Four patients suffered a significant and permanent increase in their cerebellar or brain-stem dysfunction associated with the surgical procedure. This increased deficit is believed to have occurred secondary to direct surgical trauma to the cerebellum or to the production of associated inadvertent ischemia in three patients; the fourth patient suffered a delayed hemorrhagic infarction within the cerebellar hemisphere, probably secondary to strangulation of the venous drainage from the hemisphere during a surgical procedure performed in the sitting position. These cerebellar deficits are mild in two patients, of moderate severity in one, and totally disabling in the fourth patient. Two additional patients suffered a transient increase in their cerebellar deficit during the 1st postoperative week, a deficit that cleared within the first 6 months after the surgical resection in both patients.

These figures translate into an operative mortality of 7% and an operative morbidity of 13%. The overall significant mortality and morbidity is thus 20% (Table 5).

Discussion

Cerebellar and brain-stem AVM’s are in essence the last of the intracranial malformations to be managed successfully by surgery. The first successful extirpation of a cerebellar AVM was performed in 1932, and several authors, subsequently reported small series of surgically treated patients. Since the introduction of microvascular surgical techniques, these lesions have been more routinely submitted to surgical removal with acceptable rates of morbidity and mortality. 7-8,11,17,21

It is difficult to determine if AVM’s located in the posterior fossa have a special propensity to spontaneous hemorrhage or if the strongly disproportionate number of patients presenting with intracranial bleeding episodes simply represents a selection phenomenon. The Cooperative Study of Subarachnoid Hemorrhage and Intracranial Aneurysms documented intracranial hemorrhage as the presenting complaint in over 72% of infratentorial AVM’s in comparison to 66% of supratentorial lesions. 14 Studies subsequent to this cooperative effort, including the exhaustive pathological study by McCormick, et al., 12 suggest that the incidence of posterior fossa malformations presenting with spontaneous hemorrhage is well above 50%. 4,10,11,21 McCormick’s study of angiomas of the posterior fossa identified 70 true AVM’s, of which 42 (60%) presented with hemorrhage. A summary of the five largest clinical series that describe the surgical treatment of over 100 patients suggests that more than 80% presented with intracranial hemorrhage. 3,7,10,14,20 Several of the authors of these clinical series also comment on the frequency of recurrent intracranial hemorrhage, a finding which occurred in almost 50% of our cases. Additionally, we have noticed a very unique propensity of these lesions to bleed principally into the ventricular system rather than into the cerebellar parenchyma or subarachnoid space. This tendency almost certainly reflects the common intraventricular extension or presentation of all but the most peripherally located cerebellar AVM’s that we have encountered.

Deficits relating to the cerebellum, brain stem, or cranial nerves have for some time been recognized as nonhemorrhagic evidence of posterior fossa AVM’s, and have been attributed by various authors to occult hemorrhage or to compressive or ischemic effects of these lesions on the surrounding parenchyma; however, only recently with the availability of dependable noninvasive radiographic procedures has the relative frequency of this clinical presentation become more apparent. 3,4,10,18,21 Stahl, et al., 18 identified 18 such cases in their excellent review of the reported literature in 1980, and in 1984, Martin, et al., described an additional 13 patients with similar presentations. Many clinicians currently attribute such deficits to ischemia, but it is possible that they represent pathological mechanisms other than ischemia associated with shunting or stealing of normal flow by the low-resistance arteriovenous fistula. As Drake has pointed out, it is common when removing an AVM unassociated with prior clinical hemorrhage to identify one or several cortical or parenchymal areas of hemosiderin staining, suggesting episodes of previous bleeding. However, symptoms do occur in some patients without even microscopic evidence of old hemorrhage, and it is improbable that fluctuating or progressing neurological deficits in the brain stem and cerebellum can be attributed to seizure activity. In fact a strong case can be made for the reverse argument; that is, that epileptiform discharges and the development of seizure foci in supratentorial malformations may be caused by chronic neuronal ischemia adjacent to the fistulous connection, if they are not the result of microscopic injections of hemoglobin into the cortex. Furthermore, increasing data from sophisticated cerebral blood flow evaluations of AVM patients routinely demonstrate the extensive presence of large ischemic territories surrounding even small malformations, and in addition provide surprising documentation of impairment in normal cerebral vasoreactivity — defects that in many cases are reversed by malformation resection (FJ Bonte, et al., unpublished data, 1984).
This present series suggests a relative exclusivity of the clinical syndrome in patients harboring posterior fossa AVM’s; that is, malformations that bled did not seem to be frequently associated with progressive neurological deficit due to “steal,” and those lesions that produced ischemia only occasionally ruptured into the brain or adjacent subarachnoid space. This suggestion, which is supported by the more statistically sound clinical data currently available, but should be kept in mind when considering surgical versus nonoperative therapy.

The third presenting symptom complex reported in other previous series (that of increased intracranial pressure, most commonly due to hydrocephalus) was only found associated with prior bleeding episodes in this series. We believe that this discrepancy relates principally to the inclusion in previous reports of patients with vein of Galen fistulas, and secondarily to the prior lack of recognition of hydrocephalus as a late result of previous subarachnoid or intraventricular bleeding.

No patient in our series presented with a cerebello-pontine angle syndrome such as that described by Verbiest and subsequently reported in a very small percentage of Jannetta’s extensive experience with cranial nerve compressive syndromes. In light of the plethora of normal and abnormal vasculature present in the cerebellopontine angle and ambient cisterns in patients harboring AVM’s of the posterior fossa, this absence of associated neurological deficit is somewhat surprising but is consistent with the previous findings reported by Martin.

The true anatomical distribution of posterior fossa AVM’s is somewhat difficult to identify. This difficulty is due in some measure to the differences in anatomical terminology employed by the authors of several series, and in part because of the relative nonspecificity of some of the previous angiographic evaluations of patients whose malformations were never anatomically examined. Despite these drawbacks, by relying heavily on the extensive pathological review by McCormick and colleagues, a general appreciation of the common distribution of these lesions can be obtained. A total of 120 verified posterior fossa AVM’s have been reported by these several authors; each AVM could be classified as involving either the brain stem, cerebellum, or both (included here are the combination lesions and those which Drake and others have termed “cerebello-pontine angle malformations”). In general, two-thirds of AVM’s located beneath the tentorium were found to lie solely within the cerebellum, another 20% were found to involve the medulla or pons exclusively, and the remaining 15% to 20% were combined lesions, usually located at least in part in the cerebellopontine angle. Further discrete localization of these lesions is not possible from the published data, but review of selected surgical material suggests that a large percentage of the cerebellar malformations reported were located entirely or in part within the midline cerebellar structures, including the superior and inferior vermis and the adjacent cerebellar tonsils. Exact anatomical localization is of more than academic importance: not only do these malformations have a rather characteristic arterial supply, depending on their location, but also the considerations for surgical positioning and exposure vary according to the position of the AVM nidus.

In clinical practice, radiographic evaluation of these malformations must be tailored to provide critical information regarding vascular supply and exact localization. Here the contrast-enhanced CT scan has proven of tremendous value, both in initially suggesting the diagnosis in many of these patients (some of whom presented with non-localizing subarachnoid or intraventricular hemorrhage) but also in localizing the malformation itself. Of almost equal importance has been the CT scan’s ability to clearly elaborate the relationship of the malformation to the adjacent ventricular system. While little information is available from older series, our own experience suggests that a thoughtful melding of the CT and angiographic data is perhaps more important in these lesions than in other more routinely located malformations for assessing the true size and extent of the malformation and for planning an appropriate operative exposure. The significant incidence of intracranial aneurysms in association with posteriorly located malformations reported by Drake and McCormick, and documented by our series, emphasizes the importance of detailed high-quality angiographic investigation of the entire intracranial arterial circulation of these patients.

Prior to the advent of microsurgical techniques, several neurosurgeons had demonstrated that cerebellar hemisphere malformations could be surgically excised without an unduly high risk of neurological devastation. The recent developments in neuroangiographic diagnostic techniques coupled with coincident advances in anesthetic and surgical capabilities have broadened the feasibility of surgical excision to include more intricate and involved lesions of the midline structures, cerebellopontine angle, and some brainstem malformations. As yet, the specific peculiarities of the proximal vertebrobasilar system and its AVM’s have prevented recent interventional radiographic techniques from exerting a therapeutic influence similar to that seen in treatment protocols with large supratentorial malformations. As a result, surgical
excision remains the mainstay of operative treatment. As is true with intracranial malformations in general, proximal ligation of arterial feeders to these lesions does not appear in and of itself to convey any protection from recurrent and often fatal intracranial hemorrhage, but frequently does serve to complicate and enhance the difficulty of any subsequent attempt at definitive malformation resection. The encouraging recent results reported by several authors suggest that the application of modern neurosurgical techniques to the removal of posterior fossa AVM’s offers significant improvement over the natural history of the disease process, especially in patients who present with intracranial hemorrhage.

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References


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