Chapter 41: Epistaxis and Nasal Trauma

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Foreign Bodies

Foreign bodies of the nasal cavity, like those of the ear, are generally found in the pediatric or institutionalized population. The literature is replete with case reports documenting an incredible list of nasal foreign bodies, both animate and inanimate. Most primary practitioners and otolaryngologists - head and neck surgeons have anecdotal reports of sensational or exotic foreign bodies that they have encountered. Management can be quite challenging, not only from the standpoint of diagnosis but also from the standpoint of removal. The importance of this section is to remind the reader that in pediatric and institutionalized patients foreign bodies must be of primary consideration in the differential diagnosis of unilateral nasal obstruction, fetid purulent serosanguineous rhinorrhea, unilateral sinusitis, and widespread infection of the face, orbit, and periorbital areas.

The parent or custodian will sometimes witness or suspect insertion of a foreign body by the patient or a sibling or playmate. However, most cases are unwitnessed, and the chief complaint is fetid discharge, purulent rhinorrhea, or sinusitis. Diagnosis in these cases is not always straightforward. Metallic foreign bodies can usually be seen on radiographs of the sinuses and soft tissues. Even with the aid of contrast material, however, radiographs are of little help with items such as food particles, pieces of plastic, wood, rubber, cloth, crayons, or chalk. In addition, many objects have been in place for substantial periods of time and swell because of water absorption. This fact makes their identification and removal difficult. At times distinguishing a foreign body from altered nasal anatomy, such as a benign polyp or a neoplasm, is challenging.

The loose object in the nose must be dealt with carefully. Mobile foreign bodies can be easily lodged in the nose or pushed posteriorly into the nasopharynx and aspirated into the larynx or trachea, thereby creating a potentially life-threatening situation.

The following outline gives the major types of nasal foreign bodies. Dividing them into animate and inanimate foreign bodies is customary, and the discussion that follows makes that separation:

I. Animate foreign bodies

A. Myasis

1. Screwworms (*Cochliomya hominivorax, C. macellaria*)

2. Fly larval forms (*Oestrus, Hypoderma, Dermatobia*)

B. *Aspergillus* infections

C. Rhinosporidiosis (nasal polyps)

D. *Ascaris lumbricoides* (roundworms)

II. Inanimate foreign bodies

A. Anything that can fit in the nose

B. Rhinoliths.

Etiology

Animate foreign bodies

The nose infested with insects, fly larvae (maggots), or intestinal worms is not a healthy or hygienically clean nose. The conditions that predispose to animate foreign body infestation are characteristically found in hot, humid, or dry climates. Close living conditions, poor sanitation, and poor hygiene are the rule in most cases. The condition known as myasis (infestation with fly larvae) is seen in some parts of the southwestern USA. Flies of many different species produce myasis in humans. Some require an intermediate host for larval development, whereas others are opportunistic and cause infestation only if the host is debilitated or has suffered an injury to tissues. Infestation may result from egg deposition, with penetration of normal mucosal lining of the nose, or through a previously injured site.

The Texas screwworm fly (*Cochliomyia hominivorax, C. americana, C. macellaria*) produces one specific form of myasis in humans. Brown (1945) details the life cycle of the Texas screwworm fly. The infestation begins when the female deposits a cluster of eggs (up to 400), which hatch in 6 to 12 hours. The freshly hatched larvae are infectious and survive up to 6 days. The larvae burrow into the tissues, forming pockets where they mature. The larval stage causes disease in the nose and paranasal sinuses, resulting in tissue destruction, infection, and even penetration of bone. The process can ultimately involve the meninges, leading to meningitis, encephalitis, brain abscess, and death. Treatment involves instilling oil or chloroform into the nose. The chloroform irritates the larvae, which then crawl out of the nose and can be killed.

Some amebae can cause opportunistic infection via nasal entry. These free-living amebae comprise a large group that inhabit brackish water. The *Naegleria* is an ameboflagellate in which the ameboid stage alternates with one possessing two flagella. Pathogenicity of this ameba is well documented (Butt, 1966). Most cases occur during the summer months in young people who have recently swum or dived in brackish water. The amebae may enter the central nervous system via defects in the cribriform plate. Signs and symptoms of meningitis with olfactory, frontal temporal, and cerebellar involvement are noted. Patients become irrational, lapse into coma, and may be dead within 1 week as a result of overwhelming meningoencephalitis.

Rhinosporidium seeberi, although quite rare in the USA, causes a fungal disease that is responsible for significant nasal parasitism among the natives of India or Sri Lanka. Nasal polyps form that contain spores of the fungus in all stages of development. These polypoid masses have a filiform, leaflike process that is dull pink. Their surface is studded with many tiny pale spots, which are the sporangia. These polypoid growths are vary friable and bleed easily when touched. Treatment of rinosporidiosis is surgical, although diaminodiphenylsulfon (DDS, dapsone) is effective in nasal and nasopharyngeal rhinosporidiosis (Nair, 1979). The use of intravenous amphotericin B should also be considered when surgical removal is incomplete.

Aspergillus is another fungus that causes nasal disease. This organism can produce inflammatory granulomatous lesions in the nose and paranasal sinuses. The fungi are ubiquitous in nature, and most humans are immune to infection or are asymptomatic carriers. The disease is usually seen in immunocompromised patients, although it occurs more and more frequently in individuals without underlying disease. *Aspergillus fumigatus* and *A. niger* are the common fungi cultured in true infection. Material suspected of harboring yeast should be examined histologically in fresh and stained (silver) preparations. In addition, material should be cultured on Sabouraud's glucose agar. On microscopic examination the cultured specimens show conidiophores that expand into large vesicles at the end. In tissue the diagnosis is based on finding mycelia with branching septate hyphae.

Ascaria lumbricoides infestation is one of the most common parasitic diseases of humans. Ascariasis is found in unsanitary conditions in temperate and tropical areas of the world. Ingested eggs hatch in the duodenum where the larvae penetrate the mucosa and enter blood vessels and lymphatics. The larvae are carried either to the liver or the heart, but eventually they end up in the lungs. Larvae penetrate into the alveolar spaces where they grow and molt. After about 10 days, a migration up the trachea into the esophagus occurs, and ultimately they return to the small intestine. During migration and regurgitation larvae may enter the nose and paranasal sinuses. *Ascaris* infestations are treated with piperazine citrate or with surgery in cases of complete intestinal obstruction.

Inanimate foreign bodies

The list of inanimate foreign bodies that can enter the nasal passages is far more extensive and considerably more dramatic and exotic than that of animate foreign bodies. Virtually any item that can be placed into the nose has been found there. The case reports in the literature are vast and varied. The mechanism of entry is usually at the hands of a child or a playmate.

The more common foreign bodies that otolaryngologist - head and neck surgeon or pediatrician sees are beans, nuts, peas, pieces of chalk, erasers, crayons, marbles, cloth, pieces of meat, vegetable matter, fruit pits, jewelry, small pieces of plastic, wood, and hardware (screws, nuts, bolts), especially those used in toy building sets.

An example of an unusual nasal foreign body is the hearing aid battery. The alkaline battery causes tissue damage by flow of a low direct current that leads to tissue lysis and necrosis

(Babu, 1981). Malhotra et al (1970) reported finding a broken handle from the door of an Army vehicle in the nose of one of his patients. This arrowhead-shaped, metallic foreign body was lodged in the nose and maxillary sinus for 24 days and remained unnoticed by the patient and his physicians until facial swelling developed.

Failure to recognize a foreign body is not unusual. Guthrie (1926) cites two cases of interest. The first was a 34-year-old man who had had a 2-inch bolt in his nose for 17 years; the second patient had a calcified cord in his nostril for 16 years. In each case the foreign body remained asymptomatic and was discovered during a routine physical examination for another problem. Marrone et al (1968) reported a case of a patient having a tooth in the nose resulting from avulsion during endotracheal intubation for a nondental procedure. McAndrew (1976) reported the displacement of a lower third molar into the posterior choana during dental extraction. The missing tooth went unnoticed until a radiograph was taken.

Iatrogenic foreign bodies include pieces of broken instruments, gauze and cotton sponges, nasal packing, and needles left behind during routine nasal or sinus surgery. When applying topical anesthesia for nasal work, the surgeon should use neurosurgical cottonoid that has radiopaque stripes and strings for handling. These items are usually counted in the operating room, which eliminates the chance for loss.

Rhinoliths are calcified masses found in the nasal passages. They consist of salts of calcium and magnesium in the form of phosphates, oxalates, and carbonates. The salts crystallize around a small piece of organic or inorganic material and after several years can reach formidable size. Removal usually requires general anesthesia and may necessitate crushing the rhinolith into fragments before removal.

Diagnostic assessment

Diagnosis may be difficult if no history is available. Nevertheless, considering a foreign body in the differential diagnosis of unilateral nasal obstruction and purulent, fetid, serosanguineous rhinorrhea, with or without sinusitis, is imperative. Abscess of the nasal septum, nasal dorsum, and cheek can be the result of a foreign body as well. In most cases the child never volunteers that he put something in the nose, and it may not be until the presenting infection has been treated that an intranasal examination will divulge the cause of infection.

Radiographic analysis of the nose and paranasal sinuses is helpful if the foreign body is metallic or calcified and to rule out any accompanying sinusitis. At times, contrast material placed in the nose will outline a space-occupying object. The mainstay of diagnosis, however, remains the physical examination. Good lighting, nimble fingers, and, most of all, a cooperative patient are essential. In children or uncooperative adults, general anesthesia may be necessary. Pretreatment of the nose with a topical vasoconstrictor shrinks the mucosa to improve stability and decrease bleeding.

Management

The management of inanimate foreign bodies is, of course, removal. The key is to be able to solve the problem without creating new ones. Specifically, the uncooperative patient should be given general anesthesia. Repeated attempts at removal in a child thrashing about will ultimately lead to trauma of the surrounding tissues, epistaxis, and possibly further impaction of the foreign body into the nose. In the case of a posterior foreign body the object can be pushed into the nasopharynx where aspiration will cause obstruction of the airway. The instruments used for removal are subject to the preference of the otolaryngologist - head and neck surgeon. Hartmann alligator forceps, bayonet forceps, or wire loops are common choices. The treatment of rhinoliths, as mentioned previously, may require breaking the mass into fragments and removing the pieces individually. Occasionally lateral rhinotomy is necessary. With animate objects appropriate cultures and histologic studies should be obtained. The pathologist must examine tissue samples of polypoid masses or granulation tissue to rule out neoplastic disease. In most cases of animate foreign bodies surgical removal is adequate.

Complications

The clinical manifestations of a nasal foreign body that cause the patient or parent to seek medical attention are frequently the complications brought on by the untreated foreign body. The foreign body sets up local inflammation and edema of the nasal mucosa. The body's defense mechanisms try to "wall off" the foreign object, and soon rhinorrhea becomes purulent. As local tissue necrosis develops, the discharge becomes foul smelling and serosanguineous. This local reaction can lead to ethomoid, maxillary, and sphenoid sinusitis with their associated complications. Periorbital and orbital cellulitis, orbital abscess, meningitis, encephalitis, brain abscess, and cavernous sinus thrombosis are all potential complications of sinus disease. Nasal septal abscess and destruction of the cartilaginous nasal structure are also possible if treatment is not sought.

Summary

The population affected most by inanimate nasal foreign bodies is the pediatric or institutionalized patient. The child will usually not volunteer information about the placement of a foreign body, and the parent may not have witnessed the event. The physician must consider the possibility of a foreign body when confronted with a child who has a foul smelling, unilateral, nasal discharge. The diagnosis and removal of the foreign body may require general anesthesia. Repeated attempts at removal in the awake, uncooperative patient are discouraged. During removal care should be taken to avoid impacting the object further in the nose or creating a nasopharyngeal foreign body that can be aspirated and obstruct the airway.

Epistaxis

Most patients who develop nasal bleeding handle the problem without the need of a physician. Because of inexperience, improper equipment, or inadequate knowledge of nasal anatomy and physiology, the primary care physician frequently unsuccessfully treats cases

intractable to home remedies. The 5% to 10% of patients requiring an otolaryngologist to control expistaxis can be a significant challenge to the most experienced physician. These patients may require hospitalization, blood transfusions, and in some cases surgical interruption of the nasal blood supply. If not successfully managed, they can be victims of life-threatening cardiac and respiratory abnormalities an sepsis, which may lead to death.

Epidemiology

Epistaxis affects all age groups without sex predilection. Anterior epistaxis is more common in the child or young adults, whereas posterior nasal bleeding is more often seen in the older adult with hypertension or arteriosclerosis. The incidence is somewhat higher during the colder winter months when upper respiratory infections are more frequent and temperature and humidity fluctuations are most dramatic. Epistaxis is also common in hot dry climates with low humidity. Patients who suffer from sinus disease, nasal inflammation, and allergy are more prone to epistaxis because the nasal mucosa is more inflamed, hyperemic, and friable. In addition, changes from a bitter cold outside environment to a warm, dry, heated house result in variations of the normal nasal cycle of alternating congestion and decongestion. These changes can result in poor ventilation of the sinuses, infection, nasal congestion, engorgement of the mucosal linings, and ultimately epistaxis. The following list gives the more common factors predisposing to epistaxis:

1. Infection

a. Rhinitis

b. Nasopharyngitis

c. Sinusitis

2. Trauma

a. Accidental or self-induced

b. Iatrogenic

3. Allergy

4. Hypertension and atherosclerotic vascular disease

5. Hereditary hemorrhagic telangiectasia

6. Blood dyscrasias

a. Iatrogenic (drug induced)

b. Disease mediated

c. Alcoholism

7. Atrophic rhinitis

8. Tumor

a. Primary

b. Secondary

9. Congenital or acquired nasal defects.

Trauma to the nose distorts the bony cartilaginous framework and may tear the nasal mucosa. Bleeding occurs from raw bone edges or lacerations of the vascular mucosa. Common sites for such mucosal tears are along the septum, at the lateral recesses of the piriform aperture, and at the junction of the upper lateral cartilages with the nasal bones. A direct blow may result in laceration of the mucosa at the lateral margins of the inferior turbinate or at the junction of the quadrilateral cartilage with the bone septum. Patients with a grossly distorted nasal framework may require closed reduction of the fractures before epistaxis can be controlled. Another source of trauma in today's society is the nonmedical use of cocaine in the nose. The nasal lining is chronically irritated and frequently infected, leading to septal perforation, crusting, and epistaxis. Foreign bodies are another form of trauma that lead to direct lacerations of mucosa or secondary infection, granulation tissue, or bleeding.

Hereditary hemorrhagic telangiectasia (Osler-Weber-Rendu disease) is an autosomal dominant disease. The pathologic condition lies in the inherited lack of contractile elements in the walls of blood vessels. The telangiectasias are dilated venules and capillaries or small arteriovenous malformations that are found in the skin or mucosal linings of the entire aerodigestive tract. The coagulation parameters in these patients are entirely normal, including absolute platelet counts and tests of platelet function. The telangiectasias bleed from the slightest trauma, usually on a daily basis. These patients experience recurrent epistaxis and gastrointestinal hemorrhage and during their lives require hundreds of blood transfusions. Treatment is very difficult and usually unsuccessful. The mainstay has been resurfacing the nasal septum and lateral walls of the nose with dermis obtained from the thigh. Saunders (1968) reported excellent results from this technique. Recently the use of laser photocoagulation has shown promising results. Parkin and Dixon (1981) reported laser treatment of eight patients using both the argon and neodymium:yttrium aluminum garnet (Nd:YAG) lasers, with substantial improvement in symptoms. Treatment must be repeated every 4 to 6 months because of the development of new lesions.

Blood dyscrasias are usually seen in the alcoholic patient or the patient with a debilitating systemic disease, immunodeficiency, or a lymphoproliferative disorder. These patients have decreased clotting factors and low platelet counts, which results in epistaxis that is extremely

difficult to control.

The atrophic rhinitis known as ozena is caused by a chronic bacterial infection from *Klebsiella ozaenae.* Nasal crusting and a mucosa that is atrophic, fibrotic, and scarred characterize it. Treatment of the underlying disease prevents epistaxis.

Neoplastic diseases, both benign and malignant, cause epistaxis indirectly from erosion of normal sinonasal structures or directly as in tumors of high vascularity, such as nasopharyngeal angiofibroma or hemangioma. The differential diagnosis of nasal tumors is extensive and beyond the scope of this chapter. However, when biopsy of an intranasal mass is planned, the physician should be in a controlled setting where packing materials, cautery, and blood for transfusion are available.

The list of diverse causes of epistaxis serves as a remainder to the physician that a thorough history and physical examination and the appropriate laboratory studies to rule out previously undiagnosed coagulopathy are essential in the diagnostic assessment of persistent epistaxis.

Anatomy and blood supply

The nasal cavity is one of the most important physiologic components of the upper aerodigestive tract. This fact frequently goes unrecognized until some insult alters normal breathing. The epithelial lining of the nasal cavity is quite specialized and diverse. Keratinizing stratified squamous epithelium lines the nasal vestibule. Respiratory epithelium covers the majority of the septum, floor, and lateral walls, and olfactory epithelium lines the superior turbinate and uppermost septum.

The nasal mucosa is endowed with a rich blood supply derived from both the external and internal carotid arteries. The external carotid system provides two main sources of blood to the nose: the maxillary artery and the facial artery. The facial artery is less significant than the maxillary artery. The superior labial artery, which arises from the facial artery, has two nasal branches: the septal branch ramifies on the anterior nasal septum and vestibule, and the alar branch supplies the nasal ala. The most significant external carotid contribution is the maxillary artery. Several terminal branches of the maxillary artery exist in its third, or pterygopalatine, portion. The sphenopalatine and the descending pharyngeal arteries are important to the otolaryngologist dealing with epistaxis. The sphenopalatine artery enters the nasal cavity through the sphenopalatine foramen at the posterior limit of the middle turbinate. The artery has two branches, the posterior lateral nasal branch, which supplies the turbinates as well as the ethmoid and maxillary sinuses, and posterior septal branches, which cross over the nasal roof beneath the sphenoid bone to supply the entire septum inferiorly and anteriorly.

The internal carotid artery supplies the nose via the ophthalmic artery as it arises from the carotid at the cavernous sinus (Fig. 41-1). The ophthalmic artery has two branches to the nose, the posterior and anterior ethmoidal arteries. The posterior ethmoidal artery is the smaller of the two and enters the posterior ethmoidal foramen to supply the cells of the posterior

ethmoid sinus. Nasal branches of the posterior ethmoidal artery descend into the nose through the cribriform plate and ultimately anastomose with branches of the sphenopalatine artery. The anterior ethmoidal artery enters the anterior ethmoidal foramen with the nasociliary nerve to supply the anterior ethmoidal air cells, frontal sinus, and dura. Nasal branches of the anterior ethmoidal artery enter the nose along the crista galli and supply the anterior superior septum and lateral walls.

Several areas are of interest within the nose because of a high frequency of associated epistaxis. The first is located along the septum where branches of the anterior ethmoidal, sphenopalatine, and superior labial arteries anastomose. This site is known as Kiesselbach's or Little's area and is the site of most anterior nosebleeds (Fig. 41-2). The site associated most frequently with posterior epistaxis is located at the entry zone of the sphenopalatine artery behind the middle turbinate (Fig. 41-3). Accurately defining the incidence of anterior versus posterior epistaxis is difficult, but most authorities would agree that anterior epistaxis accounts for more than 90% of nasal bleeding, especially in children and young adults.

Management

Patients with nasal bleeding usually control the problem with direct pressure. When individual efforts fail, patients contact their primary physician or seek help in a hospital emergency room. The otolaryngologist - head and neck surgeon is asked to see patients when initial packing or cautery has failed. The initial step in control of the nosebleed is establishing rapport with the patient. Assessing the degree of blood loss and determining the packed cell volume and the need for transfusion are also important. Underlying medical problems should be addressed and treated appropriately. In trauma cases appropriate treatment of life-threatening injuries takes priority. Management of the airway and fluid replacement are mandatory and at times requires stopgap measures to control epistaxis and protect the airway. One or two intranasal Foley catheters can be inflated in the nasopharynx and pulled from the anterior nostrils to compress potential bleeding sites posteriorly or to protect the airway from blood.

In uncomplicated cases management starts with the physical examination. This requires a cooperative patient, good lighting, a nasal speculum, suction, and a topical agent that combines anesthesia and vasoconstrictive properties (4% cocaine hydrochloride is an excellent agent for this).

At times having the patient blow his nose is helpful to evacuate clots and debris. Next, the precise bleeding site is carefully identified so that packing can be placed against it. Before applying any packing or cautery, cotton pledgets saturated with cocaine solution should be placed in the nose to provide vasoconstriction and topical anesthesia. As with any drug, dosage and potential toxic side effects must be known before use. The dose of cocaine for adults is 2 to 3 mg/kg of body weight, which means that a person weighing about 70 kg would be given 5 mL of a 4% solution (40 mg/mL x 5 mL = 200 mg; 3 mg/kg x 70 kg = 210 mg). For children the solution should be no stronger than 1% to 2%. Physiologic disturbances from high doses of cocaine include pyrexia, dilated pupils, tachycardia, irregular respirations, abdominal pains, vomiting, and major convulsive seizures.

The majority of nosebleeds are located anteriorly on the septum. The vasoconstriction diminishes the blood flow and makes either silver nitrate cautery or electrocautery more effective. The anesthesia provided by the topical cocaine may be inadequate if a substantial anterior pack is to be placed. In most cases failure of the anterior pack to control epistaxis means that the pack was improperly placed because of poor visualization, anatomic constraints, or pain and discomfort for the patient. This can be overcome by supplementing the topical anesthesia with injectable 1% lidocaine hydrochloride with epinephrine added. The nasal dorsum, septum floor, and infraorbital nerves are blocked with no more than 10 to 15 mL of solution. In addition, a palatal nerve block with lidocaine can be helpful. This is administered into the greater palatine foramen located approximately 1 cm anterior and 1 cm medial to the hook of the hamulus of the pterygoid plate. Anterior packs are made from long, 1-inch wide strips of gauze. Plain gauze in 3-yard lengths is fine for this purpose. The gauze is heavily impregnated with antibiotic ointment and placed in the nose in pleated fashion, starting inferiorly along the floor and packing superiorly, taking care to push the gauze under the inferior turbinate. Only closed loops of gauze are placed posteriorly to prevent strands of packing from dangling down from the nasopharynx. Previously fashioned Telfa or gauze strips, 1/2 inch x 4 inches, can be substituted for gauze strips (Fig. 41-4, C). Anterior packs are left in place for 3 to 5 days.

If an adequately placed anterior pack fails to stop bleeding, the bleeding site is possibly located posteriorly and this may or may not be verified by nasopharyngoscopy. A posterior pack is placed for true posterior bleeding or in some cases to enhance ineffective anterior packs. This can be accomplished by inflating a No. 16 or 18 Foley catheter in the nose with 10 to 15 mL of saline and pulling it anteriorly against the vomer and sphenoid rostrum in the posterior naris. A formal anterior pack that abuts against the catheter is then placed posteriorly in the nose in the standard fashion. A piece of plastic tubing is slid over the Foley catheter before it is placed in the nose (Fig. 41-4, A). This plastic can then be slid tightly against the anterior pact, which applies a countertension against the Foley balloon. The tension is secured by a compression clamp (Fig. 41-5). An alternate type of posterior pack can be fashioned as a 3 x 5 cm roll of lamb's wool. Previously placed passing sutures secure it in position. To place this pack the physician places two small, red, Robinson catheters through the nose and brings them out the mouth. Two sutures previously tied to a gauze roll (Fig. 41-4, B) are tied individually to the catheters and the catheters pulled out the nose, thereby placing the sutures through the nose. The pack is guided around the uvula while the sutures are pulled through the nose. Once the pack is positioned (Fig. 41-6), the nose is packed anteriorly as previously described. The sutures are tied over a gauze roll in front of the columella to provide tension. The small suture previously tied to the lamb's wool pack is used to retrieve the pack at the time of removal. Both types of posterior packs stay in place for 4 to 5 days.

Posterior nasal packs are very uncomfortable for the patient as well as being associated with potential risks and complications. Abnormalities in respiratory function can lead to hypoventilation, hypoxemia, cardiac arrhythmias, and possible cardiac arrest. Therefore patients with posterior packs are always admitted to the hospital for observation. Moist oxygen supplementation by mask and careful analgesia are provided. Prophylactic antibiotics are customary to prevent potential infections of the middle ear and sinuses as well as aspiration

pneumonia and septicemia. Care must be taken when using a Foley catheter balloon as a posterior pack. Overinflation may cause soft palate problems with possible through-and-through necrosis.

Commercially manufactured nasal packs are available that have multiple balloons with a single or double lumen for saline administration. Some physicians advocate the use of these balloons, and perhaps they have a place in the management of uncomplicated epistaxis. However, the use of such devices in a training program is not recommended because of the importance of learning to pack a nose properly as well as the fact that these items are not always available.

The patient with epistaxis who has severe thrombocytopenia or an iatrogenic coagulopathy from chemotherapy agents presents a special problem to the consulting otolaryngologist - head and neck surgeon. These patients are frequently immunosuppressed, and there is the definite possibility of life-threatening bacterial or fungal infections. Packing the nose in these patients is difficult and potentially harmful, and on removal the packing can be the source of new trauma and epistaxis. In cases such as these, a pack of oxidized cellulose, topical thrombin, or microfibrillar collagen (Avatene) is recommended because it does not require removal. The microfibrillar collagen is an excellent material to use since it can be molded into a cigar shape and placed into the nasal cavity. The material swells and compresses the mucosa while providing a clotting surface that eventually dissolves.

For most patients epistaxis is a new problem and a past history of coagulopathy or trauma is absent. However, the diagnostic assessment should include evaluation for hypertension, allergy, hereditary bleeding disorders, and drugs that alter blood clotting (such as aspirin and other antiinflammatory agents).

Kiley et al (1982) studied the problem of recurrent epistaxis in children, searching for evidence of coagulation disorders. They concluded that mild congenital hemorrhagic disorders occur far more frequently than commonly realized from the results of routine coagulation screening tests. Patients with mild forms of von Willebrand's disease, for example, fluctuate between normal and abnormal test results, having either a normal bleeding time or normal factor VIII levels. This type of patient must be studied more thoroughly with postaspirin bleeding times, factor VIII-related antigen, and ristocetin aggregation studies for platelet function.

Nonsurgical techniques

Montgomery and Reardon (1980) reported that conventional packing techniques are associated with a 25% failure rate. When packing fails to control epistaxis, a procedure to decrease the pressure gradient that feeds the nasal capillary bed should be considered. Several nonsurgical techniques to control persistent epistaxis have been reported. Padrnos (1968) reported a successful method of epistaxis control by injecting the pterygomaxillary space via the greater palatine foramen with a solution of anesthetic and epinephrine. This action was felt to cause constriction of the vessels in the pterygopalatine fossa, as well as to provide sensory and afferent nerve anesthesia to the nose. Bluestone and Smith (1967) reported the use of cryotherapy to control severe epistaxis in 21 patients. This technique proved to be cumbersome, required special equipment, and was less than predictable. Angiographic arterial embolization has been used successfully in the treatment of intractable posterior epistaxis and is currently experiencing more widespread acceptance and use (Robertson and Reardon, 1979). This is due to the fact that there are many well-trained neuroradiologists experienced in highly selective catheterization of small arterial branches of the carotid system. A complete discussion of this technique is found below.

Surgical techniques

Surgical arterial ligation of nasal feeding vessels is usually the last resort in control of intractable epistaxis. However, early ligation has been advocated from the standpoint of cost-effectiveness, improved control, and safety. Wang and Vogel (1981) found a surgical failure rate nearly half that associated with packing (14.3% versus 26.2%), a decreased complication rate with surgery (40% versus 68%), and the average length of hospital stay for surgical patients to be 2.2 days less than those treated with packing techniques. Whether surgical treatment of epistaxis is better for the patient than conventional packing techniques remains controversial. Each patient must be individually considered with respect to potential morbidity, underlying medical status, and the emergent nature of the case. This section focuses on the technique and indications for arterial ligation for intractable expistaxis. The use of early ligation must reflect the patient's medical status, the success of previous packing efforts, and the surgeon's familiarity and results with the operation of choice.

Ethmoidal artery ligation. Treatment of persistent superior and anterior epistaxis often requires ligation of the anterior and posterior ethmoidal arteries. In cases of epistaxis in which the source of bleeding is ill defined, the ethmoidal arteries are frequently ligated in conjunction with the maxillary artery.

The anterior and posterior ethmoidal arteries are branches of the ophthalmic artery, which derives from the internal carotid artery. The ethmoidal arteries enter the anterior and posterior ethmoidal foramina and traverse the ethmoid sinus to ultimately ramify on the lateral nasal walls and septum. Montgomery (1979) described surgical access to the ethmoidal arteries from a standard external ethmoidal incision. Kirchner et al (1961) detailed the surgical

landmarks of this area and noted that the anterior ethmoidal artery enters the ethmoidal foramen, which is approximately 1.5 cm posterior to the lacrimal fossa at the level of the frontoethmoid suture. The posterior ethmoidal artery is located approximately 10 mm behind the anterior foramen and 5 to 7 mm anterior to the optic nerve. The proximity to the optic nerve and small contribution to the nasal blood supply make identification and ligation of the posterior ethmoidal artery less important than that of the anterior artery. Once identified, the arteries can be ligated with silk suture or small, vascular, hemostatic clips.

Management of posterior epistaxis. Posterior epistaxis that is not controlled by packing can be treated by one of three surgical techniques: (1) external carotid artery ligation, (2) transantral ligation of the maxillary artery, or (3) intraoral ligation of the maxillary artery.

Ligation of external carotid artery. External carotid artery ligation can be a successful technique for posterior epistaxis because it reduces the blood flow to the capillary bed of the nasal mucosa. The technique is simple, can be done with the patient under local anesthesia, and deals with anatomy familiar to the otolaryngologist - head and neck surgeon. Two important points should be made about the procedure: (1) two branches of the external carotid artery must be identified before placement of the ligature in order to avoid ligation of the internal carotid artery; and (2) care must be taken to avoid injury to the vagus nerve, superior laryngeal nerve, hypoglossal nerve, sympathetic chain, or mandibular branch of the facial nerve.

The major disadvantage of external carotid artery ligation is that significant potential exists for colateral blood flow to feed into the maxillary artery distal to the point of ligation on the external carotid artery. In addition, it carries a risk of potential harm to vital neck structures. Also, extracranial-to-intracranial bypass surgery to revascularize the middle cerebral artery from the superficial temporal artery cannot be done if the external carotid system has been ligated. This fact may be important in patients of advanced age who have arteriosclerosis since posterior epistaxis occurs in these patients.

Transantral ligation of maxillary artery. Transantral ligation of the maxillary artery is the most widely used arterial ligation procedure to control intractable epistaxis. Seiffert (1928) first described the technique; Gergely (1935) performed the procedure on cadavers and concluded that this was the method of choice for most cases. However, it was not until 1965 that Chandler and Serrins popularized the procedure. Since then, many authors have written regarding various modifications, but Seiffert's original procedure has endured with minimal change.

The operation usually requires general anesthesia and a Caldwell-Luc procedure to gain access to the pterygomaxillary space. The posterior wall of the maxillary sinus is removed to gain access to the third, or pterygopalatine, part of the maxillary artery. The pterygopalatine portion is tortuous with many branches and frequently requires the operating microscope for isolation of vessels, since pulsations can rarely be seen by direct vision. The most distal branches, the sphenopalatine and greater palatine arteries, can be of formidable size and must be doubly clipped. The larger vessels can be pulled anteriorly with a nerve hook into the sinus to facilitate placement of the hemostatic clips (Figs. 41-7 and 41-8).

Persistent pain in the maxillary dentition, potential damage to the sphenopalatine ganglion and vidian nerve, and oral antral fistula are potential complications. In addition, the transantral technique has the limitations of not being feasible in children, patients with hypoplastic maxillary sinuses, patients with tumors of the maxillary sinus, or patients with severely comminuted facial fractures. In some instances chronic maxillary sinusitis may be a contraindication to the transantral technique.

Intraoral ligation of maxillary artery. A technique involving intraoral ligation of the maxillary artery for intractable posterior epistaxis is an alternative to transantral ligation (Maceri and Makielski, 1984). This technique provides surgical access to the first and second parts of the maxillary artery as it arises from behind the ramus of the mandible. The technique is applicable to all ages, does not require Caldwell-Luc operation or the microscope, and can be used in a patient with maxillary fractures as well as cancer patients.

After a general anesthetic has been administered, an incision is made in the gingivobuccal sulcus at the level of the third molar, then inferiorly along the ramus of the mandible (Fig. 41-7). The surgeon inserts a finger into the wound and bluntly dissects some of the alveolar tissue. The buccal fat pad may be dissected free or retracted medially to enhance exposure. The temporal muscle belly is split and partially dissected from the medial surface of the mandible. The artery can be directly visualized at the base of the wound (Fig. 41-8) or palpated and brought into the field with a nerve hook. Hemostatic clips are applied as shown in Fig. 41-9. The intraoral technique frequently results in varying degrees of trismus for several days or weeks because of temporal muscle manipulation. Also, the potential for damage to the inferior alveolar nerve or infection in the intratemporal space exists.

The intraoral approach to the maxillary artery has been modified and further described by Stepnick et al (1990). This study included cadaver dissections and three clinical cases of intractable posterior epistaxis managed successfully by the intraoral approach. Stepnick et al advocate minimal dissection of the maxillary artery in the infratemporal space to minimize trauma to the muscles of mastication and subsequent trismus. The technique is accomplished by using a "fishhook" device to pull the artery up into the field in a blind fashion. In cases where the dissection is difficult and the artery hard to find, the authors recommend the more traditional transantral approach.

One must realize that no surgical procedure will render the nose avascular, and the purpose of arterial ligation is to sufficiently decrease the pressure gradient of the nasal capillary bed to stop the epistaxis. Of course, the closer to the bleeding site the ligation is performed, the less likely the chance that collateral vessels will maintain an elevated filling pressure and thus sustain the epistaxis. Fig. 41-9 shows the location of the hemostatic clips placed by the transantral and intraoral techniques. In some cases, Pearson et al (1969) point out, the descending palatine and pharyngeal arteries are of substantial size and anastomose freely within the nasal cavity. In such cases more specific ligation may be needed if main-stem maxillary artery ligation fails.

Transarterial embolization

Advances in selective angiography and digital subtraction techniques have elevate transarterial embolization to a new position in the management scheme of intractable posterior epistaxis. The single factor that most commonly limited more widespread use of embolization was the availability of an experienced neuroradiologist. Since more training programs in radiology now focus on teaching highly selective angiography and invasive radiology, experienced neuroradiologists are more readily available.

The present options for treatment of intractable epistaxis are being reconsidered in favor of more aggressive early surgery and arterial embolization in lieu of cumbersome posterior nasal packs in certain patients. This is especially true for embolization when adequate packing and arterial ligation have failed to control nasal hemorrhage. In fact, if angiography demonstrates the middle meningeal or facial arteries to be a significant part of the local nasal circulation, embolization may be the only recourse. Therefore angiography before arterial ligation is recommended in the following circumstances (Welsh et al, 1990):

1. Evidence of flow from the contralateral carotid system.

2. History of transient ischaemic episode or cerebrovascular accident or physical evidence of carotid artery disease (muscular paresis or paralysis).

3. Vascular compromise of the globe (amaurosis fugax).

There are many advantages to arterial embolization, including direct visualization of the bleeding site close to the source. In addition, the vessel can be occluded closer to the site of bleeding, thereby decreasing the effects of collateral blood flow. In experienced hands, the success rate of embolization including repeat attempts is reported at 90% (Hicks and Vitek, 1989). The procedure is rapidly performed with the patient under local anesthesia and is an excellent option in patients with bleeding disorders such as Rendu-Osler-Weber disease (Strutz and Shumacher, 1990). Embolization cannot be used for anterior epistaxis since the blood supply to the anterior septum is from the anterior and posterior ethmoidal arteries (branches of the internal carotid system) in most cases.

The complication rate in experienced hands has been demonstrated to be less than 0.1% (Merland et al, 1980). Potential problems include cerebrovascular accidents caused by internal carotid embolization and facial nerve paralysis (Metson and Hanson, 1983. Arterial embolization is contraindicated in the face of angiographic evidence of dangerous anastomoses with the internal carotid system, severe atheromatous disease, and allergy to angiographic contrast material.

The technique uses computerized digital subtraction angiography to localize the bleeding site and to define the vascular anatomy. Specifically, the internal maxillary artery is selectively studied to its most terminal branches. The material used for embolization can be polyvinyl alcohol spheres (150 to 590 microm in diameter), Gelfoam particles of various sizes, and in

some cases coil springs.

The benefits of arterial embolization are well demonstrated in the following example. A 35-year-old man with persistent posterior epistaxis was referred for ligation of the internal maxillary artery after anterior and posterior nasal packing failed to control hemorrhage. The patient underwent intraoral ligation of the maxillary artery with initial control of bleeding. Two days after ligation he again began to hemorrhage from the left posterior nares. The neuroradiologist was consulted, and immediate angiography of the left external carotid and internal maxillary arteries was performed. The digital subtraction preembolic angiogram (Fig. 41-10) showed two vascular clips on a branch of the internal maxillary artery and a persistent vascular blush on the septum fed by unclipped branches of the internal maxillary artery along the floor of the septum. The patient was embolized with polyvinyl alcohol spheres until the bleeding was abolished (Fig. 41-11). The bleeding has remained controlled without complications.

Successful arterial embolization in the hands of an experienced angiographer provides the patient with a highly effective alternate to vascular arterial ligation and should be strongly considered the treatment of choice if conventional packing techniques fail to control bleeding. It is quite reasonable to assume that arterial embolization will become a first-line option for the treatment of posterior epistaxis in lieu of packing in selected patients. Such an acceptance requires that the morbidity and mortality of embolization remain lower than that of packing and that the efficiency continues to be extremely high.

Septal Hematoma

Hematoma of the nasal septum is exclusively a consequence of nasal trauma. The most common causes are sports injuries, motor vehicle accidents, altercations, and rhinologic surgery. It is mandatory that primary care physicians and physicians dealing with emergency patients be able to diagnose septal hematoma. Diagnosis is made by inspection of the nose. Identification of a dislocated septum or hematoma requires visual recognition.

The appearance of a hematoma can be confused with nasal polyps, a deviated nasal septum, or enlarged turbinates. With hematoma one or both nostrils may be obstructed by a large, soft, red or bluish mass. Needle aspiration will usually yield blood or liquefied clot. Difficulty comes when the hematoma is posterior and a severely dislocated septum precludes adequate inspection. The major symptom of septal hematoma is progressive nasal obstruction. In most patients (children excepted) a history of antecedent trauma can be elicited. Pain is usually present in the form of an anterior midline headache. Epistaxis is not commonly found with septal hematoma.

Anatomy

The anatomy of the septum is demonstrated in Fig. 41-12, showing the bony and cartilaginous contributions. Blood supply to the cartilaginous septum depends on the integrity of an intact mucoperichondrial membrane. Septal trauma and dislocation can lacerate blood vessels in the perichondrium, leading to collection of blood between the membrane and cartilage. This

blood collection dissects the mucoperichondrium off the septal cartilage from one or both sides. The cartilage is totally dependent on the mucoperichondrium for its nutrition, and when the membrane is separated from the septum for more than 48 hours, necrosis of the cartilage occurs.

Management

Treatment of septal hematoma starts with diagnosis. In an adult definitive therapy can be provided using local anesthesia in the office or emergency room. Children will most likely require general anesthesia for treatment and possibly for diagnostic evaluation as well.

When local anesthesia is used, a topical vasoconstrictor such as 4% cocaine hydrochloride is applied via cotton pledgets to provide both anesthesia and vasoconstriction. Supplemental anesthesia, when needed, is provided by injection of 1% lidocaine hydrochloride with epinephrine added. In early cases the hematoma can be aspirated under direct vision with an 18-gauge needle. More organized hematomas are treated by incision of the mucoperichondrium in two or three parallel locations. The clot is evacuated and any necrotic septal cartilage removed. The mucoperichondrial flap is repositioned in direct contact with the cartilaginous septum. An absorbable suture of 4-0 chromic catgut can be used as a running horizontal mattress suture that quilts the mucoperichondrial flaps against the septal cartilage. In cases in which the mucoperichondrial flap is edematous and enlarged, septal splints can be used to provide a large uniform area of pressure against the cartilage. Finally, both sides of the nose are packed with standard anterior nasal packs. In some cases of acute trauma performing a closed reduction of the fractured nose and septum may be advisable to facilitate adequate removal of the clot and make the packing more effective. Packs are removed in 24 hours and the nose examined to be certain the clot has not reaccumulated. Packing is then replaced and left in position for 4 to 5 days. If splints have been used, they will remain in place for 2 weeks. Patients should be given appropriate antibiotics to cover nasal flora as well as *Staphylococcus aureus.* In cases of accompanying sinusitis, appropriate culture and sensitivity determinations are necessary. Most patients can be managed on an outpatient basis.

Unrecognized septal hematoma

An unrecognized septal hematoma will cause necrosis of the septal cartilage. The result is a gelatinous, decomposed septum that results in scar tissue and contraction on restoration. This ultimately leads to loss of dorsal nasal support and the saddle-nose deformity. In young children this condition may be associated with cessation of nasal growth and a permanently infantile nose. Subsequent correction usually requires cartilage or bone grafts to reconstruct the nose.

The septal hematoma will not resolve on its own. It will lead to a saddle-nose deformity, or it may be interrupted by abscess formation if the hematoma becomes infected. Septal abscess can also result from acute infectious sinusitis, nasal foreign bodies, and, of course, nasal surgery. Treatment of septal abscess involves incision and drainage, culture and sensitivity studies, appropriate IV antibiotics, and nasal packs. At times a slip of Penrose drain can be placed beneath the flap for 24 hours to facilitate drainage. These patients should be hospitalized so that antibiotics can be given intravenously to prevent retrograde thrombophlebitis, meningitis, brain

abscess, or cavernous sinus thrombosis and possible death. It may be necessary to reopen the abscess cavity on several occasions in more difficult cases.

The problem of nasal septal hematoma is complicated by failure to diagnose the situation early enough. The neglected hematoma, especially in a child, can lead to unsightly cosmetic deformity, airway obstruction, and the need for subsequent reconstructive surgery. Diagnosis is made by direct inspection of the nose and a high index of suspicion.