Intracranial Hypertension After Resection of Cerebral Arteriovenous Malformations
Predisposing Factors and Management Strategy

Issam A. Awad, MD; Michele Magdinec, RN; Armin Schubert, MD

Background and Purpose Surgical excision of cerebral arteriovenous malformations (AVMs) may be complicated by postoperative breakthrough edema and hemorrhage and by intracranial hypertension. Embolization, staged resection, and meticulous surgical technique have decreased but not completely eliminated this complication. In this study we prospectively assess the prevalence of intracranial hypertension after excision of cerebral AVMs, examine factors predisposing to this complication, and document the outcome of aggressive monitoring and treatment of elevated intracranial pressure (ICP).

Methods During a 4-year period at a single institution, 32 consecutive patients with cerebral AVMs underwent surgical excision after staged embolization. All patients underwent postoperative monitoring of ICP and a uniform management protocol of intracranial hypertension.

Results Intractable intracranial hypertension was encountered after resection of 9 of 32 cerebral AVMs, including 3 of 20 (15%) AVMs 6 cm or less in maximum diameter and 6 of 12 (50%) AVMs greater than 6 cm in maximum diameter. This complication occurred in 5 of 10 (50%) lesions located in distal or border-zone locations, in 4 of 9 (44%) AVMs arising directly off proximal cerebral arteries, and in none of 13 AVMs in other locations. Preoperative single-photon emission-computed tomography perfusion scans were performed in 17 patients and demonstrated parenchymal hyperperfusion beyond the AVM nidus in 5 of 10 AVMs 6 cm or less in maximum diameter, none of which manifested postoperative intractable ICP. Hyperperfusion was observed on single-photon emission-computed tomography in 7 of 7 AVMs greater than 6 cm that were studied by this modality, and intractable ICP was observed postoperatively in 5 of these cases despite preoperative staged embolization in every case. Patients with symptomatic intractable ICP were treated with intravenous short-acting barbiturates under a strict critical care protocol. There was no instance of uncontrollable intracranial hypertension or breakthrough edema while on barbiturate therapy. There was no permanent morbidity related to this treatment and no mortality or new disabling morbidity in this series.

Conclusions We conclude that intractable intracranial hypertension remains a common complication after resection of a subgroup of cerebral AVMs despite preoperative embolization, modern neuroanesthesia and critical care management, and microsurgical technique. A proactive management protocol aimed at ICP control is safe and effective in the management of this complication. (Stroke. 1994;25:611-620.)

Key Words • cerebral arteriovenous malformations • hypertension • intracranial pressure

Postoperative brain edema and breakthrough hemorrhage is a potentially lethal or severely disabling complication following successful surgical resection of cerebral arteriovenous malformations (AVMs).1-25 Preoperative embolization, staged resection strategies, intraoperative (or early postoperative) angiography ensuring complete AVM excision, and meticulous surgical and neuroanesthetic techniques have decreased but not completely eliminated this complication. Lesions at high risk of this complication are thought to be larger AVMs, AVMs in distal or border-zone locations, and AVMs causing significant hemodynamic cerebral steal on preoperative diagnostic studies.2-4,10,12,14,15,25-26 However, the prevalence of this complication has not been clearly evaluated in prospective studies using a uniform management protocol.

Treatment of this complication has typically consisted of induced systemic hypotension and attempted control of intracranial pressure (ICP). Outcome after this complication has generally been disappointing, with these cases representing the majority of mortality and serious morbidity in large series of cerebral AVMs.* There have been anecdotal reports of successful management of intractable ICP elevations after resection of AVMs with barbiturate-induced coma.1,2,4,6,12,14,20,29 There have been no published reports of systematic analysis of this therapy, including indications, complications, and/or outcome.

In 1988 a uniform management strategy was instituted by the Cerebrovascular Surgery Service at the Cleveland Clinic Foundation aimed at the prevention, early detection, and aggressive treatment of intracranial hypertension after resection of cerebral AVMs. Barbiturate therapy was instituted in patients with symptomatic ICP elevations not responsive to more conventional measures of osmotic and loop diuretics and hyperventilation. We analyzed the prevalence of intractable intracranial hypertension after resection of cerebral

*References 5, 13, 14, 16, 17, 19, 20, 27, 28.
AVMs, factors predisposing to this complication, and the role and outcome of its management using barbiturate therapy.

Subjects and Methods

From 1988 to 1992, 32 supratentorial cerebral AVMs were excised by the Cerebrovascular Surgery Service at the Cleveland Clinic Foundation. These included 7 lesions less than 3 cm in maximum nidus diameter, 13 AVMs 3 to 6 cm in maximum diameter, and 12 AVMs greater than 6 cm in maximum diameter. Infratentorial AVMs, dural AVMs, angiographically occult AVMs, and lesions operated emergently after catastrophic hemorrhage were excluded from this series.

Preoperative Preparation

Surgical excision was delayed by 1 month or more after overt cerebral hemorrhage. All lesions were studied preoperatively with computed tomography (CT) and magnetic resonance imaging. Diagnostic cerebral panangiography was performed in every case and documented the maximum nidus diameter, pattern and tortuosity of arterial feeders, rapidity of shunting, paucity of filling of normal cerebrovascular territories, and the pattern of venous drainage.

Preoperative embolization was considered in all cases except small superficial cortical AVMs with slow arteriovenous shunting. In lesions greater than 3 cm in maximum nidus diameter or with more than two embolizable arterial feeders, the embolization was staged to allow 3 to 10 days between embolization sessions and from the last embolization to surgical resection. All embolizations were performed extraoperatively using femoral puncture, retrograde aortic cannulation, and a variety of flow-directed and other cerebral microcatheters. A variety of embolic materials were used at the discretion of the interventional neuroradiologist, so as to accomplish maximal decrease of flow through the AVM nidus before surgical excision.5-9,11,36-34 Serial diagnostic angiograms were performed during the embolization procedures and helped to guide the extent of and staging of these procedures.5-7,31 Arterial feeders passing toward eloquent brain locations or feeding AVMs in such locations were test-injected with amobarbital sodium before embolization, while closely monitoring the respective cortical function in the awake patient.

Preparatory embolization was performed in 2 of 7 AVMs less than 3 cm in maximum nidus diameter, in 9 of 13 AVMs 3 to 6 cm in maximum diameter, and in 11 of 12 AVMs with maximum diameter greater than 6 cm (embolization was not possible in 1 patient with a giant left temporal lobe AVM because of the pattern of arterial feeders arising directly from sylvian vessels). More than one embolization session was performed in 16 patients, including all AVMs with maximum nidus diameter greater than 6 cm. The mean number of embolization sessions per patient was 2.2 (range, 1 to 4 embolization sessions per patient). The intervals between embolization sessions and until surgery ranged from 1 to 16 days (mean, 5.2 days).

Preoperative Single-Photon Emission-Computed Tomography

Seventeen patients were subjected to perfusion single-photon emission-computed tomography (SPECT) studies before embolization or resection. Eleven of these patients underwent additional subsequent SPECT scans to monitor cerebral perfusion between embolization sessions and before resection. In 6 patients the SPECT tracer consisted of radioactive iodine–labeled N-iodoammonium.23,24 In the remaining patients it consisted of hexamethylpropyleneamine oxime labeled with radioactive technetium.37,38 In all instances the SPECT scans were evaluated blindly by a nuclear medicine staff physician who specifically indicated the presence or absence of parenchymal hyperperfusion beyond the borders of the AVM nidus.23,24 Delayed control scintillation scan was often performed to assist with the delineation of the AVM nidus, which persisted as an area of hyperperfusion after redistribution of the tracer in the rest of the brain. Parenchymal hyperperfusion was considered as adjacent, remote ipsilateral, or remote contralateral to the AVM nidus. Persistence, worsening, or normalization of areas of hyperperfusion was noted in cases undergoing serial SPECT scans after staged embolization.

Perioperative Management

Excision of the AVM was performed at a single stage under general anesthesia in all instances. Systolic arterial pressure was maintained below 100 mm Hg throughout surgery and the early postoperative period. Maximal brain relaxation was ensured before dural opening and commencing excision of the AVM. Osmotic and loop diuretics and hyperventilation (arterial Pco2 less than 30 mm Hg) were used to accomplish this objective. Intravenous barbiturates (thiopental sodium) were used during anesthetic induction and intermittently throughout the procedure as needed, especially in the setting of brain swelling or with temporary vessel occlusion. Electroencephalographic (EEG) leads were applied to the scalp in a global 10-20 montage except in the area of craniotomy, and the EEG was monitored continuously throughout the procedure.

Since 1989 all craniotomies have been performed in a radiolucent head holder, and intraoperative angiography has been performed after excision of the AVM (and before cranial closure) to ensure complete resection of the nidus. Before the availability of intraoperative angiography patients underwent early postoperative angiography, usually before awakening from anesthesia. Digital subtraction techniques allowed satisfactory angiography despite the EEG scalp leads.

In all instances and regardless of the complexity of the operation, an ICP monitoring device was placed before closure of the craniotomy. In cases where the ventricular system was entered, a ventricular catheter was used for this purpose. In other instances a subdural Camino device (Camino Corporation) was used, and this was exited through a separate stab wound remote from the main incision. The ICP was monitored continuously during the reversal of anesthesia and for at least 2 days postoperatively in every case.

After resection of the AVM the arterial Pco2 was normalized unless there was severe brain swelling or intracranial hypertension during the later stages of craniotomy closure. In cases with adequate brain relaxation and satisfactory ICP during craniotomy closure, the patient was awakened and extubated with judicious attention to prevent coughing, strain, and elevations of systolic arterial pressure above 100 to 110 mm Hg. In cases of intraoperative brain swelling or postresection ICP elevation, the patient was kept intubated and sedated as described below.

Management of Intracranial Hypertension

Sustained elevations of ICP above 25 mm Hg were managed with intermittent doses of osmotic and/or loop diuretics. In intubated patients hyperventilation was instituted to keep the arterial Pco2 below 30 mm Hg.

Patients with ICP greater than 30 mm Hg not responding to osmotic and/or loop diuretics and who exhibited progressive signs of altered sensorium were sedated, intubated, and hyperventilated. A CT scan was performed to rule out focal hematoma or oedematous anemic to surgical intervention.

Patients with sustained ICP elevations greater than 30 mm Hg associated with compromised neurological status despite osmotic and/or loop diuretics and hyperventilation were considered to have intractable ICP and were selected for barbiturate therapy. Arterial blood pressure was vigorously controlled below 100 to 110 mm Hg or to maintain cerebral perfusion pressure greater than 45 to 50 mm Hg. Pentobarbital was used as the agent of choice.
for barbiturate therapy in the neurosurgical intensive care unit. A loading dose of 10 mg/kg was administered over 30 minutes followed by 5 mg/kg per hour for the next 3 hours. Barbiturate therapy was maintained when needed using a pentobarbital drip at a dose of 1 to 2 mg/kg per hour adjusted to achieve ICP and EEG end points as discussed below.

Vigorous multisystem medical support was instituted during barbiturate therapy, including prophylactic antibiotics against hospital-acquired pneumonia and close monitoring of cardiac function using a Swan-Ganz catheter in every case. Frequently there was decreasing requirement of antihypertensive drips to maintain the desired arterial hypotension after institution of barbiturate therapy. Hemodynamic and cardiac support with pressors or inotropic agents was occasionally required to maintain peripheral and cerebral perfusion in the desired range.

Normovolemia was maintained in all patients and occasionally required infusion of colloids in view of peripheral vasodilation in prolonged barbiturate therapy. Laboratory parameters of normonatremia, renal function, coagulopathy, cardiac function, and liver function were closely monitored. Pneumatic antiembolic stockings were used in every case for deep vein thrombosis prophylaxis. A CT scan of the head was performed frequently during barbiturate therapy (on the average every 2 days), especially in the setting of increasing ICP, pupillary changes, or asymmetrical changes in the burst activity on the EEG. The latter were the only parameters that warned of a possible significant neurological change during barbiturate therapy. The CT scan was evaluated for evidence of mass effect including sulcal effacement, blurring of the junction of gray matter and white matter, ventricular symmetry, and patency of the basal cisterns. The CT scan also excluded ventriculomegaly, hematomas, and hygromas that might have contributed to elevated ICP.

The primary end points of barbiturate therapy were ICP trends in the desired range or EEG burst suppression of 2 to 8 bursts per minute. In some cases a lighter level of barbiturate sedation was sufficient for ICP control (Fig 1). Barbiturate therapy was gradually tapered after 24 to 48 hours if ICP showed better control trends and CT scans showed decreasing edema or decreasing mass effect and absence of hemorrhage. Pentobarbital levels were monitored as the patient emerged from barbiturate coma. The patient was allowed to awaken spontaneously and was evaluated for extubation based on respiratory parameters.

Results

Prevalence of Intracranial Hypertension
In all but 2 patients there were one or more periods of elevation of ICP above 25 mm Hg in the first 48 hours after AVM resection. This usually responded to analgesics, blood pressure control, and/or osmotic and/or loop diuretics. In intubated patients, hyperventilation was also a useful adjunct to ICP control. In 9 of the 32 patients (28%), there were sustained ICP elevations above 30 mm Hg not responding to these measures and associated with neurological deterioration. The size, pattern of venous drainage, and location of AVMs that manifested intractable ICP are compared in Table 1 with those of AVMs that did not exhibit this complication. Intractable ICP was observed in 1 of 7 (14%) AVMs with maximum nidus diameter less than 3 cm, in 2 of 13 (15.3%) AVMs with maximum nidus diameter greater than 5 cm (P<.05). There was no correlation between the pattern of AVM venous drainage (deep or superficial) or the eloquence of AVM location and the prevalence of intractable intracranial hypertension.

Table 1. Comparison of Arteriovenous Malformation Size, Venous Drainage, and Location in Patients With and Without Postoperative Intractable Intracranial Hypertension

<table>
<thead>
<tr>
<th>AVM size, cm</th>
<th>Intractable Intracranial Hypertension (n=9)</th>
<th>No Intractable Intracranial Hypertension (n=23)</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;3</td>
<td>6</td>
<td>1</td>
</tr>
<tr>
<td>3-6</td>
<td>11</td>
<td>2</td>
</tr>
<tr>
<td>&gt;6</td>
<td>6*</td>
<td>6*</td>
</tr>
<tr>
<td>Venous drainage</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cortical only</td>
<td>2</td>
<td>9</td>
</tr>
<tr>
<td>Deep</td>
<td>14</td>
<td></td>
</tr>
<tr>
<td>Eloquent brain</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>6</td>
<td>19</td>
</tr>
<tr>
<td>No</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>AVM location</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Distal border zone</td>
<td>5</td>
<td>5</td>
</tr>
<tr>
<td>Proximal in parallel feeders</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>Other</td>
<td>0</td>
<td>13*</td>
</tr>
</tbody>
</table>

AVM indicates arteriovenous malformation. *P<.05, χ² test.
Giant posterior parietal arteriovenous malformation (AVM) requiring postoperative barbiturate therapy for control of Intracranial pressure. Top left, Preoperative angiogram; Internal carotid injection lateral view. Note long ectatic feeders from the middle cerebral artery toward the distal border zone where the AVM nidus is located. Top right, Preoperative angiogram; vertebral injection lateral view. Hypertrophic posterior cerebral artery feeds a large compartment of the AVM. Bottom left, Preoperative angiogram; venous phase, lateral view. Note direct drainage into the superior sagittal sinus via a giant cortical vein (arrow). Bottom right, Preoperative single-photon emission-computed tomography perfusion scan (left) revealing significant hypoperfusion beyond the region of the AVM (closed arrow). A delayed scan performed 2 hours later (right) shows redistribution of the tracer except in the region of the AVM nidus itself (open arrow). The patient had severe papilledema. Despite preoperative embolization in three stages, the patient required 2 days of barbiturate therapy for control of Intracranial pressure postoperatively. He made a full neurological recovery except for nondisabling visual field defect. The papilledema resolved after surgery.

Intractable ICP occurred in 5 of 10 (50%) patients with AVMs in distal or border-zone locations (Fig 2) and in 4 of 9 (44%) patients with AVMs deriving feeders directly (in parallel) from basal or sylvian major vessels (Fig 3). This complication was observed in none of 13 patients with AVMs in other locations (including lesions of all sizes) (P<.05). While the number of cases was too small for statistical power in multivariate analysis, the impact of AVM location on postoperative intracranial hypertension was clearly not accounted for solely by AVM size. The one AVM less than 3 cm in maximum diameter that was associated with intractable postoperative ICP elevations was in a distal border-zone location. Conversely, 5 of the 6 giant (greater than 6 cm) AVMs that were not associated with postoperative intracranial hypertension were in lobar locations without distal border-zone feeders or direct (in parallel) feeders from basal or sylvian vessels.

Of the 17 patients who underwent perfusion SPECT studies before embolization and resection, 5 were without any evidence of cerebral parenchymal hypoperfusion (Table 2). None of these cases involved AVMs greater than 6 cm in maximum diameter. Embolization was performed in 2 of these 5 patients, and postoperative intractable ICP did not occur in any case.

Twelve patients exhibited cerebral hypoperfusion on SPECT, including 5 with AVM nidus diameter less than 6 cm and 7 with AVM nidus diameter greater than 6 cm. Remote contralateral hypoperfusion (interhemispheric steal) was documented in 5 of the 7 patients with AVMs greater than 6 cm in maximum diameter and in none of the patients with smaller AVMs. Remote ipsilateral hypoperfusion (intrahemispheric steal) was demonstrated in 2 of the smaller AVMs and in all 7 AVMs greater than 6 cm in maximum diameter. None of the 5 patients with AVMs less than 6 cm in maximum diameter that exhibited preembolization cerebral steal developed postoperative intractable intracranial hypertension (although embolization was performed in 3 of these patients). The 7 patients with giant AVMs all exhibited cerebral hypoperfusion on SPECT, and all underwent prepa-
ratory embolization. Five of these patients developed intractable ICP after excision.

The impact of embolization on cerebral hypoperfusion could only be addressed in an anecdotal fashion (Fig 4) because SPECT scans were not routinely performed after embolization in all instances, and their timing in relation to the most recent embolization was highly variable. In some instances cerebral hypoperfusion was greatly improved by embolization, and some of those patients still required postoperative barbiturate therapy for control of ICP. In other patients there was persistent hypoperfusion beyond the AVM nidus after several sessions of embolization, and some of these patients did not develop intractable intracranial hypertension postoperatively. Therefore, while the SPECT scan allowed the documentation of the extent of cerebral hypoperfusion and in some instances its response to embolization, it was a sensitive but nonspecific predictor of postoperative intracranial hypertension.

Management and Outcome of Patients with Intractable Intracranial Hypertension

Barbiturate therapy was required immediately postoperatively or on the first postoperative day in 7 of the 9 patients; it was required on the second postoperative day in the remaining 2 patients. Duration of barbiturate therapy was 2 to 5 days (mean, 3.2 days). There were no instances of uncontrollable ICP after institution of barbiturate therapy, as described in our protocol. One patient developed new contusional hemorrhages on the third postoperative day while in barbiturate-induced coma, but this did not adversely affect ICP control and did not result in focal mass effect. All patients developed pneumonia while in barbiturate-induced coma, despite vigorous antibiotic prophylaxis. Modification of the antibiotic regimen was performed after individualized culture results. There were no medical complications that resulted in permanent sequelae. Outcome at 2 months was good (no neurological deficit) or fair (minor non-
TABLE 2. Perfusion Single-Photon Emission-Computed Tomography Studies and Subsequent Postoperative Intractable Intracranial Hypertension

<table>
<thead>
<tr>
<th>Preembolization SPECT Studies</th>
<th>AVM Size, cm</th>
<th>Preoperative Embolization</th>
<th>Postoperative Intractable ICP</th>
</tr>
</thead>
<tbody>
<tr>
<td>No cerebral hypoperfusion</td>
<td>&lt;3</td>
<td>No 1 1 1 1 1</td>
<td>Yes 0 0 0 0 0</td>
</tr>
<tr>
<td>beyond AVM nidus (n=5)</td>
<td>3-6</td>
<td>Yes 1 3 4 0 0</td>
<td>Yes 0 0 0 0 0</td>
</tr>
<tr>
<td>One or more zones of cerebral hypoperfusion beyond AVM nidus (n=12)</td>
<td>3-6</td>
<td>Yes 1 3 4 0 0</td>
<td>Yes 0 0 0 0 0</td>
</tr>
</tbody>
</table>

SPECT indicates single-photon emission-computed tomography; AVM, arteriovenous malformation; and ICP, intracranial pressure.

Discussion

Postoperative Intracranial Hypertension

Postoperative intracranial hypertension may result from a variety of intraoperative or postoperative sequelae of resection of cerebral AVMs. Postoperative hemorrhage in the AVM bed has often been implicated, and this may be minimized by meticulous surgical technique, compulsive hemostasis in the AVM bed, and verification of complete AVM excision by intraoperative or early postoperative angiography. Neuroanesthetic technique, judicious control of arterial hypertension, and correction of coagulopathy may further decrease the prevalence of this complication. However, there remain instances of unexplained focal or global cerebral edema, hemorrhage in the AVM bed, and contusional hemorrhages in nearby or distant brain substance, all of which may create a situation of uncontrollable intracranial hypertension. Controversy as to the etiology of this complication remains. It has been postulated that in at least some cases the complication may be related to redistribution of parenchymal blood flow resulting from elimination of the arteriovenous shunt.1 In other instances there may be a problem of venous outflow occlusion.42 Similar focal hyperemic complications have been described after extraoperative embolization of AVMs.6,11,30-32 Undoubtedly, the trauma of surgery may further exacerbate this phenomenon.

References

1, 4, 5, 7, 8, 14, 16, 17, 20-22, 28.

References 2-4, 10, 12, 15, 18, 23-26, 41.

Disabling neurological deficit) in 8 of the 9 patients. The ninth patient recovered to the same level of preexisting hemiparesis and dysphasia that had resulted from a previous hemorrhage. The outcome of the remaining 23 patients who did not manifest uncontrollable ICP elevations was good in 22 patients and fair in 1. There were no instances of new disabling neurological deficit or mortality in this series.

Fig 4. Left frontal giant arteriovenous malformation (AVM) in distal border-zone location. Left, Preoperative angiogram; carotid injection anteroposterior view. Note the tortuous feeders from the middle cerebral and anterior cerebral arteries and the giant nidus in a distal border-zone location. Right, Preembolization perfusion single-photon emission-computed tomography (SPECT) scan (left) revealing areas of parenchymal hypoperfusion beyond the AVM nidus in the same hemisphere (closed arrow) and in the contralateral hemisphere (open arrow). A postembolization preresection repeat perfusion SPECT scan (right) reveals normalization of hypoperfusion in the contralateral hemisphere (resolution of interhemispheric steal) and improvement but persistence of ipsilateral parenchymal hypoperfusion (closed arrow) (persistence of intrahemispheric steal). This patient did not develop postoperative uncontrollable intracranial hypertension.
Numerous investigators have correlated the prevalence of postoperative breakthrough complications with AVM size, location, and hemodynamic features. These factors are not totally independent. Our results are consistent with previous reports and confirm a greater prevalence of this complication in larger AVMs and in AVMs in distal border-zone locations or in locations deriving direct (in parallel) feeders from basal or sylvian vessels. These features might predispose to a greater degree of hemodynamic impact of the AVM on nearby brain.

In fact, larger AVMs have been shown to result in a greater degree of intrahemispheric and interhemispheric steal than smaller AVMs. Our own SPECT perfusion studies are in agreement with these findings. SPECT studies represented a sensitive index of hemodynamic features that might predispose to postoperative intracranial hypertension. However, it is not clear whether SPECT perfusion scanning provides additional information in this regard beyond AVM size and location. It is also not clear whether SPECT scans predict postoperative hyperemic complications or merely indicate which lesions may be at risk for such complications. Our preliminary experience with this modality indicates that a negative SPECT scan may be a reliable predictor of favorable control of ICP postoperatively (within the limits of the management protocol adopted at our center). However, an abnormal SPECT scan does not necessarily predict uncontrollable ICP.

While SPECT abnormalities tended to normalize with serial embolizations, this did not always occur and did not predict or exclude subsequent postoperative intracranial hypertension. There may have been situations in which embolization caused additional infarcts or hypoperfusion in nearby brain from embolization itself; these may have obscured any normalization of hypoperfusion on SPECT resulting from decrease of steal through the AVM nidus. In other instances, normalization of SPECT hypoperfusion did not prevent postoperative intractable ICP. Further information is needed to address these issues and to further document the role of SPECT scanning in AVM management.

**Prevalence and Management of Postoperative Intracranial Hypertension**

Postoperative intracranial hypertension is an undesirable consequence of AVM resection. It may reflect complications of surgery, including hemorrhage and brain swelling, and can result in compromised cerebral perfusion. Numerous anecdotal reports have associated this complication with a poor outcome. However, postoperative intracranial hypertension does not reflect a single pathophysiological phenomenon. Because of this, we adopted a multifaceted strategy aimed at the prevention, early detection, and aggressive treatment of this complication. Our protocol included consideration of preparatory embolization whenever possible and staging of embolization sessions so as to allow a period of cerebral hemodynamic adjustment before final resection of the AVM. During and after surgical excision, we aimed at strict control of systemic arterial pressure, judicious correction of coagulopathies, and verification of complete excision of the AVM nidus.

Despite these measures and meticulous surgical and neuroanesthetic techniques, many patients exhibited undesirable ICP elevations in the postoperative period. In a fraction of these cases, ICP elevations were associated with impaired level of consciousness or other neurological deficits and were uncontrollable with somatic and/or loop diuretics and hyperventilation. This complication was more prevalent in subjects with AVMs with a profound hemodynamic impact on the brain because of size and/or location. The prevalence of postoperative intracranial hypertension in published series of subjects with AVMs cannot be ascertained in view of the variable case selection and inconsistent monitoring and management strategies. This information should allow better comparisons of management protocols in patients with similar lesions and a heightened clinical awareness about this potential complication in patients with AVMs of specific sizes and locations.

Anecdotal experience from our center and numerous published reports indicate that uncontrollable intracranial hypertension after AVM resection may be a catastrophic complication. This was associated with nearly all instances of severe complications after resection in every large series of AVM surgery. However, barbiturate therapy has been shown to be effective in other situations of intractable intracranial hypertension. Barbiturates have also been shown to be protective in certain situations involving focal cerebral ischemia and to result in metabolic and blood flow alterations that might be favorable in presumed postresection hyperemia. This therapy was only considered in cases in which other treatable causes of postresection intracranial hypertension were excluded and those in which more conventional therapeutic measures for control of ICP had failed.

**Management and Outcome of Barbiturate Therapy**

Barbiturate therapy was instituted only in situations involving truly intractable ICP and accompanying neurological deterioration despite all other more conventional interventions. The lowest barbiturate dosage was used to accomplish ICP control, under continuous EEG monitoring. Deeper barbiturate-induced coma beyond burst suppression (ie, isoelectric EEG) has been shown in numerous studies to be associated with greater therapeutic toxicity and no significant added benefit. This therapy was only considered in cases in which other treatable causes of postresection intracranial hypertension were excluded and those in which more conventional therapeutic measures for control of ICP had failed.

As in previous reports of barbiturate therapy, there were numerous medical complications, including pneumonia. The hemodynamic effects of barbiturate therapy assisted in maintaining systemic arterial pressure in the desired range, although a few patients required pressors. Meticulous protractive multisystem critical care management prevented permanent complications from the therapy. There was not a single instance of lasting morbidity attributed to barbiturate therapy in this series. The outcome of this management strategy was gratifying. All patients who did not develop uncontrollable ICP elevations did well. No patient with intracranial hypertension...
ble intracranial hypertension has died, and all but one (with preexisting major disability) have had a good or fair outcome. In the absence of a controlled trial, we cannot attribute any beneficial effect to barbiturate therapy alone. However, this multifaceted management strategy appears to have contributed to a favorable outcome in cases that were likely to have a dismal outcome, including cases of uncontrollable ICP elevations after resection of giant AVMs. It is possible that other anesthetic agents may have been equally effective, including isoflurane, etomidate, and other barbiturates. All these agents may result in EEG burst suppression, but they have been noted to have varying other metabolic and hemodynamic effects. There have not been any reports documenting the effectiveness of these agents in intractable intracranial hypertension and after resection of cerebral AVMs.

Conclusions
Postoperative intracranial hypertension is a prevalent complication after resection of cerebral AVMs. This complication is more common in larger AVMs and in lesions with more profound hemodynamic impact on nearby brain. Staged preparatory embolizations, meticulous microsurgical and anesthetic techniques, and verification of complete excision of the AVM nidus have not completely eliminated this complication. A strategy aimed at the rapid detection and aggressive treatment of elevated ICP appears desirable. Barbiturate therapy is a safe and effective modality that allows control of previously intractable intracranial hypertension in every case. Vigorous intensive care management is required during barbiturate therapy, including vigilant management of frequent medical complications. However, there were no instances of permanent medical morbidity attributed to this therapy. Management outcome was rewarding, including the absence of mortality or new disabling neurological morbidity in a series including a large number of complex lesions.

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