

**TITLE: Epistaxis**

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**RESIDENT PHYSICIAN: Glen T. Porter, MD**

**FACULTY PHYSICIAN: Francis B. Quinn, Jr., MD**

**SERIES EDITORS: Francis B. Quinn, Jr., MD and Matthew W. Ryan, MD**

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Epistaxis (Greek for nosebleed) is a problem which has been a part of the human experience from earliest times. It's occurrence was the subject of folklore and myth, and has been treated by physicians from the earliest times. Hippocrates commented that holding pressure on the nose helped to abate bleeding. Others tried writing magical words on the forehead with the patient's own blood, having the patient sniff his/her own dried blood into their nose, and requiring the patient to wear red-colored amulets. Carl Michel (1871), James Little (1879), and Wilhelm Kiesselbach were the first to identify the nasal septum's anterior plexus as a source of nasal bleeding. Pilz was the first to surgically treat epistaxis with ligation of the common carotid artery (1869). This was followed by Seiffert ligating the internal maxillary artery via the maxillary sinus in 1928. Henry Goodyear performed the first anterior ethmoid artery ligation in the treatment of epistaxis. The medical community's understanding of epistaxis has increased dramatically. Our treatment, though somewhat modified over the years, has continued to include techniques first noted several thousand years ago.

Epistaxis is very common in America. In fact, 5-14% of Americans can be expected to have a nosebleed each year. Of those, only about 10% will see a physician. 10% of that number will eventually be evaluated by an otolaryngologist. This generally means that by the time a patient is referred to a specialist his/her epistaxis should be taken seriously.

## **Anatomy and Physiology**

Each nasal cavity is bounded on its medial and lateral aspects by the lateral nasal wall (consisting of three turbinates and a complex system of ostia, sinuses, and fossae), and the nasal septum. Anteriorly and posteriorly it is bounded by the nares and the choana. The roof is made up of the cribriform plate and the fovea ethmoidalis, and the floor consists of the maxilla bone. The cavity is lined with pseudostratified ciliated epithelium, and serous and mucin-secreting glands. A rich vascular supply underlies this thin mucosal covering. Nasal erectile tissue results in a large number of venous sinuses, AV anastomoses, and venules. Multiple arterial vessels fed by the high-pressure carotid system course through the nose. The respiratory mucosa with its underlying vascular supply serve to regulate heat exchange and humidification during respiration.

The nasal cavity is extremely vascular. Blood is supplied via the internal and external carotid systems. The first intracranial branch of the internal carotid artery is the ophthalmic artery which gives off the anterior and posterior ethmoid arteries. These structures course medially and cross the roof of the nasal cavity, supplying the superior septum and lateral nasal wall. The anterior artery is consistently larger, whereas the posterior ethmoid artery is often smaller or branched. The External Carotid artery provides blood to the nose via the Internal Maxillary artery (IMAX) and the Facial artery. The IMAX enters the pterygopalatine fossa and gives off several branches to the nose. These include the sphenopalatine, the posterior nasal, and the greater palatine artery. The sphenopalatine is the largest of these and enters the nose via the sphenopalatine foramen located most commonly at the level of the posterior middle turbinate/ethmoid crest. The greater palatine artery provides blood to the anterior septum by a circuitous route. It emerges from the greater palatine canal via the greater palatine foramen. It then courses anteriorly along the underside of the hard palate and then turns superiorly to enter the nose via the incisive canal. The facial artery supplies the anterior nose via the superior labial artery.

There are two areas that are often implicated in nose bleeds--Keisselbach's plexus (giving rise to anterior bleeds), and Woodruff's plexus (giving rise to posterior bleeds). Keisselbach's plexus is located over the anterior nasal septum and is formed by anastomoses between the sphenopalatine, greater palatine, superior labial, and anterior ethmoid arteries. Woodruff's plexus is located over the posterior middle turbinate and is made up of anastomoses between branches of the IMAX, namely, the posterior nasal, sphenopalatine, and ascending pharyngeal. The maxillary sinus ostium serves as the dividing line between "anterior" and "posterior" epistaxis. Anterior bleeding is usually easier to access and is, therefore, less dangerous. Posterior epistaxis is more difficult to treat as visualization is more difficult and blood is often swallowed, making it more difficult to gauge the amount of blood loss.

## **Etiology of Epistaxis**

The etiology of epistaxis is not always straightforward. Multiple disease processes can result in bleeding from the nose. In order to properly diagnose the patient, it is important that the clinician be aware of underlying abnormalities that can result in the clinical picture. The multiple causes of epistaxis can be divided into two broad categories—local and systemic disease states. Local factors leading to epistaxis include vascular anomalies, infectious/inflammatory states, trauma, iatrogenic injuries, neoplasm, desiccation and foreign bodies. Systemic factors leading to epistaxis include hypertension, atherosclerosis, infectious/inflammatory diseases, blood dyscrasias, platelet deficiencies or dysfunction, coagulopathies, as well as kidney and liver disease. Elucidation of a good medical history as well as a thorough physical exam should allow the physician to significantly narrow his differential.

### **Local Factors**

A rare, but extremely dangerous, vascular etiology is that of carotid aneurysm. Aneurysms can result from cranial surgery or head trauma and develop over a period of years.

Extradural and cavernous sinus aneurysms may burst into the sphenoid sinus with little warning and may be accompanied by sudden blindness or other cranial nerve deficits. Treatment is emergent embolization.

A more common cause of nose bleeds is local infection and inflammation. Sinusitis, upper respiratory tract infections, and even allergies can damage the respiratory epithelium to the point that it becomes friable and irritated. Trauma to the nasal mucosa or the underlying bony and cartilaginous structures can result in bleeding. Nose picking is one form of mucosal trauma thought to give rise to many anterior bleeds. Certainly, traumatic deformation and fractures of the nose and surrounding structures can cause bleeding. Both damaged mucosal surfaces and lacerated vascular structures may bleed. Trauma to the sinuses, orbits, middle ear and the base of skull can result in epistaxis. Bleeding from the nose is also common after sinonasal surgeries as well as orbital floor procedures.

Neoplasms of the nasal cavity, sinuses, and nasopharynx can also give rise to epistaxis. These tumors include nasal hemangioma, hemangiopericytoma, papilloma, squamous cell carcinoma, adenoid cystic carcinoma, adenocarcinoma, juvenile angiofibroma, and melanoma. Tumor-related epistaxis is a result of the tumor erosion of normal structures and is usually unilateral. Juvenile Angiofibroma is nearly always seen with epistaxis, nasal obstruction, and a nasal mass in teenage males. Nasopharyngeal carcinoma should always be suspect with epistaxis and a unilateral middle ear effusion.

Anything that results in disruption of the normal nasal physiology can result in epistaxis. Nose bleeds are more common during the winter months and in hot, dry climates as these climates lead to drying of the nasal mucosa, stasis of secretions, infection and inflammation. This quickly leads to increased vascularity as well as cracking of the mucosa, both of which increase epistaxis. Foreign bodies and intranasal parasites lead to an inflammatory reaction often characterized by purulent drainage, friable mucosa, and bleeding. Anatomical anomalies such as a deviated septum or septal spur can disrupt nasal airflow with resulting desiccation of adjacent tissues. Chronic septal perforations rimmed with delicate granulation tissue can also give rise to bleeding.

### **Systemic Factors**

Hypertension, though not conclusively tied to epistaxis, is a factor in the development of atherosclerosis. Accumulation of the atherosclerotic plaques in the arterial walls with fibrous replacement of the muscular vessel wall layers lead to decreased hemostatic abilities and more fragile vessels. This is thought to be the reason that posterior nose bleeds are seen more often in older persons.

Hereditary hemorrhagic telangiectasia is the most common systemic disorder of the vascular system which affects the nasal mucosa. This disease results in the lack of contractile elements in vessel walls and multiple telangiectasias (dilated venules and capillaries or small AV malformations) throughout the skin and mucosa of the aerodigestive and urinary system. Epistaxis is difficult to control as the vessels do not spasm normally. Onset of symptoms is

usually at puberty and progressively worsens with age. These patients usually die from gastrointestinal bleeding.

Systemic diseases such as syphilis, tuberculosis, and Wegener's granulomatosis can lead to epistaxis as they tend to ulcerate normal tissues. Typhoid fever, nasal diphtheria, whooping cough, scarlet fever, rheumatic fever, and leprosy can also cause epistaxis.

Epistaxis as a result of blood dyscrasias can be found in patients with lymphoproliferative disorders, immunodeficiency, systemic disease, or in the alcoholic patient. A low platelet count is one dyscrasia that can lead to bleeding. Thrombocytopenia is technically a platelet count less than 100,000. However, clinically significant thrombocytopenia is usually around 40-50,000. There can be spontaneous mucous membrane bleeding at levels of 10-20,000. This platelet deficiency can be the result of chemotherapy agents, malignancies, hypersplenism, DIC, drugs, and multiple other disorders. In contrast, platelet dysfunction is seen when there are enough platelets, but they do not function normally. This can be seen in liver failure, kidney failure, vitamin C deficiency and in patients taking aspirin and NSAIDs. Clotting factor abnormalities can result in frequent, recurring epistaxis. VonWillabrand's disease (most common), Factor VIII deficiency (Hemophilia A), Factor IX deficiency (Hemophilia B), Factor XI are all common primary coagulopathies. Disorders of fibrinogen, prothrombin, and the other clotting factors are rarely seen. Drug-related coagulation disorders are often seen in patients taking aspirin, coumadin, NSAIDs as well as in patients with Vitamin C and K deficiencies. Patients taking herbal supplementation such as Ginkgo Biloba and Vitamin E may increase their risk of bleeding.

Nose bleeds can also be divided by age group. Epistaxis is rarely seen in infants. Children with nosebleeds are often diagnosed with anterior septal bleeds, often secondary to nose picking. They are also more likely to have a nasal foreign body. One study of chronic nose bleeds in children showed that 1/3 of these patients can be expected to have a coagulation disorder. The etiology of epistaxis in young adults is often trauma or idiopathic. Nosebleeds in the middle-age patient are more often the harbinger of neoplastic disease. Finally, older persons have nosebleeds as a result of hypertension and atherosclerosis (more prone to posterior bleeds) These are not hard rules, but generalities that can help to direct the search for a diagnosis.

## **Evaluation of Epistaxis**

When evaluating a patient with epistaxis it is important to pursue an extensive medical history and physical examination. Initial management should always begin with evaluation of the "ABC's" of ATLS initial resuscitation. This should include evaluation of the patient's blood pressure and heart rate. Fluid resuscitation may be necessary in the epistaxis patient. Replacement with Lactated Ringers or Normal Saline is usually sufficient. If the patient is estimated to have lost more than 30% of his/her blood volume (1.5 liters in a normal adult), or if he/she shows signs of severe blood loss (tachycardia, low blood pressure, tachypnea) blood products should be transfused. Once the patient is stable, a full history and physical examination can be pursued. In most situations, the specialist will not be involved until after the patient is stabilized.

Once the patient is stable and the medical history has been obtained, the physical exam may proceed. Care should be taken to address systemic disorders (hypertension, thrombocytopenia, etc.) before proceeding with any sort of treatment. The physical exam can then proceed while these systemic problems are being addressed. Anterior rhinoscopy is sometimes sufficient, but most of the time a nasal endoscopic exam should be pursued. Visualization is often poor and requires suctioning of clots and any active bleeding (some otolaryngologists recommend having the patient vigorously blow the clots out themselves, claiming that doing so will stop many bleeds). If possible, it should be ascertained from which quadrant the patient is bleeding. This will help dictate subsequent treatment. Special attention should be paid to the plexus areas as these are areas that often bleed. There are patients who are bleeding so profusely that any exam is difficult. These patients require more immediate intervention.

## **Treatment of Epistaxis**

### **Non-surgical**

Traditionally, patients are initially managed with topical vasoconstrictors, packing or cauterization. These interventions are able to stop the bleeding 80-90% of the time. Rebleeding is common, however. Topical treatments include Afrin, Pseudoephedrine, Lidocaine with epinephrine, and cocaine. For mild nose bleeds this is often all that is needed. Packing is the placement of an intranasal device which will apply constant local pressure. The material may be nugaue, merocels, epistats, a rapid rhino, or any one of a hundred different sort of nasal tampons—they all essentially do the same thing (in fact, some otolaryngologists have reported endoscopically applied local pressure can be effective in the treatment of epistaxis). There are essentially three locations for packing—anterior, posterior, and anterior/posterior. Once inserted (usually coated in antibiotic suave), these devices can be inflated with air or expanded with vasoconstrictive agents or antibiotic drops. The exception to this is the “old-fashioned” anterior/posterior nasal pack. The anterior pack is performed either separately or in conjunction with the posterior pack. It is placed by using forceps to pack the nose with six feet of gauze with the first layer placed on the nasal floor and subsequent layers placed on top of the underlying layer until the nose is tightly packed. The posterior pack consists of a gauze pad which is launched into the posterior nasopharynx and lodged against the choana via the oral cavity (a red rubber catheter is inserted through the nose and a gauze pad is tied to the catheter with suture which is then used to pull it into the back of the nose). It is important to recognize the potential for alar necrosis, toxic shock syndrome, hemodynamic changes, apnea, and discomfort that nasal packing of any sort can induce. Any patient receiving a posterior pack should be admitted to the hospital. It is generally understood that older patients, children, and those with comorbidities should be admitted to an ICU for cardiopulmonary monitoring if a posterior pack is being placed. Newer devices have “airways” built in which allow airflow through the nasal cavity despite the packing. There are no studies looking at their effect on the known risks of nasal packs. Although antibiotics are probably not necessary, they are usually given to patients with anterior packs in place. These patients can often be discharged home with follow up arrangements.

Most nasal tampon devices must eventually be removed (usually within three days). This presents a problem in the patient with an irreversible coagulopathy. In these patients it may be

more wise to pursue cautery or packing with absorbable material (surgicel, gelfoam, etc.). Some of the newer pneumatic packs may also be good candidates as they are coated with a biologic substance which may allow atraumatic removal. Fibrin glue application was noted to be effective in these patients (n=10) in one study. Some feel that these patients should become embolization/surgical candidates if these preliminary attempts fail to stop the bleeding.

Nasal cautery can be very effective. First, it is important to be able to see the location of the bleeding tissue and be able to access it. Cauterization can be performed using silver nitrate sticks (these should be held in place for approximately 30 seconds, but avoided for bilateral septal bleeds), Trichloroacetic acid, bipolar, and the suction bovie. There have been several articles addressing bipolar treatment that show good efficacy (about the same as packing—70%) and low complication rates. Some otolaryngologist have used a red rubber catheter in the contralateral nasal cavity on continuous suction to facilitate visualization when performing intranasal cautery. However, it is important to remember that cautery is NOT effective in a heavily bleeding field. In such a situation one of the other management options should be opted for.

There are many other nonsurgical means of managing nosebleeds. Most have not been subject to clinical trials (please see [Dr. Quinn's Epistaxis Pearls from the Internet](#)). A few exceptions bear mentioning. One is hot water irrigation which, with the right device (to avoid aspiration), appears to be effective in a majority of patients and compares favorably with nasal packs (less pain, slightly better efficacy, shorter hospital stay). Another study showed that the use of daily antibiotic salve with and without initial silver nitrate cautery showed no increased efficacy (short or long-term) with silver nitrate. One author reported short-term control of epistaxis with a greater palatine foramen injection of saline or 1% lidocaine with epinephrine (2-3 cc). His study showed that the local pressure exerted by the increased fluid volume was the mechanism of action (not the vasoconstrictive agent).

Indications for surgery have been widely debated, but usually include failure of medical treatment after 72 hours, nasal anatomy that precludes local treatments, patient refusal of medical management, initial hematocrit of <38% (males), and the need for transfusion. Many authors have argued that a posterior bleed that will necessitate a posterior pack is indication enough to pursue surgical treatment. They cite the morbidities of posterior packs and the high percentage of patients who go on to receive surgical treatment after posterior pack application. A hotly debated issue is the relative hospital cost—studies have shown both increased and decreased hospital costs with surgical treatment of epistaxis. Future studies looking at short-term and long-term costs specific to different treatment protocols for posterior nasal bleeds would be helpful.

## **Surgical**

Once the medical management of the patient's epistaxis has been deemed a failure surgical management and/or embolization are the next steps in control of the bleeding. The surgical options include transmaxillary internal maxillary artery ligation, transoral internal maxillary artery ligation, anterior/posterior ethmoid ligation, external carotid ligation, transnasal endoscopic sphenopalatine artery ligation, and submucosal supraperichondrial septoplasty. For patients with HHT septodermoplasty, laser, and nasal closure are surgical options.

Transmaxillary IMAX ligation was first proposed in the 1920's. It soon became the surgical option of choice for recalcitrant epistaxis. This procedure is performed via a Caldwell-Luc approach to the maxillary sinus. The posterior sinus wall is carefully removed and the pterygopalatine fossa contents exposed. Dissection under the operating microscope allows the surgeon to ligate the distal branches of the internal maxillary artery (ascending pharyngeal, sphenopalatine, posterior nasal) under direct vision. There is a 10-15% technical failure rate. Complications (25-30%) include pain in teeth, damage to teeth, damage to sphenopalatine ganglion or vidian nerve, damage to infraorbital nerve, oro-antral fistula, and sinusitis. It also requires a general anesthetic. The transoral approach is reserved for patients with no maxillary sinus and is generally less effective as the ligation is more proximal to the bleeding.

Anterior/Posterior Ethmoid artery ligation is performed for patients with superior bleeding or in conjunction with IMAX surgery for patients with an unknown bleeding source. The approach is made via a Lynch incision and the periorbita is carefully dissected free from the medial orbital wall. The anterior artery is located approximately 8-12 mm from the lacrimal crest. The posterior artery is located approximately 10-12mm from the anterior artery. The optic nerve is only 4-6 mm posterior to the posterior ethmoid artery. The effectiveness of this procedure is not always known, but complications (25-30%) include stroke, blindness, ophthalmoplegia, and epiphora. External carotid ligation is seldom performed as it is very proximal to the bleeding site and often is not as effective as more distal treatments and carries more severe complication risks. One author has proposed submucosal supraperichondrial septoplasty for recurrent septal bleeds (fibrosis results in decreased vascularity). Septoplasty may be indicated for patients in whom a nasal deformity is judged to be the cause of their epistaxis. A treatment that has received more attention in the recent literature is transnasal endoscopic sphenopalatine artery ligation. There are multiple studies which report an efficacy rate of 87-100% efficacy and no complications. The approach is via an endoscopic dissection of the posterior middle turbinate (in a subperiosteal plane) and exposure and ligation of the sphenopalatine artery with a clip. The mucosa is then replaced. This is perhaps the most distal treatment and is very effective in the case of bleeding from this artery. Although early authors found this approach to be fairly difficult, but recent authors indicate that this approach should be fairly straightforward for the skilled endoscopist. Finally, the management of Hereditary Hemorrhagic Telangiectasia has been studied and at least two studies indicate that two epistaxis populations exist—mild and severe. Patients with mild disease are most effectively treated with laser ablation and topical estrogen cream. More severe recurrent epistaxis should be treated with septodermoplasty (removal of nasal mucosa and split-thickness skin grafting of the area) or even nasal closure (almost complete resolution). These patients always have ingrowth or regrowth of the telangiectasias, but they are always less severe.

Embolization of the internal maxillary artery is effective, safe, and quick. A catheter is introduced via the femoral artery and advanced to the level of the external carotid. Angiography identifies the target arteries, and may show the location of the nasal bleed (>.5ml/min). Platinum coils are then released to cause embolization of the target arteries. Initially, this intervention was thought to be indicated only in those patients who failed surgery. However, recent literature supports embolization as first-line treatment with or without surgical management. This change is primarily due to safer procedures (coils and pre-embolization angiography), and accumulation of experience. Currently, embolization is effective in 82-100% of the cases with minor complications seen in 0-45% of patients (facial pain, paresthesias, etc.—resolve in one week), and 0-3% major complications (CVA, paraplegia, blindness—all of which resolved (recent

literature)). Embolization is indicated in patients who cannot tolerate anesthesia, those who have failed surgery, generalized bleeding disorders, or in patients with bleeding sites that are difficult to access. It is contraindicated in patients with severe atherosclerosis. In many centers this has become the procedure of choice for patients with severe refractory epistaxis. However, not all hospitals employ interventional radiologist who can perform embolization. In such a situation surgical management would be the only option.

Epistaxis is the manifestation of many different disease processes. Its treatment is as varied as its etiologies. Treatment will be most effective when underlying medical problems are understood, nasal anatomy is appreciated, and the patient's response to treatment and general medical status are taken into account. The otolaryngologist should be familiar with treatment options and be able to offer surgical intervention, if necessary.

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