Chapter 3: Interventional Neuroradiology of the Head and Neck

Lee H. Monsein

Interventional neuroradiology originated in 1930 when Brooks attempted to treat a carotid-cavernous fistula by inserting a piece of muscle into the internal carotid artery (ICA). Since then, detailed studies of the vascular anatomy, advancements in catheterization techniques, and the introduction of new embolic materials have contributed to the evolution of interventional neuroradiology.

Vascular lesions of the head and neck are challenging therapeutic problems. Endovascular techniques have played a role in the management of these lesions since selective embolizations of the external carotid artery (ECA) were described by Djindjian et al (1972). Today the interventional neuroradiologist can offer significant assistance to the head and neck surgeon faced with a difficult case. Such assistance may include embolotherapy of tumors and vascular malformations, endovascular control of bleeding, or temporary or permanent carotid artery occlusion.

Embolotherapy

General principles

To provide safe and effective treatment, an understanding of normal, variant, and pathologic vascular anatomy is crucial for the interventional neuroradiologist. Excellent reviews have been written on the subject (Lasjaunis and Berenstein, 1987g; Newton and Potts, 1974; Osborn, 1980; Russell, 1986).

Lasjaunis (1981) popularized the term *hemodynamic equilibrium* to describe the way in which many areas of the head and neck, including the brain, are fed by a variable number of complementary intracranial and extracranial vascular supplies. Where there is acquired or iatrogenic occlusion of the internal carotid or vertebral arteries or their branches, these connections may rescue cerebral perfusion. However, these same channels can provide the route by which ischemic complications can occur during an embolization procedure. Similarly, vessels that supply tumors of the head and neck may also supply the brain, cranial nerves, and skin.

Important potential anastomoses exist between these arteries: the internal maxillary and the internal carotid via the artery of the foramen rotundum; the middle meningeal and the ophthalmic via recurrent meningeal, meningoophthalmic, and meningolacrimal branches; the ascending pharyngeal and the C5 portion of the internal carotid via lacerum branches; the ascending pharyngeal and the third cervical level of the vertebral via musculospinal branches; the ascending pharyngeal and the second and third cervical levels of the vertebral via hypoglossal branches; and the occipital and vertebral via first and second cervical level collaterals (Plate 2; Table 3-1). In addition, a number of anatomic variations may exist such as the ophthalmic artery originating directly from the middle meningeal artery.

"Dangerous" anastomoses may be opacified before, during, or after embolization. Their visualization, however, is an absolute contraindication to embolotherapy. Certain maneuvers can be performed in order to protect against embolic material traversing these channels and reaching the intracranial circulation. An embolic material whose physical characteristics preclude its traversing these channels is one option, or the channels themselves may be blocked before embolization of the pathologic area. With "flow control", the direction of the blood flow is stopped or reversed in an anastomosis so that embolic material does not go toward vital tissue during embolotherapy. The parent artery at or above a potentially "dangerous" anastomosis may be temporarily or permanently occluded to prevent delivery of embolic material to more distant vital structures. A tourniquet can be placed around the head to prevent embolic material, which can cause skin necrosis, from entering the vessels of the scalp.

Caution is required when performing embolization of vessels that may supply the cranial nerves. The transcranial nerves are supplied primarily by three branches of the internal maxillary artery: the cavernous branch of the middle meningeal artery, which supplies the gasserian ganglion; intracranial branches of the middle and accessory meningeal arteries, which supply the third division of the fifth cranial nerve; and the artery of the foramen rotundum, which supplies the second division of the fifth cranial nerve (and less frequently its first division as well as the third, fourth, and sixth cranial nerves). The intrapetrous portion of the seventh cranial nerve can be supplied by the stylomastoid artery or the petrosal branches of the middle meningeal and accessory meningeal arteries. The neuromeningeal division of the ascending pharyngeal artery supplies part of the sixth cranial nerve, the ninth through eleventh cranial nerves via the jugular branch, and the twelfth cranial nerve via the hypoglossal branch (Plate 2; Table 3-2).

In general, an artery is considered safe for embolization if it does not supply the brain and cranial nerves directly or indirectly via "dangerous anastomoses" or if it supplies an area with a preexisting deficit such as the ophthalmic artery in a patient who is already blind. To predict whether embolization of an extracranial vessel will lead to peripheral nervous system cranial nerve deficits, a provocative lidocaine test has been recommended (Horton and Kerber, 1986). A mixture of 2% lidocaine with an equal volume of contrast is injected, opacifying the vessel for 2 to 4 seconds. The appearance of a transient cranial nerve deficit may predict the development of a permanent one after subsequent injection of embolic material. Similarly, the amobarbital sodium test may be performed to determine whether embolization of an intracranial vessel will lead to a CNS deficit. Neurologic testing is performed immediately after injection of 40 to 50 mg of amobarbital sodium.

A complete angiographic workup is recommended before attempting embolization. The embolization may be performed immediately following the diagnostic study or at a later time. A carefully planned angiographic protocol is necessary. More distal arteries should first be injected because spasm or thrombosis may prevent entrance into these arteries later.

Diagnostic and therapeutic angiography with local anesthetic is preferred, allowing for continual neurologic testing during the procedure. This is facilitated by the use of one of the newer nonionic or low-osmolality contrast agents that offer better patient tolerance than traditional ionic iodinated contrast agents. General anesthetic should be used only in patients who are uncooperative or who cannot tolerate such an extended procedure.

We use digital subtraction and conventional angiography for most embolization procedures. Digital subtraction angiography can decrease the time, cost, and radiation required to perform an evaluation. It also demonstrates some tumor blushes better than conventional angiography. Conventional angiography can display vascular anatomy with higher resolution - an important consideration when vessel invasion is suspected. It also appears to be better received by some surgeons, particularly with regard to vessel mapping.

A number of agents are available for embolization of hypervascular head and neck tumors (see box). Several factors determine the correct agent for a particular case. Because most of these hypervascular lesions are benign, a relatively safe agent should be chosen. In general, smaller and more permanent embolic agents are more dangerous.

Box: Modern embolic agents

Absorbable particulate agents

Gelfoam powder
Gelfoam particles
Microfibrillar collagen (Avitene)
Autologous clot
Dura mater

Nonabsorbable particulate agents

Polyvinyl alcohol (PVA)
Detachable balloons
Coils

Nonabsorbable liquid agents

N. butyl cyanoacrylate (NBCA)
Ethibloc (outside USA)

Cytotoxic agents

95% ethanol
Chemotherapeutic agents.

Particles less than 100 microm have a greater tendency for aberrant migration into the vasa nervorum of the cranial nerves or through dangerous anastomoses into the intracranial circulation. Although it has been reported that meningiomas, paragangliomas, and juvenile nasopharyngeal angiofibromas rarely demonstrate arteriovenous shunting (Wickbom, 1974), we have encountered this phenomenon frequently. In addition, we have injected radioactive macroaggregated microspheres measuring between 60 and 100 microm into the feeders of some of these tumors before resection and demonstrated significant shunting to the lungs. The embolic agent should, however, be small enough to be delivered through a microcatheter and reach the interstices of the tumor. For these reasons, particles of at least 100 to 140 microm are recommended for the presurgical embolization of most of these tumors. Absorbable

embolic agents are preferable to decrease the severity and longevity of potential ischemic complications. Nonabsorbable agents, however, are indicated when a tumor is inoperable or the embolization procedure is palliative.

The goal of the embolization procedure depends on the aggressiveness and operability of the tumor or degree of epistaxis. In the usual case, embolization is terminated when there is a significant decrease in abnormal blush, when washout in the feeding vessel becomes stagnant, or when dangerous anastomoses become visible. Pain in the territory supplied by the embolized vessel and fever are quite common after embolization. Other more serious complications are infrequent and are usually related to improper technique. These are predictable depending on the vessels embolized and include brain infarction and transient or permanent cranial nerve palsies.

After tumor embolotherapy, the patient is given corticosteroids and supportive care; sometimes anticonvulsants are administered. Surgery is performed 1 to 5 days later. Surgery performer earlier than 1 day after embolization may not take full advantage of progressive necrosis. Surgery later than 5 days after embolization may allow recanalization. After epistaxis treatment or carotid occlusion it is important that the patient remain hemodynamically stable.

Tumors

Preoperative tumor embolotherapy of the head and neck is performed most commonly for meningiomas, paragangliomas, or juvenile nasopharyngeal angiofibromas. The principles described, however, are similar for most hypervascular or aggressive tumors of the region.

Meningiomas (Fig. 3-1)

Meningiomas are benign, potentially curable lesions. However, they can be locally invasive, particularly in the skull base region, and have a propensity to recur if not totally removed. They are usually slow growing and evoke minimal symptoms; therefore any treatment, including embolotherapy, must be of limited risk.

The major determinant in recurrence of meningiomas is the extent of surgical resection. This is influenced by tumor vascularity, size, location, and involvement of major dural venous or arterial structures. Tumor vascularity is most amenable to alteration before surgery. For this reason, Manelfe et al, as early as 1973, before a number of subsequent investigators, recommended endovascular embolization before surgical resection or for palliation in inoperable cases. Surgery after embolization offers several advantages over surgery with ligation of arterial feeders. Access may be gained to surgically unapproachable vessels. Contralateral blood supply may be controlled without a bilateral surgical approach. Delivery of emboli directly into the vascular bed of the tumor results in a greater degree of ischemia and necrosis that can result in less blood loss, mass effect, and time in the operating room.

The indications for preoperative embolization of a meningioma are determined by the tumor's size, location, and arterial supply. Large, highly vascular tumors of the skull base and middle cranial fossa, in which the arterial supply is difficult to reach before surgical removal,

can benefit the most from preoperative embolization. Unfortunately, however, the feeding arteries of skull base lesions are often too small to catheterize. The objectives of preoperative embolization of meningiomas are to facilitate surgical excision by decreasing blood loss and to lessen the chance of recurrence by causing necrosis at the site of dural attachment.

Meningiomas arise from the arachnoid villi and therefore receive their initial blood supply from dural branches of the meningeal arteries, most commonly the ECA. As they enlarge, they can recruit additional blood supply from pial branches of the cerebral arteries. Meningeal vessels therefore usually supply the center of a meningioma while the periphery is supplied by cerebral vessels. Pure dural or pial blood supplies are less common. The vascular supply may be unilateral or bilateral.

Meningiomas of the middle cranial fossa, planum sphenoidale, and lesser wing of the sphenoid bone can receive blood from the ECA via the middle meningeal artery, accessory middle meningeal artery, or meningeal branches of the anterior deep temporal branch of the internal maxillary artery. Meningiomas can be supplied by the ICA via meningeal branches of its cavernous segment or the recurrent meningeal branch of the lacrimal branch of the ophthalmic artery. Meningiomas of the posterior fossa, cerebellopontine angle, and foramen magnum can receive blood from many sources. This includes the ECA via the posterior meningeal branch of the middle meningeal artery, the neuromeningeal branch of the ascending pharyngeal artery, and the meningeal branches of the occipital artery. The vertebral artery via anterior and posterior meningeal branches and the cerebellar arteries can also supply meningiomas.

In addition to determining feasibility and safe routes of embolization, angiography of a meningioma can provide other potentially important information. Tumor vascularity, the site of dural attachment, arterial and venous displacement, encasement, anatomy, and the adequacy of collateral blood supply can be demonstrated by angiography. The difficulty of surgical resection can sometimes be predicted by angiographic demonstration of arterial venous encasement. Drainage through cortical veins may suggest cortical infiltration resulting in a poor surgical cleavage plane. Supply by the superficial temporal artery or drainage through the diploic and superficial scalp veins may indicate bone or extracranial soft tissue involvement. Lesions that can stimulate the appearance of a meningioma, such as a giant carotid aneurysm of the sella, can be excluded.

Our protocol includes catheterization of the common, internal, and external carotid arteries bilaterally, which can be accomplished with a conventional angiographic catheter. Distal arteries that potentially feed the tumor are then selected with a microcatheter. Permanent occlusion of the ICA may be necessary under certain circumstances.

In addition to angiography, computed tomography (CT) and magnetic resonance imaging (MRI) have been used preoperatively to assess the efficacy of embolization. Large areas of diminished contrast enhancement appear after embolization. Histopathologically, the degree of tumor necrosis can vary considerably. Polymorphonuclear cell infiltration surrounds regions of ischemic or hemorrhagic necrosis ranging from microscopic proportions to less than 5 mm in size, alternating with areas of normal vascularity. In a small number of specimens, the embolic agent is seen, which is associated with thrombosis.

In a large series of meningioma embolizations, temporary neurologic deficits were reported in 2.7% of 185 patients (Lasjaunis and Berenstein, 1987d). Permanent neurologic deficits occurred in 1.6% of patients. There was no morbidity. There have been rare reports of peritumoral, intratumoral, and subarachnoidal hemorrhage after embolization of meningiomas (Hayashi et al, 1987; Suyama et al, 1987).

Paragangliomas (Figs. 3-2 and 3-3)

Paragangliomas are a fascinating and diverse group of tumors that, although relatively rare, have generated significant multidisciplinary interest. They are usually benign but can be multicentric in 10% of cases, malignant in 3% to 18% of cases, and can exhibit neurosecretory activity in 1% of cases. Because of their complex vascular supply and relation to vital structures, paragangliomas can present difficult therapeutic challenges. The mortality and morbidity associated with surgical resection have been reported to be as high as 33%and 50%, respectively. Since Hekster et al (1973) described preoperative embolization of paragangliomas, a number of investigators have demonstrated its benefit. By limiting blood loss, preoperative embolization can significantly improve surgical conditions. Definitive cures have also been reported with embolization alone (Iaccarino et al, 1985).

Paragangliomas may be divided into two general groups based on location: temporal, including tympanic and jugular types; and cervical, including vagale and carotid body types. Rare tumors have also been reported in the orbit, larynx, and upper airway. The primary blood supply of intratemporal paragangliomas derives from the inferior tympanic and the neuromeningeal branch of the ascending pharyngeal artery bilaterally. The stylomastoid branch of the occipital or posterior auricular artery, anterior tympanic branch of the superficial temporal artery, superior tympanic branch of the middle meningeal artery, and caroticotympanic branch of the ICA may also participate. The neuromeningeal branch of the ascending pharyngeal artery at the level of the jugular foramen and hypoglossal canal, mastoid branch of the occipital artery, dural branches of the vertebral artery, clival branches of the cavernous portion of the ICA, and dural branches of the petrosal branch of the middle meningeal artery may supply paragangliomas with extradural intracranial extension. Intradural intracranial extension may be supplied by the anterior and posterior inferior cerebellar arteries.

The major blood supply to the larger superior compartment of carotid body tumors arises from the musculospinal branch of the ascending pharyngeal artery. The smaller inferior component is supplied by the carotid body branch of the ICA. Additional blood supply may be derived from the recurrent lingual, superior laryngeal, ascending cervical, and deep cervical arteries, and the muscular branches of the occipital arteries. Direct supply of the tumor from small branches of the ICA implies malignancy, infiltration of carotid wall, and the need for occlusion of the carotid artery. The blood supplying vagal tumors principally arises from the musculospinal branch of the ascending pharyngeal artery bilaterally and muscular branches of the proximal segment of the occipital artery. Larger lesions may derive supply from ascending cervical and vertebral arteries.

Our angiographic protocol is determined by the location of the tumor but usually includes a study of the vertebral and common, internal, and external carotid arteries including the ascending pharyngeal, occipital, internal maxillary, and posterior auricular branches bilaterally. The ascending and deep cervical arteries are included for a low cervical lesion.

This is followed by superselective catheterization of potential feeding vessels. Paragangliomas have multiple noncommunicating vascular compartments in up to 85% of cases. To obtain the most benefit from preoperative embolization, the vessels supplying all compartments of the tumor should be considered.

Histopathologically, embolization has been shown to cause cell damage (which can be irreversible and related to ischemia), necrosis, and fibrotic transformation. Minor complications (eg, pain and fever) can occur in up to 80% of patients. Rare fatalities have been reported secondary to uncontrollable fluctuations in blood pressure after embolization of secretory active tumors (Pandya et al, 1978).

Juvenile nasopharyngeal angiofibromas (Fig. 3-4)

Juvenile nasopharyngeal angiofibromas (JNAs) are rare tumors that occur almost solely in adolescent males. They are benign, locally invasive tumors with a strong tendency to recur. Surgical resection when possible, is considered the treatment of choice. JNAs are highly vascular lesions that are difficult to remove, and hemorrhagic fatalities have been reported. Preoperative embolization of JNAs, first described by Roberson et al in 1972, can significantly reduce the volume of blood loss. Blood loss without embolization can be over 2000 mL.

These tumors are usually supplied exclusively by branches of the ECA. Most commonly this includes branches of the distal maxillary, accessory meningeal, superior pharyngeal division of the ascending pharyngeal, and the ascending palatine arteries. Dural branches of the ICA may supply the lesion if there is intracranial extension. Ethmoidal or inferior muscular branches of the ophthalmic artery can contribute if the lesion invades the ethmoid sinus or orbit. Vascular anastomoses from the vertebral and thyrocervical arteries may fill previously ligated branches of the external carotid artery.

Angiography is usually not necessary unless preoperative or palliative embolization is required. A suggested angiographic protocol would begin with evaluation of the internal and external carotid arteries bilaterally followed by selective study of the distal internal maxillary, accessory meningeal (pharyngeal branch), ascending pharyngeal (eustachian branch), and ascending palatine arteries. The procedure is completed with a facial artery injection, used to opacify the collateral supply to the internal maxillary artery and check for the completeness of the embolization procedure. Although not routinely done, safe and effective embolizations of branches of the petrous and cavernous portions of the ICA have been reported (Halbach et al, 1989a). Permanent occlusion of the ICA may be necessary under certain circumstances.

The largest reported series of preoperative embolization included 58 JNA patients. No mortality and no permanent morbidity were reported (Garcia-Cervignon et al, 1988). There was one case of transient hemiparesis that occurred in association with temporary balloon occlusion of the carotid artery.

Epistaxis (Fig. 3-5)

Epistaxis can be caused by venous, arterial, or arterialized venous bleeding. If venous bleeding does not stop spontaneously it can usually be effectively controlled by compression or anterior nasal packing. Arterial bleeding constitutes a medical emergency, and more aggressive therapy may require posterior nasal packing, ligation of the maxillary or ethmoid arteries, or embolization of arterial feeders, which was initially described by Sokoloff et al in 1974. There are numerous causes for severe uncontrollable epistaxis (see box).

Epistaxis can be caused by arterial bleeding from branches of the internal carotid, maxillary, facial, or ascending pharyngeal arteries. The ICA can be responsible for bleeding from the ethmoid sinus via the anterior and posterior ethmoid branches of the ophthalmic artery and the sphenoid sinus via capsular branches or the carotid siphon itself. Bleeding from the following sources can be attributed to the maxillary artery: (1) conchae and nasal septum via medial and lateral nasal branches from its sphenopalatine segment; (2) the posterior septum and conchae via an anterior branch of the descending palatine artery; (3) the maxillary sinus via alveoloantral and infraorbital branches; and (4) the eustachian meatus, nasopharynx, and soft palate via pterygovaginal and accessory meningeal branches.

Box: Causes of epistaxis

Idiopathic origin (spontaneous or hypertensive)
Hereditary hemorrhagic telangiectasia
Benign tumors (for example, juvenile nasopharyngeal angiofibroma)
Malignant tumors (for example, primary squamous carcinoma)
Arteriovenous malformations
Trauma (direct or from pseudoaneurysm rupture)
Aneurysm rupture
Hemostasis disorders
Postsurgical origin.

The facial artery is usually not responsible for primary epistaxis. It is, rather, a major source of recurrent epistaxis through revascularization of other previously occluded arteries to which it provides collateral blood supply. These include the maxillary artery via infraorbital and alveoloantral branches, the transverse facial artery, and the ophthalmic artery via nasoangular and nasoorbital branches. The facial artery can also be responsible for bleeding from the ala and external nose via the alar branch, the palate via the palatine branch, and the lower nasal septum via the superior labial artery. The ascending pharyngeal artery is usually not an important source of epistaxis unless bleeding is caused by juvenile nasopharyngeal angiofibromas or angiomatosis. Superior, middle, and inferior pharyngeal branches, from the anterior division of the ascending pharyngeal artery, supply the medial and paramedial nasopharynx. The middle pharyngeal branch can supply the soft palate through the ascending palatine artery. It may make anastomotic connections with the internal carotid and internal maxillary arteries.

Angiography should include injection of the internal carotid, internal maxillary, and facial arteries on the presumed site of bleeding, followed by injection of the contralateral side. Embolization of the internal maxillary artery is followed by injection of the facial and

ascending pharyngeal arteries to check for residual or collateral supply. Embolization of the internal maxillary artery is often successful in halting epistaxis even when no source of bleeding is identified at angiography. A posttraumatic aneurysm of the petrous (Willinsky et al, 1987) or cavernous (Gilbert et al, 1986) portion of the ICA may be permanently occluded by balloons in some cases of massive epistaxis (Willinsky et al, 1987; Simpson et al, 1988).

Control of epistaxis with embolization can be very successful. Riche et al (1979) reported immediate postembolization cessation of bleeding in 53 of 54 patients and no further bleeding in 29 of 42 patients in 3 months to 6 years follow-up. Four major complications occurred in their series.

Vascular abnormalities

Patients with intracranial and extracranial vascular abnormalities often present with auditory symptoms. These abnormalities can be classified according to the type of lesion: primary vascular lesions consisting of congenital abnormalities of vascular elements and secondary vascular lesions caused by acquired abnormal connections between arteries and veins.

Primary vascular lesions

Primary vascular lesions of the head and neck region consist solely of abnormal vascular elements. Mulliken and Glowacki (1982), Lasjaunis and Berenstein (1987b, 1987c), and Mulliken and Young (1988) combined the clinical characteristics of these lesions with the results of tissue culture, histochemical, and electron microscopic studies to identify these lesions as *hemangiomas* that demonstrate proliferative and usually involutional behavior or as hemangiomas and *vascular malformations* that typically do not exhibit involutional behavior (Table 3-3). It is difficult to interpret the literature regarding these lesions because most authors do not make a clear distinction between these very different entities.

Hemangiomas. Hemangiomas are tumors that grow through proliferation of endothelial cell elements. Hemangiomas occur predominantly in females with a reported female/male ratio of 5:1. Although only 30% are found at birth, the majority appear by 3 months of age. Typically there is a rapid phase of growth at about 6 months of age, with involution beginning at 12 months and complete by 7 years of age. Involution occurs in about 95% of patients. Involution can result in total disappearance of the lesion or in a smaller mass of fibrofatty tissue that can be surgically resected.

Therapy is indicated for lesions that do not experience involution and for those that are associated with oral, orbital, or subglottic functional impairment, complications such as hemorrhage, congestive heart failure, hypoprothrombinemia, and gross disfigurement. Systemic corticosteroids are the initial treatment of choice. Embolization with particles can sometimes be helpful in arresting the proliferative phase and promoting involution. The principles of embolization for these lesions are similar to those for any tumor of the head and neck region.

Vascular malformations (Fig. 3-6). Primary extracranial vascular malformations are caused by inborn errors of vascular morphogenesis and are therefore almost always found at birth. They consist of abnormal capillary, arterial, venous, or lymphatic channels of varying morphology. The majority of these lesions expand. However, this is caused by hemodynamic factors rather than cellular proliferative elements. They rarely undergo involution.

Conservative therapy is recommended in children unless there is significant dysfunction or life-threatening complications. The aggressiveness of treatment in adults is dictated by degree of the debilitation, the expertise of the treatment team, and whether the goal of therapy is palliative or curative.

Table 3-3. Primary extracranial vascular lesions

Features	**Hemangiomas**	**Vascular malformations**
Cellular		
Mast cells per high-power field	25	0.8
Factor VII antigen	Yes	Yes
Tissue culture	Yes	No
In vitro angiogenesis	Yes	No
Capillary formation		
Cell culture	1-2 months	No
Clot culture	5 days	No
Endothelium	Plump; increased turnover	Flat; slow turnover
Basement membrane	Thick, multilaminated	Thin, normal
Clinical		
Present at birth	30% (only as red macule)	90% (may not be apparent)
Female/male ratio	3:1	1:1
Tendency for involution	Yes	No
Requires treatment	Infrequently	Frequently
Hematologic	Platelet trapping Thrombocytopenia	Venous stasis Localized
consumptive coagulopathy		
Skeletal	Rare mass effect and hypertrophy	Destruction, hypertrophy, distortion
Angiographic	Well circumscribed; intense parenchymal staining	Diffuse; no parenchyma, phleboliths, AV shunting.

Treatment of arterial lesions usually includes embolization of arterial feeders (Berenstein et al, 1984). If possible, embolization should precede surgery so that the interventional neuroradiologist has access to the entire external carotid system. More permanent agents such as liquid adhesives are recommended when possible. Surgical ligation of feeders is discouraged.

Venous lesions respond to direct in situ injection of ethanol or other sclerosing agents. When coupled with surgery most of these lesions can be improved and sometimes totally ablated.

Secondary vascular lesions

Secondary vascular lesions can be intracranial or extracranial. Intracranial lesions are caused by several small abnormal connections between the arteries and veins of the dura and are referred to as *dural vascular malformations.* These intracranial lesions are similar to slow-flow dural vascular malformations of the cavernous carotid region. Abnormal connections can also develop between the extracranial arteries and veins, similar to high-flow direct vascular malformations of the cavernous carotid region. These extracranial lesions are referred to as *arteriovenous fistulas.*

Dural vascular malformations. Dural vascular malformations (DVMs) associated with auditory symptoms usually occur in or around the posterior fossa. Like DVMs of the cavernous carotid region, they are considered acquired lesions that probably are initiated by developmental (venous hypoplasia or aplasia) or acquired (thrombosis, infection, trauma, or surgery) venous occlusive disease. Presumably, microscopic arteriovenous shunts, normally present in the dura, enlarge until they become symptomatic in an attempt to maintain venous drainage in the face of venous occlusive disease. This can also occur in the supratentorial compartment, most commonly in the carotid-cavernous region; however, these usually do not present with auditory symptoms.

A bruit is probably the most common symptom associated with a DVM of the posterior fossa. However, headaches, cranial nerve palsies, focal central nervous system deficits, seizures, increased intracranial pressure, and congestive heart failure may also occur (Lasjaunis et al, 1986). DVMs associated with cortical venous drainage have a propensity to bleed.

DVMs are not static lesions. Spontaneous regression or progression has been reported. DVMs that are associated with high-flow shunts or cortical venous drainage and those that occur in children should not be expected to regress. Dural vascular malformations usually have a benign course. Therapy is therefore not indicated unless a bruit or associated symptoms can no longer be tolerated or unless there is cortical venous drainage.

The goal of DVM therapy should be the obliteration of enough of the abnormal arteriovenous shunts to make symptoms tolerable and to eliminate cortical venous drainage (Halbach et al, 1987; 1989c; Lasjaunis and Berenstein, 1987e; Picard et al, 1990). If the DVM has an arterial supply accessible by endovascular techniques, embolization is indicated. If arterial feeders are not accessible or embolization is inadequate, endovascular treatment via the venous route is a possibility in some cases (Halbach et al, 1989b). Surgery, radiation

therapy, and estrogen therapy have also been recommended.

Embolization should be preceded by a thorough angiographic evaluation. Liquid adhesive is the preferred embolic agent in most cases. Particles may be indicated when there is concern about delivery of the embolic agent to vital structures. Coils or balloons are used when the endovenous route is chosen (Fig. 3-7).

When performing embolization of a DVM it is important that the complete venous outflow not be abruptly occluded because this may precipitate bleeding or worsening of symptoms. Systemic anticoagulation for several weeks after embolization is therefore appropriate in some cases when there is not significant cortical venous drainage. A staged procedure should be considered when there is a concern about abrupt total occlusion. Complications, including infarcts, cranial nerve palsies, proptosis, and hemorrhage, are predictable, and are related to the territories of embolization and the venous drainage.

Arteriovenous fistulas. Arteriovenous fistulas (AVFs) represent abnormal high-flow connections or fistulas between arteries and veins. The cervical carotid and vertebral arteries or their major branches are most commonly involved. AVFs usually have a posttraumatic etiology like the more common intracranial "direct" vascular malformations of the cavernous carotid artery. Infrequently they are congenital or are caused by vessel erosion by a malignant tumor.

Like arteriovenous fistulas anywhere in the body, symptoms related to AVFs are caused by arterial deprivation in the territory distal to the fistulas or venous hyperpressure or congestion in the draining veins. The clinical presentation of an AVF depends on which arteries are involved. In addition to tinnitus and a bruit, AVFs involving the vertebral artery can produce signs and symptoms of vertebrobasilar insufficiency, spinal cord ischemia, and nerve root compression. AVFs involving the common, internal, or external carotid arteries present with tinnitus and a bruit. Occasionally, cranial nerve palsies are seen and rarely, congestive heart failure is seen if the shunt is large.

The angiographic evaluation includes the study of both vertebral, internal carotid, and external carotid arteries. Main branches of the ECA, especially the occipital artery, are injected depending on the suspected vessels involved. The assessment of collateral blood supply through the circle of Willis is crucial if vessel trapping is considered. This requires cross compression or test occlusion maneuvers.

AVFs are usually caused by large channels between the arterial and venous systems and therefore require an embolic agent of large caliber (Debrun et al, 1979; Lasjaunis and Berenstein, 1987a; Reizine et al, 1985; Scialfa et al, 1979). Successful treatment have been described with a number of agents; however, detachable latex or silicone balloons filled with iodinated contrast are the agents of choice. The balloons are initially introduced via the arterial system, and if possible, are negotiated through the shunt into the venous system. They are then inflated until there is closure of the shunt. Sometimes the balloon will not enter the venous side or the shunt is too big. In this case the parent artery may have to be sacrificed or "trapped". If arterial access to an AVF is inadequate, introduction of balloons or coils via an intravenous route may be attempted in selected cases. Occasionally a liquid adhesive or surgery may also be required (Fig. 3-8).

The success rate of AVF closure varies and depends on which vessels are involved and the size of the shunt. Symptom relief will depend on the degree of closure and the longevity and severity of clinical manifestations. In general, most AVFs can be totally or partially cured with a combination of modern endovascular and surgical techniques.

Temporary and Permanent Occlusion of Carotid Artery

Permanent occlusion of the ICA often becomes necessary in the treatment of certain skull base lesions (Hibbert, 1989). Nishioka (1966) demonstrated that occlusion of the common carotid artery or ICA in patients with intracranial aneurysms carries a 30% risk of ischemia of the ipsilateral cerebral hemisphere. In 21% of these cases, onset of deficits is delayed for more than 48 hours after occlusion. The risk of carotid occlusion in a patient with a head and neck malignancy is thought to be similar. Inadequate collateral circulation and thromboembolism are thought to be the two causes of ischemic complications (Fox et al, 1987).

Although intraoperative means have been advocated to assess a patient's tolerance of permanent carotid artery occlusion, preoperative testing is preferred to identify those patients who will have inadequate collateral circulation after this alteration in cerebral blood flow. As early as 1910, Matas (1911) recommended a temporary occlusion test to predict a patient's tolerance for carotid occlusion.

The ICA may be temporarily occluded by a balloon catheter, which is more effective and reproducible than digital compression (Matas, 1911; Matsuda et al, 1988; McKissock et al, 1960; Tolle and Bevilacqua, 1963; Webster and Gurdjian, 1958) and less invasive than a ligature or clamp. Gradual occlusion with ligatures or clamps has not been shown to be superior to abrupt occlusion with a balloon catheter (Brice et al, 1964; Heros, 1984; Landolt and Millikan, 1970).

In addition to clinical examination, with or without provocative hypotension, several methods of assessing the adequacy of collateral circulation during the occlusion test have been recommended. Angiography (Jeffreys and Holmes, 1971), electroencephalography, somatosensory evoked potentials (Momma et al, 1987), and stump pressures have been used. Cerebral blood flow (CBF) assessment with 133Xe and external probes (Boysen, 1971; Holmes et al, 1971; Jennet et al, 1966; Leech et al, 1974) or stable xenon with CT scanning (De Fries et al, 1990; Erba et al, 1988; Johnson et al, 1991) improves the sensitivity of test occlusions and gives quantitative information. However, the equipment required for CBF determinations with these techniques is not widely available, and there has been some concern about the effect of xenon itself on CBF (Giller et al, 1990). External probe measurements with 133Xe are easily performed in the angiography suite and offer reproducible quantitative measurements but provide no information about regional perfusion. Stable xenon with CT gives regional information but is cumbersome and requires transfer of the patient to another room with a carotid catheter in place. Recently, 99mTc-hexamethyl-propyleneamine oxime (99mTc-HMPAO) single photon emission computed tomography (SPECT; Matsuda et al, 1988; Monsein et al, 1991; Szabo et al, 1990) and transcranial Doppler ultrasound (Feaster et al, 1990) have also been recommended. These techniques are commonly available, are easily utilized, and offer quantitative information.

Before the occlusion test, a complete diagnostic angiogram is performed. The tip of a balloon occlusion catheter is then positioned at the first cervical level of the ICA whose occlusion is being contemplated. The balloon is inflated for 20 to 30 minutes or less if the patient becomes symptomatic. During this time, we obtain a neurologic examination, electroencephalogram (EEG), contralateral ICA and vertebral angiograms, and stump pressures, and we also inject an intravenous dose of 99mTc-HMPAO for laser SPECT scanning (Fig. 3-9).

Once it has been shown that a patient will tolerate occlusion of the ICA, it may be permanently occluded. This is performed at a later time after all data have been analyzed. Permanent occlusion is achieved by endovascular placement of detachable balloons filled with iodinated contrast. The reason for occlusion of the carotid artery will dictate where the balloons will be placed. If the entire ICA is to be sacrificed, balloons are placed just below the ophthalmic artery, in the siphon, and at its origin. The patient's tolerance for ICA occlusion is again assessed by neurologic examination and EEG before the initial balloon is detached.