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Bharat A. Mehta

Clifford R. Jack Jr.

Roushdy S. Boulos

Suresh C. Patel

James I. Ausman

See next page for additional authors

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Interventional Neuroradiology: Henry Ford Hospital Experience with Transcatheter Embolization of Vascular Lesions in the Head, Neck, and Spine

Authors

Bharat A. Mehta, Clifford R. Jack Jr., Roushdy S. Boulos, Suresh C. Patel, James I. Ausman, and Ghaus M. Malik

Interventional Neuroradiology: Henry Ford Hospital Experience with Transcatheter Embolization of Vascular Lesions in the Head, Neck, and Spine

Bharat A. Mehta, MD,* Clifford R. Jack, Jr, MD,* Roushdy S. Boulos, MD, PhD,* Suresh C. Patel, MD,* James I. Ausman, MD, PhD,† and Ghaus M. Malik, MD†

Innovative developments over the past 15 years have resulted in increased sophistication and effectiveness of therapeutic neuroembolization procedures. As a result, percutaneous transcatheter embolization has assumed an increasingly important role in the management of patients with vascular lesions of the head, neck, and spine. By reducing bleeding during surgery, this technique can markedly facilitate the surgical approach to difficult vascular lesions. The technique may also be applied in cases where surgery is contraindicated.

Therapeutic neuroembolization has been performed in 23 patients with vascular lesions of the head, neck, or spine at Henry Ford Hospital. Devascularization of the extraaxial blood supply was achieved in all cases, and no neurologic complications were encountered. Eighteen patients with vascular tumors of the head, neck, or spine and three patients with arteriovenous malformations (AVM) were embolized preoperatively, resulting in marked facilitation of the surgical procedures. One patient with a dural AVM and one with a spinal metastasis were embolized without surgery. (Henry Ford Hosp Med J 1986;34:19-30)

Transcatheter embolization procedures have assumed an increasingly important role in the management of vascular lesions of the head, neck, and spine in recent years. A variety of innovative developments over the past 25 years have fueled the rapid development of neuroembolization techniques.

In the 1960s, Luessenhop (1) pioneered the injection of silastic spheres into the internal carotid artery (ICA) to embolize intracranial arteriovenous malformations (AVM). Embolization of the extraaxial blood supply of head, neck, and spine lesions was reported by several groups in the early 1970s (2,3). New vascular occlusive agents directed primarily at the extraaxial blood supply of head and neck vascular lesions as well as vascular spinal cord lesions were developed in the late 1970s and early 1980s. These include microspheres, autologous clot, Gelfoam, polyvinyl alcohol sponge (PVA), collagen, lyophilized dura, and liquid polymers (3-7).

Improvements in catheter delivery system technology as well as a more thorough understanding of both the vascular anatomy and the pathophysiology of head, neck, and spine lesions have resulted in the increasingly wide applicability of neuroembolization procedures (3,7-11). The development of digital subtraction angiography (DSA) in the 1970s and the current widespread availability of this technology represents a significant step forward.

Neuroembolization therapy is a rapidly changing field with many areas of active research. Modern treatment of many vascular lesions of the head, neck, and spine crosses many medical disciplines. This necessitates close communication between the

referring clinician, neuroradiologist, and various specialists, particularly the neurosurgeon, otolaryngologist, neurologist, plastic surgeon, radiation therapist, and oncologist. It is important for clinicians to be aware of developments in this field because select patients serve to benefit greatly from the application of these techniques.

This paper discusses only the neuroembolization procedures performed at Henry Ford Hospital. Our nonembolization interventional neuroradiologic procedures are discussed in a separate paper (12). Goals, risks, and techniques for effective neuroembolization are also discussed.

Patients, Methods, and Results

Embolization technique

Technical considerations in therapeutic embolization of vascular tumors and AVMs are nearly identical. A diagnostic angiogram must be obtained before consideration of a therapeutic embolization procedure. The angiogram is reviewed, and a specific therapeutic approach is developed by the team of specialists, including the neuroangiographer, caring for the patient.

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*Department of Diagnostic Radiology, Division of Neuroradiology, Henry Ford Hospital.

†Department of Neurosurgery, Henry Ford Hospital.

Address correspondence to Dr Mehta, Department of Radiology, Henry Ford Hospital, 2799 W Grand Blvd, Detroit, MI 48202.

The patient is admitted to the hospital the evening before the therapeutic embolic procedure. At that time the neuroangiographer will outline for the patient and family the technical considerations of the procedure, the risks, benefits, and goals.

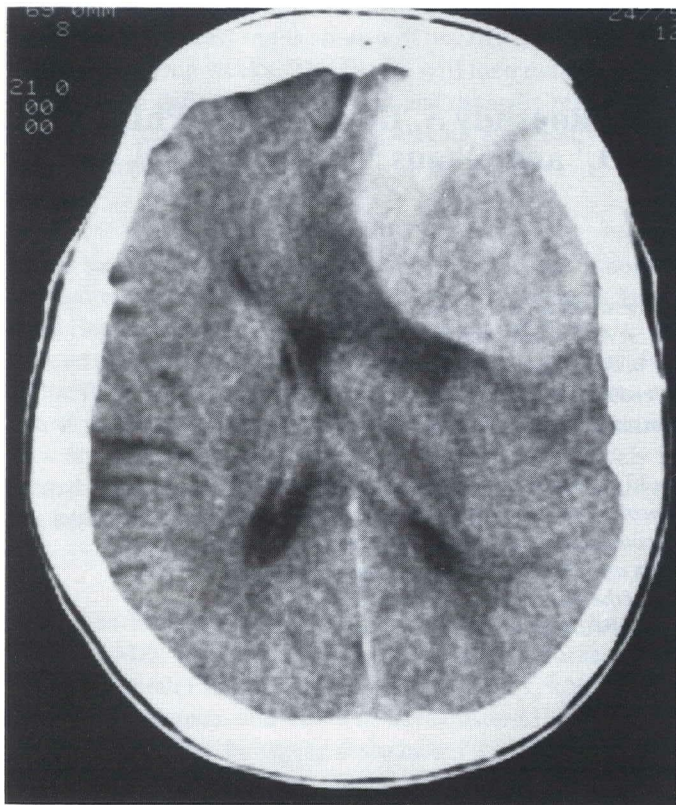


Fig 1(A)—A 60-year-old woman with a large left frontal meningioma: Computed tomography (CT) scan.

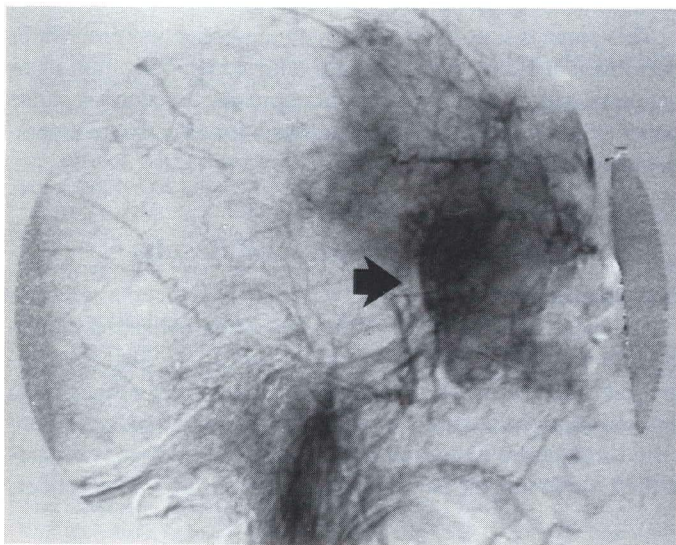


Fig 1(B)—A 60-year-old woman with a large left frontal meningioma: Preembolization left external carotid artery injection late venous phase demonstrating diffuse tumor stain (arrow).

The therapeutic procedure is performed with the patient awake, under mild sedation and mild analgesia. The procedure is done by introducing a thin-walled nontapered angiographic catheter, usually via the transfemoral approach, into branches of the external carotid artery (ECA). Beginning with the largest, the individual arterial branches supplying the lesion are superselectively catheterized, then embolized. Embolization is performed by careful hand injection of a slurry, which is a mixture of embolic particles, heparinized saline, and x-ray contrast material. The injection is done under fluoroscopic monitoring to prevent reflux of the slurry into proximal arteries supplying normal tissue. The ideal end point is defined by obliteration of the abnormal capillary vascular stain in the lesion while preserving the integrity of the main feeding arteries. Frequent test (DSA) runs are obtained to evaluate the approach to the end point.

Case material and results

Twenty-three patients have undergone neuroembolization procedures at Henry Ford Hospital from 1981 through 1985. All procedures were performed by one of the authors (Mehta) in the Department of Radiology, Division of Neuroradiology. These 23 patients can be conveniently grouped into four categories based on the therapeutic goal of the interventional procedure and the underlying pathology.

Preoperative tumor embolization—Eighteen patients underwent preoperative particulate embolization for devascularization of head, neck, and spine tumors. Patients' ages ranged from 5 to 74.

Tumor blood supply derived from external carotid artery branches was successfully embolized in five patients with meningioma (Fig 1), one with a temporal malignant fibrous histiocytoma, and one with a gliosarcoma (Fig 2). The arterial

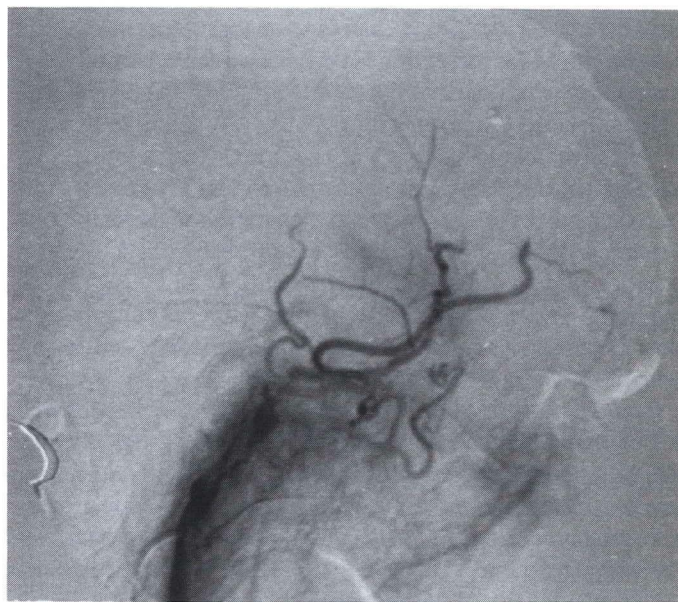


Fig 1(C)—A 60-year-old woman with a large left frontal meningioma: Postembolization left external carotid artery injection demonstrating obliteration of the tumor vascular stain with preservation of the main external carotid arterial trunks.

supply (intercostal arterial branches) to a metastatic hypernephroma involving the T3 vertebral body was also successfully embolized (Fig 3).

Embolization was successfully performed in five patients with juvenile nasopharyngeal angiofibroma (Fig 4), three with glomus tympanicum (Fig 5), one with glomus jugulare, and one with a carotid body tumor.

A slurry of Gelfoam particles (UpJohn, Kalamazoo, MI) was the embolic agent used in all 18 cases. Devascularization of the extraaxial blood supply to the tumor was achieved in all cases and no neurologic complications were encountered. All patients had surgery within 24 hours. In all 18 cases, preoperative tumor devascularization decreased blood loss during surgery and enabled the surgeon to achieve a more complete and less complicated tumor resection.

Tumor embolization without surgery—A patient with a single tumor was treated with embolization and radiation therapy without surgery. This elderly man had a highly aggressive metastatic hypernephroma to the osseous structures comprising the craniocervical junction. This unresectable metastasis had rapidly destroyed the C1 vertebral body, part of C2, and the foramen magnum, which resulted in marked craniovertebral instability. Each of the three ECA branches feeding the tumor mass was superselectively catheterized by femoral approach and injected with 1 mL of dehydrated ethyl alcohol (Abbott Laboratories, Chicago, IL) (Fig 6). This resulted in complete ablation of tumor vascularity and immediate pain control without neurologic complication. The patient eventually died of unrelated problems.

Preoperative arteriovenous malformation embolization—The extraaxial blood supply was embolized preoperatively in three patients with head and neck arteriovenous malformations, without neurologic complication. Embolization resulted in facilitation of the ultimate surgical resection in each case.

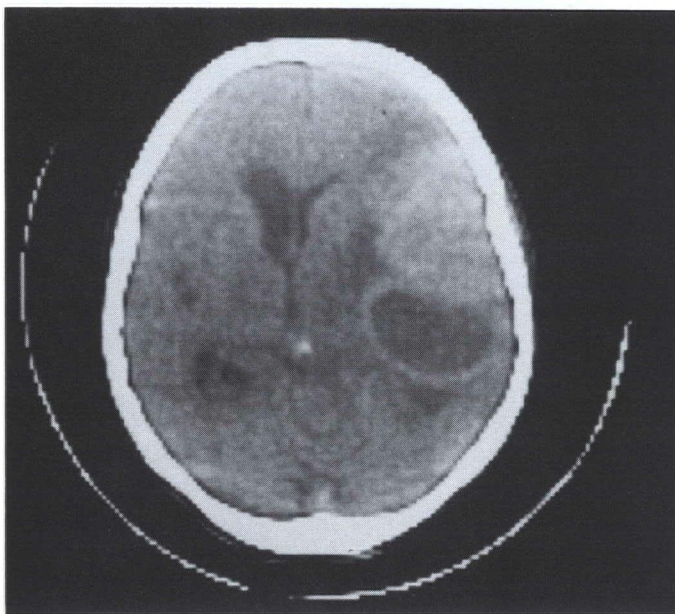


Fig 2(A)—A 55-year-old woman with a left frontotemporal gliosarcoma: CT scan.

A 29-year-old man had a facial AVM, a cirroid aneurysm of the scalp, involving the forehead, with both ECA and ophthalmic artery supply. The ECA supply was successfully embolized with polyvinyl alcohol sponge (PVA) (Unipoint Ind., Highpoint, NC). This resulted in immediate and marked improvement in what had been a significant cosmetic facial deformity (Fig 7).

A 34-year-old woman had recent intracranial hemorrhage and dense left hemiparesis from a known high-flow right frontal AVM. This huge AVM had both intraaxial and extraaxial supply. The latter arose from branches of the external carotid arteries bilaterally. The ECA branches supplying the AVM were embolized bilaterally with PVA, resulting in reduced ECA flow and facilitation of the AVM resection (Fig 8).

A 48-year-old woman had a sudden onset of right hemiplegia and aphasia due to intracerebral hemorrhage. Angiography revealed a dural AVM along the left tentorial leaf which drained into the vein of Galen. The ECA branches were embolized with several Giantro coils (Cook, Bloomington, IN), which resulted in facilitation of surgical resection.

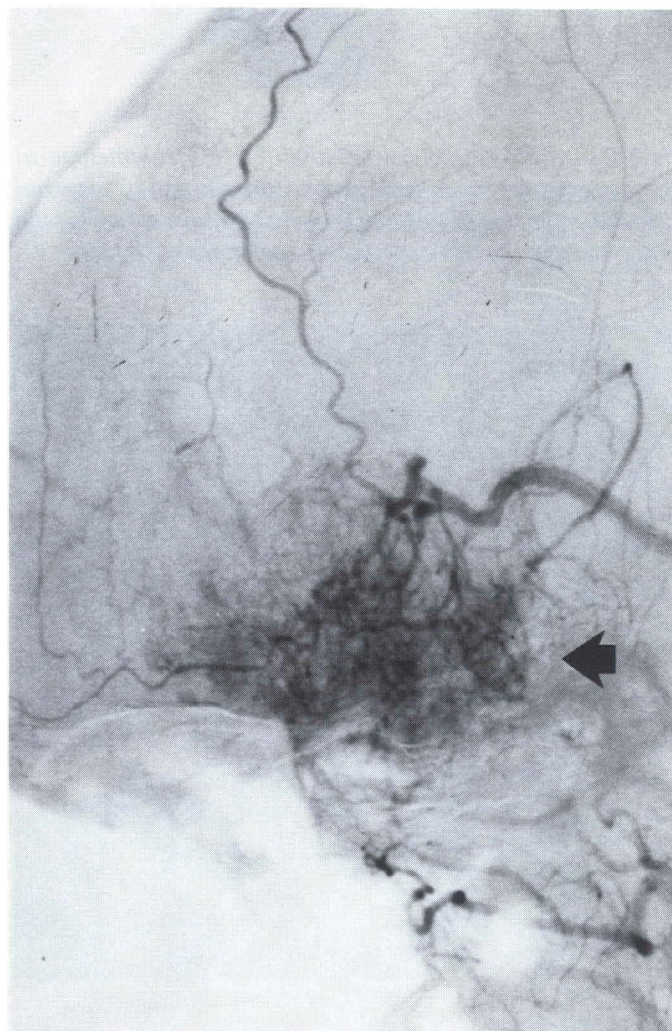


Fig 2(B)—A 55-year-old woman with a left frontotemporal gliosarcoma. Lateral view: preembolization left external carotid artery injection shows extraaxial tumor supply (arrow).

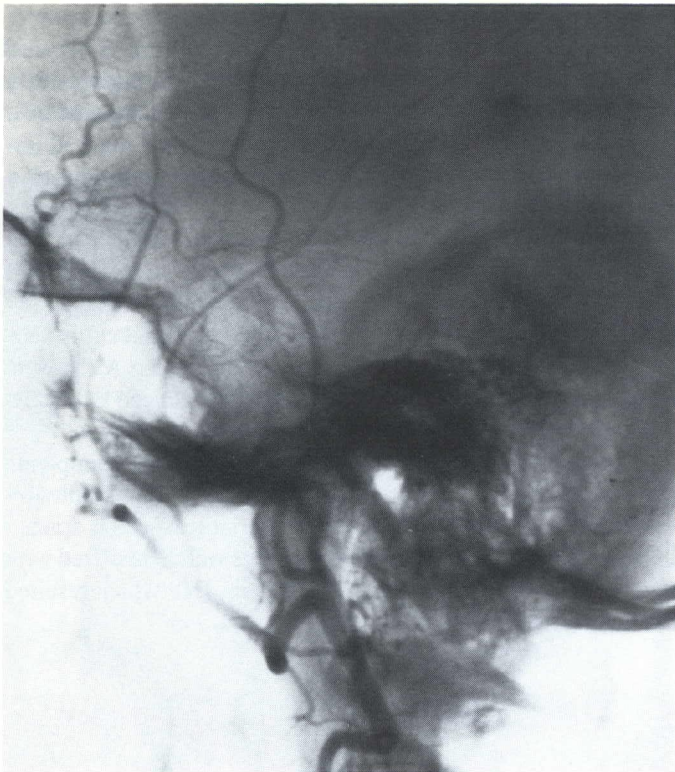


Fig 2(C)—A 55-year-old woman with a left frontotemporal gliosarcoma. Lateral view: postembolization left external carotid injection demonstrating patient's main external carotid artery trunks with obliteration of the tumor vascular stain.



Fig 3(A)—Postmetrizamide myelogram CT in a 58-year-old man with metastatic hypernephroma to the right side of the T3 vertebral body (larger arrow) with partial effacement of the metrizamide filled subarachnoid space (smaller arrow).

Arteriovenous malformation embolization without surgery—A single AVM patient was treated with embolization alone without surgery. She had a subjectively intolerable intracranial bruit due to a large dural AVM of the right transverse sinus and was deemed a nonsurgical candidate due to advanced age (85) and the extensiveness of the lesion. Embolization without surgery was felt to be the safest therapeutic alternative for this patient. The ECA branches supplying this lesion were embolized with PVA, which resulted in nearly complete but temporary angiographic occlusion and cessation of symptoms. The symptoms, however, recurred at 48 hours, and the patient refused further angiography which would have provided an understanding of the recurrent symptoms and treatment options. No neurologic complication was encountered (Fig 9).

Discussion

In recent years, percutaneous transcatheter embolization has played an increasingly important role in the management of AVMs and vascular tumors of the head, neck, and spine (2-4,9). The goals, risks, and technique of embolizing vascular head, neck, and spine tumors are essentially identical to those for embolizing AVMs.

Anatomic considerations

The normal blood supply of head and neck structures is categorized as either intraaxial, extraaxial, or both. Intraaxial supply consists of internal carotid artery (ICA) and basilar artery branches which supply brain parenchyma. Extraaxial supply

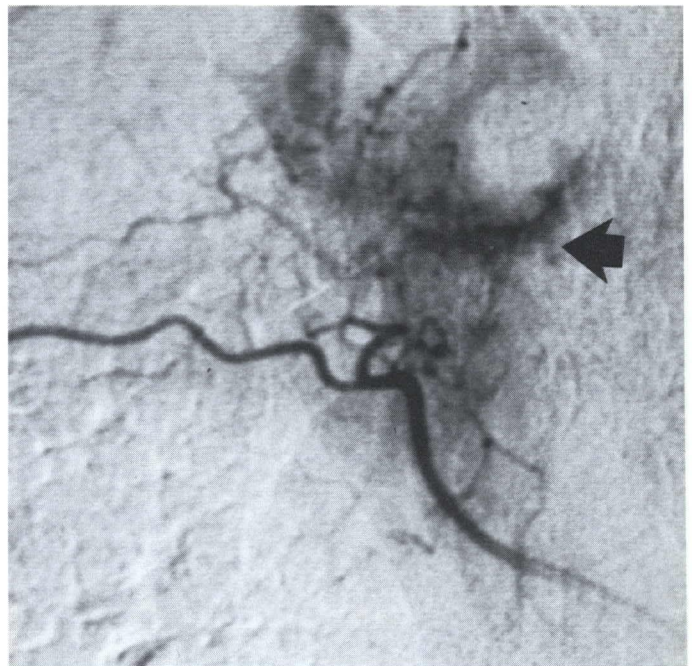


Fig 3(B)—Preembolization injection of the right supreme intercostal artery demonstrating tumor vascular stain (arrow).

consists of external carotid artery (ECA) branches and specific small branches of the ICA and vertebral artery which supply nonbrain tissue, eg, skin, bone, dura, etc. (13). The extraaxial blood supply of head and neck lesions can be embolized without damage to neurologic tissue.

In the spine, branches of the intercostal arteries (radiculomedullary branches) which supply normal spinal cord tissue (most notably the artery of Adamkiewicz) should not be embolized. The arterial supply to bone and paraspinous soft tissue can be embolized.

The most important guideline in all embolization procedures is to achieve selective deposition of particulate embolic material in abnormal tissue while sparing normal tissue (13). This is particularly critical in head, neck, and spine embolization procedures where accidental embolization of the central nervous system could cause a devastating neurologic deficit. Knowledge of primary and collateral vascular pathways to the brain, cranial nerves, and spinal cord is essential.

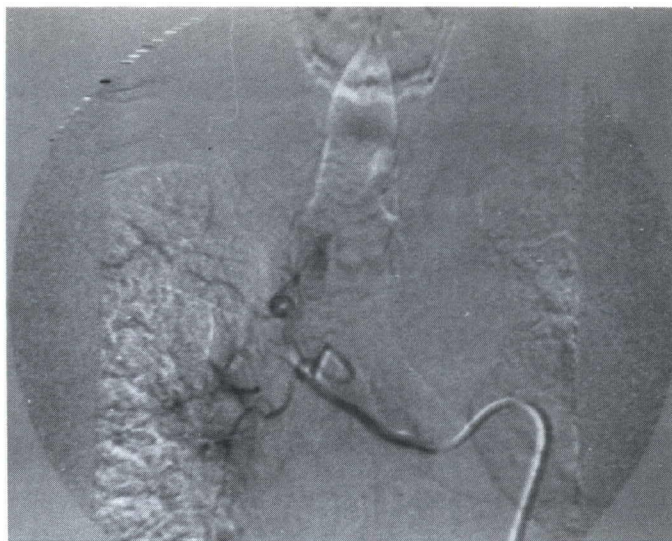


Fig 3(C)—Postembolization injection of the right supreme intercostal demonstrating obliteration of tumor vascular stain.



Fig 4(A)—CT scan of a 14-year-old boy with a large right-sided juvenile nasopharyngeal angiofibroma (arrow).

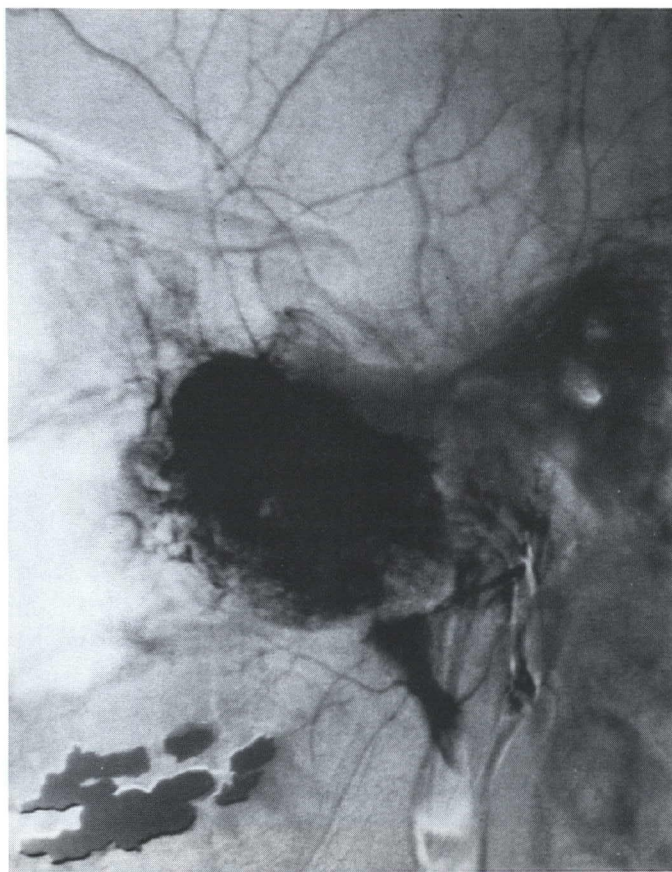


Fig 4(B)—Preembolization. Lateral view right external carotid injection showing intense tumor vascular stain.

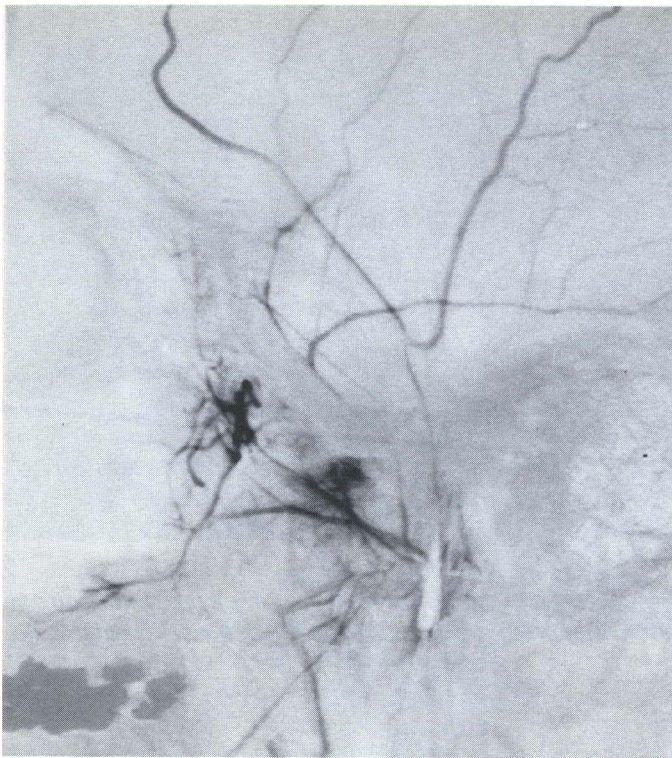


Fig 4(C)—Postembolization injection showing tumor devascularization.

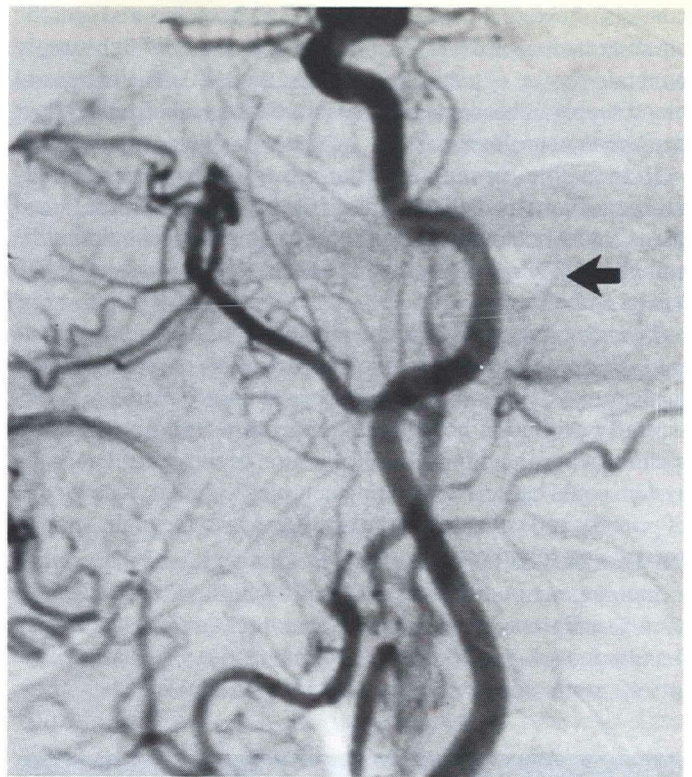


Fig 5(B)—A 56-year-old man with a right-sided glomus tympanicum. Postembolization: lateral view right common carotid injection showing obliteration of tumor vascular stain (arrow).

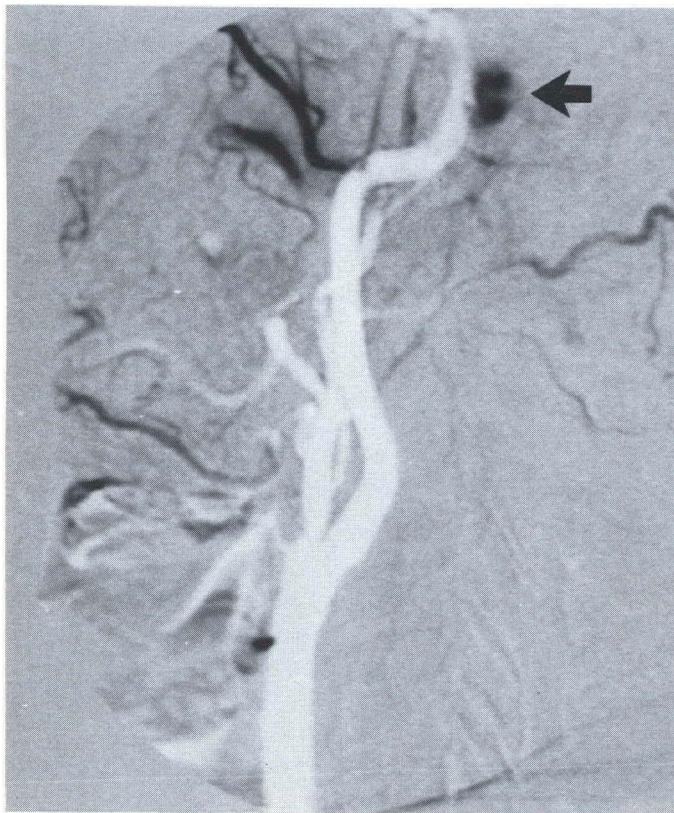


Fig 5(A)—A 56-year-old man with a right-sided glomus tympanicum. Preembolization: lateral view right common carotid injection showing obliteration of tumor vascular stain (arrow).

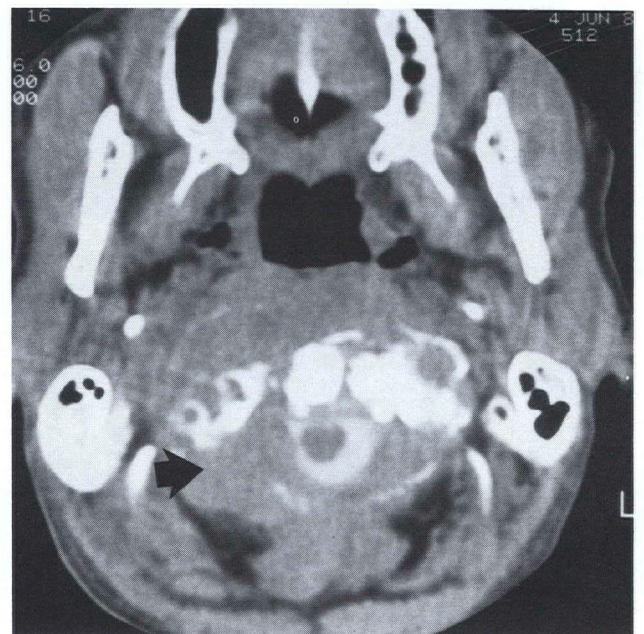


Fig 6(A)—Postmetrizamide myelogram CT of a 78-year-old man with metastatic hypernephroma destroying the body of the C1 vertebral body (arrow).

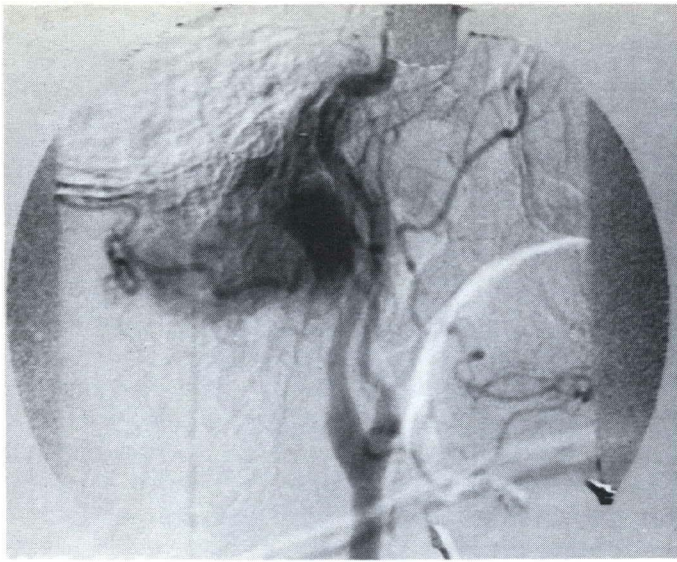


Fig 6(B)—Lateral view: preembolization right common carotid artery injection showing obliteration of tumor vascular stain involving C1 vertebral body and surrounding soft tissues.

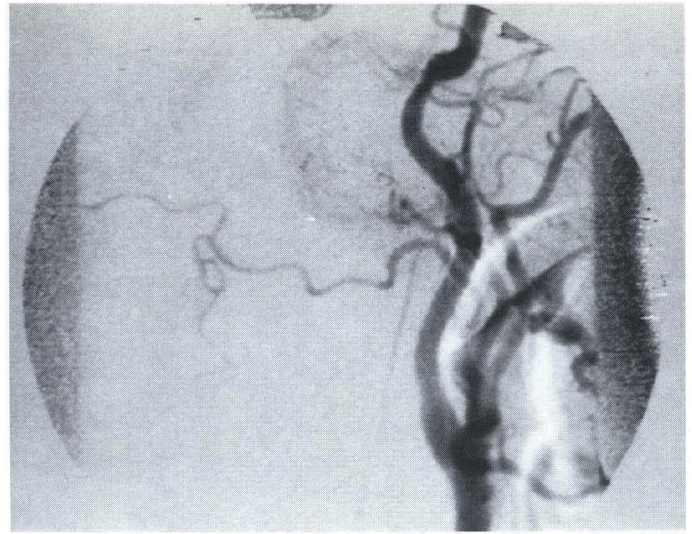


Fig 6(C)—Lateral view: postembolization right common carotid artery injection showing obliteration of tumor vascular stain involving C1 vertebral body and surrounding soft tissues.



Fig 7(A)—A 29-year-old man with a facial AVM of the scalp. Lateral view: preembolization left external carotid injection arterial phase showing arterial supply from ECA branches as well as the AVM nidus (arrow).

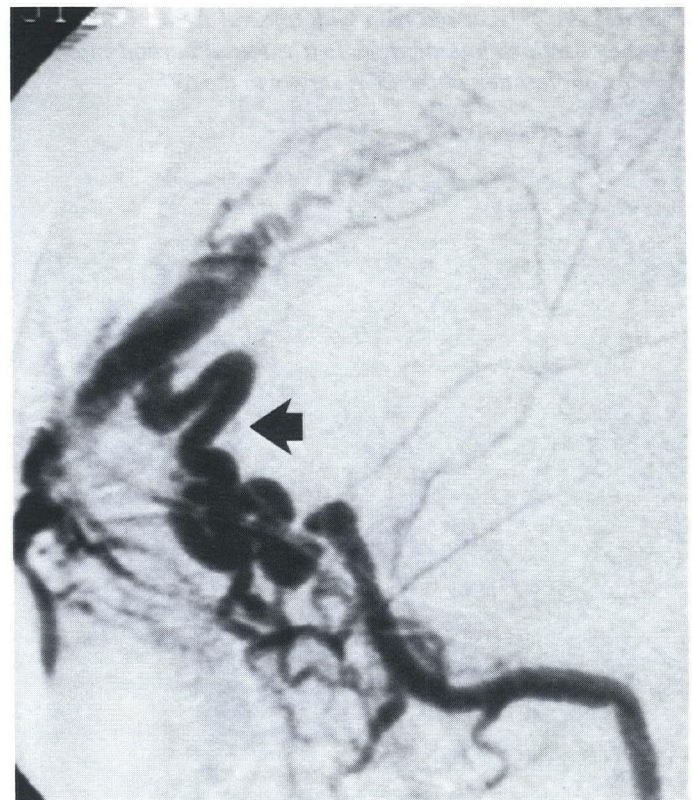


Fig 7(B)—A 29-year-old man with a facial AVM of the scalp. Venous phase of Fig 7(A) injection lateral film showing large scalp vein draining the AVM (arrow).

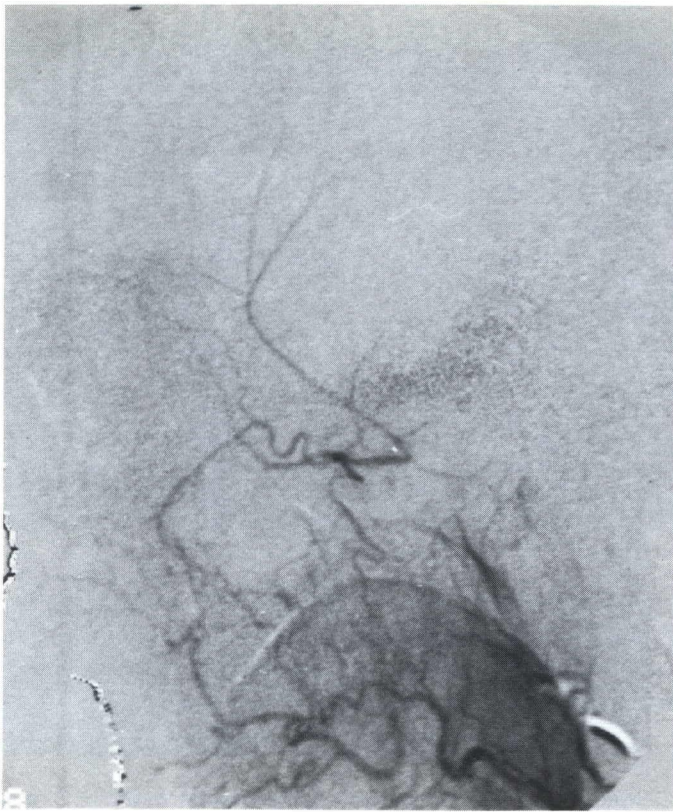


Fig 7(C)—A 29-year-old man with a facial AVM of the scalp. Postembolization: lateral view. Left external carotid injection showing obliteration of the ECA supply to the AVM.



Fig 8(A)—A 34-year-old woman with a huge right frontal AVM with both intraaxial and extraaxial supply. Lateral view: right ICA injection showing intraaxial supply to AVM.

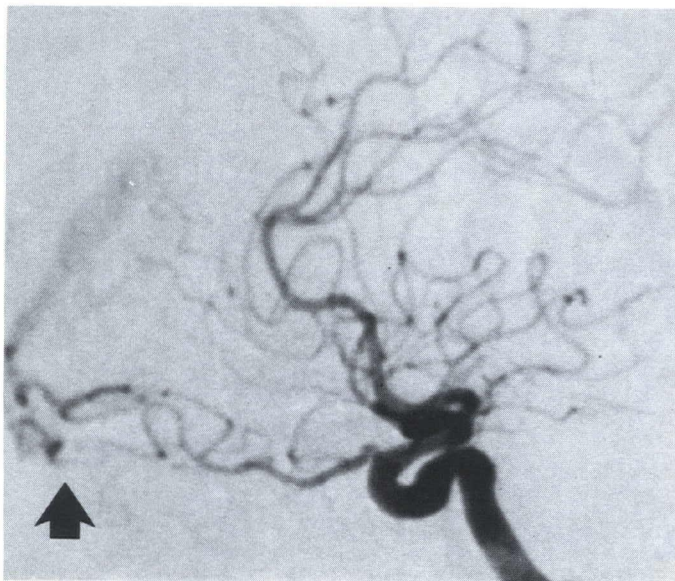


Fig 7(D)—A 29-year-old man with a facial AVM of the scalp. Postembolization: lateral view. Left internal carotid injection showing residual ICA AVM supply from the ophthalmic artery which could not be embolized.

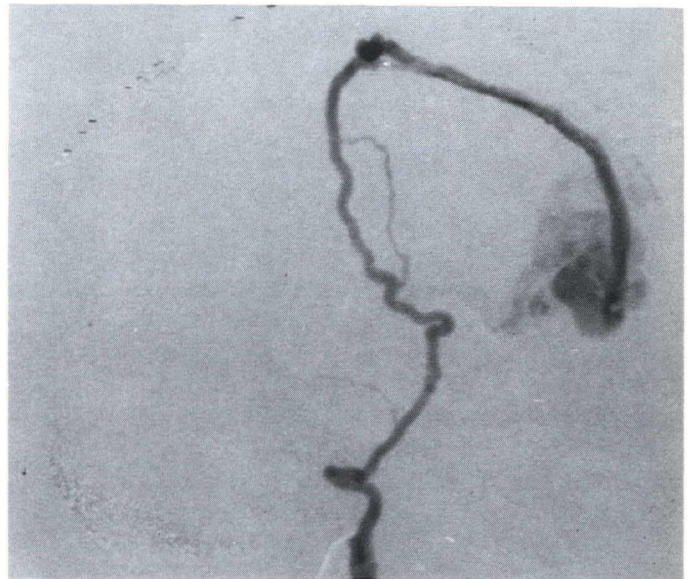


Fig 8(B)—A 34-year-old woman with a huge right frontal AVM with both intraaxial and extraaxial supply. Preembolization left selective middle meningeal artery injection showing obliteration of this extraaxial feeder.

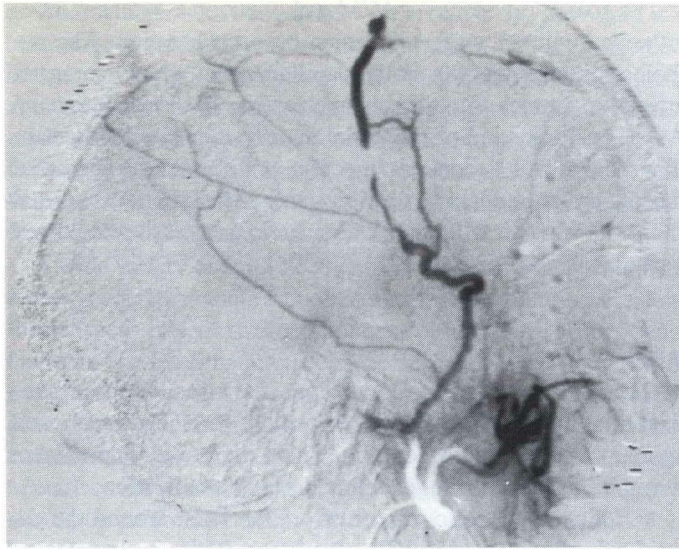


Fig 8(C)—A 34-year-old woman with a huge right frontal AVM with both intraaxial and extraaxial supply. Postembolization left selective middle meningeal artery injection showing obliteration of this extraaxial feeder.



Fig 8(E)—A 34-year-old woman with a huge right frontal AVM with both intraaxial and extraaxial supply. Postembolization left selective superficial temporal artery.



Fig 8(D)—A 34-year-old woman with a huge right frontal AVM with both intraaxial and extraaxial supply. Preembolization left selective superficial temporal artery.

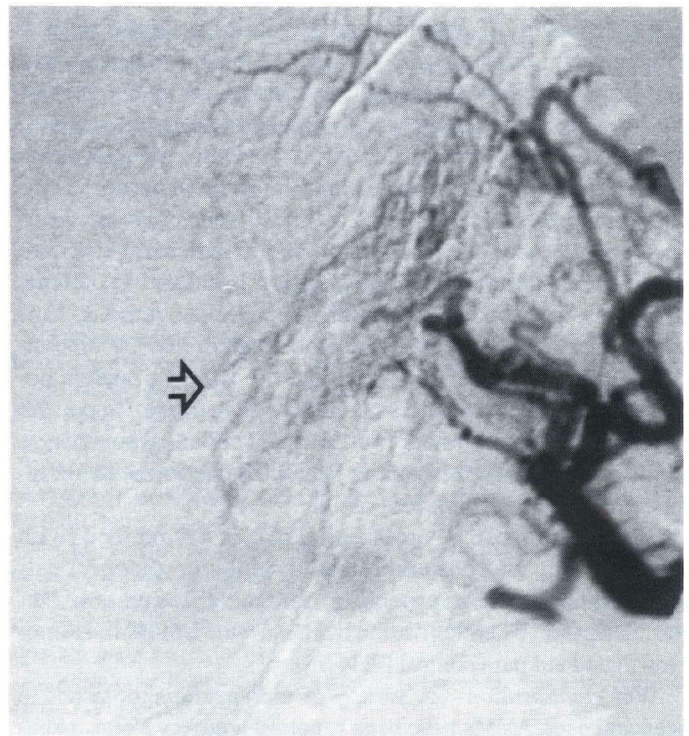


Fig 9(A)—An 85-year-old woman with a large inoperable dural AVM of the right transverse sinus. Postembolization: lateral view. Selective right MMA injection showing devascularization of the arterial rete (arrow) which had supplied the AVM nidus.

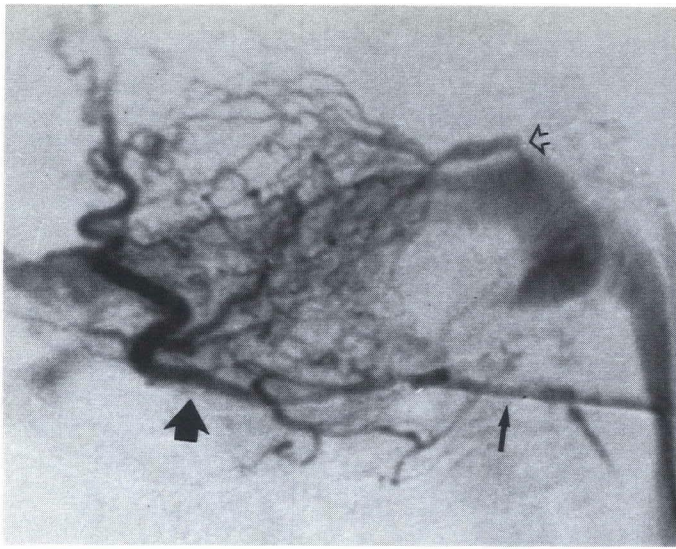


Fig 9(B)—An 85-year-old woman with a large inoperable dural AVM of the right transverse sinus. Preembolization: selective percutaneous needle (smaller arrow) puncture of the right occipital artery (larger arrow) from which a rete of small arterial feeders supply the dural AVM nidus (hollow arrow).

Goals

Tumor embolization—The goal of preoperative embolization of head, neck, and spine tumors is tumor devascularization. Selective deposition of embolic material within the capillary bed of a tumor can promote thrombosis within the tumor vascular bed. This greatly facilitates surgical resection primarily by decreasing bleeding during surgery. In addition to devascularization, preoperative embolization may shrink the tumor. In many cases preoperative embolization may make complete or more complete tumor resection possible (2,8,13). Hospital stays are shortened and the need for transfusion and its attendant risks are diminished. Preoperative embolization is helpful in patients who refuse transfusion for personal or religious reasons. This beneficial aspect of preoperative embolization may become increasingly important if concern about the safety of random donor blood product transfusion continues to increase. To prevent revascularization of tumors from adjacent collateral sources, as would occur with AVMs, surgery should be performed after tumor embolization whenever possible.

Embolization can be an essential part of a combined (with chemotherapy and postembolization radiation therapy) palliative therapeutic approach to unresectable tumors. Devascularization by embolization has been shown to inhibit tumor growth and aid pain control (2,14,15).

AVM embolization—A variety of therapeutic approaches to head and neck AVMs have been reported (surgery alone, radiation therapy, injection of sclerosing agents, and systemic steroids) (9). Surgical resection of facial AVMs is often technically difficult. Current controversy centers on whether surgery alone, embolization alone, or a combined approach will yield the best results.

The goal of preoperative AVM embolization as well as embolotherapy without surgery is lesion devascularization. The pathology in an AVM lies at the capillary level where abnormal arterial-to-venous shunting channels exist. To devascularize an AVM, occlusive embolic material must be deposited at the capillary level. This is done by injecting emboli into the abnormal feeding artery and letting the flow carry emboli to the abnormal shunt site where it will lodge and result in progressive occlusion of shunt channels (2,3,11,13).

Risks

Damage to normal neurologic tissue due to inadvertent embolization is the most serious risk associated with head, neck, and spine embolization procedures. Three different mechanisms may give rise to neurologic deficit during embolization:

1. Reflux of embolic material from the ECA around the carotid bifurcation into the ICA circulation. This can be avoided by using a slow hand injection technique (under fluoroscopic guidance) so as not to elevate the pressure in the ECA vessel being embolized (13). The risk of reflux increases as the end point is approached due to increasing stasis in the artery being embolized (8). Liberal use of DSA test runs to carefully monitor approach to the end point also will reduce the possibility of reflux.

2. Passage of embolic material through normally present extraaxial to intraaxial collateral channels may also produce stroke. An example of this type of normal communication is anastomoses between the occipital and vertebral artery at the C1 level. Knowledge of the anastomotic pathways and careful attention to their possible presence on the preembolization angiogram is a must. Superselective placement of the catheter tip beyond these communications will greatly reduce the risk of stroke by this means (13,16).

3. The blood supply to the extracranial portions of most cranial nerves is derived from ECA branches. Palsy of the 2nd and 3rd division of the trigeminal nerve and cranial nerve 7 has been reported following embolization of the middle meningeal artery (MMA). Palsy of cranial nerves 3, 4, 6, and 5 (first division) have been reported following embolization of normal variants of the MMA. Embolization of the neuromeningeal branch of the ascending pharyngeal artery can result in cranial nerve 7, 9, 10, 11, and 12 palsy (8,17). These cranial nerve palsies nearly always resolve in less than one year. A positive test injection of lidocaine into the artery to be embolized prior to embolization will alert the neuroangiographer to this potential complication.

Skin necrosis has been reported when too forceful an injection of fine particulate embolic material is employed (13). Complications which are seen in most cases include postembolization fever and local pain. These are easily treated with preprocedural and postprocedural steroids and analgesics. The symptoms nearly always resolve within days.

Technical considerations

Technical procedures are frequently long and tiring for both the angiographer and patient and may require several sittings before ideal results are achieved. The use of DSA has proved to be

an incalculable asset during these procedures. By providing instant angiographic feedback with subtraction, DSA greatly decreases the length of the procedure, the attendant's discomfort, and fatigue of both patient and angiographer. DSA also has made possible the practice of multiple test runs during the procedure for meticulous monitoring of progress toward end point. Prior to the availability of DSA, this practice while desirable from a safety standpoint was not realistic.

A wide variety of embolic agents have been made available in recent years. Particulate embolizations reported herein were performed with one of two compounds, either Gelfoam or PVA. Gelfoam is a temporary vascular occlusive agent. The material is degraded by proteolytic enzymatic pathways. The dissolution of the intravascular thrombus it produces begins 24 hours after embolization (6). Therefore, this material is used only for preoperative embolization. For optimum hemostatic effect, surgery should be performed within 24 hours of embolization (6). The key advantage of this material is ease of handling. Due to low viscosity, a Gelfoam slurry will flow smoothly through the catheter, allowing controlled physiologic embolization (6,13). An additional advantage is that normal vessels inadvertently embolized will eventually recanalize due to the temporary nature of this occlusive material.

Conversely, PVA is a permanent occlusive agent. PVA particles serve as a "scaffolding" which promotes the ingrowth of fibroblasts (5,6). This fibroblastic proliferation results in permanent vascular occlusion, with PVA an "integral" part of the body (5,6,18). PVA is used if surgery is not planned immediately or at all. The disadvantage of PVA is that it is more difficult to handle. A slurry of PVA particles is very viscous (5,6). Therefore, it is difficult to achieve a smooth, controlled introduction through the catheter and the risk of inadvertent embolization of normal vessels is higher. Also, accidental embolization of normal vessels will be permanent.

Transcatheter embolization of an unresectable metastasis to the craniocervical region was performed with ethanol in one patient. Ethanol has been shown in animal studies to be an excellent neurovascular sclerosing agent. When perfused directly into the middle cerebral artery of monkeys, focal segmental vascular sclerosis is produced with no distal endothelial injury and no seizure activity (19). Ethanol fulfills all the requirements of an ideal vascular occlusive agent: low viscosity, nonadhesive, inexpensive, readily available, and easy to sterilize (19).

Ethanol has been proven effective in human use for ablation of renal cell carcinoma (14). Used in this setting, it produces complete tumor necrosis and permanent arterial occlusion. This was the effect desired in the patient with end stage metastatic disease to the craniocervical junction. The mechanism of action appears to be direct cellular damage to the endothelium which is followed later by thrombosis (14,15).

The one dural AVM embolized with intravascular coils was performed early in our embolization experience. This technique results in arterial occlusion proximal to the AVM nidus and would not be our method of choice today (13).

Much remains unknown in the field of therapeutic neuroembolization. Initial vascular occlusion postembolization depends on a variety of factors including particle size, thrombogenicity of embolic material, the method of injection (selectively and

control of blood flow), and hemodynamic characteristics of the lesion to be embolized. The precise interrelationship of these variables warrants further study (3,6,13). Little is known about embolization dynamics. The status of the embolized vascular bed changes throughout an embolization procedure. The relative roles of vessel spasm, thrombosis, and histologic reaction of the vessel wall in producing vascular occlusion need to be evaluated (3,6,13).

Summary

Therapeutic embolization techniques can be applied to a variety of vascular lesions of the head, neck, and spine. Our experience with these procedures has been summarized in four broad categories: preoperative tumor embolization, tumor embolization without surgery, preoperative arteriovenous malformation embolization, and arteriovenous malformation embolization without surgery. With careful attention to technique, these procedures can be performed with minimal risk and with excellent therapeutic results. This is a rapidly changing field with many areas of active research. A broad range of clinical specialists should be aware of these techniques, as potential embolization candidates may be seen in many different clinical specialty areas.

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