Management of arteriovenous malformations of the brain stem

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In a series of 250 intracranial arteriovenous malformations (AVM's), 12 malformations involved the brain stem. Nine of these lesions were treated surgically, with complete obliteration in eight. There were no operative deaths, and only two patients were made worse by surgery. In the nonsurgically treated group, one patient had embolization therapy and two patients underwent radiation therapy. Results in these three patients have not been satisfactory. These data indicate that brain-stem AVM's can be surgically removed with acceptable morbidity.

Key Words □ arteriovenous malformation □ brain stem □ operative approach □ vascular anatomy

Arteriovenous malformations (AVM's) of the brain stem present a unique technical challenge for the neurosurgeon. Surgical removal of these lesions is considered hazardous due to their obscure location in proximity to vital portions of the brain and the presence of complex vascular anatomy which may be imperfectly defined by current angiographic techniques. The arterial supply to these malformations is often contiguous to that of the brain stem. Resection of these AVM's has been reported infrequently. 2-4,6,7,9,10,14-16,18,19,25 In a series of 250 intracranial AVM's, we have encountered 12 malformations involving the brain stem. On the basis of this experience, we conclude that these AVM's can be surgically obliterated with a reasonable rate of morbidity and mortality.

Summary of Cases

Clinical Material

A summary of the clinical aspects of these 12 patients is presented in Table 1. The age at onset of the first symptom ranged from 9 to 65 years, with a mean of 31.8 years. Seven of the 12 patients were treated within 6 months of their first symptom, 10 patients were treated within 2 years, one patient was first treated 10 years after the initial diagnosis, and one patient was treated 36 years after her first symptom but only 4 months after a diagnosis was established. There were seven males and five females.

Eleven of the 12 patients came to medical attention because of posterior fossa hemorrhage. One of these patients (Case 6) had a transient ischemic episode 2 years before she suffered a pontine hemorrhage; the rest of the patients had no premonitory signs. Only one patient (Case 10) had a slowly progressive neurological course with no evidence of acute hemorrhage. This patient's brain-stem symptoms were presumably due to mass effect and vascular steal from the AVM. The location of the AVM was frequently indicated by the neurological abnormalities following hemorrhage. Four patients developed unilateral lower cranial nerve palsies (Cases 1, 4, 5, and 6). In all of these patients the AVM's were situated at the lateral aspect of the brain stem; three AVM's were located in the cerebellopontine angle. Only three patients (Cases 3, 7, and 12) had signs of primary cerebellar hematoma without cranial nerve palsies. These patients had large AVM's centered in the cerebellum but extending to the brain stem either via the middle cerebellar peduncle or from the vermis to the tectum of the midbrain. Three patients (Cases 2, 8, and 9) bled into the pontine or midbrain tegmentum with secondary rupture into the fourth ventricle. These hemorrhages were from small intrinsic brain-stem AVM's which left the patients with a neurological syndrome compatible with destruction of the dorsolateral pons or midbrain. One patient (Case 11) had a subarachnoid hemorrhage (SAH) with no residual neurological deficit. This AVM was located in the tectum of the midbrain and quadrigeminal cistern.
## TABLE 1
Clinical characteristics of patients with brain-stem AVM’s*

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Age (yrs),†</th>
<th>Sex</th>
<th>First Symptoms</th>
<th>Preop Location of Arterial Findings</th>
<th>Location of AVM</th>
<th>Arterial Supply</th>
<th>Approach &amp; Treatment</th>
<th>Postop Angio Status</th>
<th>Follow-Up Status</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>29, M</td>
<td>SAH</td>
<td>ataxia; CN: Lt V, VI, VII</td>
<td>Lt CPA</td>
<td>Lt AICA lateral</td>
<td>no AVM</td>
<td>6 mos: same as preop</td>
<td></td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>37, M</td>
<td>SAH</td>
<td>ataxia; hemiparesis; nystagmus</td>
<td>pons, floor of 4th vent</td>
<td>Lt AICA &amp; middle peduncle</td>
<td>sitting: vermis split</td>
<td>no AVM</td>
<td>Lt IX, X; weakness, resolved in 8 mos</td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>25, M</td>
<td>cerebellar hem</td>
<td>ataxia only</td>
<td>Lt AICA, Lt PICA</td>
<td>Lt CPA &amp; middle peduncle</td>
<td>1st op: sitting; 2nd op: lateral</td>
<td>no AVM</td>
<td>Lt VI CN &amp; increased ataxia, resolved in 6 mos</td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>45, F</td>
<td>SAH</td>
<td>ataxia; CN: Lt V, VI, X; nystagmus</td>
<td>cerebellum &amp; superior peduncle</td>
<td>Lt CPA</td>
<td>Lt AICA lateral</td>
<td>no AVM</td>
<td></td>
<td></td>
</tr>
<tr>
<td>5</td>
<td>40, F</td>
<td>SAH</td>
<td>ataxia; Lt V, VII, VIII, IX, X; rt hemisensory loss</td>
<td>Lt CPA</td>
<td>Lt AICA lateral</td>
<td>no AVM</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>6</td>
<td>22, F</td>
<td>pontine hem</td>
<td>ataxia; CN: rt V, VI, VII, IX, X; Lt hemisensory loss</td>
<td>Lt PICA</td>
<td>Lt AICA lateral</td>
<td>no AVM</td>
<td>same as preop</td>
<td></td>
<td></td>
</tr>
<tr>
<td>7</td>
<td>65, M</td>
<td>SAH</td>
<td>ataxia only</td>
<td>Lt tectum</td>
<td>Lt vermis &amp; tectum</td>
<td>sitting</td>
<td>no AVM</td>
<td>increased ataxia; Parinaud’s syndrome</td>
<td></td>
</tr>
<tr>
<td>8</td>
<td>29, M</td>
<td>4th vent hem</td>
<td>ataxia; CN: rt VI–XI; Lt hemiparesis; bilat INO</td>
<td>Lt tectum</td>
<td>Lt PICA</td>
<td>lateral</td>
<td>no AVM</td>
<td>same as preop</td>
<td></td>
</tr>
<tr>
<td>9</td>
<td>18, F</td>
<td>SAH</td>
<td>ataxia; bilat CN: IV</td>
<td>Lt tectum</td>
<td>Lt PICA</td>
<td>lateral</td>
<td>no AVM</td>
<td>same as preop</td>
<td></td>
</tr>
<tr>
<td>10</td>
<td>51, F</td>
<td>gradual onset of symptoms</td>
<td>ataxia; CN: Lt V–X</td>
<td>Lt tectum</td>
<td>Lt PICA</td>
<td>lateral</td>
<td>no AVM</td>
<td>same as preop</td>
<td></td>
</tr>
<tr>
<td>11</td>
<td>20, M</td>
<td>SAH</td>
<td>normal</td>
<td>tectal plate region</td>
<td>Lt PCA, Lt PICA</td>
<td>embolization with Silastic pellets; 85% reduction of AVM</td>
<td>both PCA’s occluded</td>
<td>rt hemianopsia; Lt INO; bilat ataxia</td>
<td></td>
</tr>
<tr>
<td>12</td>
<td>54, M</td>
<td>SAH</td>
<td>severe ataxia, bulbar incoordination</td>
<td>tectal plate region</td>
<td>Lt AICA, Lt PICA</td>
<td>conventional RT 3000 rads</td>
<td>5 yrs later: 75% reduction of AVM</td>
<td>6 yrs: devastating SAH; severely disabled</td>
<td></td>
</tr>
</tbody>
</table>

* Abbreviations: AVM = arteriovenous malformation; SAH = subarachnoid hemorrhage; CN = cranial nerve dysfunction; roman numerals = cranial nerves; CPA = cerebellopontine angle; AICA = anterior inferior cerebellar artery; vent = ventricle; INO = intranuclear ophthalmoplegia; hem = hemorrhage; ant = anterior; PICA = posterior inferior cerebellar artery; SCA = superior cerebellar artery; PCA = posterior cerebral artery; RT = radiation therapy.
† Age at treatment.

### Results of Treatment

Nine of the 12 patients in this series underwent surgery. Eight of these patients had complete obliteration of the malformation as confirmed by postoperative angiography. One patient (Case 3) underwent a two-stage procedure before the AVM could be completely removed. Another patient (Case 5) was to have a second operation after her malformation appeared incompletely removed during the initial procedure; however, repeat angiography demonstrated that the residual malformation had thrombosed. In this group of nine surgically treated patients, four had no change in their neurological condition. Three patients had increased deficits immediately following surgery, but all recovered at least to their preoperative status within 1 year. At present, two patients (Cases 2 and 7) have additional neurological deficits related to the operative procedure. Both patients are ambulatory and capable of self-care. The follow-up period for Case 2 is 3 months, and the current rate of improvement in this patient would indicate further recovery.

One patient (Case 11) was treated by embolization only. He had a considerable neurological deficit as the result of the procedure, and the lesion was not obliterated. Two months following embolization, he could walk with assistance, but surgical treatment was not...
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Representative Cases

Case 1
This 29-year-old man was seen 4 months after he had suffered a left pontine hemorrhage. As a result of the hemorrhage, he had a severe left facial weakness, weakness of the left masseter muscle, difficulty in swallowing and speaking, left cerebellar ataxia, and a left lateral rectus nerve palsy. Angiography demonstrated an AVM traversing the brain stem, extending from the left lateral margin of the pons to the ependymal surface of the fourth ventricle (Fig. 1 left). The arterial supply to the malformation arose from the anterior inferior cerebellar artery (AICA).

The lesion was resected via a left suboccipital craniectomy performed in the lateral or park-bench position. The flocculus was resected to afford adequate exposure of the lower cranial nerves and the lateral side of the brain stem. A large arterialized vein was encountered which was intimately associated with the seventh and eighth cranial nerves and was coiled around filaments of the ninth and 10th cranial nerves, where it was joined by numerous branches of the AICA. Proximally the vein arose from the lateral surface of the pontomedullary junction, while distally it drained toward the incisura. The feeding arteries were cauterized, and the vein was followed to the substance of the brain stem, where feeding vessels that appeared on the pial surface of the pons were cauterized. At this point the vein became smaller and turned blue. No further intramedullary dissection was necessary.

Postoperatively, the patient had more impairment of the function of the lower cranial nerves. He required gastrostomy and tracheostomy for temporary control of pulmonary toilet and nutrition. These aids were removed within 2 months of surgery, but the patient was left with complete palsy of the fifth through the eighth cranial nerves on the left. A follow-up angiogram demonstrated no filling of the original malformation (Fig. 1 right).

Case 2
This 37-year-old man was seen 21 months after he had suffered a pontine hemorrhage. He required rehabilitation but eventually was able to return to work. Neurological examination demonstrated vertical nystagmus on upward gaze. He had decreased sensation over both sides of the face with a mild left hemiparesis and hemisensory loss. He exhibited ataxia on the left side, but was nonetheless able to work as an engineer. A computerized tomography (CT) scan demonstrated the lesion in the floor of the fourth ventricle (Fig. 2 left). Angiography revealed a small AVM in the region of the right rostral pons, fed by penetrating branches from the basilar artery (Fig. 2 center), and magnetic resonance imaging documented that the malformation lay in the floor of the fourth ventricle, just to the right of midline (Fig. 2 right).

A suboccipital craniectomy was performed with the patient in the sitting position and the vermis was split in the midline. Exposure of the fourth ventricle revealed an area of discoloration on the right side of the ventricular floor (Fig. 3 left). A circumscribing incision was made around this area of cystic encephalomalacia and discoloration. A number of small arterial loops were encountered immediately below the surface. These represented penetrating arteries supplying the malformation. These vessels were cauterized, as was the entire malformation. The draining vein was transected at the superior pole of the AVM, and the lesion was removed.

Postoperatively, the patient had more severe left-sided ataxia, further decrease of left-sided sensation, and a right sixth nerve palsy with a left intranuclear ophthalmoplegia. Except for the ataxia, these deficits improved with time and physiotherapy. A postoperative angiogram demonstrated no malformation (Fig. 3 right).

Case 6
This 22-year old woman was seen 2 years after she experienced a transient episode of numbness on the left side of the body. A CT scan at that time was normal. Five months prior to our evaluation she suffered an acute right pontine hemorrhage. Neurological deficits after the hemorrhage included a left hemiparesis, complete right seventh cranial nerve palsy, deafness of the right ear, decreased sensation in the distribution of the
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FIG. 2. Preoperative studies in Case 2. Left: Contrast-enhanced computerized tomography scan showing an arteriovenous malformation (AVM) centered in the tegmentum of the midbrain and pons (arrow). Center: Subtraction vertebral arteriogram, anteroposterior view, showing the AVM (closed arrow) fed by penetrating branches of the basilar artery and drained via an enlarged mesencephalic vein (open arrow). Right: Magnetic resonance image showing the AVM in the floor of the fourth ventricle (arrow).

first division of the fifth cranial nerve on the right, with absent corneal reflex, moderate right-sided cerebellar ataxia, right sixth cranial nerve palsy, absent right-sided gag reflex, paralyzed right vocal cord, and left hemisensory loss of pain and temperature sensation. Despite these problems, she was able to walk with the help of a cane. Angiography showed an AVM in the right pons fed primarily by the AICA (Fig. 4 left). A contrast-enhanced CT scan localized the lesion in the right brachium pontis.

Surgery was performed via a right suboccipital craniectomy with the patient in the lateral or park-bench position. The cerebellum was retracted to expose the cerebellopontine angle, and a large arterialized vein was

FIG. 3. Case 2. Left: Operative exposure demonstrating a site of old hemorrhage and the arteriovenous malformation (closed arrows) in the floor of the fourth ventricle at the rostral pontine level. The acoustic stria is demonstrated by an open arrow. Right: Postoperative angiogram showing no residual malformation.
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The first successful surgical treatment of a brain-stem AVM was reported by Mount in 1965. Postoperative angiography confirmed obliteration of the malformation after clipping of the main feeding artery. Green and Vaughan later reported the subtotal removal of an extensive AVM involving the cerebellum, brain stem, and cranial nerves. Two brain-stem AVM’s were totally removed by Chou, et al.; both patients made excellent recoveries, but postoperative angiography was not performed. Drake reported operating on five patients with brain-stem AVM’s, three of which were located primarily in the cerebellopontine angle. The clinical results in four of the five patients were gratifying and indicated that surgical intervention in these lesions is a feasible approach. There are also several sporadic reports of the surgical treatment of brain-stem AVM’s, 

The diagnosis of brain-stem AVM’s is frequently made after the patient experiences a posterior fossa hemorrhage. Eleven of the 12 patients in the present series presented with SAH or cerebellar hemorrhage. In the series of Stahl, et al., 14 of the 21 cases had an SAH. Four of seven patients described by Logue and Monckton had an SAH and 80% of patients with posterior fossa AVM’s reported in the Cooperative Study had an SAH. The morbidity and mortality rates associated with these hemorrhages are high, and the goal of therapy must be to eliminate the risk of recurrent hemorrhage.

Approximately 50% of patients with supratentorial AVM’s present with acute SAH or intracerebral hemorrhage. This figure is considerably lower than the 80% to 90% incidence of hemorrhage associated with brain-stem AVM’s. There are two plausible explanations for this difference. Seizures are the presenting symptom for 28% to 67% of patients with supratentorial AVM’s. This symptom is not observed with malformations of the brain stem or cerebellum. Other modes of presentation (such as headache, and vascular steal syndrome) are rare, even in the group with supratentorial AVM’s. The second explanation for the high incidence of hemorrhage as the presenting symptom in cases of brain-stem AVM’s is that malformations in this location tend to be small. Morello and Borghi reported hemorrhage in 86% patients with small supratentorial AVM’s, but in only 46% of those with giant AVM’s.

Vascular Anatomy

The arterial supply and venous drainage of brain-stem AVM’s vary depending on the site of the malformation. Three AVM’s in this series were located in the superior aspect of the vermis and quadrigeminal cistern, indenting the tectum of the midbrain. The fourth cranial nerve is often involved with these malformations, and its proximity presents a special hazard during surgical intervention. In this location, nutrient arteries arise primarily from the superior cerebellar artery with variable contributions from the posterior cerebral artery.

Discussion

Review of the Literature

Brain-stem AVM’s are rare lesions, and their treatment has been infrequently reported in the literature. Of a total of 453 cases of intracranial vascular malformations reviewed in the Cooperative Study of SAH, 11 malformations (2%) involved the brain stem. None of these lesions was surgically removed. Logue and Monckton discussed seven cases of brain-stem AVM’s. They concluded that total removal of an AVM is not feasible when the brain stem is involved. McCormick, et al., performed neuropathological studies on 346 cerebral angiomas. Sixty-six of these malformations involved the brain stem, but only 18 were AVM’s. There is no reference to surgical treatment in this series of patients. Stahl, et al., reported on 21 cases of brain-stem vascular malformations, 18 obtained from the literature. Nine of these cases were AVM’s and there is no mention of surgical treatment.

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and its posterior choroidal branches. These malformations usually drain into the vein of Galen, the straight sinus, and (to a lesser extent) the pontomesencephalic veins.

Three malformations were located in the cerebellopontine angle and involved the lateral surface of the pons and adjacent cranial nerves. The AICA provides the primary blood supply to these AVM's. Hypertrophied feeding arteries from the AICA tend to be extensively intertwined with the fifth through the 12th cranial nerves. This association explains the vulnerability of the lower cranial nerves to damage after hemorrhage from these AVM's. Venous drainage from these AVM's is via the petrosal vein, superior petrosal sinus, and (to a lesser extent) the pontomesencephalic system.

Two malformations in this series were located in the lateral cerebellar hemisphere and extended to the brain stem via one of the cerebellar peduncles. A malformation that traversed the superior cerebellar peduncle to the midbrain in Case 4 was fed by the hemispheric branches of the superior cerebellar artery. An AVM in the middle cerebellar peduncle of Case 6 was more closely allied with the malformations of the cerebellopontine angle, and received its blood supply from the AICA as well as from the cerebellar branches of the posterior inferior cerebellar artery. Drainage of these laterally placed lesions is via the petrosal venous system.

Five malformations in this series were within the brain stem. All of these AVM's were supplied by paramedian or circumferential penetrating arteries from the basilar artery. These malformations tend to be smaller, less surgically accessible, and bleed with more devastating effects than malformations with subarachnoid or cerebellar extension. Venous drainage is into the lateral mesencephalic and pontomesencephalic veins.

Surgical Considerations

Although the surgical considerations for brain-stem AVM's are similar to those for other AVM's, there are some important exceptions. Whereas supratentorial AVM's are usually removed by means of circumscribing incisions, it has been possible to extirpate some brain-stem AVM's by interruption of dedicated feeding arteries or to eliminate the shunt by simply cauterizing a small AVM.

Successful surgical treatment of brain-stem malformations begins with careful evaluation of the neurodiagnostic studies. A thorough understanding of the arterial supply, venous drainage, and anatomical location of these lesions is essential to the planning of any operative intervention. There are three basic surgical approaches to AVM's involving the brain stem (Fig. 5). In rare instances, a staged procedure requiring two different approaches may be used to insure safe removal of the AVM.

Malformations that involve primarily the lateral surface of the brain stem, cerebellum, and cerebellopontine angle are best approached through a unilateral suboccipital craniectomy. With the patient in the lateral or park-bench position, the bone removal is extended laterally into the mastoid air cells until the sigmoid sinus is visible, and then inferiorly to the rim of the foramen magnum. The dura is opened in a cruciate fashion, allowing retraction on the lateral surface of the cerebellum with exposure of the cerebellopontine angle. Arteriovenous malformations of the superior aspect of the vermis, quadrigeminal region, and dorsal midbrain are best reached via the supracerebellar infratentorial approach. The patient is placed in the sitting position and a full suboccipital craniectomy is performed from the transverse sinus to the level of the foramen magnum. The dura is opened in a cruciate fashion, allowing retraction on the lateral surface of the cerebellum with exposure of the cerebellopontine angle.

![Fig. 5. Drawing showing the three basic surgical approaches to arteriovenous malformations (AVM's) of the brain-stem. Route A is over the anterior cerebellum directed toward the rectum of the midbrain. Route B is via the posterior fossa into the cerebellopontine angle or lateral side of the pons and midbrain. Route C involves a vermis-splitting incision. The approach is via the fourth ventricle to the medullary pons and lower end of the aqueduct. These basic surgical approaches should provide exposure to all operable brain-stem AVM's.](image-url)
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stem, or arterial branches that may arise from the lower two-thirds of the basilar artery. A safe operation on vascular malformations requires control of all elements of the arterial supply to these lesions.

Malformations that are within the confines of the fourth ventricle or that involve the cerebellar peduncles require a midline approach through a complete suboccipital craniectomy. The sitting position is preferred for patients undergoing these operations, especially when the vermis is split and exposure of the lower aqueduct is desired, as in Case 2.

In all cases where cerebellar retraction is to be utilized, spinal drainage is invaluable in affording relaxation of the cerebellum. Microsurgical technique, bipolar cautery, coaxial illumination, and the microscope are essential elements for the safe removal of these lesions. Feeding arteries are divided as they enter the malformation, and major draining veins are preserved until the total arterial supply to the malformation has been eliminated.

The cases reviewed in this paper form a large series of brain-stem AVM’s operated on by one surgeon. These lesions represent a grave threat since most of our patients had devastating neurological deficits following hemorrhage. The goal of therapy in these patients was to obviate the risk of recurrent hemorrhage without producing disabling neurological deficits. In our opinion the only way of achieving this goal is by total obliteration of the malformation. This was accomplished in eight of the nine patients who underwent surgery. There were no operative deaths in this series. Two patients were made worse by the operation; however, both of these individuals are reasonably independent and continue to improve. Nevertheless, our statistics for the operative treatment of brain-stem AVM’s do not compare with morbidity and mortality rates for the supratentorial AVM’s. Notwithstanding the 11% surgical mortality rate reported by the Cooperative Study on AVM’s, recent series have proved that cerebral malformations can be removed with a mortality of under 2% and a morbidity of under 10%. Given the technical difficulty of the surgical approach and the serious threat to neurological function posed by these lesions, we believe that our operative morbidity of 22% is acceptable.

We used three basic approaches to obliterate the brain-stem AVM’s in these patients (Fig. 5). These approaches are dependent upon the location of the malformation. Malformations located close to the tectum of the midbrain are approached via an infratentorial supracerebellar approach (Route A) that may be midline or lateralized if the lesion tends to one or the other side. For lesions that involve the midline, and to some extent both sides, we have not used a transtentorial approach either from the occipital or temporal region because of obvious anatomical considerations. For lesions that are located along the lateral aspect of the brain stem, especially those in the cerebellopontine angle, the approach should be paramastoid as though approaching an acoustic tumor (Route B). We have generally preferred the lateral or the park-bench position for these patients since it allows maximum relaxation of the cerebellum. For lesions located primarily in the floor of the fourth ventricle, either caudal or rostral, a vermis-splitting approach (Route C) via the midline is necessary. Not all of these approaches permit the surgeon to reach the AVM from the arterial side first; however, considering the obscure location of many of these lesions and the confining nature of the posterior fossa, the best exposure for the various lesions will be obtained by using these three basic routes.

Embolization of supratentorial AVM’s, which we consider an important preparation of these patients for surgery, has not been of significant utility in the treatment of brain-stem AVM’s. Case 11 demonstrates the risk of damaging vital neural structures when emboli are directed into vessels supplying the brain stem. Furthermore, brain-stem AVM’s are supplied by major branches of the basilar artery which generally originate at right angles from the parent artery. Consequently, it is difficult to selectively occlude the feeding arteries without risking the patency of arteries to normal structures. At the present state of our technology, these factors contraindicate the use of embolization in the preoperative preparation of most patients with brain-stem AVM’s. However, future improvement in intravascular techniques may define a role for embolization treatment of these lesions.

Gamma-beam irradiation for the treatment of small AVM’s has been advocated by Steiner. Successful treatment of a large group of patients has been reported with complete obliteration of the malformations. The major drawback to this method is the long latent period between treatment and obliteration of the malformation. In many instances this period exceeds 2 years, during which time the patient is at risk for hemorrhage. Additionally, the long-term deleterious effect of this high-energy radiation is unknown. Therefore, patients should be chosen carefully for radiosurgery.

Patients who have had hemorrhage from a brain-stem AVM are at risk for renewed rupture of the malformation, characteristically with devastating results. These patients should be treated surgically unless the AVM is situated deep in the substance of the brain stem. Patients with AVM’s that have not ruptured and are not situated in a favorable location for surgical removal can be considered for radiosurgery. One patient in this series (Case 10) was treated in this fashion.

Conclusions

Our experience in treating 12 patients with AVM’s of the brain stem supports the contention that surgical obliteration of malformations in this region is not only feasible but is the treatment of choice. Conservative treatment leaves the patient susceptible to recurrent hemorrhage which often results in devastating neurological deficits. Embolization therapy in the vertebro-
basilar system can be hazardous. Radiation therapy is
effective, but the patient continues to be at risk for
repeat hemorrhage during the time that is required to
shrink the malformation, and the long-term side effects
of this treatment are unknown.

References


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