Endovascular procedures are rapidly expanding as treatment options for cerebrovascular diseases and neoplasms of the head and neck and are becoming less invasive but more effective. There are potentially dangerous anastomoses between the extracranial and intracranial circulations; hence, thorough knowledge of the anatomy is essential to minimize the risk of cranial nerve palsies, blindness, or neurologic deficits. It is essential to understand the scientific basis of treatment rationale based on advancing new neuroimaging techniques to better serve patients. An interdisciplinary approach and treatment in high-volume centers are vital to obtain maximal benefit for patients.

Endovascular therapy has continuously evolved and improved since it was first described in 1904. Rapid advances in the field have resulted in the ability to safely embolize and effectively treat craniocervical neoplasms and vascular malformations. Preoperative embolization of tumors reduces intraoperative blood loss, shortens surgical time, and decreases surgical morbidity. This technique involves superselective catheterization of the feeding arteries to the tumor bed with infusion of embolic particles to saturate the tumor bed in the hopes of inducing necrosis. For some malignant head and neck tumors, selective infusion of chemotherapeutic agents has been performed as part of combined therapy.

The endovascular therapy of brain arteriovenous malformations (AVMs), dural arteriovenous fistulas (DAVFs), carotico-cavernous fistulas, and vein of Galen malformations is part of the therapeutic strategy. This article presents the indications, techniques, results, and potential complications of endovascular embolization of craniocervical vascular malformations, and craniocervical tumors.

BRAIN ARTERIOVENOUS MALFORMATIONS

Indications for Treatment

Embolization of an AVM is mainly used as an adjunct to surgical treatment to reduce the size of an AVM or make it safer for surgery, although in some patients with small AVMs and a few arterial feeders, “angiographic cure” of an AVM may
be possible. With surgically inaccessible AVMs, embolization may be used to eliminate perinidal or flow-related aneurysms as a prelude to radiosurgery. With large AVMs that are not candidates for surgical treatment, embolization may be performed in stages to reduce the size of an AVM and render it suitable for radiosurgery, in a protocol-driven fashion (the efficacy of this modality is as yet unproven). Several centers, however, have reported good preliminary experience.

In general, any treatment of an AVM must offer patients a significant advantage over the natural history. In this regard, the combined mortality and morbidity of procedures used must be considered in making a treatment decision and discussing options with patients.

The authors classify AVMs as ruptured and unruptured, because the natural history is different. The rebleeding rate of a ruptured AVM is 6% for the first year and 2% to 4% per year thereafter, whereas the bleed rate of an unruptured AVM has been estimated from 2% to 4% (as low as 2% and as high as 10%). In addition, AVMs are generally classified according to Spetzler-Martin (S-M) grade (Table 1). Although controversial, especially in regards to grade 3, which encompasses a variety of AVMs, it is the most widely used grading system. In this regard, the authors believe that the size of an AVM and its location (perirolandic, posterior fossa, or deep) are the most important variables determining outcome. The role of embolization-related complications has been well correlated with increasing AVM grade. For S-M grades 1 and 2 AVMs, embolization may be offered to mark an AVM for a surgeon (the black-colored Onyx [eV3, Irvine, California] is a good marker) to occlude an important feeding vessel that is surgically less accessible, to occlude a perinidal aneurysm, or to attempt to cure an AVM endovascularly. With grades 3 and 4 AVMs, embolization is usually performed in a staged fashion to reduce the risk of postoperative normal pressure perfusion breakthrough bleeding and to make surgery easier. Grade 5 AVMs are operated only under exceptional circumstances. For grades 4 and 5 AVMs, embolization may be performed as a prelude to radiosurgical treatment.

With ruptured AVMs, the timing of embolization is a matter of variability and controversy. In the authors’ institution, patients with large clots are often operated emergently (if they are herniating and if an AVM is small) or early (within a few days). In such patients, embolization may be performed urgently if a surgeon desires. In other cases, embolization is started as soon as possible (without waiting for the clot to clear) and continued in stages (at approximately 2-week intervals) until the desired level of obliteration is needed.

### Technique of Embolization—Brain Arteriovenous Malformations

Many centers have reported the safety and efficacy of n-butyl cyanoacrylate (NBCA) and particle embolization for AVM treatment. Because of the well-documented handling characteristics of

<table>
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<tr>
<td>Spetzler-Martin grading score of AVM</td>
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<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Points</th>
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<tbody>
<tr>
<td><strong>Size of lesion</strong></td>
<td></td>
</tr>
<tr>
<td>Small (&lt;3 cm)</td>
<td>1</td>
</tr>
<tr>
<td>Medium (3–6 cm)</td>
<td>2</td>
</tr>
<tr>
<td>Large (&gt;6 cm)</td>
<td>3</td>
</tr>
<tr>
<td><strong>Location</strong></td>
<td></td>
</tr>
<tr>
<td>Non eloquent</td>
<td>0</td>
</tr>
<tr>
<td>Eloquent site(^a)</td>
<td>1</td>
</tr>
<tr>
<td><strong>Pattern of venous drainage</strong></td>
<td></td>
</tr>
<tr>
<td>Superficial only</td>
<td>0</td>
</tr>
<tr>
<td>Any deep</td>
<td>1</td>
</tr>
</tbody>
</table>

Based on S-M grading system estimating the risk of surgery, which consists of three elements: size, venous drainage, and location. From Spetzler RF, Martin NA. A proposed grading system for arteriovenous malformations. J Neurosurg 1986; 65(4):476–83; with permission.

\(^a\) Sensorimotor, language, or visual cortex; hypothalamus or thalamus; internal capsule; brain stem; cerebellar peduncles or cerebellar nuclei.
Onyx, however, the authors’ institutional experience focuses primarily on Onyx embolization.

The majority of brain AVMs are now embolized with the liquid embolic agent, Onyx, in two viscous concentrations, Onyx-34 or Onyx-18. All embolization procedures are performed with patients under general anesthesia in a biplane angiographic unit. Systolic blood pressure during the procedure is controlled at less than 120 mm Hg. Post embolization, the blood pressure is kept 10% lower than the baseline systolic pressure in children or less than 110 to 120 mm Hg in adults after the procedure for 24 hours. After preparing and draping the groins in a sterile fashion, the femoral artery is accessed by an 18-gauge needle and, using the Seldinger technique, a 6-French (Fr) femoral sheath is inserted into the vessel and secured. Diagnostic angiography is then performed using a 4-Fr vertebral catheter with views of the intracranial (IC) vessels in various projections, including 3-D views, to delineate all feeders to an AVM and understand the dynamics of blood supply and venous drainage pattern. In some AVMs with rapid shunts, filming at high speed (6 frames/s) may be required. Intra- and perinidal aneurysms are noted.

Patients are heparinized before embolization, usually with 5000 U intravenously, and 1000 U per hour are repeated as needed to maintain the activated clotting time at 300 seconds (50 mg/kg in children). The heparin is not reversed at the end of the procedure unless there is intraoperative perforation of the vessel or rupture of AVM. The guiding catheter is constantly flushed via a pressure bag with saline, which does not contain any heparin.

The first vessel chosen for embolization is usually a direct feeder to the AVM (not feeding through collaterals and not an en passage vessel) with at least 2 cm of safe distance available for reflux before cortical branches are reached (Figs. 1 and 2A). A dimethyl sulfoxide (DMSO)-compatible microcatheter (Marathon 1.3-Fr eV3) is navigated into the artery close to the nidus of the AVM with aid of a 0.008-in micro–guide wire (Mirage, eV3); multiple reshapes of guide wire may be needed to reach the target site. In some cases, when an artery is large, a catheter may be able to be advanced along the vessel close to the AVM with the wire retracted inside, to avoid perforation of a thin-walled artery. It is important to have the microcatheter tip as close to the nidus as possible and to have a good distance between the catheter tip and any normal branch proximal to it (safe reflux distance). The safe reflux distance ideally should be at least 2 cm and clearly noted in the roadmaps before embolization. The micro–guide wire is removed and biplane superselective angiography is performed through microcatheter to analyze the anatomy of the nidus segment and verify that only the AVM nidus is filled. During embolization, good angiographic views of AVM in two projections should be shown on the screen to see the tip of the microcatheter clearly, to appreciate the reflux

![Fig. 1. Various types of feeder vessels to the AVM. (A) En passage vessel giving branches on its way to the cortex. (B) Direct feeder to the AVM nidus. (C) Network of enlarged collaterals supplying the AVM.](image-url)
readily, and to appreciate the anatomy of the AVM and the surrounding brain. Once embolization from this position is decided on, the microcatheter is over flushed with 10 mL of normal saline to clear any remaining contrast, then filled with 0.23 mL DMSO, and the catheter hub is also bathed in DMSO to clear the saline, because Onyx precipitates in contact with saline. Onyx is aspirated into a 1-mL syringe and a connection is made to quickly form a meniscus between the Onyx and DMSO. Onyx is injected slowly at a flow rate of 0.1 mL per minute to fill the microcatheter and replace the DMSO in the dead space. Embolization is usually started with Onyx-34 (twice as viscous as Onyx-18) in order to form a plug around the catheter tip. Free flow of the Onyx is noted into the AVM nidus until there is a reflux noted. Embolization is performed with a gentle and steady push, or gentle small puffs, with progress viewed all the time. In small AVMs, significant penetration may occur with the first push, before reflux occurs, so care should be exercised. When reflux is noted, the operator stops for varying periods of time (30 seconds to 1 minute) before restarting. After first reflux, anywhere from 2 to 10 minutes may be needed to form an adequate plug before the second forward penetration occurs again. As this point is about to be reached, forward and retrograde (bidirectional) flow often is observed.

Progress of embolization is followed by noting single radiographic exposures of the Onyx cast (and comparing with the AVM images) and intermittent angiographic runs to evaluate the AVM filling (Figs. 3–10). For each embolization sequence, a blank road map is obtained. Avoiding venous embolization is important. When passing through a vein, Onyx tends to laminate initially along the venous wall or quickly pass through to the venous sinus. During embolization, filling of the veins is noted by streaking or pooling, which is an indication to stop for up to 1 minute. The operator must have a good understanding of where the veins are inside the AVM and in its periphery to identify and avoid venous embolization. Reflux into normal brain areas around the AVM perimeter is an indication to stop for 30 seconds. The maximal safe reflux distance along the feeding vessel should be judged on the initial angiogram (usually no more than 2 cm back or 1 cm distal to a cortical branch of the feeding artery) and the fluoroscopy tube is appropriately positioned during embolization to judge this rapidly, because the embolic agent filling the AVM may interfere with the initial views. After the initial embolization epochs, with the plug well formed, the operator may switch to Onyx-18, which is less viscous and penetrates the AVM more easily.

**Endpoint of embolization**

Operators should use judgment and experience to end an embolization session. With small AVMs, the entire AVM or the majority of the AVM is embolized in one session. In such cases, the endpoint is reached when the major veins are filling (Fig. 2B). One should not try to achieve a perfect result with the sacrifice of safety, because this is when complications occur.

With large and giant AVMs, embolization is usually done in stages, with approximately 25% to 40% of the AVM embolized in the first session. The endpoint of an embolization session is reached when, after repeated pushes of embolic agent, no significant progress is noted. The considerations are to embolize larger AVMs (>3 cm) in a staged fashion to achieve maximal but safe reduction of the AVM volume and to embolize areas that are surgically difficult. The operator starts with direct feeders to the AVM (not en
passage feeders) and progresses to more surgically difficult areas. During subsequent sessions, the goal of an embolization should be to embolize regions that are more difficult for surgery (e.g., the more posterior region in a perimotor AVM, the part supplied by the PCA branches in a parieto-occipital AVM, or the portion supplied by ACA feeders in a medial frontal AVM). With large AVMs, 2 to 3 days before surgical excision, a larger volume of the AVM may be embolized, but patients must remain in the hospital with strict control of the blood pressure until surgical resection.

Although it is preferable to embolize the apical portions of the AVM to make surgical excision

Fig. 3. (A) MRI scan showing a 7-cm AVM in the frontal lobe bordering the sensorimotor cortex. (B) Angiograms showing a S-M grade V AVM. (C, D) Angiograms showing Onyx cast obliterating the posterior portion of the AVM bordering the rolandic fissure after four stages of Onyx embolization in 3 weeks. (E, F) Plain skull radiographs show the embolized AVM.
easier, this is often difficult, in the authors’ experience. As much as possible, intra- and perinidal aneurysms must be obliterated in the first embolization session, especially in cases of ruptured AVMs. An embolization session is stopped when (1) the desired volume of embolization has been reached, (2) there is repeated opacification of draining veins, (3) the maximum safe distance for reflux is exceeded, or (4) despite a moderate injection pressure, there is no forward flow of Onyx.21

Management of vessel perforation
During initial navigation of the microcatheter, penetration of the artery may rarely occur by guide wire or catheter. It can be recognized by an unexplained position (relative to the arterial anatomy) of the wire or catheter or, in extreme cases, by changes in a patient’s vital signs. It can be confirmed by a small-volume contrast injection through a microcatheter or by angiography through a guiding catheter. Once this is recognized, the catheter (or wire) must be left in place while the heparin is reversed with protamine. After reversal, while the microcatheter is withdrawn, a small (0.1 to 0.2 mL) volume of Onyx is injected into the artery to seal the perforation. Further embolization may be done through another vessel if the feeding artery is well sealed, the

![Fig. 4. Angiograms at 9 months showing complete removal of the AVM.](image)

![Fig. 5. MRI scans showing an AVM in the right sylvian fissure and frontal lobe. (A) Axial; (B) Coronal.](image)
extravasation is minimal, and there are no changes in vital signs in a case of ruptured AVMs, or deferred to another session in cases of large extravasations, unstable vital signs, or unruptured AVMs. During embolization, extravasation of Onyx also may occur from the feeding artery, one of the draining veins, or inside the AVM nidus. This can be recognized by pooling and is an indication to stop the embolization.

**Microcatheter withdrawal**
At the end of the procedure, after aspirating with the Onyx syringe, the microcatheter is pulled back slowly, increasing the tension on the tip during withdrawal, holding the tension for a few seconds, and repeating this maneuver a few times until the microcatheter pulls out of the cast of Onyx around it. During retrieval, protamine is kept ready for reversal of anticoagulation should an accidental rupture occur, but the authors have never had to use it in their series of patients. Immediately after retrieval of the microcatheter, the position of the guiding catheter must be checked because it can easily migrate cranially. The guiding catheter should be thoroughly aspirated to remove any clots before performing post procedural angiography.

**Postoperative care of patients**
Patients are monitored in an ICU, and the blood pressure is kept 10% lower than the baseline systolic pressure in children or less than 120 mm Hg in adults for 24 hours to minimize the risk of postembolization hemorrhage. Femoral sheaths
are usually removed on the following day and most patients are discharged home after 48 hours.

**Hemorrhage from arteriovenous malformations during or after embolization and management**

Hemorrhagic complications are devastating and probably occur due to inadvertent occlusion of the venous outflow during embolization without elimination of the arteriovenous shunt. Congestion of the nidus with slow-standing dye might be observed. Embolization of multiple pedicles at a single setting may result in too rapid an alteration of the hemodynamic of the AVM and may result in subsequent hemorrhage. This can be avoided by staged embolization. Finally, catheter/wire manipulation may cause vessel perforation resulting in intracerebral hemorrhage (ICH) or subarachnoid hemorrhage (SAH). This can be minimized by limiting the use of guide wire to selecting the desired proximal pedicle while letting the natural flow characteristics of the AVM carry the pliable flow-directed microcatheter to the distal position for embolization. Immediate reversal of heparin with protamine and termination of procedure is required in arterial perforation with unstable parameters.

**Arteriovenous fistulae**

With very high-flow AVMs and arteriovenous fistulae, it may be necessary to use a larger Onyx-compatible microcatheter (Echelon 10 or Echelon 14, eV3) to allow the placement of coils into the vessel to arrest or slow the flow. This may be followed up with the liquid embolic agent. During such procedures, significant hypotension (down to 60-mm systolic blood pressure) or adenosine-induced brief cardiac arrest may be used. In

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Fig. 7. Angiograms (A, B) and plain skull radiograph (C) showing the obliteration of the medial portion of the AVM by Onyx cast.
such cases, monitoring of somatosensory evoked potentials (SSEP) and motor evoked potentials (MEP) is helpful.

Clinical Experience and Outcome

In a series of patients treated by the authors’ team from August 2005 to October 2008 at their institution, a total of 128 patients were treated definitively, 4 by embolization alone, 60 by embolization and microsurgery, 14 by embolization and radiosurgery, 7 by all three modalities, and 43 by only surgical removal or radiosurgery.

From the total of 128 patients, 60 presented with rupture of the AVM and ICH or SAH. In a subset of 75 patients treated with embolization, complications were noted in 10 (Table 2). One patient developed left-sided hemiplegia 8 hours after embolization of a S-M grade V AVM in the occipitotemporal region through feeders from the anterior choroidal artery. She had an infarct in the right occipitoparietal and thalamic region on MRI. The AVM was surgically removed, but the patient remains disabled (modified Rankin score [mRS] 3). A second patient had an arterial perforation by the micro–guide wire that was managed by immediate reversal of heparin with protamine and termination of the procedure. She was successfully embolized the next day and had complete microsurgical resection after 2 more days. She had no neurologic deficits. Two patients had stuck microcatheters after embolizations that were left in situ; they were identified intraoperatively and extracted in both cases. There were two deaths of patients who did not recover after their bleed, unrelated to the treatment.

At 3 months, 63% recovered with no or mild disability. The outcome as measured by mRSs were slightly better in the radiosurgery series than in the microsurgery series in AVMS with S-M grades I to III but this difference was not significant ($P = .583$). One patient from the radiosurgery group had recurrent bleed in the interim period (Table 3).

Case Examples of Embolized Arteriovenous Malformations

Patient 1

An 8-year-old girl presented with recurrent tingling sensations on the left side of the body and weakness of the left arm and leg. She was found to have a S-M grade V AVM measuring $7.0 \times 4.5 \times 2.8$ cm in size, involving the motor area. Embolization was aimed at the apex and the posterior part bordering the sensorimotor cortex parts of the AVM that were more difficult to manage with surgery. Onyx embolization was performed in four sessions over 3 weeks. The posterior portion of AVM bordering the Rolandic fissure was embolized, but the apex could not be embolized. Surgical resection was elected because of her young age, which creates a greater potential for cerebral plasticity because she was likely to compensate for any deficits sustained at the time. The AVM was excised completely 4 days after the last stage of embolization. Postoperatively, she had increased weakness that gradually improved. At follow-up 3 years later, she had normal school and intellectual performance but a slight weakness of her left upper extremity with
mRS 1, and angiography showed complete removal of the AVM.

**Patient 2**
A 52-year-old woman presented with recurrent severe headaches. She was found to have a S-M grade IV AVM in the right sylvian fissure, the basal ganglia, and the frontal lobe (see Figs. 5 and 6). Embolization was performed with Onyx in four sessions over 4 weeks, aiming at the medial portion of the AVM in the basal ganglia (see Fig. 7). Surgery was performed 2 days after the last embolization, and the AVM was removed completely. The patient had left hemiparesis postoperatively and had inpatient rehabilitation. At a follow-up examination 8 months after surgery, power in all four limbs was normal, and she had returned to her normal activities and work (mRS 1). Angiography 8 months after surgery showed complete obliteration.

**Patient 3**
A 22-year-old man presented with seizures. On imaging, he was found to have left frontal S-M
grade 3 AVM in the premotor area. He was subjected to two sessions of embolization with Onyx followed by surgical resection. Patient had a complete recovery with no symptoms at follow-up (mRS 0).

**Patient 4**
A 39-year-old man presented with intraventricular hemorrhage and on imaging was found to have thalamic AVM. The source of hemorrhage was a small perinidal aneurysm, which was then embolized to eliminate immediate rebleeding risk. He was subsequently referred for radiosurgery and patient is being followed up.

**Patient 5**
A 52-year-old man developed severe headache secondary to SAH with left cerebellar intraparenchymal hemorrhage due to AVM rupture. Angiography demonstrated a left cerebellar complex, racemose hemispheric arteriovenous malformation, predominantly fed by branches of the left posterior inferior cerebellar artery (PICA) with a perinidal aneurysm on the superior branch of the left PICA feeding into the AVM. Because of this, there was high risk of immediate rebleed and patient was planned for immediate embolization. He underwent preoperative embolization in two stages followed by surgical resection of the AVM and clipping of the superior cerebellar artery (SCA) aneurysm. Patient had a good recovery and was mRS 2 at follow-up.

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**DURAL ARTERIOVENOUS FISTULAS**
IC DAVFs are acquired abnormal arteriovenous connections within the dura that account for 10% to 15% of all IC AVMs (Figs. 11–23). Most lesions may develop as a consequence of venous sinus thrombosis and the attempted recanalization but some congenital DAVFs have also been described. There is a female preponderance with symptoms usually developing during middle to late adulthood. A presentation with aggressive neurologic symptoms is more common in men.

Previous surgery, ear infection, and head trauma have all been cited as potential causes, although the common predisposing factor seems to be venous sinus thrombosis. Venous thrombosis promotes venous hypertension, which acts as the initiating factor opening up microscopic vascular connections within the dura. Maturation of these channels secondary to progressive venous stenosis or occlusion results in the development of direct shunts between the arteries and dural veins. In addition, a second complementary mechanism of DAVF evolution may occur with the release of angiogenic growth factors, such as vascular endothelial growth factor (VEGF) and basic fibroblast growth factor, promoting neovascularization and development of a DAVF. Lasjaunias and Berenstein have suggested “an underlying dural weakness” facilitating dural shunts in some individuals. In children, DAVFs may occur as congenital lesions, with
a presumed cause of venous thrombosis in fetal life or abnormal development.

**Symptoms**

DAVFs may be asymptomatic or present with symptoms that are mild to severe. Severe symptoms include ICH, visual loss, focal neurologic deficits, dementia, and eye pain or diplopia secondary to venous hypertension. Minor symptoms may include headache, tinnitus, dizziness, trigeminal neuralgia, hemifacial spasm, and so forth. Drainage of a petrous region DAVF to the transverse or sigmoid sinus commonly produces pulsatile tinnitus or hearing loss, sometimes in association with an audible bruit. Other cranial nerve (CN) symptoms (trigeminal neuralgia and hemifacial spasm) may be caused due to compression by an enlarged artery or draining vein. Cavernous sinus DAVFs may develop orbital signs, such as congestion, chemosis, and ophthalmoplegia. More aggressive behavior may manifest as focal neurologic deficits, dizziness, a dementia-type syndrome or cerebral hemorrhage, including subarachnoid, subdural, or intraparenchymal bleeds. Such features are usually considered due to venous hypertension.

The most common location of cranial DAVF is transverse-sigmoid sinus followed by the cavernous sinus. In a meta-analysis of 360 DAVFs, the tentorial incisura was the most ominous location, with 31 of 32 cases associated with hemorrhagic or nonhemorrhagic stroke. The venous drainage pattern is the most important predictor of the clinical behavior, however, and those with retrograde leptomeningeal drainage

<table>
<thead>
<tr>
<th>Embolization</th>
<th>No. of Patients</th>
</tr>
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<tbody>
<tr>
<td>Thromboembolic event leading to hemiplegia</td>
<td>1</td>
</tr>
<tr>
<td>Vessel perforation</td>
<td>1</td>
</tr>
<tr>
<td>Infection</td>
<td>1</td>
</tr>
<tr>
<td>Vein of Labbé stenosis</td>
<td>1</td>
</tr>
<tr>
<td>Stuck catheter</td>
<td>2</td>
</tr>
<tr>
<td>Failed catheterization</td>
<td>3</td>
</tr>
</tbody>
</table>

Table 2

<table>
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<th>Complications of embolization</th>
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<td>Embolization</td>
</tr>
<tr>
<td>Thromboembolic event leading to hemiplegia</td>
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<td>Vessel perforation</td>
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<td>Infection</td>
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<td>Stuck catheter</td>
</tr>
<tr>
<td>Failed catheterization</td>
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Table 3

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<th>Modified Rankin score outcome based on modality</th>
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<tr>
<td>Modified Rankin Score</td>
</tr>
<tr>
<td>-----------------------</td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td>0. No symptoms at all</td>
</tr>
<tr>
<td>1. No significant disability despite symptoms; able to carry out all usual duties and activities</td>
</tr>
<tr>
<td>2. Slight disability; unable to carry out all previous activities, but able to look after own affairs without assistance</td>
</tr>
<tr>
<td>3. Moderate disability walks without assistance</td>
</tr>
<tr>
<td>4. Moderately severe disability; unable to walk without assistance and unable to attend to own bodily needs without assistance</td>
</tr>
<tr>
<td>5. Severe disability requiring care</td>
</tr>
<tr>
<td>6. Dead</td>
</tr>
</tbody>
</table>
exhibit a much higher incidence of hemorrhage or venous infarction. The annual mortality rate for a DAVF with cortical venous reflux (CVR) may be as high as 10.4%, whereas the annual risks for hemorrhage or nonhemorrhagic neurologic deficits during follow-up are 8.1% and 6.9%, respectively, resulting in an annual event rate of 15%. Moreover, rebleeding rates may be as high as 35% over the first 2 weeks after the initial hemorrhage.37,38 Thus, these formidable lesions need careful evaluation and rapid treatment.

The preliminary diagnosis of DAVF is based on clinical presentation. After initial suspicion, CT and CT angiography or magnetic resonance angiography may be performed. Angiography is needed for definitive diagnosis. The projections

Fig. 11. After two stages of embolization, more than 70% obliteration of the AVM with only a small residual portion seen inferiorly (arrows). (A) Anteroposterior view, left ICA injection; (B) Lateral view, left ICA injection.

Fig. 12. Postoperative angiography shows complete resection of AVM with no residual AVM nidus seen. (A) Anteroposterior view; (B) Lateral view.
must include bilateral external carotid artery (ECA) runs, internal carotid arteries (ICAs), and both vertebral arteries (VAs) if necessary. For lesions around the foramen magnum, upper cervical area, thyrocervical, and ascending cervical arteries should be studied after an initial subclavian injection.

The Borden system of classification is commonly followed for classification and treatment indication, but the Cognard system is more detailed (Tables 4 and 5).

**Treatment Philosophy and Algorithm**

The primary goal of treatment is the complete obliteration of the lesion. In some cases, however, palliative treatment for control of symptoms may be performed. The therapeutic armamentarium includes conservative monitoring, arterial embolization, transvenous occlusion, surgical excision, and radiation therapy. In current practice, many lesions can be eliminated by an endovascular technique. The main goal of endovascular treatment of aggressive DAVFs is to eliminate the cortical venous drainage and the resulting risk of ICH. Different endovascular approaches can be used in the treatment of these lesions.

In Borden 1 cases, treatment is needed only for severely symptomatic cases, and palliative (incomplete treatment) is an option. The aim of treatment in such lesions is cure, but palliation can be accepted to reduce the morbidity. In Borden 2 and 3 cases, treatment is mandatory, and when a patient presents after an acute ICH,
treatment should be provided as soon as possible, to prevent the prospect of rebleeds. The goal is to occlude the fistula in all DAVFs, and especially to occlude the origin of the draining vein to prevent recurrence in Borden 3 fistulas. When endovascular treatment is difficult or has failed, then microsurgery is done as an alternative.39,40 In some difficult cases, radiosurgery may follow the embolization procedure. In the authors’ center, transarterial embolization with Onyx is used as the primary treatment modality unless there are arterial branches supplying CNs, predominant ICA feeders, or potential extracranial (EC)-IC collateral anastomosis. The transvenous technique is used in cases of type 2 DAVFs where transarterial access is not possible or where combined transarterial and transvenous treatment is needed. The goal of transvenous access is to enter the fistula pouch from the venous side for coil embolization at the point of the fistula itself. In some cases of Borden 3 fistulas, the fistula can be accessed through the transvenous route.41 Surgical treatment is reserved in the authors’ center for emergent clot evacuation and for cases where endovascular treatment is not possible or incomplete. A complete devascularization of the fistula or the occlusion of all the draining veins/vein of the fistula must be feasible for surgery to be successful.41 Radiosurgery is performed as a last resort, for some cases of fistulae that cannot be optimally treated by one of the techniques described previously, usually after embolization or surgical resection. Radiosurgery takes 2 to 3 years to obliterate the DAVFs and, until the lesion is completely obliterated, there is still a risk of bleeding or other symptoms.42,43

Fig.14. (A) Carotid angiogram; reduction in the size of AVM and presence of Onyx cast (white arrow) post embolization. (B) CT angiogram; (C) CT scan without contrast.
Case Examples of Embolization Technique for Dural Arteriovenous Fistulas

Case 1: transvenous embolization of an indirect carotid-cavernous fistula

A 49-year-old man presented with headache and proptosis and was found to have an indirect carotid cavernous fistula (Borden 2) with bilateral meningohypophyseal trunk (MHT) feeders and left ECA feeders draining into the cavernous sinus, superior ophthalmic vein, and superior petrosal sinus. Complete obliteration of the fistula and relief of all symptoms was achieved after transvenous coil embolization of the cavernous sinus through the left superior petrosal sinus. This procedure was under general anesthesia, a 6-Fr sheath was placed in the femoral artery, and angiography was performed as needed during the procedure, using a 4-Fr vertebral catheter. The femoral vein was punctured, a 6-Fr sheath inserted, and a 6-Fr main pancreatic duct catheter negotiated into the internal jugular vein. An Excelsior SL-10 microcatheter (Boston Scientific, Natick, MA) with a Synchro 14 microwire (Target Therapeutics/Boston Scientific, Fremont, California) was then used to superselectively catheterize the left superior petrosal sinus and ultimately the left cavernous sinus. The catheter was positioned anteriorly at the junction of the left ophthalmic vein and multiple entering arterial fistula pedicles and multiple guglielmi base detachable (GDC) 360 platinum coils were deployed within the anterior left cavernous sinus. Occlusion of the fistula was confirmed by transarterial angiography.

Fig.15. S-M Grade 3 AVM within the left cerebellar hemisphere with arterial feeders from left AICA/PICA trunk, with small feeding branches from left superior cerebellar, and posterior cerebral arteries (A, B). Multiple small feeding artery perinidal aneurysms located close to the AVM nidus posteriorly (C).
The cavernous sinus is usually entered by navigating a microcatheter through the inferior or superior petrosal sinus. Rarely, when such access is not possible, and when there is predominant anterior drainage, catheterization can be via the superior ophthalmic vein or a facial vein. The superior ophthalmic vein can be exposed by a cut-down technique.

**Case 2: transarterial Onyx embolization of convexity dural arteriovenous fistula (Borden 3)**

A 60-year-old man presented with headache and was found to have an ICH. Cerebral angiogram showed a convexity osteo-DAVF with feeders from bilateral occipital artery, middle meningeal trunk, and right posterior cerebral artery. The venous drainage was predominantly into meningeal and cortical vein and into the middle third of superior sagittal sinus. There was partial narrowing of the superior sagittal sinus and with venous congestion on bilateral ICA injections. Transfemoral access was achieved using a 6-Fr sheath under anesthesia. Superselective catheterization of the ECA was done using a 5-Fr Envoy catheter advanced over a 0.035 in glidewire. Using road mapping guidance, a Marathon 1.3-Fr microcatheter was advanced over a Mirage 0.008 microwire, and transarterial Onyx embolization was performed in three sessions through bilateral posterior branches of middle meningeal trunks and right occipital artery. There were no complications or CN palsies and the fistula was completely obliterated.

![Fig. 16](image-url) *(A) Postembolization showing significant reduction in the size of AVM. Postoperative angiography shows a complete resection with aneurysm clip seen in situ (arrow) (B, C).*
Fig. 17. Cerebral angiogram showing an indirect carotid-cavernous fistula (Borden type 2) with bilateral MHT feeders (A, B) and left ECA feeders (C, D) draining into the cavernous sinus, superior ophthalmic vein and superior petrosal sinus (arrow).

Fig. 18. (A) Tranvenous access to the fistula site through the left superior petrosal sinus. (B) Postembolization cerebral angiogram showing complete obliteration of the fistula.
Case 3: Onyx embolization of tentorial arteriovenous fistula (Borden 3)
An 85-year-old woman presented with headache and was found to have a posterior fossa hemorrhage on CT scan. Cerebral angiogram showed a tentorial DAVF supplied by feeders from bilateral occipital artery and left posterior meningeal branches. The venous drainage was into a tentorial vein that had a venous aneurysm and drained into the transverse sinus. A 6-Fr KSAW Shuttle-Select Fig. 19. (A) Noncontrast head CT showing an intracerebral hemorrhage. Cerebral angiogram showing a convexity osteo-DAVF with feeders from bilateral occipital artery, middle meningeal trunk (B, C) and right posterior cerebral artery (D). Venous phase of cerebral angiogram showing partial narrowing of the superior sagittal sinus and with venous congestion on bilateral ICA injections (E, F).
(Cook, Bloomington, Indiana) introducer sheath was placed in the left ICA and using a Echelon 10 and Mirage 0.008 inch guide wire, complete obliteration of the fistula was achieved after transarterial embolization with Onyx-18 through the dural branch of the occipital artery. The catheter was stuck in the occipital artery, broke at a point 10 cm from the tip, and was deposited in

Fig. 20. (A) Postembolization angiogram showing Onyx cast (arrow) and (B) complete obliteration of the fistula.

Fig. 21. (A) Noncontrast head CT showing posterior fossa hemorrhage. Cerebral angiogram showing a tentorial DAVF supplied by feeders from bilateral occipital artery (B, D) and left posterior meningeal branches. The venous drainage was into a tentorial vein that had a venous aneurysm and drained into the transverse sinus (C).
the occipital artery after cutting the catheter just under the femoral skin edge.

Results and Complications

In the authors’ institution, 32 patients with DAVF (Table 6) were treated over 3.5 years between May 2005 and December 2008 by endovascular embolization (transarterial embolization with Onyx, transvenous embolization GDC coils), surgery, or radiosurgery. Most common presentation was hemorrhage (n = 11), headaches, tinnitus, and orbital symptoms. Twenty-eight patients were treated by endovascular embolization. Five patients (after incomplete-1 patients/failed-4 patients embolization) had surgical excision of the fistula. Three patients were treated with gamma knife radiosurgery after partial obliteration of the fistula with Onyx.

The distribution of patients according to Borden classification was I, 6 (18.8%) patients; II, 13 (40.6%) patients; and III, 13 (40.6%) patients.

Twenty-six of 32 (81%) patients had their fistulas completely obliterated after multimodality treatments at 1 to 36 months’ follow up. Of the seven patients with residual after endovascular embolization, four patients had surgical obliteration of the fistula and three underwent gamma knife radiosurgery. Surgical complications included cognitive deficits with word-finding difficulty in one and seizures in another. At follow-up (3–36 months), 15 patients had mRS 0–2, four patients had mRS 3–5 (three due to the initial insult of the hemorrhage), and two patients were dead. One patient with a tentorial DAVF was attempted to be embolized by a transvenous technique, but the microwire was stuck to the vein during attempted extraction and caused its rupture resulting in massive hemorrhage and death. The other patient died after an accidental fall and consequent subdural hematoma (Table 7).

Transarterial embolization with Onyx has increased obliteration rates with low morbidity.
Surgical obliteration of the draining vein remains an important treatment option for fistulas not accessible through the endovascular route or after failed endovascular treatment.

**EMBOLIZATION OF CRANIOCERVICAL TUMORS**

Tumors of the craniocervical area are embolized mainly to make surgical resection easier, by reducing the bleeding during surgery, and sometimes, also by making them softer by inducing intratumoral necrosis (Figs. 24–34). Rarely, embolization can be used as a palliative modality to cause symptomatic improvement.²,⁴⁴–⁴⁶ Because embolization is part of the multimodal treatment, it should be done with minimal risk, avoiding new CN deficits, stroke, or hemorrhage. Adequate devascularization of a tumor can rarely be produced without embolizing all the important feeders and the nidal portions of the tumor due to the presence of intratumoral collateral circulation. The vascularity of the tumor is often multicompartmental, as it is supplied by different arteries, but the intratumoral vessels do communicate with each other. At the conclusion of embolization, the feeding arteries may be occluded close to the tumor.⁴⁷ Tumors that are considered for embolization are usually large and extra-axial, although embolization also may be performed of intra-axial tumors, such as hemangioblastoma, choroid plexus papilloma, or intraventricular meningioma, when the feeding arteries can be safely accessed. Extra-axial tumors considered for embolization can be divided into (1) convexity and parasagittal and (2) skull base tumors, the latter being technically more difficult.³⁴ In addition to intratumoral embolization, an endovascular surgeon may be asked to assess the risk of arterial sacrifice before or during surgery by the performance of a carotid occlusion test. Occlusion testing of a venous sinus is not safe and has been accompanied by a disastrous

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<td><strong>The Borden classification system</strong></td>
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<td>3</td>
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complication in the senior author’s experience (L.N. Sekhar, MD, unpublished data, 1992).

As with embolization of AVMs, endovascular surgeons and microsurgeons must communicate effectively to understand the goals of the embolization and the permissible risk before the procedure.

**Embolization Technique**

**Diagnostic angiography**

General endotracheal anesthesia is preferred for all embolization procedures, although in cooperative patients, and when a single vessel is embolized, local anesthesia with conscious sedation can be used. Using the Seldinger technique, a 4-Fr sheath is inserted into the femoral artery and secured. With the assistance of a microcatheter and micro-guide wire, the appropriate first- or second-order vessel is selected and catheterized with a 4-Fr vertebral catheter. Standard practice, for most large tumors, is to perform a diagnostic angiogram of both ICAs, both ECAs, and one dominant VA to document the status of the anterior and posterior communicating arteries. Based on a tumor’s location, all potential sources of blood supply must be studied, and any dangerous anastomoses between ECA and ICA or VA branches must be ruled out. 3-D angiography may be used to better delineate the feeding vessel but, in some cases, superselective angiography of the feeding vessel with a microcatheter may be required, although this is usually best performed once a decision to proceed with embolization is made. Venous assessment is important when a tumor is compromising the dural sinus or jugular vein.

**Carotid occlusion test**

If an ICA is encased by a tumor, particularly if it is encased and narrowed, then an assessment of collaterals may be requested by the operating surgeon. Two levels of assessment may be performed. In the authors’ center, a surgical sacrifice of the ICA is rarely done without a concurrent bypass procedure. In such a case, the authors perform only a carotid compression angiogram. Cerebral angiography with manual ipsilateral common carotid compression with contralateral ICA injection (anteroposterior view needed, focus on venous phase, filling, and emptying) demonstrates the potential cross flow through the anterior communicating artery. A delayed venous filling and emptying is the best evidence of poor collateralization. An Alcock’s maneuver, which involves carotid compression during injection of the dominant VA (anteroposterior and lateral views show the entire skull for lateral view), can show the posterior communicating artery and potential collateral

### Table 5

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<tr>
<th>Type</th>
<th>Description</th>
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<tbody>
<tr>
<td>Type I</td>
<td>Drainage into a dural sinus, with normal antegrade flow</td>
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<tr>
<td>Type II</td>
<td>Drainage into a dural sinus, with reflux into II a: (other) sinuses</td>
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<td></td>
<td>II b: cortical veins</td>
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<td></td>
<td>II a + b: sinuses + cortical veins</td>
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<tr>
<td>Type III</td>
<td>Drainage into cortical veins</td>
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<tr>
<td>Type IV</td>
<td>Drainage into cortical veins with cortical ectasia</td>
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### Table 6

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<th>Location</th>
<th>No. of Patients</th>
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<tr>
<td>Transverse-sigmoid</td>
<td>10</td>
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<tr>
<td>Petrotentorial</td>
<td>8</td>
</tr>
<tr>
<td>Parasagittal/falcine</td>
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</tr>
<tr>
<td>Anterior fossa</td>
<td>1</td>
</tr>
<tr>
<td>Middle fossa</td>
<td>2</td>
</tr>
<tr>
<td>Torcular</td>
<td>1</td>
</tr>
<tr>
<td>Carotico-cavernous fistula</td>
<td>7</td>
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flow. A carotid occlusion test is performed with the patient awake. The ICA is occluded in the C1-C2 segment with a 7-mm HyperForm balloon catheter (Micro Therapeutics, Irvine, California) through a 6-Fr guiding catheter for 30 minutes after systemic heparinization, and patients are observed for any neurologic deficits. The authors also use transcranial Doppler monitoring of the ipsilateral middle cerebral artery during the balloon test occlusion. A 30% drop in velocities with the balloon inflated indicates only borderline collateralization, and a 40% decrease indicates that the degree of collateral flow is probably inadequate to support cerebral perfusion. It is also helpful to have patients on aspirin (325 mg) for 1 day preoperatively.

**Embolization**

Once a decision is made to proceed with embolization, patients are heparinized. For ECA embolization, a 4-Fr vertebral catheter is used as a guiding catheter. Usually, a Renegade 0.021 inch ID microcatheter (Boston Scientific) with a 0.014 inch or 0.016 inch hydrophilic micro-guide wire (for 350- to 500-μm polyvinyl alcohol [PVA] particles) or a Marathon microcatheter with a Mirage 0.008 inch guide wire (for 45- to 150-μm PVA particles) is navigated into the vessel of interest, as distally as possible, and beyond any important potential collaterals. In case of the middle meningeal artery, the operator needs to get past the petrosal branch, which supplies the facial nerve. With any ECA embolization, all collaterals to the ophthalmic artery should be studied carefully and avoided, because sometimes the ophthalmic artery has an aberrant origin from the MMA. With the microcatheter in place, superselective angiography is performed to study the microvasculature, intratumoral collaterals, and the ease of reflux. The microcatheter is flushed with saline before the embolic agent is introduced. For ICA or VA branch embolization, arterial road maps are needed; therefore, the 4-Fr sheath is exchanged for a 6-Fr sheath, and a 6-Fr guide catheter is used.

The most common embolic agent is PVA particles, mixed with contrast material. Particles, sizes 150 to 250 μm, are used for better penetration of tumor supply.

### Table 7

<table>
<thead>
<tr>
<th>Endovascular Complications</th>
<th>No. of Patients</th>
</tr>
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<tr>
<td>Stuck catheter</td>
<td>3</td>
</tr>
<tr>
<td>CN palsy (V/VII)</td>
<td>1</td>
</tr>
<tr>
<td>Rupture of vein with death</td>
<td>1</td>
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Fig. 24. An irregular area of moderate tumor blush is projected over the right frontal lobe supplied primarily from the frontal division of the right middle meningeal artery (A). A large area of filling defect is present within the adjacent superior sagittal sinus (B).
neuromeningeal trunk supplying the CNs 9, 10, and 11, 350- to 500-μm particles are used. This avoids penetration of the fine blood vessels (vasa nervosum) supplying the nerves and attendant CN paralysis. Embolization is performed in small puffs, with repeated road maps as necessary and continuous fluoroscopic observation, until stasis of contrast within the tumor is noted. At this point, the embolic syringe is replaced with saline, which is used to gently push out the contrast-particle mixture remaining inside the dead space of the microcatheter. In some cases of ECA embolization, the feeding vessel is occluded with Gelfoam torpedoes using Rendezvous microcatheter. Gelfoam supplied in sheets is cut into strips, approximately 1- to 2-mm wide and 2- to 3-mm long, and then rolled so that they are very thin. They are then loaded into the hub and the distal part of a 1-ml syringe, which is connected to the microcatheter, and injected. They produce rapid occlusion of the feeding arteries, but recanalization occurs in approximately 6 weeks. The liquid embolic agent, Onyx, may also be used in cases of very vascular tumors; in such cases, the embolization technique is similar to that used for AVMs.

Embolization through the MHT is important in devascularizing clival meningiomas. When successful, and combined with embolization of any ECA feeders, this is effective in reducing the blood loss from these tumors. The cannulation of the MHT may be technically demanding, often requiring 3-D angiography to understand the exact point of origin of the artery from the cavernous ICA and some probing with a microwire, with reshaping as needed. A Marathon 1.3-Fr microcatheter and a Mirage 0.008 microwire are used to cannulate the MHT, taking the microwire at least 5 mm past the origin of the MHT. Once cannulated with the microwire, difficulty may be encountered with the introduction of the microcatheter due to the size of the vessel or the angle of take off. When the microcatheter is well into the artery, it should be wedged at its branch point or slightly pulled back. Embolization is performed carefully, and with the smallest particles, to avoid reflux into the ICA and downstream embolization.

**Embolization by Direct Puncture of the Tumor**

This technique is rarely used when a tumor is difficult to access by the transarterial route but can be easily accessed by direct puncture. Examples of this case are angiofibromas, paragangliomas, hemangiopericytomas, some tumors of the convexity, some jugular-mastoid tumors, and juvenile nasopharyngeal angiofibromas, particularly when the feeding arteries are not accessible due to previous procedures. The lesion is accessed through a prior craniotomy or a preauricular or precondylar area. After selective transarterial angiography, the lesion is punctured and direct angiography is performed with and without manual compression of the venous drainage in the region and under fluoroscopic or ultrasound guidance. After the tumor is punctured, a contrast medium is injected through the needle to identify the corresponding tumoral compartment and its draining vein. Embolization is then performed with a mixture of NBCA (Cordis Neurovascular, Miami, Florida) and lipiodol (Guerbet France, Paris, France), injected using a 3-ml syringe under fluoroscopic control. NBCA and lipiodol are mixed in 1:3 proportions to which some powdered tungsten is added. With lipiodol, the progressive filling of the tumoral compartment can be followed visually with fluoroscopy. Several intratumoral injections are necessary to fill the tumor as completely as possible. Hence, the needle is withdrawn after each injection to reach another tumoral compartment. Each intratumoral injection must be slow and progressive, lasting from 20 to 30 seconds according to the size of the

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*Fig. 25.* Near complete stasis of flow within the frontal division of the middle meningeal artery with significant decrease in the previously noted tumor blush overlying the right frontal lobe.
tumoral compartment. The injection is stopped when the NBCA starts to flow into the draining vein. In some cases, protection of ICA or VA is necessary with a balloon while using direct puncture embolization.

When Not to Embolize

The presence of dangerous EC-IC anastomoses, shared arterial supply between tumor and normal structures, the caliber of tumor vessel, and the potential for the embolic particles to reflux into the parent vessel are all factors that need to be considered before a decision to embolize. Pial vessels are fragile and thus associated with a high risk of arterial perforation and IC hemorrhage. The potential for stroke due to thromboembolism when embolizing through pial vessels is also higher than when embolizing through ECA branches. If a major tumor blood supply is derived from small ICA branches or the ophthalmic artery, complete devascularization using microcatheters is not possible without unacceptable risk.

Complications of Intratumoral Embolization

Most common complications noted are fever and localized pain. Potentially more devastating complications include focal neurologic deficits due to inadvertent passage of embolic material.
through reflux or from ECA branches through dangerous collateral anastomoses into the ICA or VA circulation,\textsuperscript{47,50,52,53} and intratumoral hemorrhage. Embolization in the vascular territory of the ophthalmic artery carries a risk of blindness.\textsuperscript{59} Cutaneous branches of the ECA may be occluded causing skin necrosis, thus complicating the prospective wound healing process.\textsuperscript{59,60} Transient CN palsies (provided particulate reabsorbable embolic materials are used instead of polymerizing fluid materials) can also occur due to interruption of the vascular supply (vasa vasorum), which is often derived from ECA.\textsuperscript{47,52} The petrous branch of MMA supplies the facial nerve and the neuro-meningeal branch of the ascending pharyngeal artery supplies CNs 9 through 12 and can be inadvertently embolized. Tumor swelling post embolization may compromise the airway or increase the IC mass effect and this may be avoided by administration of steroids and early surgery after the procedure.\textsuperscript{46,59–61} Shunting of the embolic material through the tumor in the setting of patent

Fig. 27. Particle embolization of the right distal internal maxillary artery, right middle meningeal artery (A), right accessory meningeal artery, and right MHT feeding branch (B) with no residual significant tumor blush from the right ECA branches and very little residual tumor blush from the right ICA postembolization.

Fig. 28. MRI showing a hypervascular recurrent hemangiopericytoma in the middle cranial fossa. (A) Axial; (B) Coronal.
Formane ovale may permit paradoxical embolization to occur systemically. A hypertensive response due to release of vasoactive peptides from large chemodectomas may occur and cause death. This can be avoided by careful anesthetic preparation and management.

**Case Examples of Tumor Embolization**

**Patient 1: convexity meningioma with external carotid artery embolization**

A 51-year-old woman presented with recent history of progressive headaches and memory problems. Neuroimaging demonstrated a large parafalcine meningioma, causing significant mass effect over the right frontal lobes. The tumor extended bilaterally across the superior sagittal sinus occluding it midway in its course. The patient underwent cerebral angiogram on the day before the operation and selective embolization of frontal division of the middle meningeal artery supplying the tumor was done with 150 to 250 PVA particles through a 4F Renegade with a gold-tip microcatheter (Target Therapeutics/Boston Scientific) under careful negative roadmap observation until stasis of flow was observed. She underwent a craniotomy and total resection of the tumor on the next day. Patient made a smooth recovery.

**Patient 2: clival meningioma with meningohypophyseal trunk embolization**

A 51-year-old woman presented with intractable seizure and on neuroimaging was found to have recurrence of petroclival meningioma. On angiography, there were feeders from bilateral MHT, right MMA, and distal IMA. Particle embolization was performed using a combination of 45- to 150-μm PVA particles in the right MHT and 150- to 250-μm PVA particles in the internal maxillary branches using a 6-Fr Marathon 1.3-Fr microcatheter. There was significant reduction in the tumor

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**Fig. 29.** There is marked vascular supply to lesion from two main branches arising from the internal maxillary artery and left occipital artery (A, B). Figure C is during the embolization procedure.
Fig. 30. There is vascular supply to the skull base lesion from branches of the inferolateral trunk and tumor vessels originating near the MHT. There is a prominent posterior communicating artery infundibulum. After embolization of the ILT and MHT vessels, the tumoral flush was significantly reduced (not shown).

Fig. 31. (A) Coronal gadolinium MRI showing a contrast enhancing right mastoid mass lesion up to 4.1 cm in size. (B) CT with bone windows showing significant bone erosion.
Patient 3: recurrent hemangiopericytoma with embolization

A 58-year-old man with recurrent hemangiopericytoma who underwent multiple resections and gamma knife radiosurgery for his tumor was subjected to balloon occlusion test and embolization of tumor subsequently. In two sessions, embolization was done. The 6-Fr Envoy catheter was placed into the left ECA and an Excelsior 10 microcatheter (eV3) with the assistance of Synchro 14 microwire was advanced into an anterior and posterior division of the left internal maxillary artery and occipital artery supplying the tumor, and PVA particle embolization was carefully performed using 150- to 250-μm particles and negative road-map technique. A large area of irregular tumor blush within the left aspect of the skull base blush after the procedure and she underwent surgical resection of the tumor.

Fig. 32. Angiography revealed on right carotid injection a prominent tumor stain corresponding to the known right mastoid tumor. Early venous drainage is present and suggestive of arteriovenous shunt within the tumor. The tumor angiographic appearance suggests intraosseous meningioma.

Fig. 33. Endovascular catheterization and embolization of the tumor using Onyx-18. (A) Postinjection angiography in the external carotid system demonstrated near-complete devascularization of the tumor (B).
supplied by branches of the ILT and vessels near the MHT from the left ICA and AICA were also embolized. After particle embolization of the said ECA and ICA branches, there was stasis of flow within these vessels and markedly decreased tumor blush.

Patient 4: tumor with Onyx embolization
A 62-year-old woman presented with history of headaches and was discovered to have a large hypervascular tumor. She had a previous surgery for Meniere’s disease. Preoperative CT scan demonstrated significant bony erosion. MRI demonstrated multiple vascular flow voids. Angiography demonstrated the mastoid tumor as extremely vascular with prominent blush. Early venous drainage was suggestive of arteriovenous shunt within the tumor. The tumor was embolized using Onyx-18, PVA, and Gelfoam pledgets/torpedoes before surgery to reduce the extent of bleeding and reduce CN morbidity.

Results and Complications
At the authors’ institution over the last 3.5 years, 18 convexity tumors, 42 skull base lesions, 19 intrinsic brain tumors, and 23 EC neoplasms were embolized by the endovascular team. Such embolization often, but not always, resulted in the reduction of bleeding during surgery and enhanced the ease of removal. There were no major complications encountered in the authors’ experience.

SUMMARY
Endovascular procedures are rapidly expanding as treatment options for cerebrovascular diseases and neoplasms of the head and neck and are becoming less invasive but more effective. There are potentially dangerous anastomoses between the EC and IC circulations; hence, thorough knowledge of the anatomy is essential to minimize the risk of CN palsies, blindness, or neurologic deficits. It is essential to understand the scientific basis of treatment rationale based on advancing new neuroimaging techniques to better serve patients. An interdisciplinary approach and treatment in high volume centers are vital to obtain maximal benefit for patients.

REFERENCES


