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Kenneth R. Davis[1]

This article reviews arterial embolization of epistaxis and juvenile nasopharyngeal angiofibromas. A protocol for the complete and rapid exploration of epistaxis is suggested that is based upon which arteries providing blood to the nasal fossa may be responsible for the bleeding and the most optimal way for their demonstration and evaluation. The relevant anatomy of the major arteries, their branches, and important anastomoses are described. The goals of embolization are to control severe or recurrent epistaxis, prevent recurrence if possible, and avoid occlusion of any vessels not responsible for the hemorrhage. The most appropriate embolic material is chosen, realizing that recurrence can be caused by the use of resorbable embolic material or by a very proximal or too complete embolization. Embolization may be used for specific causes of epistaxis including idiopathic, hereditary hemorraghic telangiectasia, hemorrhagic tumors, vascular malformations, trauma, disorders of hemostasis, and postsurgical problems. Juvenile nasopharyngeal angiofibromas are discussed with respect to their clinical presentation, classification, computed tomographic, MR imaging, and angiographic evaluation, treatment, future trends in treatment, and precautions and complications.

The purpose of the review of embolization of epistaxis is to describe a method for the investigation of the patient with epistaxis. This will enable the neuroradiologist to formulate an arterial map of the nasal fossa in order to choose the most appropriate site and material for embolization. The anatomy, goals of embolization, embolic materials, uses in specific diseases, postoperative cases, and overall considerations and precautions are covered.

The purpose of the review of embolization of juvenile nasopharyngeal angiofibromas (JNAs) is to emphasize the usefulness of presurgical embolization and to describe a complete and safe method of investigation. The clinical presentations; classifications; computed tomographic (CT), magnetic resonance imaging (MRI), and angiographic evaluations; treatments; future trends; precautions; and complications are covered.

Epistaxis

Anatomy

Protocols that have been established for the investigation of epistaxis require awareness of the presumed approximate site and etiology of the hemorrage, which arteries may be responsible for the bleeding, the most convenient means of demonstrating the arteries, and knowledge of the blood supply to the territory in question [1–3]. An arterial map of the nasal fossa for any given case is then generated and evaluated to make the best choice for the most appropriate site and material for embolization [3]. The vessels studied are those that arise from the internal carotid, maxillary, facial, and ascending pharyngeal arteries (Table 1). Even if there has been a previous ligation of the ethmoidal and maxillary arteries, an

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TABLE 1: **Major Vascular Supply and Branches in Epistaxis Evaluation**

Major Artery: Branch	Area of Interest Supplied	Important Anastomotic Branches
Internal carotid:		
Ophthalmic:		
Anterior and posterior ethmoidal	Ethmoid sinuses and adjacent meninges; nasal septum and conchae by anastomoses	Facial, maxillary, transverse facial
Nasoangular, nasoorbital	Malar region around ala of nose via anastomoses	Facial, maxillary, transverse facial
Capsular	Sphenoid sinus	. . .
Artery of foramen rotundum from inferolateral trunk	Distal maxillary territory	Pathway for revascularization if proximal maxillary occluded
Persistent mandibular (mandibulovidian):		
Mandibular	Nasopharynx	Inferomedial eustachian branch of accessory meningeal, eustachian branch of ascending pharyngeal, pterygovaginal
Vidian	Sphenoid sinus	Petrous internal carotid, sphenopalatine
Maxillary:		
Sphenopalatine:		
Medial nasal	Septum	Ophthalmic anterior and posterior ethmoidal, septal branch of superior labial of facial, alar branch of facial
Lateral nasal	Conchae	. . .
Pterygovaginal	Eustachian meatus (via pterygovaginal canal)	Inferomedial branch of accessory meningeal, eustachian branch of ascending pharyngeal, mandibular branch of internal carotid
Anterior branch of descending palatine	Posterior septum, conchae, and palate	Ophthalmic posterior and anterior ethmoidal
Infraorbital	Maxillary antrum	Facial, transverse facial, ophthalmic nasoangular, nasoorbital hemodynamic balance
Alveoloantral	Maxillary antrum roof	Same as for infraorbital
Accessory meningeal	Nasopharynx, eustachian meatus	Posterior: mandibular branch of internal carotid, superior pharyngeal eustachian branch of ascending pharyngeal; anteromedial: eustachian branch of ascending pharyngeal; anterior: pterygovaginal; inferior: palatine branch to ascending and descending palatine
Pterygopalatine	Palate, nasopharynx	. . .
Facial:	Distal maxillary territory	Pathway for revascularization if proximal occlusion of maxillary
Alar	Ala and external nose structures	Maxillary (infraorbital and alveoloantral), transverse facial, ophthalmic nasoangular or nasoorbital hemodynamic balance
Ascending palatine	Soft palate and nasopharynx	Descending palatine
Septal branch of superior labial	Inferior nasal septum	. . .
Transverse facial	Malar region around ala of the nose	Facial, maxillary (infraorbital and alveoloantral), ophthalmic nasoangular, nasoorbital hemodynamic balance
Ascending pharyngeal:		
Ascending palatine from middle pharyngeal	Soft palate	Descending palatine, accessory meningeal
Superior pharyngeal (eustachian)	Medial and paramedial nasopharynx	Accessory meningeal, mandibular, pterygovaginal, carotid branch to inferolateral trunk and recurrent artery of lacerum from carotid siphon
Ascending palatine:		
May arise from facial, external carotid, accessory meningeal, or middle pharyngeal branch of ascending pharyngeal arteries	Palate	. . .

evaluation of the collateral supply, which is usually from the ipsilateral side, is required.

Branches of the internal carotid artery. Branches of the internal carotid artery may be divided into two groups depending on whether the epistaxis arises primarily in the sphenoid sinus or in the ethmoid cells. The anterior and posterior ethmoidal arteries arise from the third portion of the intraorbital course of the ophthalmic artery and sometimes as a common trunk with the supraorbital artery. They run medially through the anterior and posterior ethmoid canals to reach the ipsilateral ethmoid cells. Most of their supply is to the sinuses and adjacent meninges, but through anastomoses contribute supply to the conchae and nasal septum. The nasoangular or nasoorbital branches of the ophthalmic artery may provide supply to the malar region around the ala of the nose through anastomoses with the facial, maxillary, and transverse facial arteries [1, 3].

The capsular arteries arise from the horizontal cavernous segment of the internal carotid artery to supply the sphenoid sinus and are not usually seen on angiography.

The artery of the foramen rotundum from the inferolateral trunk of the internal carotid artery is a potential collateral pathway for revascularization of the distal maxillary territory when there has been a proximal maxillary occlusion.

The persistent mandibular (mandibulovidian) artery, when present, arises from the petrosal segment of the internal carotid artery and usually divides into two branches in the foramen lacerum. The horizontal branch passes into the pterygoid canal and anastomses with the vidian artery (not usually visible at angiography), giving supply to the sphenoid sinus. The inferior branch, or mandibular artery, anastomoses with the inferomedial eustachian branch of the accessory meningeal, the eustachian branch of the ascending pharyngeal, and the pterygovaginal arteries to provide nasopharyngeal supply [1, 3].

The carotid siphon and cavernous sinus are next to the sphenoid sinus, and massive hemorrhage can result if a communication is created by trauma or rupture of an aneurysm [3].

Branches of the maxillary artery. The maxillary artery supplies the conchae and nasal septum by the medial and lateral nasal branches from the sphenopalatine segment, whereas an anterior branch from the descending palatine artery may also supply the posterior portion of the septum and conchae. The supply to the maxillary sinus is from the alveoloantral and infraorbital artery branches [1–5], and the pterygovaginal and accessory meningeal branches provide supply to the eustachian meatus, nasopharynx, and soft palate.

Branches of the facial artery. The facial artery supplies the ala and external nose. It has a more important role as a pathway for recanalization of an incompletely or completely occluded maxillary artery than as a primary source of hemorrhage and is often responsible for recurrent epistaxis since it carries blood flow to the distal portion of the maxillary artery. Therefore, postembolization studies should be made by injection of the ipsilateral facial artery rather than of the distal external carotid artery [3]. The alar branch supplies the ala and structures of the external nose, and the ascending palatine branch may originate from the proximal facial artery and provides supply to the palate. The lowest part of the nasal septum is supplied by a branch of the superior labial artery, which is not ordinarily visible at angiography and has a questionable role in the production of epistaxis [3]. There is a hemodynamic balance between the facial artery and three other systems in the malar region and around the ala of the nose, which includes the infraorbital and alveoloantral branches of the maxillary artery, the transverse facial artery, and the nasoangular or nasoorbital branch of the ophthalmic artery [1, 3].

Depending on this balance, the supply of the nasal ala may come from the facial, ophthalmic, or transverse facial/internal maxillary systems. It is only when the malar region and ala are predominantly supplied by the facial artery that embolization of this vessel is required, either for treatment of an anterior bleeding point or to prevent early revascularization of a more posterior site that is fed by sphenopalatine artery branches [3].

Branches of the ascending pharyngeal artery. The ascending pharyngeal artery anterior branch gives origin to superior, middle, and inferior pharyngeal branches that supply the medial and paramedial nasopharynx. The middle pharyngeal branch may supply the soft palate by giving origin to the ascending palatine artery, which has anastomoses with the descending palatine and accessory meningeal arteries.

The superior pharyngeal (or eustachian) branch reaches the foramen of the eustachian tube and anastomoses with the corresponding branches of the accessory meningeal and pterygovaginal arteries, as well as with the mandibular artery vestige from the internal carotid artery, when present. The carotid branch originates from the superior pharyngeal branch and anastomoses with the inferolateral trunk and the recurrent artery of the foramen lacerum from the carotid siphon [1, 3]. The vessels of the ascending pharyngeal system usually only require embolization for epistaxis caused by juvenile nasopharyngeal angiofibromas or angiomatosis, as in Rendu-Osler disease [1, 2, 3, 5].

Goals of Embolization

The goal of embolization is palliative [3, 6–11] for control of severe or recurrent epistaxis, whether idiopathic or caused by hypertension, angiomatous diseases, tumors, vascular malformations, trauma, disorders of hemostasis, or postsurgical problems, when the epistaxis is resistant to the usual treatment of packing and/or surgery. Embolization may also be used along with other techniques to reduce blood supply to hemorrhagic tumors or vascular malformations. As an emergency procedure, it can be lifesaving in cases of catastrophic bleeding [3].

Epistaxis can be classified as arterial, venous, or arteriolized venous depending on the source of bleeding. Venous bleeding is usually well controlled by manual compression or simple anterior nasal packing; venous epistaxis sometimes stops spontaneously. Arterial or arteriolized venous hemorrhages are life-threatening unless immediate control is accomplished. This type of bleeding may occur from the venous

drainage of arteriovenous malformations or arteriovenous fistulas, including carotid cavernous fistulas. The primary treatment should be nasal packing, anterior and/or posterior. If the bleeding persists or recurs, then either ligation of the maxillary and possibly the ethmoidal arteries or angiography is indicated. If angiography is chosen, then embolization may be performed at that time. Alternatively, after diagnostic angiography, the maxillary and possibly the ethmoidal arteries may be ligated. If embolization is chosen and fails to control the epistaxis, operative ligation of the maxillary and possibly the ethmoidal arteries may then be performed (Choi IS, personal communication).

Techniques

Nonresorbable embolic materials such as polyvinyl alcohol (PVA) foam or dura mater (available in Europe) should result in fewer recurrences [2, 3, 6]. The endovascular occlusion, however, must be regarded as a potentially repeatable procedure; therefore, it should be located as distal as possible so as not to compromise subsequent treatment [3]. Recurrence from revascularization is normally from vessels ipsilateral to the midline, particularly if the embolization was distal enough to occlude the territory in question [3].

The embolization technique uses superselective catheterization with 4 French and sometimes 5 French catheters. Coaxial use of an open-ended guidewire or a 2 or 3 French inner catheter may be necessary to insert through the 5 French catheter in order to enter a small vessel, such as a branch of the ascending pharyngeal artery. The small lumen, however, only permits passage of small particles or liquid embolic agents. A 4 French catheter will enter small branches that are not possible to catheterize with the 5 French catheter and permits injection of larger particles than through the open-ended guidewire. The internal carotid, maxillary, and facial arteries of the presumed noninvolved side are usually injected first (in that order) so that most of the diagnostic evaluation will be completed before the main feeding vessels to be embolized are entered. The internal carotid artery is studied best on the lateral view and the distal maxillary artery on Water's view—and on the lateral view if the catheter is placed proximal to the origin of the maxillary artery in order to see the transverse facial artery and to assess the facial artery (and therefore possibly obviate injection of the facial artery). The facial artery is then studied selectively, and the lateral view is optimal to examine the supply of this vessel. The side of the hemorrhage is studied next. The internal carotid artery is examined first and evaluated best on the lateral view. The maxillary artery is then studied, and a decision is made as to whether embolization is indicated. A piece of Gelfoam may be used to temporarily occlude the superficial temporal artery if embolization is performed from proximal to the origin of the maxillary artery. The facial artery is then studied after embolization of the maxillary artery to determine whether the embolization was distal and effective, as well as to evaluate for ascending palatine artery supply. Examination of the ascending pharyngeal artery may be required if a posterior source of bleeding is suspected and to check for residual abnormal supply from the ascending or descending palatine, accessory meningeal, pterygovaginal, or mandibular artery.

Embolic Materials

Different embolic agents should be chosen according to the etiology of an individual case. Although Gelfoam particles, which are resorbable, and PVA particles, which are not resorbable, are both commonly used in the United States for endovascular occlusion of idiopathic epistaxis, nonresorbable embolic materials such as PVA should reduce recurrence rates. Some recanalization, however, can be seen several months after PVA embolization. PVA particles have been available in various size ranges including 149–250, 250–590, and 590–1000 μm. They are prepared by washing them with sterile distilled water or saline in a blender to eliminate clumped particles. The fluid is then decanted to remove the formalin. The blender may produce a larger percentage of smaller particles than planned, so extra caution must be exercised during embolization. Contrast material is added to the PVA to provide opacification. Albumin may be mixed with the PVA to prevent clumping and achieve a more distal embolization. Gelfoam powder (40–60 μm) has also been combined with the PVA. Small Gelfoam particles, which are cut from Gelfoam strips and are considerably larger than the Gelfoam powder, generally provide a 2–4 day period of reliable, temporary occlusion. Strips of Gelfoam are usually adequate to stop posttraumatic hemorrhage or bleeding caused by a necrotic tumor from the maxillary artery territory. This permits sparing of the parent vessel and allows for the possibility of more definitive subsequent embolization therapy, if necessary. The use of a fluid embolic agent such as isobutyl-2-cyanoacrylate (IBCA) or alcohol into these arteries is not usually indicated because of the risk of complications from possible embolization of dangerous external carotid to ophthalmic and intracranial anastomoses as well as possible embolization of dangerous external carotid supply to cranial nerves [3]. Very small particles should also be used very cautiously where there are such dangerous communications. Arteriovenous fistulas, however, are often treated best by IBCA or detachable balloons, and the best form of treatment for a false aneurysm may be the detachable balloon. The choice of embolic material, therefore, depends on the type of primary lesion, the severity and site of hemorrhage, and the presence of dangerous anastomoses [6].

Uses for Specific Causes

The causes of epistaxis in which embolization can be considered include idiopathic (spontaneous or hypertensive); hereditary hemorrhagic telangiectasia (HHT) [12–16]; hemorrhagic tumors of the benign type, which include juvenile angiofibroma, hemangioma, and angiomatous polyp, or of the malignant type, which includes ulcerating primary carcinoma (Fig. 1), and hypervascular metastatic tumors such as hypernephromas and melanomas; arteriovenous malformations; trauma with rupture of an artery by direct injury or from an incomplete tear and formation of a pseudoaneurysm; disor-

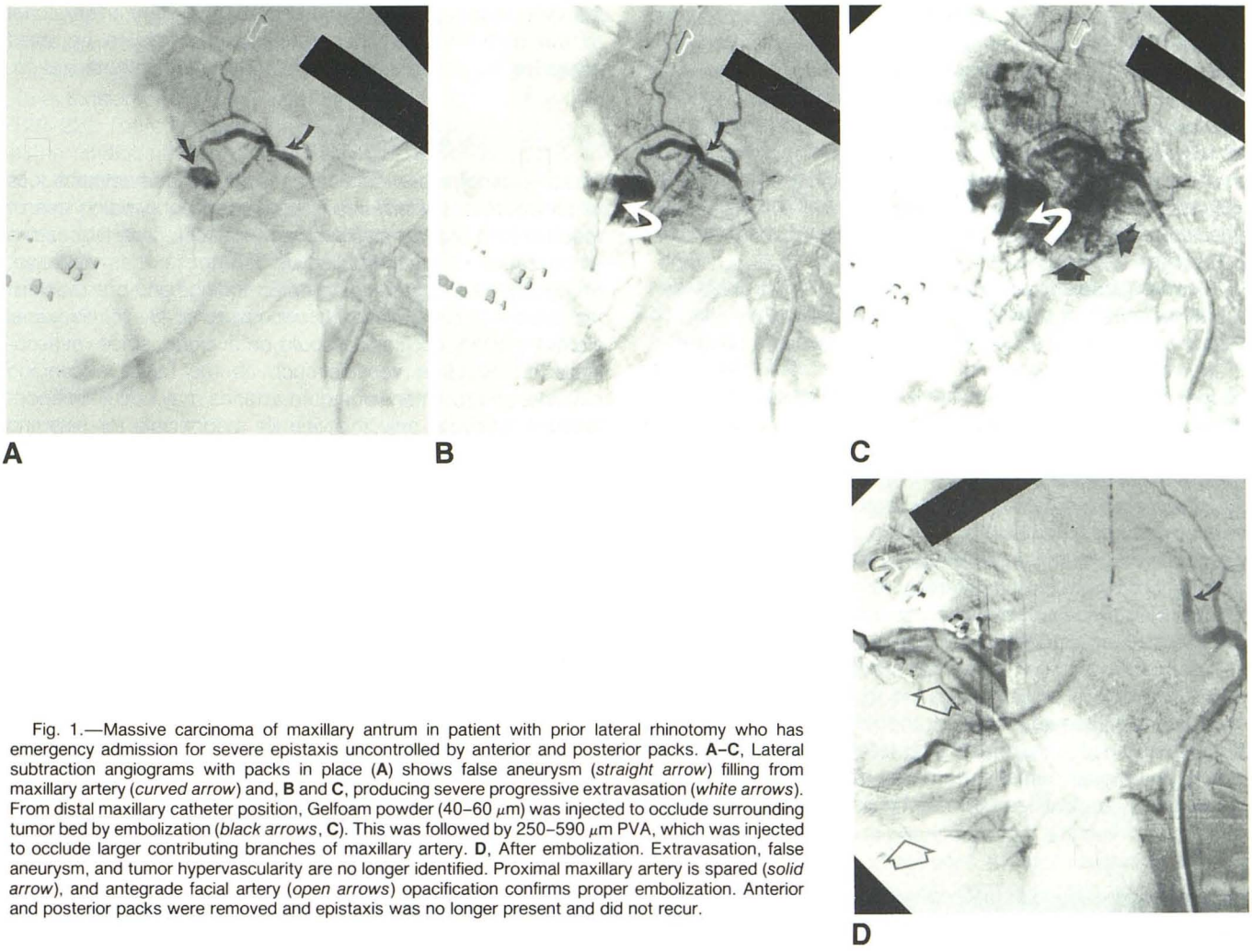

Fig. 1.—Massive carcinoma of maxillary antrum in patient with prior lateral rhinotomy who has emergency admission for severe epistaxis uncontrolled by anterior and posterior packs. **A–C**, Lateral subtraction angiograms with packs in place (**A**) shows false aneurysm (*straight arrow*) filling from maxillary artery (*curved arrow*) and, **B** and **C**, producing severe progressive extravasation (*white arrows*). From distal maxillary catheter position, Gelfoam powder (40–60 μm) was injected to occlude surrounding tumor bed by embolization (*black arrows*, **C**). This was followed by 250–590 μm PVA, which was injected to occlude larger contributing branches of maxillary artery. **D**, After embolization. Extravasation, false aneurysm, and tumor hypervascularity are no longer identified. Proximal maxillary artery is spared (*solid arrow*), and antegrade facial artery (*open arrows*) opacification confirms proper embolization. Anterior and posterior packs were removed and epistaxis was no longer present and did not recur.

ders of hemostasis; and postsurgical problems from instrumentation or poor clip position.

Idiopathic. Angiographic demonstration of the bleeding point is particularly rare in the idiopathic, spontaneous, or hypertensive type of epistaxis, and, therefore, the referring clinician should indicate the approximate presumed site of bleeding. It may be dangerous to remove the nasal packing for the angiographic injection in order to attempt to demonstrate the bleeding point since it may be difficult to immediately replace it while the patient is on the angiographic table. It is, however, reasonable to remove the packing upon completion of the embolization to check the effectiveness of the occlusion. Patients with severe idiopathic (arteriosclerotic, hypertensive) epistaxis can be treated very effectively with embolization of PVA particles when there has been recurrent bleeding after anterior and/or posterior nasal packing.

HTT. Global or selective injection may not show the angiomatous tufts of HHT; however, the maxillary artery is usually the source of the epistaxis. Bilateral facial arterial supply may also be present in certain cases. When estrogen treatment has failed, palliative endovascular occlusive treatment is necessary to control bleeding. Embolization may be performed, first with small PVA particles followed by larger particles [12, 16]. Only the distal symptomatic area should be embolized, and the proximal segment of the main feeder should be left open for possible reembolization.

Hemorrhagic tumors are discussed under Embolic Materials and Juvenile Nasopharyngeal Angiofibromas and are illustrated in Figure 1.

Vascular malformations. Epistaxis from extensive angiomatosis should probably be treated, when feasible, by operative ligation of the feeding ethmoidal branches of the ophthalmic artery prior to embolization of the branches of the external carotid artery, since external carotid embolization may only increase the internal carotid supply [6]. A considerable part of the blood supply in Wyburn-Mason syndrome, however, comes from the ethmoidal arteries, which are often inaccessible to both embolization and clipping and represent

a continuous source of danger to the patient. Embolization should always be only palliative. In these extensive nonoperative lesions, the main vessels should be left open for possible future embolizations, since subsequent and possibly emergency embolizations may be the only means to ensure survival of these patients [6]. Bilateral angiographic evaluation of a vascular malformation is performed even though there may be clinical evidence of a unilateral lesion [1, 3], because vascular malformations of the nasal fold and alar area are sometimes supplied by contralateral arteries.

Trauma. Traumatic epistaxis may occur in association with facial fractures and penetrating injuries as well as with carotid cavernous fistulas. Hemorrhage from a traumatic aneurysm in the region of the cavernous sinus or carotid siphon may occur in the acute stages of injury. Treatment of a traumatic aneurysm of the cavernous carotid performed to prevent a fatal hemorrhage into the nasopharynx (Fig. 2) is an emergency.

Balloon occlusion of the internal carotid artery is performed in acute traumatic injuries, whereas in older traumatic aneurysms fibrotic changes develop in the wall. This permits sparing occlusion of the internal carotid artery since the balloon may be detached within the aneurysm if the neck of the aneurysm is not very wide.

Postoperative cases. With poor surgical clip position, the maxillary artery is sometimes found to be patent at the site of presumed adequate ligation, and embolization may then be accomplished without difficulty. In other cases after ligature, even though direct internal maxillary artery puncture distal to the surgical ligature may be possible [3], an understanding of the collateral supply to the maxillary artery distal to the occlusion may permit these channels to be used effectively for embolization via femoral artery catheterization.

Considerations and Precautions

Occlusion of vessels not responsible for the hemorrhage is to be avoided. Recurrence is prevented by avoiding use of resorbable material, proximal embolization, and excessive embolization. Since embolization for epistaxis is palliative, proper analysis of the angiographic findings and prediction of the collateral circulation are necessary [1–3, 5]. Proximal maxillary artery occlusion should be avoided, since revascularization by other vessels such as the facial, ascending palatine, and foramen rotundum arteries may occur. In spontaneous epistaxis only the vessels responsible for bleeding are embolized to prevent local ischemia and further bleeding. Epistaxis caused by certain blood disorders may require embolization; however, if a low platelet count is present, it must be corrected with prior platelet transfusion to avoid a problem at the puncture site during and on completion of the procedure [3].

Juvenile Nasopharyngeal Angiofibromas (JNAs)

Clinical Presentation

Incidence. JNAs constitute 0.5% of head and neck tumors and are found almost exclusively in adolescent males [17–19]. Only five cases have been reported in females, and

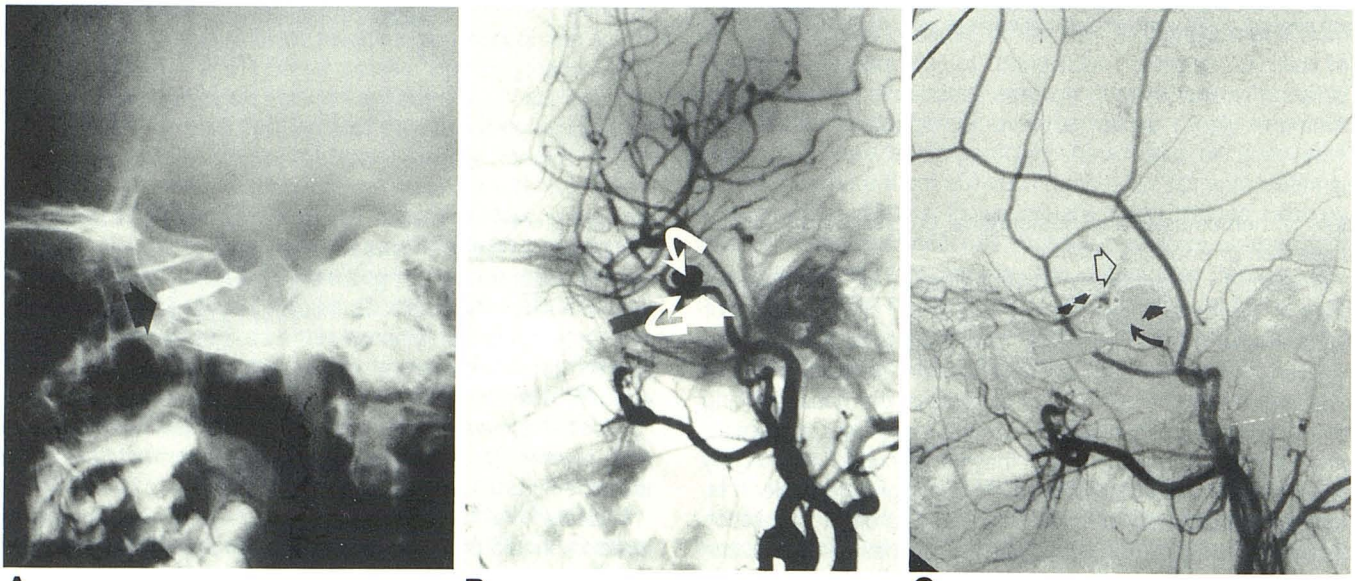

Fig. 2.—9-year-old boy just after puncture by umbrella tip through right orbit. **A**, Lateral skull film. Retained umbrella tip (*arrow*). **B**, Right carotid angiogram. False aneurysm (*curved arrows*) from cavernous internal carotid artery with narrowing and elevation of internal carotid (*straight arrow*). **C**, To prevent severe epistaxis into nasopharynx before or after removing umbrella tip, internal carotid artery was occluded by balloons. First balloon was detached within aneurysm (*single solid straight arrow*) to occupy some of dead space in order to permit tip of second balloon (*curved arrow*) to pass distal to neck of false aneurysm near ophthalmic artery origin. Second balloon was inflated across cavernous carotid artery and detached. This post–balloon detachment film shows ophthalmic artery (*double arrows*) filling retrograde to supraophthalmic internal carotid artery (*open arrow*). Umbrella tip was removed without any hemorrhagic difficulty related to internal carotid artery.

therefore chromosome analysis might be considered in the evaluation of such patients [19].

Symptoms and signs. The symptoms and signs are related to extension of the tumor into the nose, orbit, and skull base [20–22]. There is usually nasal obstruction and/or epistaxis. Other findings include nasal discharge, facial deformity, exophthalmos, anosmia, hearing loss, and a grayish red–colored mass in the nose or nasopharynx. Recurrent epistaxis has been found to result from vascular fragility and a rise in circulating estrogens. Endogenous estrogens reduce smooth muscle and elastic fibers in the vessel wall, whereas exogenous estrogens have an opposite effect [19].

Classification

The JNA is a benign, highly vascular, fibromatous or angiofibromatous hamartoma that is locally invasive, arises from the nasopharynx, and has a marked tendency to recur [13, 23]. Prepubertal adolescent tumors are comparatively less aggressive than are young adult tumors. Several surgical classifications have been proposed to stage the various patterns of tumor extension and predict the surgical resectability. Other incomplete therapeutic approaches based on morphologic classifications are not very useful since the therapeutic goal for JNAs should be complete and permanent eradication of the tumor.

Histology

The JNA has a fibrovascular stroma with the myofibroblast as the principal cell [19]. The myofibroblast interferes with synthesis of collagen fibers and elastin and can transform itself into smooth muscle cells. The stroma is invaded by fine vessels producing pseudolymphatic endothelialized spaces. The general appearance varies from cavernouslike with fibrovascular stroma to being cellular or purely fibrous. Fibrous change reduces the incidence of local invasion and the frequency of epistaxis.

CT, MRI, and Angiography

Axial and coronal CT depicts the probable nature and anatomic extent of the lesion [24, 25]. Findings may include nasopharyngeal mass, anterior bowing of the posterior wall of the maxillary antrum with a mass in the pterygopalatine fossa, opacity of one or more of the paranasal sinuses, erosion of the sphenoid bone with mass in the sinus, erosion of the hard palate, erosion of the medial wall of the maxillary sinus, deviation of the nasal septum, and intracranial extension. CT provides better demonstration of bone involvement than does MRI, but MRI has the advantage of being able to differentiate between sinus extension of tumor and sinus obstructive changes. MRI usually produces a brighter signal on T2-weighted images of the mucosal thickening and fluid within the sinus as compared with tumor. Intracranial and extracranial extension boundaries are also well shown on MRI using transverse, coronal, and sagittal planes with T1- and T2-weighted pulse sequences. Biopsy is hazardous because of the danger of massive hemorrhage. Fortunately, the characteristic CT and angiographic appearances obviate biopsies.

Combined or separate diagnostic and therapeutic sessions may be undertaken. A 4- or 5-French catheter tip is used, and an excellent fluoroscopic image, digital and conventional subtraction, and direct magnification should be available. A characteristic reticulated pattern is usually seen in the early arterial phase (Fig. 3), with a dense, homogeneous blush persisting into the venous phase [5, 19, 26–28]. The presence of early draining veins is rare. A similar angiographic appearance, however, has been reported with fibrous dysplasia and with lymphoepithelioma, which produces a dense blush as well as occasional anterior bowing of the posterior wall of the antrum [29–31].

An angiographic protocol has been developed to show the blood supply of the lesion and anatomic variations, to confirm the diagnosis when clinical findings and CT are in doubt, and to permit safe embolization [1, 2]. This involves examination of the ipsilateral internal carotid, maxillary, ascending pharyngeal, and ascending palatine arteries in that order on the lateral view. The internal carotid injection will show whether or not any abnormal vascularity indicates invasion of the base of the skull. Embolizing the maxillary artery first affords the best chance of devascularizing the tumor. Embolization of the pharyngeal branch of the accessory meningeal artery may be accomplished by selective catheterization of the accessory meningeal artery or with a proximal maxillary artery catheter position. Residual vascularity in this territory may be reached indirectly by the eustachian branch of the ascending pharyngeal artery. A facial artery injection is finally used to opacify the collateral to the maxillary artery as a check for completeness of maxillary embolization. If the JNA reaches or crosses the midline, the contralateral internal carotid artery is filmed on an anteroposterior (AP) view during temporary compression of the ipsilateral internal carotid artery. AP views are best for examining the contralateral distal maxillary and ascending pharyngeal arteries, whereas lateral images are optimal for the ascending palatine artery. The use of this sequence of branches for injection and embolization permits assessment of the branches that have just been embolized (Fig. 3). Superselective catheterization of vessels should be in the correct sequence with use of the optimal projection to prevent complications. A tumor stain seen on the internal carotid injection may correspond to intracranial or intraorbital extension but might also be due to transcranial supply and opacification of extracranial vascular territories of an exocranial lesion as an indirect rather than true tumor supply [19, 32–34]. There then may be no residual internal carotid artery opacification of the lesion after distal external carotid branch embolization. However, if the occlusion after embolization is too proximal (similar to surgical ligation), then the internal carotid artery–external carotid branch anastomotic channels may become true arterial feeders. Then, embolization of the ascending pharyngeal or ascending palatine branches becomes more critical, but may not reach the lesion [2, 19].

Extension of the lesion may be intrasphenoidal with capsular branches from both internal carotid arteries, intraethmoidal on each side with posterior ethmoidal artery supply from the ophthalmic arteries, intraorbital with supply from the

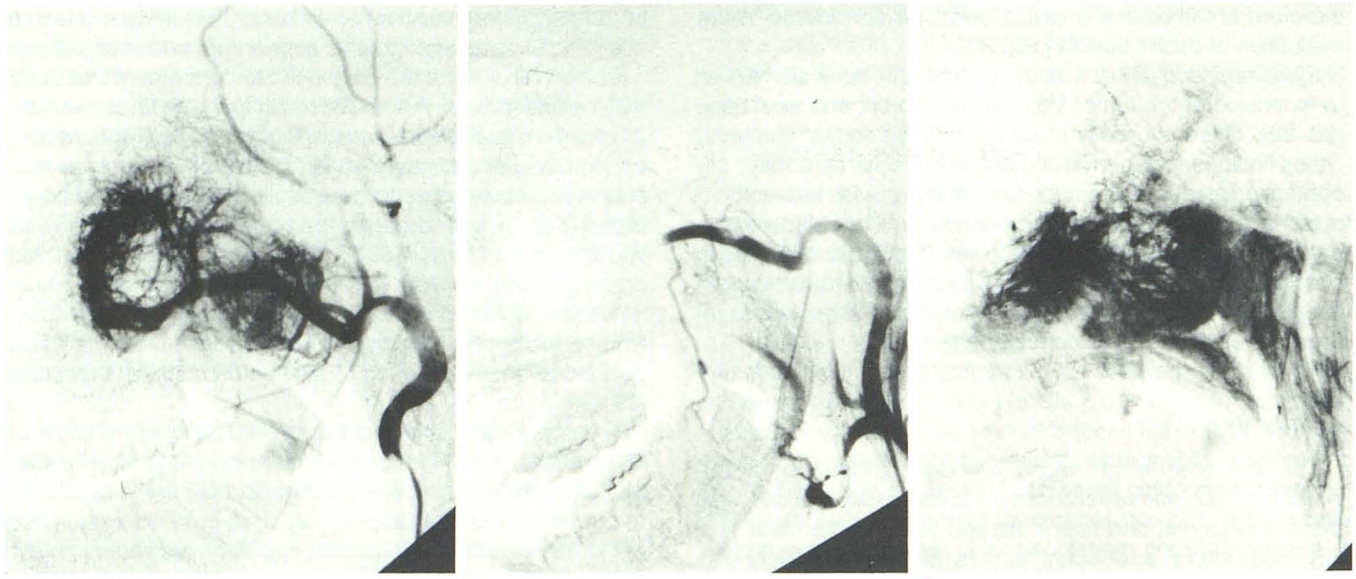

Fig. 3.—**A**, Early arterial phase of maxillary angiogram, lateral view. Reticulated hypervascular stain without early draining veins consistent with JNA. **B**, After maxillary PVA embolization. Minimal residual hypervascularity. **C**, Ascending pharyngeal angiogram after maxillary embolization reveals marked hypervascularity in ascending pharyngeal and collateral to some of the incompletely embolized distal maxillary artery.

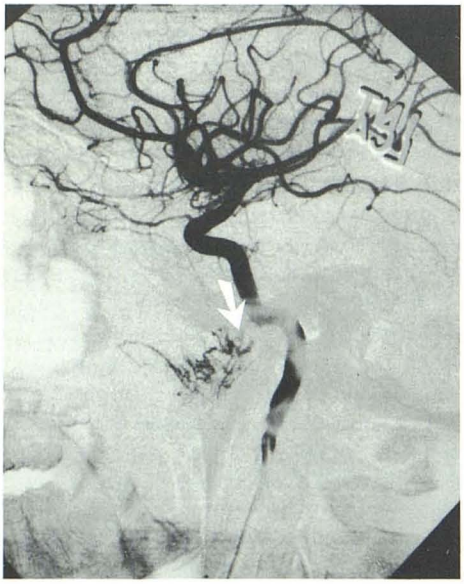

Fig. 4.—Left internal carotid angiogram after ascending pharyngeal artery eustachian branch embolization. Persistent mandibular (mandibulovidian) artery (*arrow*) supply to tumor is seen.

inferior muscular branch of the ipsilateral ophthalmic artery, or intracavernous with vascular supply shown by internal carotid, as well as middle and accessory meningeal, distal maxillary, and ascending pharyngeal artery injections [1, 19, 32, 34, 35].

The persistent mandibular (mandibulovidian) artery may arise from the petrous internal carotid artery (Fig. 4), and the surgeon should be apprised of this so that it may be ligated at the time of operative intervention [19].

Treatment

Various types of treatment have been reported [36, 37]. Surgery alone has a 25%–60% recurrence rate [19]. Radiation alone has a 25% recurrence rate and is rarely indicated because of the possibility of long-term induction of sarcomatous changes [19, 36]. Cryotherapy, electrocoagulation, hormonal therapy to reduce the size and vascularity, or injection of sclerosing agents, respectively, have also been used. Observation in the hope of unusual spontaneous regression has also been tried [19].

Surgical removal is the best and most widely accepted mode of therapy, but resection alone may be limited by significant intraoperative hemorrhage and tumor size. After embolization for devascularization, total excision may be possible with insignificant blood loss.

A therapeutic protocol [2, 19] has been designed, the objectives of which include preoperative identification of possible sources of bleeding and preoperative devascularization by embolization. Since intraoperative hemorrhage may not be controlled by packing, embolization may prevent a fatal outcome. A drier surgical field may also permit a more complete removal of the lesion, and embolization probably also reduces the incidence of recurrence regardless of the type of surgery. Because of the underlying hormonal abnormality, embolization is only effective locally, and often the tumor has extended beyond the apparent boundaries. Therefore, an intentional, more extensive embolization beyond the boundaries of the vascularity seen on the angiogram may be of value.

Superselective distal catheterization is performed, and if this is not possible, a proximal properly directed flow-con-

trolled particle injection is performed [2, 19]. A Gelfoam plug may be used to protect dangerous anastomotic vessels [2, 19]. The presence of dangerous anatomically located vessels along the course of the vessels to be embolized does not contraindicate embolization, but argues for the use of certain types of embolic material such as PVA or Gelfoam particles with a larger diameter than the lumen of the dangerous communicating vessel [19]. PVA and Gelfoam particulate agents are commonly used in the United States, whereas dura has been commonly used in Europe. The goal of post-embolization tumor necrosis may be achieved by using particles [19]. Surgery is performed 2–5 days after embolization.

Liquid embolization with IBCA is dangerous because of the nearby skull base branches to the cranial nerves and external-to-internal carotid artery anastomoses. It would only be indicated if a very aggressive embolization approach is necessary for invasion into the base of the skull. This would follow placement of a permanent balloon occlusion distal to the inferior lateral trunk of the internal carotid artery (after good circle of Willis collateral has been demonstrated) with slow IBCA injection into the internal carotid artery feeders proximal to the distal balloon occlusion. Flow of the glue can reach the tumor with sparing of the nerves (Berenstein A, personal communication). There is no role for the use of this agent in the external carotid artery branches for these tumors.

Future Trends

An alternative method of treatment has been used where, after a radial arterial line has been inserted to monitor the blood pressure and with the patient under general anesthesia, nitroprusside is used to produce vasodilatation and reduce the effect of any minimal vasospasm during a reduction in blood pressure (Moret J, Picard L, personal communication). The internal carotid artery is examined first, and then the catheter tip is placed just below the internal maxillary–superficial temporal artery junction so that flow is not disturbed. Embolization is made from this point using flow control, and steroids may be used for any later discomfort caused by particles in the superficial temporal artery area. This is followed by catheter placement in the proximal ascending pharyngeal artery and flow-controlled embolization. About 100 μm dura particles are used, and no cranial nerve palsies or ocular complications have occurred thus far.

Estrogen spheres may be available for embolization procedures within the next few years (Moret J, Picard L, personal communication). Estrogens may have an effect on the bleeding complications of these tumors. However, the problems produced by the required dose for a significant clinical effect are important in this population of adolescent males (Lasjaunias P, personal communication).

Dextran microspheres have been experimentally used in animals and may prove useful in humans. Particles of 50–150 μm or 100–300 μm can be injected through a 2-French calibrated-leak balloon catheter into the feeding vessel. These are suspended in contrast material and are more permanent and flow is better controlled than with PVA particles. However, the dextran microspheres tend to reflux more easily at the end of the procedure and, therefore, monitoring is required [38].

Precautions and Complications

Complications of ischemic cranial nerve palsies from very small particles and intracranial or ocular embolization via external carotid-to-internal carotid or ophthalmic (rarely vertebral artery from this territory) artery anastomoses may often be avoided by identifying the largest potentially dangerous vessel and placing the catheter distal to its orifice. When this is not possible, particles larger than the lumen of the dangerous vessel can be used, as well as careful flow control techniques. If the dangerous vessel is too large, occlusion of its origin by a large piece of Gelfoam may permit small particles to be injected from a more proximal position of the catheter tip.

The vascular supply to the transcranial nerves is mainly from three branches of the maxillary artery [1, 2, 5, 19, 33, 34]. These include the cavernous branch of the middle meningeal, which supplies the gasserian ganglion; the intracranial branch of the middle meningeal artery or accessory meningeal artery, which supplies the third division of the fifth nerve; and the artery of the foramen rotundum, which supplies the second division of the fifth nerve. Anatomic variants of the artery of the foramen rotundum may result in supply to the third, fourth, first division of the fifth, and sixth nerves. Other vessels that may be at possible risk include the petrosal branch of the middle meningeal artery supplying the intrapetrous segment of the seventh nerve, the recurrent meningeal or meningoophthalmic branch connecting the intraorbital ophthalmic artery with the middle meningeal artery through the superior orbital fissure, the meningolacrimal artery communication with the middle meningeal artery, and the accessory meningeal artery, which may supply the petrosal branch territory of the middle meningeal artery to the seventh nerve. To assess the middle meningeal artery supply to the seventh nerve, a lidocaine test has been suggested [39]. Even though the vascular territories are not terminal, capillary occlusion may nonetheless produce ischemic palsy. In addition, edema of the nerve within a constrictive bone foramen may result in a palsy. Important branches of the ascending pharyngeal include the neuromeningeal branch, which supplies the ninth, tenth, eleventh, twelfth, and part of the sixth cranial nerves, and the lacerum branch, which communicates with the internal carotid artery.

Minor complications such as fever and local pain may occur 12–24 hr after embolization and are treated with steroids. Transient bradycardia may occur during the injection of the maxillary artery or rarely during injection of the ascending pharyngeal artery. This is treated with intravenous atropine. Pain in the upper and/or lower teeth and analgesia without anesthesia in the skin around the mouth and distribution of the second division of the fifth nerve may be found up to 1 week after the embolization.

Major complications usually result from the use of improper embolic material, reflux of embolic material (secondary to vascular spasm, insufficient selective catheterization, or rapid injection of too many particles), or failure to recognize poten-

tially dangerous external carotid-to-internal carotid artery anastomoses because of incomplete angiographic delineation or inadequate anatomic analysis. Safe and successful embolization depends on detailed demonstration and analysis of vascular supply to the lesion and the surrounding area, knowledge of the vascular supply to the transcranial nerves, knowledge of the various external carotid–internal carotid artery anastomoses, and meticulous technique [40–42].

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