Transcatheter Embolization in the Management of Epistaxis

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Abstract

A majority of the population will experience epistaxis at some time in their life. Most cases will be from an anterior source and can be treated with pressure, anterior nasal packing, or cautery. Intractable epistaxis is generally posterior in origin and may require endoscopic cautery, posterior packing, surgical ligation, or embolization. Embolization has been used to treat epistaxis for more than 30 years and success can be achieved in approximately 90% of patients, with major complications occurring in approximately 2%. These excellent results require thorough knowledge of the regional anatomy, familiarity with the equipment and various agents used to achieve this type of embolization, as well as attention to detail and meticulous technique. There remains debate on several aspects of embolization, including the agent of choice, preferred size of the embolic, and the number of vessels to embolize. Advances in endoscopic surgery have evolved to the point that similar success rates for embolization and modern surgical techniques in treating epistaxis may be expected. This detailed review of pertinent vascular anatomy, embolization technique, and surgical alternatives should allow practitioners to formulate treatment algorithms that result in optimal outcomes at their institutions.

Keywords
- epistaxis
- embolization
- complications
- review
- internal maxillary artery
- interventional radiology

Objectives: Upon completion of this article, the reader will be able to identify the etiology and overall management of patients presenting with epistaxis. In particular, the reader will be able to demonstrate a thorough knowledge of the pertinent anatomy, angiographic appearances, transcatheter techniques, and complications associated with transcatheter embolotherapy in the setting of epistaxis.

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Historical Perspective

Epistaxis is a Greek word meaning nosebleed. Some of the earliest accounts of epistaxis date to Egyptian times and describe the use of the ashes of papyrus mixed with vinegar to treat nasal bleeds. Hippocrates said that pinching the nose "for some time" and asking the patient to breathe through the mouth stopped bleeding from the nose. In 1869, Pilz was the first person to treat epistaxis by surgically ligating the external carotid artery. Although in 1879 James Little and Carl Michel were the first to identify the vascular plexus in the anterior part of the nasal septum ("Little's area") as the common area from which nasal bleeding occurs, Wilhelm Kiesselbach's 1884 paper describing the area has led to the common usage of "Kiesselbach plexus" to describe this common source of epistaxis. Seiffert described ligation of the internal maxillary artery (IMAX) through the maxillary antrum via the Caldwell-Luc approach to control epistaxis in 1929.[1] The first report on the use of transcatheter

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embolization to control epistaxis was published by Sokoloff et al[2] in 1974; successful treatment of two patients using Gelfoam (GF) (Gelfoam-Pharmacia and Upjohn Company, Kalamazoo, MI) was described. Since that time a variety of embolic agents have been used including GF, autologous clot, fat, marrow, polyvinyl alcohol (PVA), calibrated microspheres, *N*-butyl cyanoacrylate, coils, Onyx, and other agents. The proximity to the brain and eyes makes the endovascular treatment of epistaxis somewhat more challenging and risky than embolization in other locations of the body. Interventional radiologists (IRs) who wish to work in this region must have a thorough knowledge of the regional anatomy, dangerous collaterals, pathophysiology, and available alternative treatments. Armed with this knowledge, the IR, most often in concert with the otolaryngologist, can provide optimal care to epistaxis patients.

Epidemiology and Etiology

Epistaxis is extremely common, occurring in up to 60% of the adult population at some time in their lives. Approximately 80% of cases will be caused by an anterior bleeding source[3] with a minority of cases resulting from a posterior source. Fortunately, most epistaxis resulting from an anterior source is self-limited with only 6% requiring medical attention.[4] There is a nearly equal distribution between men and women; however, posterior intractable epistaxis is more common in elderly males. Most large studies show a mild peak in epistaxis cases in the first to second decade of life, and a subsequent increase with age above 40 years. Associated factors include hypertension, inflammation/infection (allergic, bacterial, fungal, and viral), medications (e.g., aspirin, nonsteroidal anti-inflammatory drugs, warfarin, and clopidogrel), coagulopathy (e.g., hereditary hemorrhagic telangiectasia [HHT]), postsurgical/iatrogenic injuries, and trauma. Rarely, other more unusual causes such as tumor (e.g., juvenile angiofibroma), aneurysm, or vascular malformation may be causative. The typical patient presenting for evaluation and treatment at an emergency room is an elderly hypertensive patient. Epistaxis is more common in the winter months. This seasonal variation is believed to be related to reduced humidity, temperature, and increased prevalence of upper respiratory tract infections. Since the vast majority of epistaxis will be anterior in location, it is relatively easily accessible to local therapy. Less common posterior bleeding is more difficult to access and often more difficult to control.

Anatomical Considerations

The blood supply of the nasal cavity is rich and varied. The nasal fossa is supplied by both the internal carotid artery (ICA) and the external carotid artery (ECA) (see ►**Fig. 1**). The dominant supply in most individuals is from the ECA via distal branches of the IMAX. Several authors also describe a small contribution to the posterior nasal cavity from the ascending pharyngeal artery (APhA), which arises from the IMAX just beyond the facial artery (FA) origin. Important branches of the IMAX include the sphenopalatine artery (SPA) and the greater palatine artery (GPA). The GPA is the terminal branch of the descending palatine artery (DPA). The SPA provides the dominant supply to the nasal cavity walls via both medial (septal) and lateral branch; the lateral branch supplies the superior, middle, and inferior turbinates, while the medial branch supplies the nasal septum. The richly vascularized posterior aspect of the nasal cavity, sometimes referred to as Woodruff plexus, is primarily supplied by the SPA. The GPA courses via the incisive foramen to supply the inferior portion (floor) of the nasal septum and anastomoses with septal branches of the SPA. The FA generally supplies the more anteroinferior portion of the nasal cavity via the terminal alar branches of the superior labial artery (SLA) as well as the lateral nasal artery (LNA). ICA supply to the nasal cavity is most often by the ophthalmic artery (OphA) via the anterior ethmoidal artery (AEA) and posterior ethmoidal artery (PEA). The ethmoidal arteries course through the cribriform plate to supply the roof of the nasal cavity and communicate with branches of the SPA posteriorly and several other arteries anteriorly. Because of their diminutive size, the ethmoidal arteries are rarely seen on normal arteriograms. It has been suggested that their visualization portends failure of control of epistaxis using standard IMAX embolization.[5] Kiesselbach plexus is a highly vascularized region at the anteroinferior nasal septum. It is the sight of most anterior epistaxis and is richly supplied via braches of the AEA, PEA, SPA, GPA, and SLA/alar branches of the FA.

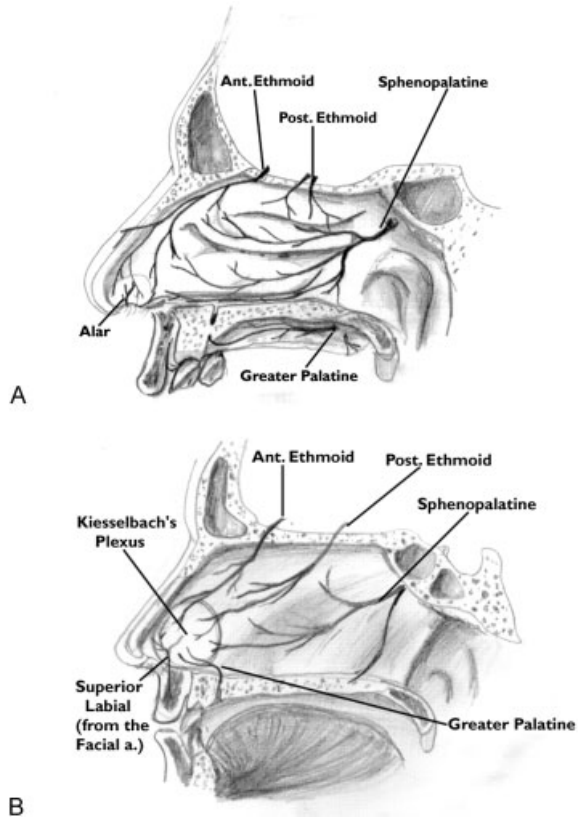

Figure 1 Schematic arterial anatomy of the nasal cavity. (A) Medial nasal wall shows blood supply of nasal septum. (B) Blood supply of lateral nasal wall. Kiesselbach plexus (or Little area), the site of most anterior epistaxis, is noted. (Reprinted, with permission, from Koh et al[5]).

As the IMAX is the critical vessel in most cases of epistaxis, it is incumbent upon the angiographer to have a complete knowledge of the anatomy of the IMAX. The IMAX is the larger of the two terminal branches of the external carotid artery, with the other being the superficial temporal artery (STA). The IMAX arises behind the neck of the mandible, travels through the substance of the parotid gland, and passes forward between the ramus of the mandible and the sphenomandibular ligament. It courses either superficial or deep to the lateral pterygoid muscle to the pterygopalatine fossa (see ►**Figs. 2** and **3**). The IMAX is classically divided into three segments termed the mandibular, pterygoid, and pterygopalatine segments. The branches of segment 1 (mandibular) include the deep auricular, anterior tympanic, inferior alveolar, middle meningeal (MMA), and accessory meningeal (AccMA) arteries. These proximal branches of the IMAX may be recalled using the mnemonic "MIADA." It is critical to identify the proximal segmental branches, as they are a common pathway for ECA–ICA collateralization, especially in cases of ICA obstruction/stenosis. The second or pterygoid portion of the IMAX courses obliquely forward and upward under the mandibular ramus and insertion of the temporalis on the surface of the lateral pterygoid muscle; it then passes between the two heads of origin of this muscle and enters the pterygoid fossa. Branches include the masseteric artery, pterygoid branches, posterior deep temporal artery, and buccal artery. This large contribution to the muscles of mastication in conjunction with proximity to branches supplying the nasal cavity explains the common incidence of jaw claudication following IMAX embolization. The anterior deep temporal artery may arise from the distal second or proximal third portion of the IMAX. The third or pterygopalatine

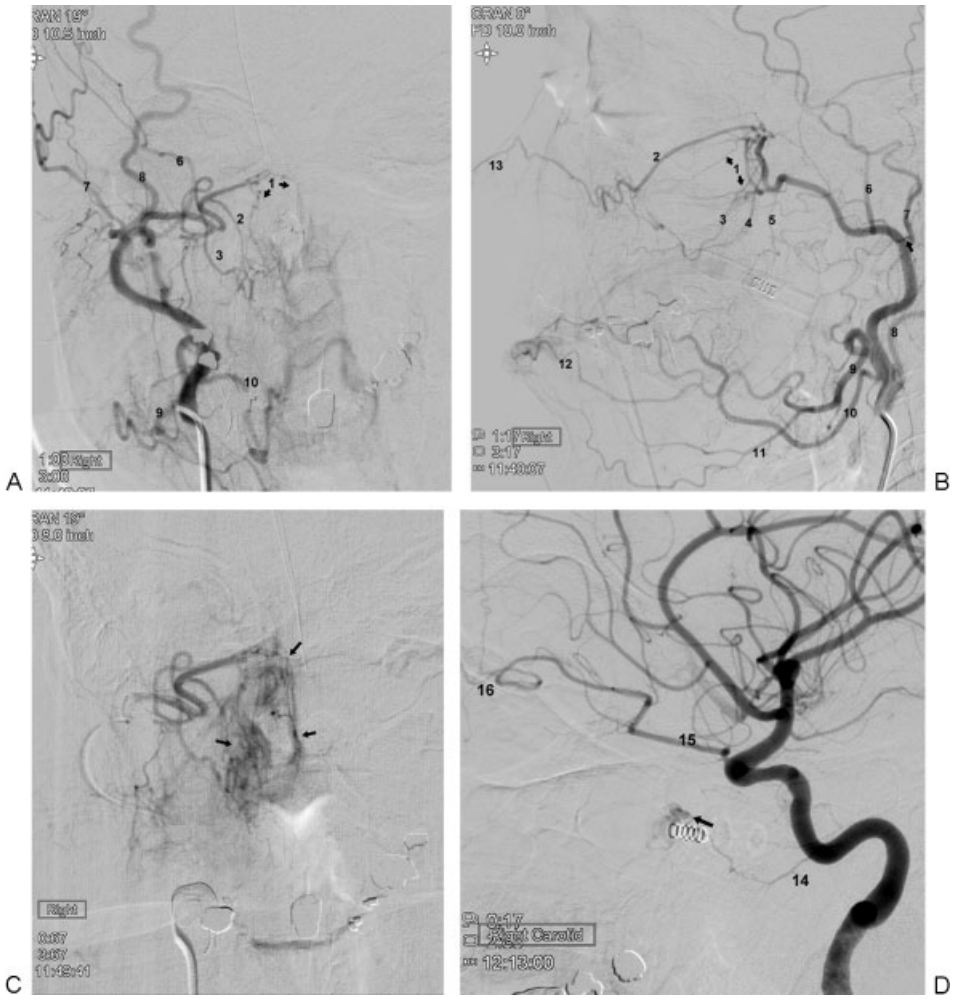

Figure 2 A 68-year-old with refractory posterior epistaxis. (A) AP and (B) lateral ECA injection shows blood supply of nasal cavity and face well. (C) Late phase AP image shows marked hyperemia (arrows) often noted in epistaxis cases without frank extravasation. (D) ICA post-embolization injection shows well the vidian artery with flow stopping at level of DPA (arrow). Note that the OphA anatomy is well defined including distal branches (15, 16). Key: 1—SPA (arrows denote medial and lateral branches), 2—IOA, 3—DPA, 4—posterior superior alveolar artery, 5—buccal artery, 6—MMA, 7—STA (note arrow pointing to most proximal branch or transverse facial artery), 8—occipital artery, 9—FA, 10—lingual artery, 11—submental artery, 12—inferior labial artery, 13—lateral nasal artery supplied in this case from IOA due to relatively hypoplastic distal facial artery, 14—vidian artery (artery of pterygoid canal), 15—OphA, 16—terminal OphA branches including the supraorbital and dorsal nasal arteries. AP, anteroposterior; DPA, descending palatine artery; ECA, external carotid artery; FA, facial artery; ICA, internal carotid artery; IOA, infraorbital artery; MMA, middle meningeal artery; OphA, ophthalmic artery; SPA, sphenopalatine artery; STA, superficial temporal artery.

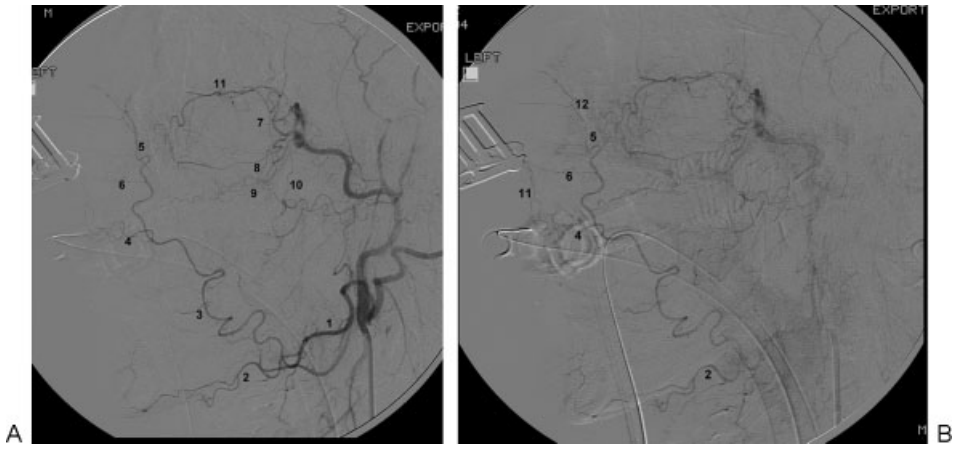

Figure 3 (A) Early and (B) late lateral ECA injections show FA and IMAX anatomy well. Key: 1—proximal FA, 2—submental branch, 3—inferior labial artery, 4—superior labial artery, 5—AngA which collateralizes distally with DNA from OphA, 6—LNA, 7—SPA and branches, 8—DPA and branches, 9—posterior superior alveolar artery, 10—buccal/masseteric branch, 11—IOA, 11—septal/alar artery arising from SLA, 12—distal AngA branches. AngA, angular artery; DNA, dorsal nasal artery; DPA, descending palatine artery; ECA, external carotid artery; FA, facial artery; IMAX, internal maxillary artery; IOA, infraorbital artery; LNA, lateral nasal artery; OphA, ophthalmic artery; SLA, superior labial artery; SPA, sphenopalatine artery.

portion of the IMAX contains the important contributors to epistaxis as well as other branches. These include the DPA (which branches into the GPA and lesser palatine artery), posterior superior alveolar artery, pharyngeal artery, artery of the pterygoid canal (vidian artery), infraorbital artery (IOA) (most anterior branch of IMAX), middle and superior alveolar arteries (both branches of the IOA), and the SPA, which is generally the terminal branch of the IMAX.

In cases of epistaxis, it is also important to consider the FA anatomy (see ►**Figs. 1–3**). The FA generally arises from the ventral surface of the ECA and courses inside the mandible. Near its origin, the FA gives rise to the ascending palatine artery (APA). The FA turns downward and forward as it passes around the mandible and gives off tonsillar and small glandular branches (i.e., submandibular artery), and the submental artery. The FA becomes superficial and subcutaneous, courses cephalad along the mandible in a variable direction, and gives rise to the inferior labial artery. The FA ascends to the maxilla and gives rise to the SLA; the SLA supplies the anterior nasal septum and gives rise to alar branches to the nasal ala. The FA courses superiorly and slightly posterior and gives rise to the LNA, supplying the ala and lateral nose. The FA gives rise to the angular artery (AngA) in its terminal segment. The AngA anastomoses with the dorsal nasal branch (DNA) of the OphA. Along its course, the FA also variably supplies muscular branches to the internal pterygoid, stylohyoid, masseter, and buccinator.

Important Anastomoses

It is critical to understand the anastomoses between the ECA and the ICA when performing arteriography and/or embolization in this region. The ECA, ICA, and OphA have important anastomoses that are always present but often not visualized or recognized on standard arteriography. Well-described anastomoses between the ECA and ICA include the vidian artery, inferolateral trunk ((ILT)—supplies the artery of foramen rotundum and the artery of foramen ovale), and meningohypophyseal trunk. Other important ECA–ICA collateral pathways, especially when the ICA is obstructed, include MMA, AccMA, and APhA. ECA collateralization to the OphA or to the ICA retrograde via the OphA is also important to assess. In a small number of individuals, the ECA may provide the dominant supply to the OphA. Well-known but often poorly visualized collateral pathways exist between the SPA, FA, and OphA. The IOA is particularly noteworthy in this regard (see ►**Fig. 2**), as it may anastomoses with the terminal branches of the FA (LNA, SLA, and AngA), DNA branch of OphA, transverse facial artery ((TFA)-most proximal branch of the STA), and buccal arteries.

The FA also has a rich network of anastomoses. Important anastomoses exist between the APA and the DPA, APhA, and AccMA. Several FA branches, including the LNA and AngA, may anastomose with the TFA. The SLA and LNA anastomose with each other. The LNA anastomoses with the septal and alar branches of the SLA, with the DNA of the OphA, and with the IOA branch of the IMAX (see ►**Fig. 2**). The AngA anastomoses with the DNA.

These collateral pathways make it imperative that the interventionalist performs high-quality, high-resolution arteriography to exclude visible anastomoses. Many times these communications are not seen on initial arteriography, so constant vigilance is needed throughout the procedure. During embolization, as pressure in vessels increases and flow is altered, collateral pathways may open up and be a source of emboli to the ICA or OphA.

Epistaxis—Basic Management

Although a careful history may reveal the underlying cause of epistaxis, unfortunately most cases remain idiopathic. An experienced practitioner should perform nasal examination

to determine the site of the bleeding, since location is critical in determining the appropriate therapy. Epistaxis originates most commonly in the anterior portion of the nasal area (Kiesselbach plexus; see "Anatomical Considerations"), which is easily accessible and amenable to local therapy. External digital pressure is the first line of control. Patients who have failed this measure require more aggressive treatment, typically consisting of chemical or electrical cautery. Cautery is directed to well-visualized sites of bleeding, generally within Kiesselbach plexus. It is imperative that the site of bleeding be identified, including both location (anterior or posterior, and lateral or medial) and side (right or left). Adequate light (headlamp or mirror), vasoconstrictor (epinephrine or topical decongestant–soaked cotton, topical cocaine), clearance of clot, adequate suction, and an experienced provider are required for best results. Unfortunately, studies suggest that these resources are not often available in many emergency rooms.[6] Nasal packing is performed when cautery fails or when a site of bleeding cannot be localized. Classically, anterior packing consisted of the placement of Vaseline impregnated ribbon gauze (Covidien, Mansfield, MA). Effective packing with gauze requires some training as it can be a difficult process and traumatic for the patient. Septal deviation or lack of experience may actually lead to mucosal trauma and more bleeding during the packing.[7] Commercially available packing devices are being widely implemented in place of traditional packing methods. Most of these devices are engineered to swell with hydration and have surfaces coated with agents that promote activation of the clotting cascade. The Merocel (Medtronic ENT, Jacksonville, FL) nasal pack is composed of hydroxylate polyvinyl acetate material, which is applied as a tampon and expands when wet. A prospective randomized study in 50 patients compared the Merocel device to traditional packing and found no difference in efficacy or patient tolerance.[8] The Rapid Rhino (ArthroCare ENT, Austin, TX) is composed of an inflatable balloon coated with a carboxymethylcellulose hydrocolloid compound that acts as a platelet aggregator and forms a lubricant on contact with water. Inflation with air allows for tamponade when placed. Deflation at the time of removal is thought to decrease trauma. Two prospective randomized controlled trials comparing Rapid Rhino with Merocel found no differences in the rate of control of anterior epistaxis but found that insertion and removal of the Rapid Rhino was easier and less painful.[9,10] Another novel concept is the intranasal injection of hemostatic substances to control epistaxis.[11,12] FloSeal (Baxter Healthcare Corp., Deerfield, IL) is a biodegradable hemostatic sealant composed of collagen-derived particles and topical bovine-derived thrombin that is applied as a high-viscosity gel. One study compared FloSeal to other commercially available nasal packs in the treatment of anterior epistaxis and found it to be significantly more effective in controlling epistaxis as well as being better tolerated.[13] Unfortunately, there is a paucity of published data on the use of virtually all of these devices in the treatment of posterior epistaxis.[14]

Posterior or combined anterior-posterior packing consists of 3 to 6 feet of meticulously placed antibiotic impregnated Vaseline gauze or bismuth–iodoform–paraffin paste ribbon. This may be used with or without a Foley or other balloon[15] that is inflated and then pulled until it engages against the posterior choana.[16] While this treatment can be effective in many cases, it has a failure rate of 25 to 60%[3,17] and is associated with several complications. These include pain, hypoxia, sinusitis, palatal edema, infection/sepsis, aspiration, and alar necrosis.[16,18,19]

Surgical Options

Surgical management of intractable epistaxis has evolved over the past 75 years. Although Pilz described the use of ECA ligation in 1869, the technique was popularized after 1925 by Hyde.[20] Transantral ligation of the IMAX (TAIMAXL) was originally described in 1928, but never gained wider clinical use until it was redescribed in 1965.[21] Transnasal SPA ligation was described in 1985,[22] and endoscopic transnasal ligation of the SPA (ETLSPA) was described in 1992.[23] Successful treatment of posterior epistaxis using the varied techniques of transantral IMA ligation has been reported in approximately 65 to 90%, with complications occurring in 10 to 40%.[17,24–26] Typical complications include cellulitis, sinusitis, infraorbital nerve injury, and epiphora; rare complications include cranial nerve palsies, skin slough, unilateral blindness, and death. Improvements in endoscopic technique and instrumentation have further improved surgical results and reduced complications compared with older techniques. Successful control of epistaxis using exclusively ETLSPA has been reported in 92 to 100%[27] of patients with an average success rate of 98% in one recent pooled analysis.[28] The addition of AEA ligation may be useful in certain cases to further improve success rates,[25] especially in the setting of trauma (see ►**Fig. 4**), difficult to localize bleeding sites, and in the elderly/medically compromised patient.[28,29] Modern surgical techniques have a relatively low complication rate (0–10%) with a trend toward lower complications in the ETLSPA as compared with TAIMAXL.[24,28,30] Complications tend to be minor such as nasal crusting, numbness of the palate, sinusitis, septal perforation, intranasal adhesions, and decreased lacrimation.[28] Unfortunately, because most surgical series are retrospective, there are significant limitations in the conclusions that can be drawn as to which is the "ideal" surgical treatment. Although one study suggested IMA ligation to be as effective as embolization with a higher minor complication rate but with lower major complications,[25] the lack of good prospective trials makes true comparison of the techniques difficult. Furthermore, expertise in ETLSPA is often not readily available at many centers on a 24-hour basis.

Embolization Technique and Results

Angiographic demonstration of active extravasation in the setting of acute epistaxis was first described by Duggan and Brylski[31] in 1970. Sokoloff et al published the earliest report of embolization to treat epistaxis in 1974.[2] The report described the use of a "Becton-Dickinson white polypropylene catheter (Becton, Dickinson, and Company; Franklin, NJ) (inner diameter, 0.13 cm; outer diameter, 0.18 cm)" to deliver

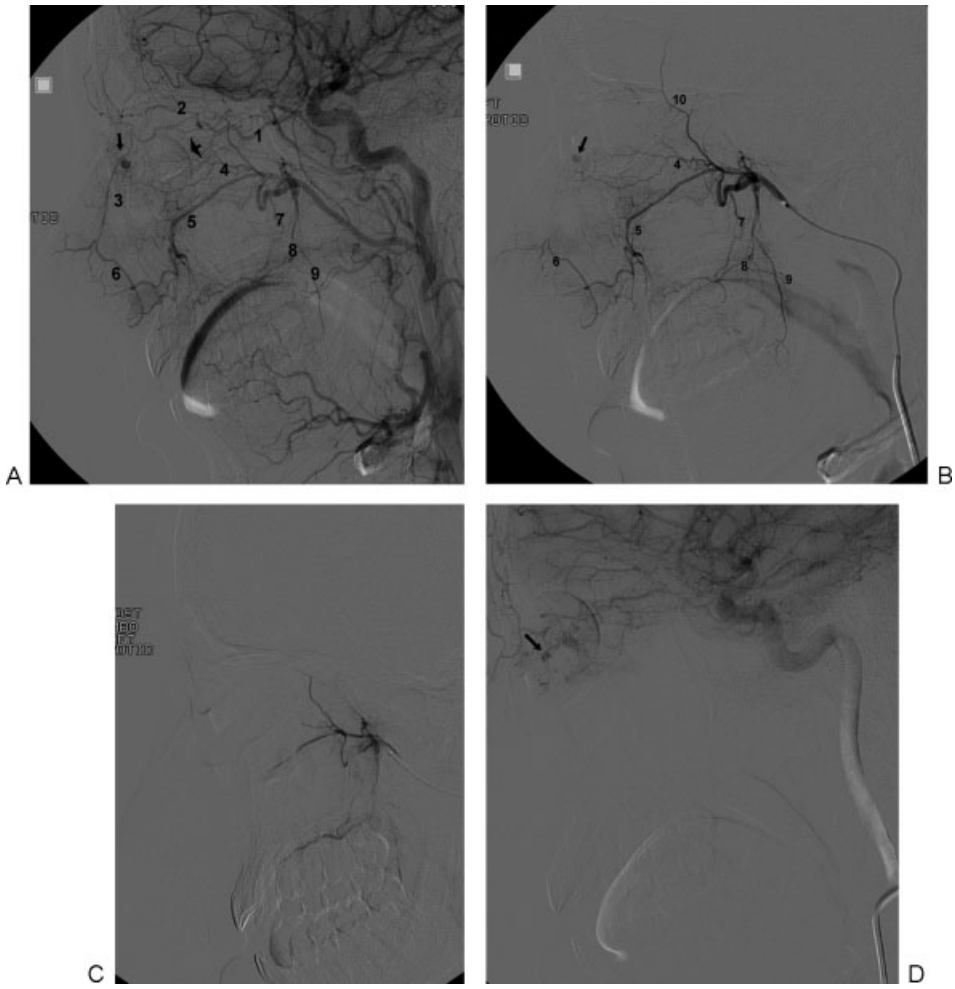

Figure 4 Trauma patient with nasal fractures and unremitting epistaxis. (A) CCA injection shows focal pooling of contrast in superior nasal region (small arrow). Note the ciliary blush of retina (large arrow). (B) Microcatheter injection in distal IMAX shows contribution from SPA branches (small arrow). (C) Postembolization IMAX injection shows complete embolization; however (D) shows late phase of injection from CCA with some persistent pooling of contrast likely resulting from persistent ethmoidal artery supply. Key: 1—ophthalmic artery; 2—extraorbital OphA branches including anterior falcine, dorsal nasal, frontal, and supraorbital branches; 3—angular artery (note communication with distal OphA branches); 4—SPA branches; 5—infraorbital artery (large in this case due to hypoplastic distal FA); 6—superior labial and lateral nasal branches; 7—posterior superior alveolar artery; 8—descending palatine artery; 9—buccal artery; 10—anterior deep temporal artery. CCA, common carotid arteriography; FA, facial artery; IMAX, internal maxillary artery; OphA, ophthalmic artery; SPA, sphenopalatine artery.

twenty 1 to 2 mm GF particles to the site of bleeding in the sphenopalatine artery." Epistaxis was successfully controlled in two patients with one sustaining a transient infraorbital nerve hypoesthesia that resolved. Since that time there have been many other reports on the use of embolotherapy for epistaxis. Controversy still exists regarding the optimal location, agent(s), and endpoints for control of epistaxis.

Successful control of intractable epistaxis hinges on control of the bleeding source. The arteriogram will generally not reveal an active site of bleeding,[32] perhaps related to the fact that nasal packing is often in place.[2] An adequate ENT examination prior to the arteriogram to assist in localization and or lateralization is therefore extremely useful. This information guides the interventionalist in directing the initial arteriographic evaluation and may limit the need for bilateral embolization in some cases. Common carotid arteriography (CCA), generally performed via a 5F catheter or 5/6F guiding catheter, may be useful to evaluate the carotid bulb and bifurcation area prior to selective catheterizations. Most practitioners strongly advocate performance of high-resolution digital subtraction angiography of both the ICA and ECA.[2,33] While active bleeding is only rarely evident on the arteriogram in idiopathic epistaxis, hyperemia, blush, or increased vascularity may be appreciated[34–37] (see ►**Fig. 2**). Arteriograms in cases of trauma or tumor will more commonly reveal abnormal findings such as pseudoaneurysm, active bleeding, or puddling of contrast (see ►**Figs. 4** and **5**). Rarely, an arteriovenous malformation (AVM) or arteriovenous fistula (AVF) may be detected (see ►**Fig. 6**). The ICA arteriogram[38] will serve to exclude dangerous collaterals and exclude rare ICA sources of bleeding as described above (e.g., carotid cavernous fistula [CCF], cavernous carotid artery aneurysm [CCAA],[39,40] and AVM[41]). A careful search should be undertaken to identify the dominant supply to the OphA and for contributions to the nasal cavity

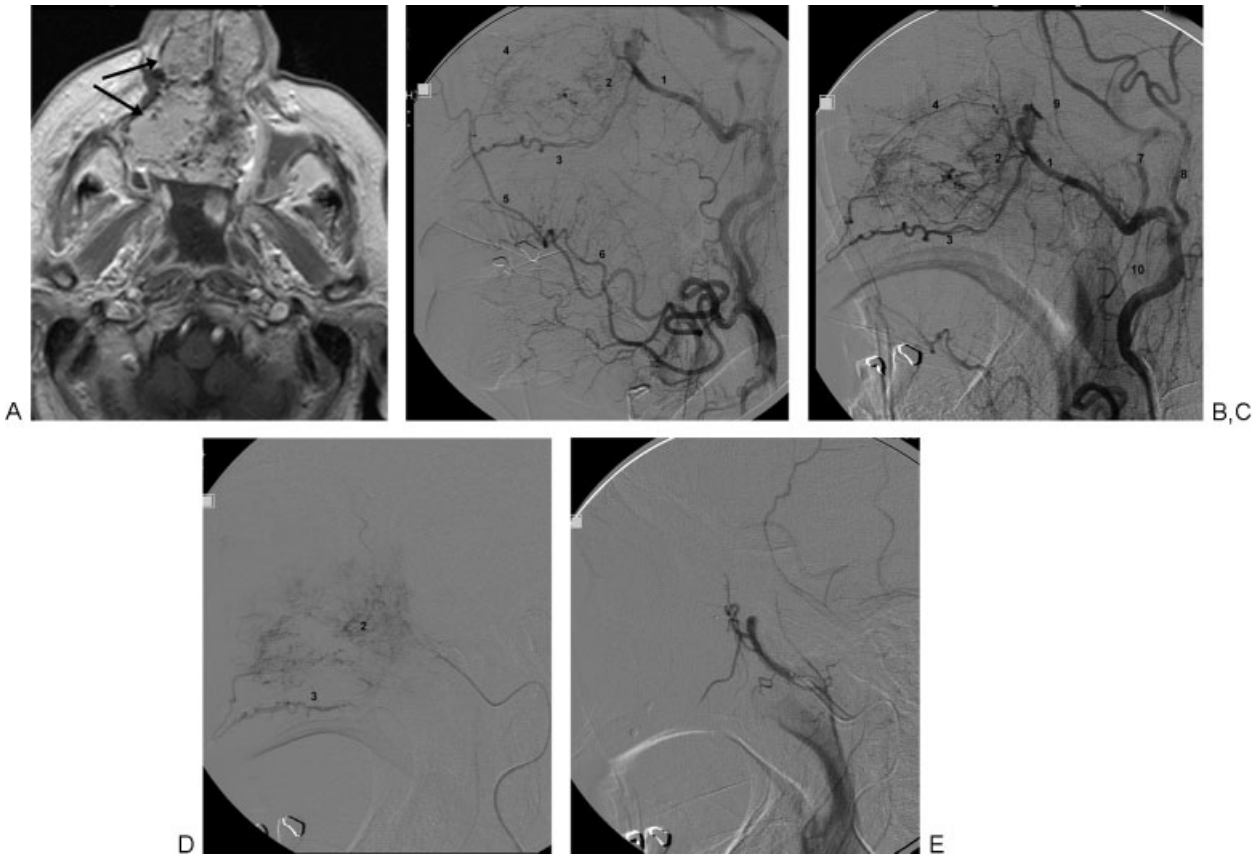

Figure 5 Patient with epistaxis and extensive infiltration of the nasal cavity from adenoid cystic tumor. (A) MRI depicts extensive infiltration by tumor (arrows). (B) and (C) Proximal right ECA injection. Note the extensive vascularity and hyperemia in the area of the SPA artery branches. (D) Progressively more selective distal IMAX injection. (E) Post-IMAX embo appearance. Key: 1—distal IMAX, 2—SPA branches, 3—DPA/GPA relatively large, 4—IOA noted to be relatively small, 5—FA with large distal branches, 6—lingual artery, 7—MMA with characteristic turn as it passes through foramen spinosum, 8—STA with typical hairpin loop as crosses zygoma, 9—middle deep temporal artery, 10—inferior alveolar artery. DPA, descending palatine artery; ECA, external carotid artery; FA, facial artery; GPA, greater palatine artery; IOA, infraorbital artery; MMA, middle meningeal artery; SPA, sphenopalatine artery.

from the AEA or PEA (see ►**Fig. 4**). The ECA arteriogram will detail the anatomy of the important supply to the nasal cavity (SPA, GPA, FA, AEA, etc.) as well as the many other potential collaterals to both the nasal cavity and the ICA. If the ECA is the dominant supply to the OphA, embolization is relatively contraindicated due to the risk of blindness.

Once the ICA and ECA have been completely evaluated, most operators will place a microcatheter (0.018–0.028 ID) into the IMAX. The microcatheter may be placed via the 5F diagnostic catheter or via a 5 of 6F guide catheter. Placement of a guide catheter or sheath offers the advantage of control catheter arteriography during the embolization. Detailed IMAX arteriography may be performed via the microcatheter. The microcatheter is advanced as distally as possible into the pterygopalatine segment of the IMAX beyond the AccMA and MMA (see ►**Figs. 4**, **6**, and **7**). Placement of the catheter beyond the muscular branches may reduce the incidence of trismus postprocedure. In the rare instance of distal SPA aneurysm or pseudoaneurysm, one might consider the use of microcoils[37,42] or even cyanoacrylate[43] (see ►**Fig. 6**); however, most of the literature describes embolization with use of PVA or GF.

The mainstay of current embolotherapy for epistaxis is PVA or GF[21,22,24,33,43–48]. Both agents have a long track record for safety and efficacy throughout the vascular system and in the treatment of epistaxis.[26,33] Multiple reports describe their favorable safety profile.[2,25,26,33,37,41–44,49] As with any embolic agent, each can lead to over-embolization or non-target embolization. Various sizes (50–750 µm) of PVA are described in the literature; however, most reports describe use of PVA particles in the 150 to 350 µm range and GF in the 1 to 3 mm range. Although the use of smaller particles (50–150 µm) is described, this may lead to increased risk of ischemia[35,42] or have a higher risk of non-target embolization via collateral vessels.[35] Slightly larger size particles may be useful when treating FAs to prevent skin necrosis.[50]

Embolization is performed with real-time fluoroscopy to ensure adequate antegrade flow throughout the procedure. The distal IMAX embolization is performed until there is near stagnation of antegrade flow and no more visualization of the nasal blush (see ►**Figs. 4–7**). Some authors have advocated for placement of slightly larger GF plug or rarely microcoil placement at the back end.[37] Microcoil placement in the distal IMAX for intractable idiopathic epistaxis[51] is generally

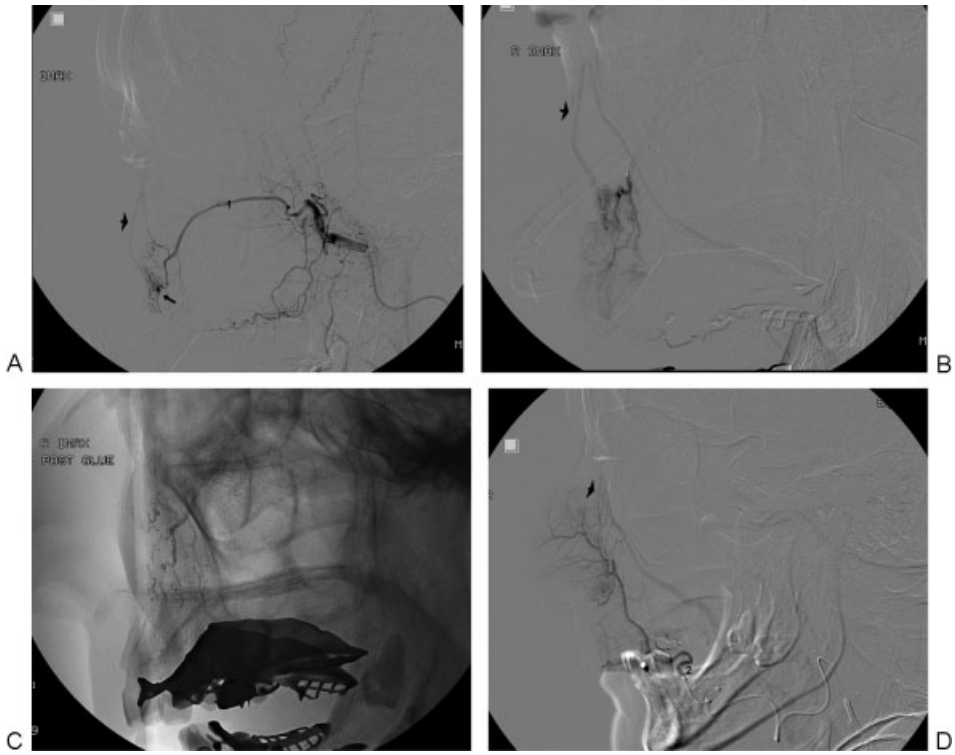

Figure 6 Patient with chronic intermittent epistaxis despite multiple ENT treatments including several cauterizations. (A) Lateral projection from IMAX injection shows abnormal tangle of vessels (small arrow) and rapid shunting (large arrow) to vein consistent with AVM. (B) Distal IOA injection confirms finding. (C) Lateral unsubtracted image shows casting from cyanoacrylate used to treat AVM due to high flow. (D) Lateral projection from distal FA injection shows persistent shunting felt to be related to continued supply to AVM. This was treated with particulate embolization. Particles of 50 to 150 μm had to be used, as the only microcatheter that could be placed this far distally would not allow larger particles. The patient awoke with complete loss of central vision which did not improve despite aggressive efforts to decrease intraocular pressure. Although OphA collaterals were not visualized during the procedure, it must be borne in mind that these collaterals exist in most patients, warranting extreme caution especially in cases where smaller particles are used. Key: 1—infraorbital artery, 2—facial artery just proximal to inferior labial artery. AVM, arteriovenous malformation; FA, facial artery; IMAX, internal maxillary artery; IOA, infraorbital artery; OphA, ophthalmic artery.

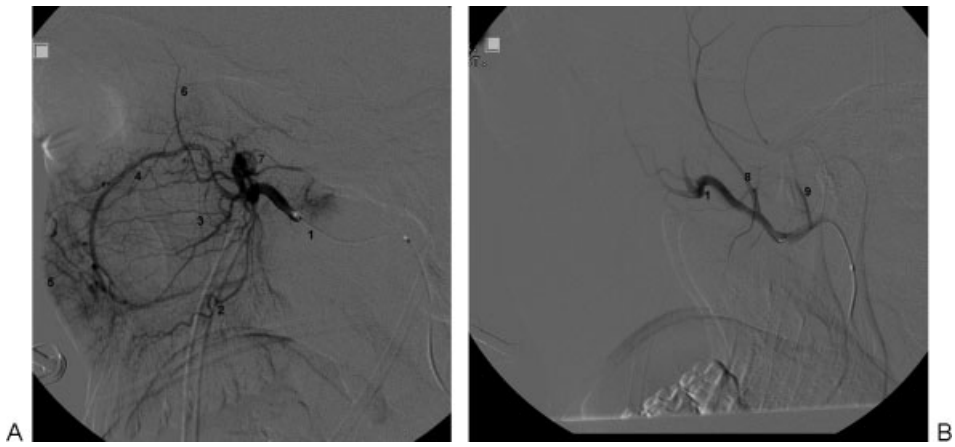

Figure 7 A 78-year-old with intractable posterior epistaxis. (A) Lateral projection from distal IMAX microcatheter injection shows some hyperemia without active extravasation. This is typical of most cases of epistaxis. (B) Lateral microcatheter injection following embolization with 250 to 350 μm PVA particles. The microcatheter has been pulled back into the first segment of the IMAX. Note the lack of filling of the branches of the distal IMAX and early opacification of some proximal branches. All of these proximal branches remain patent as only the most distal portion of the IMAX was embolized. Key: 1—microcatheter positioned in pterygopalatine segment of IMAX; 2—DPA; 3—SPA medial and lateral branches; 4—IOA; 5—distal IOA branches which may anastomose with LNA, AngA, and DNA branches; 6—anterior deep temporal artery; 7—artery of foramen rotundum; 8—middle deep temporal artery; 9—MMA. AngA, angular artery; DNA, dorsal nasal artery; DPA, descending palatine artery; IMAX, internal maxillary artery; IOA, infraorbital artery; LNA, lateral nasal artery; MMA, middle meningeal artery; PVA, polyvinyl alcohol; SPA, sphenopalatine artery.

not performed routinely today[52] except in cases to prevent non-target embolization or when particulate embolization is not possible[32,37]; avoidance of coil placement should allow the possibility of repeat embolization if needed. GF is generally thought to be temporary with recanalization demonstrated in swine model in 2 to 8 days depending on the size of GF used.[37] Many operators also advocate for embolization of the contralateral IMAX and/or the ipsilateral or bilateral FA (BFA).[33,37,41,53–55] When FA embolization is undertaken, it is recommended to perform the embolization distal to the submandibular artery. Similar to IMAX embolization, various materials have been used for FA embolization. These are generally similar to those used for IMAX embolization; however, special caution should be taken when embolizing the FA to avoid necrosis of the nasal ala[42] and non-target embolization to OphA branches. The use of GF or slightly larger particles may allow for control of bleeding without causing severe ischemia or non-target embolization. Some authors have advocated for routine coil placement in the distal FA.[34]

Embolization: Results

There is a high degree of variability in the literature with regard to whether unilateral IMAX (UMAX), bilateral IMAX (BIMAX), or IMAX and either unilateral FA (UFA) or BFA are embolized at the initial treatment. Similar high variability also exists with regard to agents that are used to achieve the embolization. Given the rich collateralization of the nasal cavity, it stands to reason that total control of epistaxis might require embolization of other branch vessels.

The original report from Sokoloff et al[2] simply describes embolization of the IMAX using GF. Shortly thereafter, embolization of the ipsilateral UFA was described in conjunction with the IMAX to reduce rebleeding rates.[44] Wehrli et al[26] reported clinical success in 74% of patients using UMAX embolization with GF or PVA with minor and major complications in up to 50% and 11%, respectively. Vitek,[33] in 1991, advocated a protocol whereby distal UMAX embolization with GF was performed on the side of the bleeding. The patient was monitored for 15 minutes and then the packing was removed. If no bleeding recurred, the case was complete. If bleeding recurred, the ipsilateral UFA was embolized. Using this protocol, he reported an increase in success rates of 87% to 97%[33] with no major complications and one single episode of transient hemiparesis (3% minor complication rate). Oguni et al[37] described a series of 37 patients utilizing a protocol whereby distal UMAX GF embolization was performed as well as the use of microcoils in some cases. If "rich collateral flow from ipsilateral FA or contralateral IMAX" was noted, additional embolization was performed. This resulted in embolization of UMAX in 33%, BIMAX in 20%, UMAX and UFA in 15%, BIMAX and UFA in 28%, and BIMAX and BFA in 5%. Short- and long-term success rates of 94.6% were reported, with 45% minor complications and 0% major complications. The authors concluded from their results and review of the literature that the addition of BIMAX or FA embolization could increase success rates from 74–87% to 88–97%.[37] Tseng reported a large experience with 107 patients undergoing a total of 114 embolizations using PVA (150–250 μm most commonly, with a range of 45–700 μm). Arteries embolized included distal UMAX (61%), BIMAX (13%), UMAX and UFA (6%), and BIMAX and BFA (2%).[56] Immediate and overall success rates were 93% and 88%, respectively, with minor complications in 17% and major in 2% (hemiparesis). Elahi et al reported on 54 patients treated with BIMAX embolization, with additional UFA or BFA embolization in some cases; they reported a 88% success rate and minor/major complications in 23%/2% of patients.[53] A very recent study[32] looked at the relationship between the number of vessels embolized, control of bleeding, and the association with complications. The study included 84 patients treated using PVA (180–200 μm). Vessels embolized included distal UMAX (10%), BIMAX (42%), BIMAX and UFA (38%), and BIMAX and BFA (11%). FA embolization was performed beyond the origin of the submandibular artery. Successful control of bleeding was reported in 88% of patients. An inverse linear association was noted between the number of vessels embolized and the chance of recurrent bleeding ($p < 0.04$). Minor complications were seen in 26% of patients overall (range 0–56%); however, the rate varied linearly ($p < 0.04$) with the number of vessels embolized. The authors concluded that embolization of bilateral IMA and ipsilateral FA on the side of bleeding will lead to fewer recurrences with a minimal increase in (primarily) minor complications. Similarly, Fukutsuji et al also reported increased minor complications (pain) after embolization of more than two vessels.[34]

►**Table 1** is modified with permission from Smith[50] and represents a review of the modern literature, primarily focusing on series with 10 or more patients. These studies include 1,045 procedures in 988 patients, and show an average primary success rate of 87% (range, 74–100%), minor complications in 20% (range, 0–75%), and major complications in 2.4% (range, 0–8%). The literature review of Christensen et al (2005) showed similar results with success rates of 88% and complications of 12%.[57] Minor complications related to epistaxis embolization are generally mild, self-limited, and related primarily to ischemia of the nasal region. These include temporofacial pain or numbness, headache, swelling, jaw claudication, and less commonly trismus. Major complications include skin necrosis,[32,41,42] facial nerve paralysis,[58] monocular blindness, and cerebrovascular accident.[41] The variability in reporting and technique make it difficult to draw conclusions regarding optimal technique. The facial nerve derives its supply from the internal auditory artery (basilar artery), the petrosal branch of the MMA, and the stylomastoid artery that usually arises from the occipital artery but may also arise from the posterior auricular artery. Facial nerve paralysis should be extremely unlikely if one prevents embolization of the petrosal branch of the MMA and AccMA by placing the catheter distal to those arteries.[58]

Embolization versus Surgery: Considerations

Strong et al compared TAIMAXL to embolization and found similar efficacy (89% vs. 94%, respectively), but found that

Table 1 Review of literature on studies using embolization to treat epistaxis

Author	Year	Patients	Procedures	Agent(s) size	Vessel(s) embolized	Initial success	Major complications	Minor complications
Merland	1984	54	54	GF, CA, other	1, 2, 3, 4, 5	51 (94%)	4 (7%)	NC
Strutz and Schumacher	1990	12	13	PVA 150–590	1, 2	10 (91%)	0 (0%)	NC
Wehrli et al	1988	18	19	PVA, GF	1	14 (74%)	2 (11%)	9 (50%)
Vitek	1991	30	30	GF 1–3 mm	1 ± 2	26 + 30 (87% ± 97%)	0 (0%)	1 (3%)
Siniluoto	1993	31	32	GF 1 mm	1	22 (71%)	0 (0%)	1 (3%)
Elden et al	1994	97	108	PVA (small-med)	2, 3, 4, 5	86 (88%)	2 (2%)	25 (23%)
Elahi et al	1995	57	54	PVA (small-med)	1, 2	49 (91%)	3 (6%)	3 (6%)
Klein	1997	26	27	PVA (1 coil)	1, 2	25 (96%)	0 (0%)	NC
Moreau et al	1998	45	46	PVA (small-med)	1, 2, 3	43 (95%)	1 (2%)	3 (6%)
Tseng et al	1998	107	114	PVA ((40)-150–250-(700)), GF 1 mm	1, 2, 3, 4	91 (88%)	2 (2%)	16 (15%)
Cullen and Tami	1998	28	33	PVA, GF, Coil	NC	22 (79%)	2 (7%)	3 (11%)
Leppänen et al	1999	37	38	PVA (150–350), Coil (early experience)	1	33 (89%)	1 (3%)	3 (8%)
Oguni et al	2000	37	40	GF(1–2 mm), Coil	1, 2, 3, 4	35 (95%)	0 (0%)	17 (45%)
Remonda	2000	47	53	PVA (150–500)	1, 2, 3	41 (87%)	2 (4%)	NC
Scaramuzzi et al	2001	12	12	Coil only	1, 2	10 (82%)	0 (0%)	2 (17%)
Ricci et al	2004	18	22	PVA (250–350)	2, 3	21 (95%)	0 (0%)	0 (0%)
Vokes	2004	28	29	GF (1–2 mm), Coil	1, 2, 3, 6, 8	24 (83%)	0 (0%)	3 (12%)
Gurney et al	2004	25	26	PVA 250–500	1, 2, 3	25 (88%)	2 (8%)	1 (4%)
Duncan	2004	51	57	PVA ((150) 250–500 (700)), GF, Coil, CA	1, 2, 3, 4, 5, 7	43 (84%)	1 (2%)	13 (25%)
Andresen	2005	22	30	PVA (150–250), ES (100–300)	NC	17 (77%)	1 (5%)	18 (75%)
Christensen et al	2005	70	70	PVA, GF, Coil	1, 2, 3	61 (87%)	1 (1%)	NC
Fukutsuji et al	2008	22	22	GF (1–2 mm), Coil (FA)	1, 2, 3, 4	17 (77%)	0 (0%)	13 (59%)
Cohen et al	2012	19	20	PVA (250–500), CA (EthA)	1, 2, 3,9	19 (100%)	0 (0%)	0 (0%)
Gottumukkala et al	2012	84	84	PVA (180–300), Coil	1, 2, 3, 4	73 (89%)	1 (1%)	22 (26%)
Totals		977	1,033			862 (87%)	25 (2.5%)	153 (20%)

Abbreviations: CA, carotid artery; CVA, cerebrovascular accident; ECA, external carotid artery; EthA, ethmoidal artery; FA, facial artery; FN, facial nerve; GF, Gelfoam; ICA, internal carotid artery; NC, not clearly stated in paper; PVA, polyvinyl alcohol; STA, superficial temporal artery.

Source: Modified, with permission, from Smith.[50]

Notes: 1, IMA unilateral; 2, IMA bilateral; 3, facial unilateral; 4, facial bilateral; 5, ascending pharyngeal; 6, lingual; 7, accessory meningeal; 8, distal ECA.

surgery was slightly less expensive than embolization ($5,941 vs. $6,783, respectively).[55] The authors suggested that the procedures are complementary and that the decision as to which procedure to choose should be made on an individual basis. Conversely, Elden et al[41] reported in 1995 that embolization was "slightly less expensive" than surgery and significantly less expensive than a trial of conservative therapy. The main cost difference between surgery and embolization was due to fewer days in the hospital for embolization compared with surgery (3.4 vs. 4.7, respectively). The authors concluded that the 2% complication rate was acceptable and that embolization should be considered as an alternative to surgery rather than reserving it for operative failures. Leppänen et al reported an average stay of 2 days in the hospital following embolization (range, 1–5),[52] while Tseng et al reported a mean stay of 1.7 days for patients undergoing embolization.[56] Similarly, Christensen et al reported an average length of stay of 2.5 days for embolization patients,[57] and Cohen et al[43] reported an average stay of 5.2 days (+/ − 3.4 days) following embolization. Cullen and Tami[25] compared IMAX ligation (various techniques) to embolization and reported similar success rates (73% vs. 79%, respectively) and no significant difference in major complications. They found that embolization, on average, was 27% less expensive than surgery ($4,544 vs. $6,183, respectively). For patients with operative failures, Sinuluoto reported successful embolization control of posterior epistaxis in six out of 8 patients (75%) previously treated with surgery, while Elahi et al reported successful embolization in six out of seven cases (86%) where surgery had failed to control the bleeding.[53]

Important Points on Less Common Causes of Epistaxis

Trauma

Maxillofacial trauma, including blunt, penetrating, and iatrogenic (transsphenoidal surgery or other procedures in this region), is an uncommon but potentially serious cause of epistaxis.[59,60] Traumatic epistaxis can be massive and may lead to exsanguination. It is most often caused by laceration of ECA branches, but rarely may be related to ICA injury. When treating such patients it is critical to first secure an adequate airway. This is imperative as patients often have multifactorial airway compromise related to bleeding in the nasal and oral cavity, facial and mandibular fractures, soft-tissue injury, and/or brain trauma with an inability to protect their airway. Basic maneuvers to control blood loss, including manual pressure, nasal packing, suture closure/cautery, and surgical ligation of obvious bleeding sites, should be performed initially. If these measures fail, consideration of embolization or more invasive surgery is recommended.[50,59,60]

Some patients treated initially with surgery may still require embolization in case of failure of ligation.[61] Ideally, angiography is used to identify the exact source of bleeding[31] and to help facilitate embolization (see ►**Fig. 4**). Particulate embolic agents may be used in many cases, but microcoils have more often been described in the setting of vessel laceration or pseudoaneurysm formation. If, however, a patient is extremely unstable or bleeding is massive, one may have to quickly sacrifice the ECA or even more rarely the ICA. Vessel sacrifice may be achieved using coils or by surgical ligation.

Trauma patients may also present in a delayed fashion. This is most often secondary to pseudoaneurysm formation and less often related to arteriovenous fistula formation. Any of the ECA branches previously described or the cervical, petrous, or cavernous segments of the ICA may be the source of bleeding. Epistaxis resulting from a traumatic cavernous ICA pseudoaneurysm is caused by rupture into the sphenoid sinus, as the cavernous ICA is intimately related to the sphenoid sinus in this region.[45] The interval between the inciting trauma and epistaxis varies from days to several decades.[46,47,62] Epistaxis in this setting may be recurrent and progressive or massive and life-threatening. As in the initial trauma setting, coil occlusion of the ICA may be performed; however, successful treatment with covered stents has also been described.[62]

Nontraumatic Internal Carotid Artery Pathology

While the majority of the aneurysms of the petrous or cavernous ICA are posttraumatic and may rarely present with epistaxis,[40,41,49] approximately 10% may be nontraumatic. Causes include mycotic, collagen vascular diseases, or idiopathic. CCAA is often detected in asymptomatic patients undergoing imaging for other reasons. Symptoms are variable and include cavernous sinus syndrome (chemosis, proptosis, ophthalmoplegia, ptosis, and trigeminal sensory loss), superior orbital fissure syndrome, or conductive hearing loss due to hemorrhagic otitis (lateral erosion of petrous bone). Less common presentations include subarachnoid hemorrhage, CCF, and epistaxis. Conductive hearing loss or hemotympanum should lead the physician to suspect the possibility of CCAA in the setting of epistaxis, as this diagnosis carries the possibility of significant mortality (up to 30%).[39] Plain radiographs or CT may show bony abnormalities in the parasellar region, and CTA/MRA may demonstrate the aneurysmal segment of the CCA. Treatment will depend on the exact location and etiology of the source of bleeding.[39,40,46,47,60–62]

Tumors

Epistaxis may be seen in a variety of neoplasms that involve the nasosinus. These include, but are not limited to, juvenile nasopharyngeal angiofibroma (JNA)[63]; nasopharyngeal carcinoma[64,65]; adenoid cystic neoplasm (see ►**Fig. 5**); acute myelogenous leukemia; malignant fibrous histiocytoma; pyogenic granuloma; hemangioma; hemangiopericytoma; meningioma[66]; and metastases. JNA, most often seen in males in the second decade of life, represent highly vascular tumors with a propensity for local spread via the skull base. JNA is one of the more common tumors that may present with epistaxis; definitive treatment of JNA relies primarily on complete surgical removal.[67,68] Embolization for JNA has been described to treat acute epistaxis[41,69] and in the preoperative setting.[68] Technique is similar to that described for idiopathic epistaxis, with the exception that when deemed safe, smaller particles (50–150 μm) may be used to affect greater

devascularization of the tumor.[70] For other tumors and postradiation patients, treatment is similar and will depend on location of vascular abnormalities[71] ranging from particulate embolization to carotid sacrifice.

Hereditary Hemorrhagic Telangiectasias

Patients with HHT (or Osler–Weber–Rendu syndrome) have telangiectasias and/or AVM of multiple organ systems including the nose, GI tract, skin, and central nervous system. Epistaxis is the most common manifestation of the disease related to nasal telangiectasias. The bleeding may be extremely mild or may be severe. Epistaxis tends to be recurrent, starting in the third or fourth decade of life, and progressively worsens over time. Because of the recurrent nature of bleeding in HHT patients, therapy is directed at reducing the frequency and severity of epistaxis.

A myriad of treatments for epistaxis in this population have been described.[48] Because of the limited numbers of patients available, there are no good comparative studies to guide one in determining the best initial therapy in HHT patients that fail conservative epistaxis treatments. The few case reports that are published on the use of embolization in HHT patients suggest significant variability in success rates (20–80%).[42,72] This variability is likely related to differences in definitions of success and length of follow-up. Embolization techniques are generally similar to those described for other causes of epistaxis, with a couple of notable caveats. First, permanent occlusion with coils is generally discouraged due to the recurrent nature of nasal bleeding in these patients.[72] Second, HHT patients will more often require the embolization of multiple vessels and repeat embolization for adequate control. Finally, it has also been noted that patients with HHT have an increased incidence of enlarged ethmoidal arteries feeding the nasal mucosa, as well as other collateral arteries, which may in part explain the increased failure rate of embolization in these patients.[72] For these reasons, Elden et al concluded that embolization for epistaxis in patients with HHT be reserved for cases that have failed all other therapies.[41]

Summary

Epistaxis is a common condition that all IRs should be familiar with from an etiologic, pathologic, diagnostic, and therapeutic perspective. Most cases will not require advanced treatments and will be treated with simple pressure application or anterior nasal packing, and less often cautery. For more severe cases, which most often will be posterior, more formal nasal packing and endoscopic cautery may be required. When these measures fail, a decision will be made as to whether surgery or embolization should be performed. This will depend on local expertise, experience, patient risk factors, and in some cases the cause of bleeding. The literature suggests similar success rates for embolization and modern surgical techniques in treating epistaxis; however, assuming both approaches are available, major complications such as stroke and unilateral vision loss may be higher in embolization. If embolization is elected or necessitated, at least UMAX embolization should be performed, with much of the literature supporting the application of BIMAX embolization. A careful search on the postembolization arteriogram for significant nasal blush or abnormalities may justify the use of FA embolization; however, the operator must accept the possibility of higher complication rates. Fortunately, most of these will be minor. To help decide if multivessel embolization is required, some authors advocate for immediate removal of nasal packs to assess for persistent bleeding; however, this is not practiced in many centers. IRs are encouraged to work closely with local otolaryngologists in developing formal pathways for evaluation and stepwise management of epistaxis patients.

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