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THE MANAGEMENT OF EPISTAXIS

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I. DIRECTIONS

Although self-instructional techniques have been effectively used in many educational settings, they have only recently been introduced into medical education. They are particularly helpful in guiding learners through a maze of confusing or contradictory information and in allowing the learners to proceed at their own pace. Self-instructional packages such as this cannot be encyclopedic in nature nor are they intended to be. They can, however, succinctly analyze a subject, present the essential content matter in a concise format, and provide a sense of direction in approaching a learning problem. Moreover, by using "Interim Quizzes" appropriately scattered throughout the package, the learner can periodically reinforce comprehension at intervals along the way. The introductory "Self-Assessment Examination" allows readers to assess their knowledge at the beginning of the package. At the end, if all the information has been understood, the learner should be able to answer all the "Posttest" items correctly. Immediate feedback provides the opportunity for the learners to test their comprehension and, if it is found to be incomplete, they may study that particular portion of the text again.

The reader is encouraged to proceed through this package, starting at the beginning and proceeding through each section in order. The content is presented in a logical sequence beginning with anatomic considerations and concluding with surgical treatment. Taking the Interim Quiz at the end of each section allows you to test your comprehension of each section. The Posttest is designed to test not only for comprehension but to provide you with the opportunity to use the information contained in this package in a simulated clinical setting. It is our hope that this Self-Instructional Package will be of value to you in understanding the subject matter and will provide you with not only an up-to-date reference source but with the incentive for future study of this topic.

Satisfactory completion of this SIPac qualifies the reader for two hours of category 1 CME credit toward the AMA Physician's Recognition Award. To satisfactorily complete the SIPac, the participant should read all the material, answer each interim quiz in turn, review the answers and, finally, complete the "Posttest."

II. SELF-ASSESSMENT EXAMINATION

(Select the one best answer.)

1. The most common site of intranasal bleeding is
 - a. inferior meatus
 - b. Kiesselbach's plexus
 - c. roof of the nose
 - d. middle turbinate
 - e. posterior septum
2. Which of the following procedures would be the most helpful in controlling profuse bleeding from the posterosuperior part of the nose when nasal packing has failed?
 - a. internal maxillary artery ligation
 - b. Gelfoam embolization
 - c. external carotid artery ligation
 - d. vitamin K administration
 - e. ethmoidal artery ligation
3. The most common consequence of posterior nasal packing is
 - a. bacterial colonization of the packing material
 - b. eustachian tube dysfunction
 - c. hearing loss secondary to otitis media
 - d. epiphora
 - e. decreased oxygen saturation
4. The most common cause of epistaxis is
 - a. trauma
 - b. septal perforation
 - c. aneurysm
 - d. arteriosclerosis
 - e. von Willebrand's disease
5. The anterior ethmoid foramen is located in which of the following structures?
 - a. lacrimal fossa
 - b. frontomaxillary suture
 - c. lamina papyracea
 - d. frontoethmoid suture
 - e. limen orbitale
6. Which one of the following is not a feature of Osler-Weber-Rendu disease?
 - a. abnormal subepidermal vessels
 - b. frequent severe epistaxis
 - c. autosomal dominant inheritance
 - d. abnormal coagulation profile
 - e. visceral telangiectasias

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7. Which of the following procedures has (have) long-term success in eliminating bleeding episodes in hemorrhagic telangiectasia?
 - a. maxillary artery ligation
 - b. maxillary artery embolization
 - c. laser application
 - d. septodermoplasty
 - e. none of the above

 8. A decrease in which of the following environmental factors primarily predisposes to epistaxis?
 - a. temperature
 - b. pollen count
 - c. humidity
 - d. pollutants
 - e. barometric pressure

 9. Posterior epistaxis is most common with which of the following disorders?
 - a. trauma
 - b. hereditary telangiectasia
 - c. septal deflection
 - d. arteriosclerosis
 - e. leukemia

 10. Following unsuccessful internal maxillary artery ligation, which of the following may be prohibited?
 - a. correction of coagulation defect
 - b. further vascular ligation
 - c. arterial embolization
 - d. endoscopic cauterization
 - e. surgical closure of the nostrils

SELF-ASSESSMENT EXAMINATION ANSWERS

1. b.
2. e.
3. a.
4. a.
5. d.
6. d.
7. e.
8. c.
9. d.
10. c.

III. OBJECTIVES

The general objective of this SIPac is to understand the management of nasal hemorrhage (epistaxis). The following specific objectives will enable the reader to meet the general objective as follows:

1. Recall basic information.
 - a. The anatomy and vascular supply of the internal nose and related structures
 - b. Local and systemic causes of epistaxis
2. Interpret the significance of abnormal findings acquired from the following:
 - a. History
 - b. Physical examination
 - c. Radiographic examination
 - d. Laboratory data
3. Generate an appropriate treatment plan for managing the patient with epistaxis.
4. Assess the efficacy of the treatment plan.

IV. INTRODUCTION

Epistaxis is one of the most common clinical problems requiring the expertise of an otolaryngologist. A significant portion of the general population report at least one episode during their lifetime.^{1,2} Most episodes of bleeding are minor, self-limited, and easily controlled by simple measures. However, there are times in which the bleeding is difficult to manage and the condition becomes serious and potentially life-threatening.

To treat the patient effectively and to avoid complications, the otolaryngologist should approach the problem systematically. Proper management of epistaxis depends upon a thorough understanding of the anatomy of the nose, the causes of bleeding, and the techniques used to control the bleeding.

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V. CONTENT

A. The Vascular Anatomy of the Internal Nose and Related Structures

The nose is richly supplied by distal branches of both the external and internal carotid arteries with widespread anastomoses between the two systems.

1. External carotid artery

a. Superior labial division of facial artery

This vessel enters the nose just lateral to the anterior nasal spine bilaterally and after ascending into the nasal vestibule, branches extensively to supply the anterior septum. The anterior septum harbors a rich anastomotic network with contributions from both the internal carotid artery (ICA) and external carotid artery (ECA) and is the source of bleeding in most cases. This plexus of vessels is commonly referred to as Kiesselbach's plexus or Little's area (Fig 1).

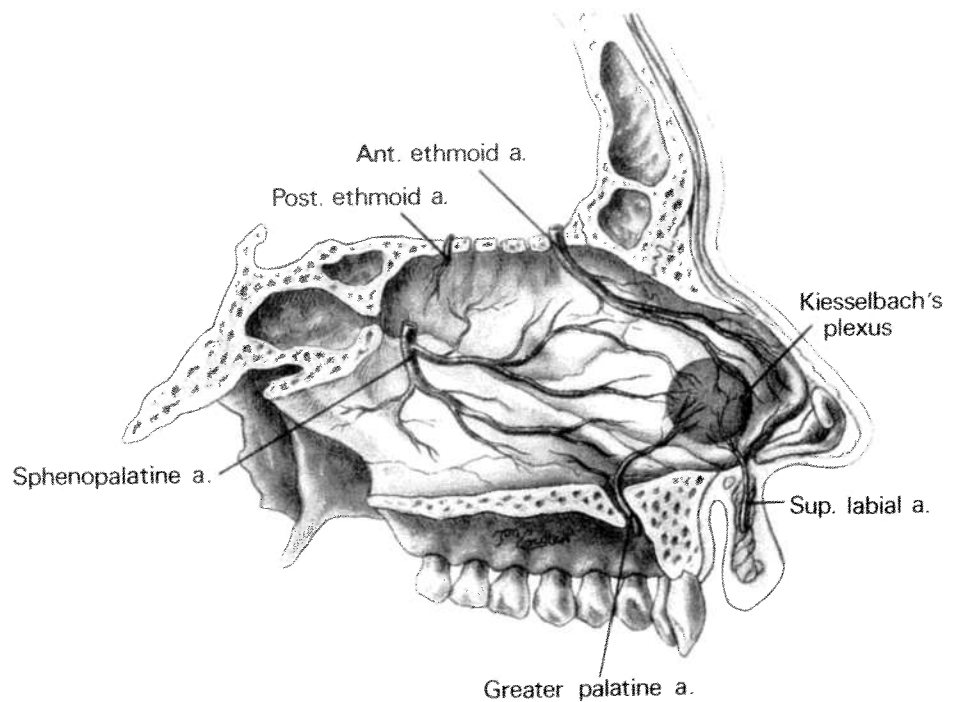


Fig 1.—Arterial supply of medial wall (septum) of nose.

b. Internal maxillary artery

A significant portion of the blood supply to the nasal cavity comes via the internal maxillary artery (IMA) and its branches (Fig 2). The IMA originates from the ECA and has classically been divided into three divisions (maxillary, pterygoid, pterygopalatine) based on its relationship to the lateral pterygoid muscle. The maxillary segment passes between the mandibular ramus and the sphenomandibular ligament. It then runs either deep or superficial to the lateral pterygoid muscle as the pterygoid segment and courses medially within the pterygopalatine fissure to enter the pterygopalatine (pterygomaxillary) fossa. There are six generally accepted terminal branches: posterior superior alveolar, infraorbital, artery of the pterygoid canal, pharyngeal, sphenopalatine, and descending palatine. The last two are most important in considering nasal blood supply.

The sphenopalatine artery enters the nose through the sphenopalatine foramen at the posterior attachment of the middle turbinate, there dividing into medial (nasopalatine) and lateral (posterior superior) branches. The nasopalatine (nasoseptal) artery courses medially over the inferior portion of the sphenoid bone. This branch then courses anteriorly and superiorly where it forms anastomoses with the greater palatine, superior

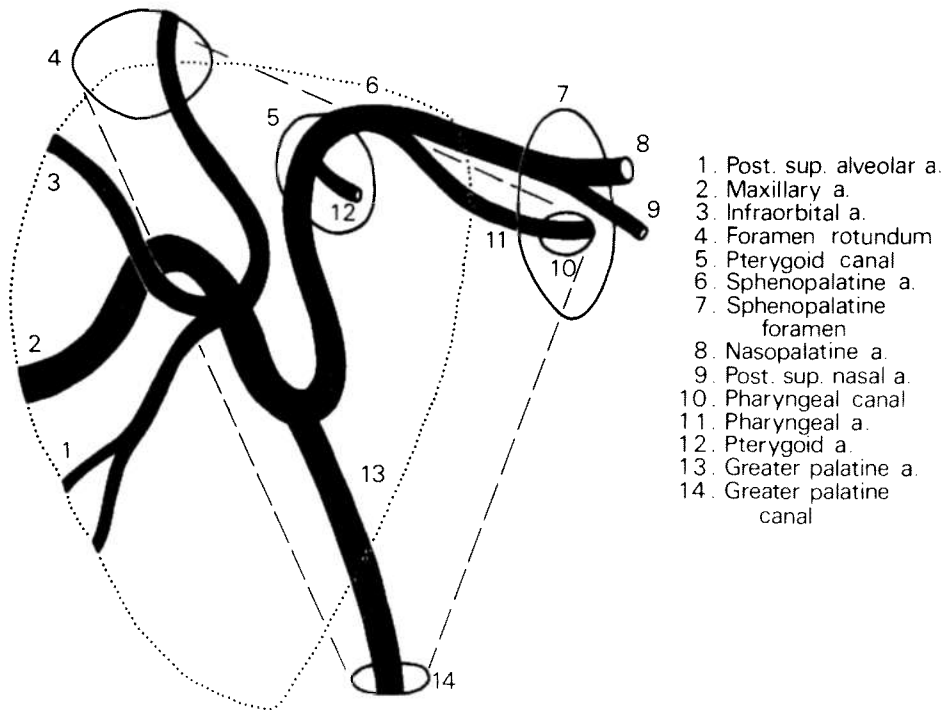


Fig 2—Schematic version of arterial anatomy of right pterygopalatine fossa as viewed through posterior wall of maxillary sinus (dotted line).

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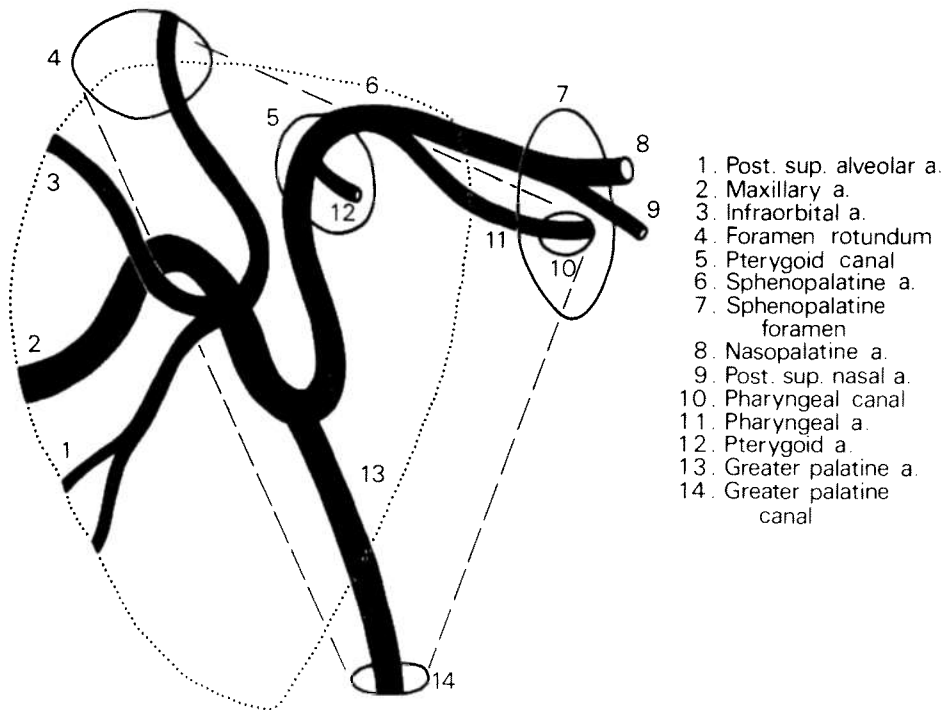


Fig 2—Schematic version of arterial anatomy of right pterygopalatine fossa as viewed through posterior wall of maxillary sinus (dotted line).

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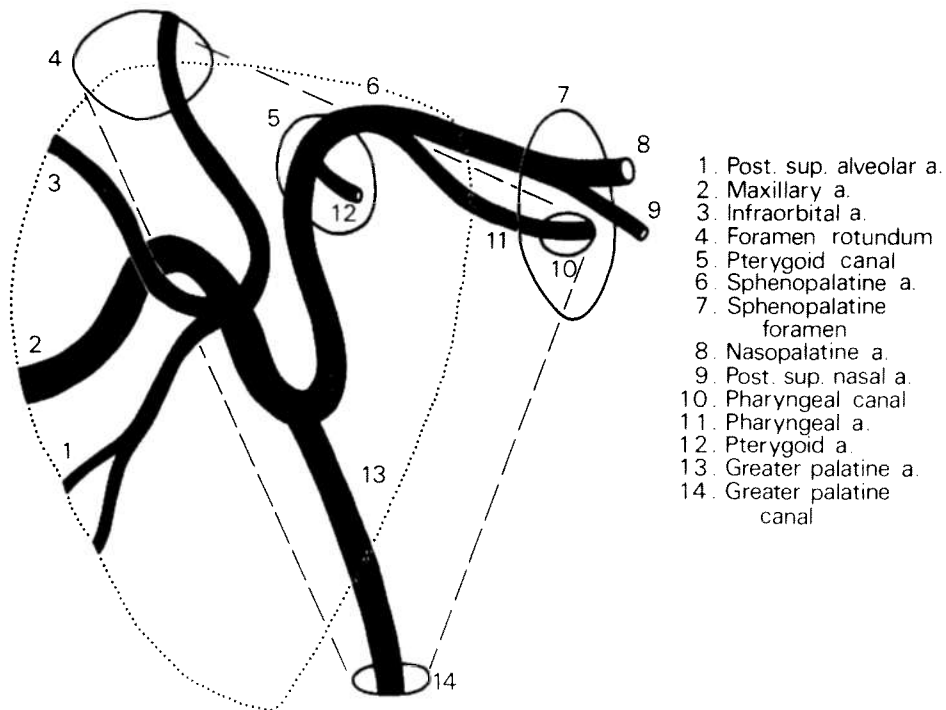


Fig 2—Schematic version of arterial anatomy of right pterygopalatine fossa as viewed through posterior wall of maxillary sinus (dotted line).

labial, and ethmoidal arteries. The lateral division of the sphenopalatine artery, the posterior superior artery, supplies the middle and inferior turbinates.

The other terminal branch of the IMA is the descending palatine artery. It descends through the greater palatine canal to emerge as the greater palatine artery. The vessel then courses anteriorly to supply the palate before turning superiorly through the incisive foramen to enter the nose. There, it supplies the septum and contributes to Kiesselbach's plexus (Figs 1 and 3).

Several common aberrations may occur in the vascular anatomy of the pterygopalatine fossa. The nasopalatine artery usually branches from the sphenopalatine artery, within the sphenopalatine foramen, and is, therefore, not normally visualized by the surgeon via the transantral approach. However, the nasopalatine artery (Fig 2) may branch directly from the end of the IMA, giving the appearance of a double sphenopalatine artery. The presence of the vidian or pharyngeal arteries may also complicate the vascular anatomy of the pterygomaxillary fossa. Although these vessels do not supply blood to the nose, they are present in the pterygopalatine fossa. Surgical ligation of these vessels instead of the IMA may be a cause of surgical failure and lead to recurrent bleeding.

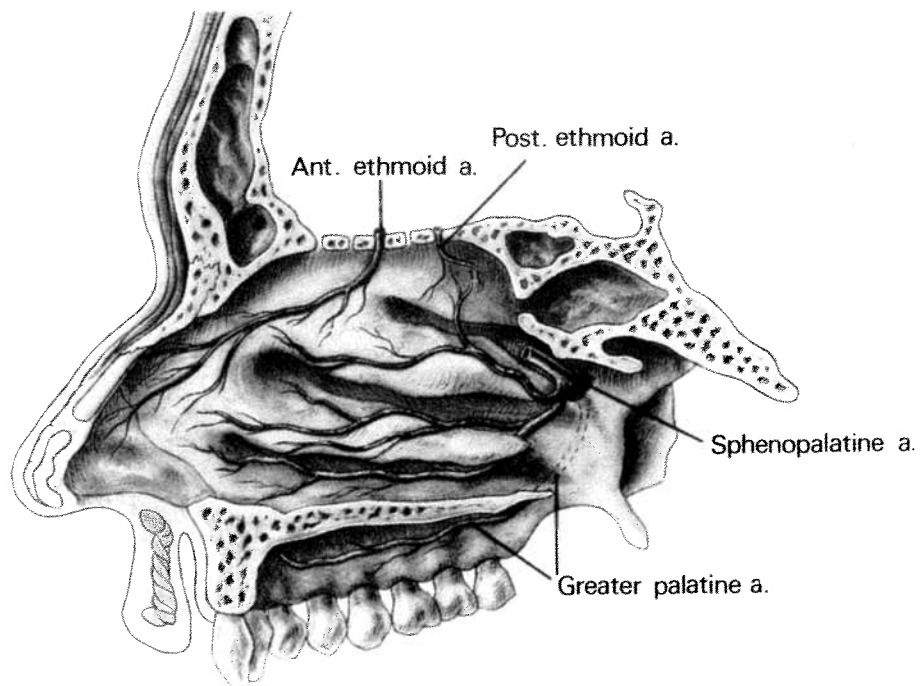


Fig 3.—Arterial supply of lateral wall of internal nose.

2. Internal carotid artery

The blood supply to the nose derived from the ICA comes from the ethmoid branches of the ophthalmic artery. After passing through the petrous portion of the temporal bone, the ICA turns sharply and passes near the lateral surface of the sphenoid bone. As it passes near the cavernous sinus, it has branches that communicate with the IMA via the artery of the pterygoid canal and the ascending pharyngeal artery. As the ICA pierces the dura it gives off its first branch, the ophthalmic artery. After entering the orbit through the superior orbital fissure, the ophthalmic artery divides into approximately ten branches. Two of these, the posterior and anterior ethmoid arteries, contribute a portion of the blood supply to the nasal cavity.

a. Anterior ethmoid artery

Anteriorly within the superior aspect of the orbit, the ophthalmic artery divides to form the anterior ethmoid artery. This vessel enters the anterior ethmoid canal through its foramen (Figs 3 and 4) and enters the nose through the open nasal slit (a space between the crista galli and cribriform plate) where it divides into septal and lateral branches (Figs 3 and 4). The septal division of this artery contributes branches to Kiesselbach's plexus.

b. Posterior ethmoid artery

More posteriorly, the posterior ethmoid artery leaves the ophthalmic artery within the orbit and enters the posterior ethmoid canal. The artery then passes into the superior aspect of the nose through the junction of the fovea ethmoidalis and the cribriform plate where it divides into lateral and medial (septal) branches (Figs 3 and 4).

c. Venous drainage of nose

Venous drainage of the nose generally parallels the arterial supply. Six routes of drainage are usually described³: (1) anterior ethmoidal vein tributaries to cavernous sinus; (2) posterior ethmoidal vein tributaries to cavernous sinus; (3) sphenopalatine vein tributaries to maxillary vein or cavernous sinus via emissary veins; (4) greater palatine vein tributaries to maxillary vein to posterior facial vein or cavernous sinus via emissaries; (5) septal vein to anterior facial vein; and (6) small channels from vestibule to tributaries of anterior facial vein.

3. Incidence of epistaxis

Using a self-administered medical history, Weiss¹ found that 11% of 6672 patients had at least one episode of epistaxis during their lifetime. Other clinicians have estimated that approximately 60% of the adult population will experience some form of nasal bleeding.² Men are generally affected more often than women and it is unusual for an infant to have nasal bleeding. Children and young adults usually bleed from the anterior portion of the nasal cavity which is minor and easily controlled. More severe bleeding occurs from the posterior aspect of the nasal cavity and is associated with arteriosclerosis of advancing age.

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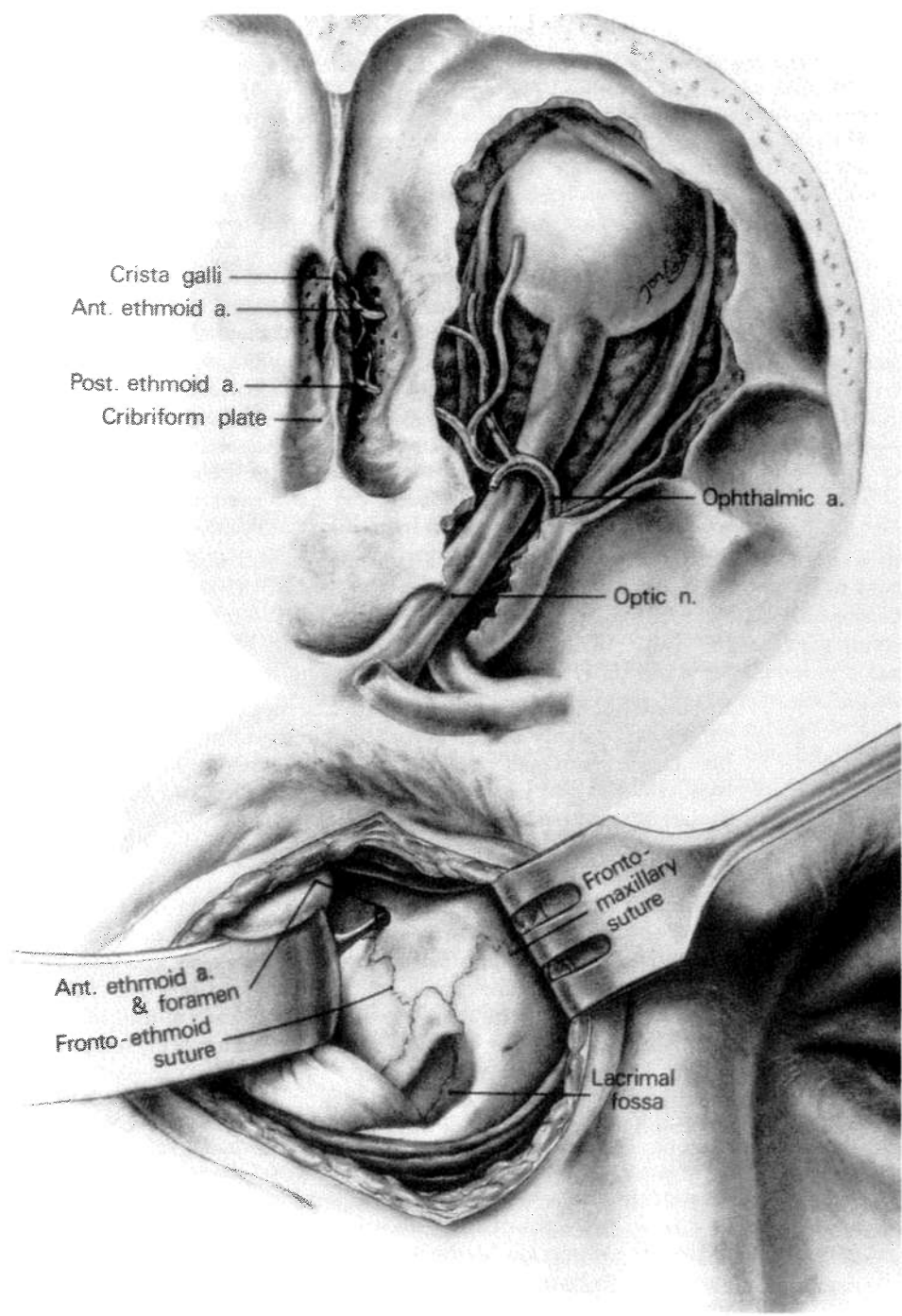


Fig 4.—(Upper panel) Superior view of orbit as seen after removal of orbital roof. Note branching from ophthalmic artery of posterior and anterior ethmoidal arteries and their subsequent course to cribriform plate. (Lower panel) Placement of anterior ethmoidal artery and its foramen as seen during anterior ethmoidal artery ligation procedure.

4. Etiology

Epistaxis is caused by a variety of local and systemic factors that alter the local environment of the nose. These factors may affect the nasal vasculature directly. In other cases, the nasal vessels may be affected indirectly through alterations in the nasal mucosa.

a. Local factors

i. Trauma

Direct trauma to the nose, either accidental or self-inflicted (nose picking), is thought to be the most common cause of epistaxis. Digital trauma is the most common cause of epistaxis in the pediatric population. Irritation and ulceration of the mucosa of the anterior nasal septum is produced by habitual rubbing and nose picking, causing crusting of the mucosa. When the crusts are elevated during vigorous nose blowing, bleeding may occur. External nasal trauma often produces epistaxis by shearing the soft tissues of the nose over its bony framework causing lacerations in the nasal mucosa. Epistaxis from nasal fractures usually results from injury at the dorsal bony-cartilaginous junction or at the piriform aperture. More extensive fractures of the maxillofacial skeleton or high-velocity missile injuries may produce severe and rapid blood loss from shearing of vessels as they pass through bony foramina. Epistaxis of this type is often life-threatening and requires prompt attention.

Epistaxis following nasal surgery (i.e., septoplasty, endoscopic sinus surgery) is usually minor and self-limited where the origin of the bleeding is from freshly cut mucosal edges. Bleeding of this type is less frequent with the use of endoscopic techniques and the improved design of nasal packing.

ii. Inflammation

Inflammatory conditions such as upper respiratory tract infection, allergic rhinitis, and sinusitis are well known to be associated with blood-streaking of nasal mucus. Increased vascularity and greater friability of the vessels are characteristic of inflamed mucosa. Epistaxis may ensue with forceful nose blowing under these conditions. It has been shown that some bacterial strains common to the nasal cavity produce fibrinolytic enzymes (e.g., streptokinase, staphylokinase) capable of lysing blood clots.¹ Production of these enzymes may lead to prolonged and recurrent bleeding.

Environmental conditions of low humidity and dry heat, typical of winter months, are associated with a higher incidence of epistaxis. The mucosa of the nasal cavity desiccates, leading to increased frequency of bleeding. Epistaxis has been found to be unrelated to meteorologic changes in barometric pressure⁵; however, rapid decreases in pressure and ascents to high altitude commonly cause epistaxis.

Local inflammation may also result from the introduction of toxins into the nose. Chronic cocaine use, topical corticosteroids, and expo-

sure to toxic irritants (e.g., ammonia, chlorine, or wood dust) induce hyperemia of the nasal mucosa with subsequent bleeding. Septal perforations may also be the cause of recurrent epistaxis. The bleeding source is frequently located at the septal margin where there is absence of normal mucosa and the exposed cartilage is partially covered by friable granulation tissue. Crusting is common, and may be associated with an underlying infection that perpetuates the process of necrosis, granulation tissue, crusting, and more infection.

iii. Nasal septal deformity

Epistaxis occurs frequently in patients with nasal septal deviation. The deflection of the nasal septum interrupts the normal flow of the air stream and produces eddy currents that dry the adjacent nasal mucosa. This leads to crusting and bleeding. Septal spurs alone can lead to higher epistaxis rates for the same reason. Correlation between the side of nasal bleeding and septal deviation has been reported.⁶

iv. Foreign bodies

Epistaxis in children and mentally disturbed individuals may be the result of an intranasal foreign body. This should be suspected when there is a unilateral foul nasal discharge. The foreign body usually has one of the following characteristics: (1) sharp edges, (2) irritating chemical properties, or (3) porosity (e.g., sponge). Bleeding occurs from the inflamed mucosa and granulation tissue surrounding the foreign body. In some parts of the world parasites, such as leeches, may lodge in the nose and nasopharynx and cause bleeding.⁷ The leech enters the mouth or nose when the patient drinks contaminated water from open streams.

v. Tumors

Neoplasms should always be included in the differential diagnosis in a person who presents with progressive and/or recurrent bleeding. Although rare, neoplasms of the nose and paranasal sinuses typically present with unilateral nasal obstruction associated with progressive and recurrent bleeding. Recurrent epistaxis in an adolescent male should raise the suspicion of a juvenile nasopharyngeal angiofibroma. Other tumors to consider in a patient with recurrent epistaxis include nasal septal hemangioma, hemangiopericytoma, papilloma, squamous cell carcinoma, adenoid cystic carcinoma, adenocarcinoma, and melanoma. The otolaryngologist should inquire about prolonged exposure to wood dust, which is a risk factor for the development of sinonasal adenocarcinoma.⁸ If a mass is discovered in the nose after the bleeding has been controlled, imaging studies, such as computerized tomography, should precede biopsy. Premature office biopsy of a vascular intranasal mass may be complicated by severe epistaxis.

vi. Aneurysms

Aneurysms and pseudoaneurysms of the ICA are rare causes of epistaxis. These usually arise from the cavernous portion of the ICA and there is typically an antecedent history of maxillofacial trauma. Deficits of the first six cranial nerves may also be present. Bleeding from an internal carotid aneurysm or pseudoaneurysm is brisk and life-threatening, with mortality rates approaching 50%.⁹ Bleeding from a pseudoaneurysm of the descending palatine artery has also been described.¹⁰ Additionally, posttraumatic carotid-cavernous sinus fistulae may present with severe epistaxis.¹¹

b. Systemic factors

i. Arteriosclerosis and hypertension

The accumulation of atheromatous material in arterial walls compromises the integrity of the vessel. Fibrous and collagenous elements in maxillary and ethmoid arteries have been identified histologically.¹² Vessels weakened by arteriosclerotic changes may help to account for the increased incidence of posterior nasal bleeding in the elderly population.

Hypertension has also been identified as a potential contributing factor in epistaxis.¹³ The distressed and apprehensive patient who presents with epistaxis is likely to have at least transient elevation of his blood pressure. However, the blood pressure should normalize after the bleeding has been controlled. Studies correlating nasal bleeding with diastolic pressure, systolic pressure, and status of the retinal vessels have failed to demonstrate significant differences in the prevalence of epistaxis between patients with and without hypertension.^{4,5,12} This suggests that the observed association between hypertension and epistaxis may reflect the increased incidence of nasal bleeding and elevated blood pressure with aging. Nevertheless, the acute treatment of epistaxis should include monitoring of blood pressure and treatment if necessary. If previously undiagnosed hypertension persists after the resolution of epistaxis, the patient should be referred for medical evaluation.

ii. Blood dyscrasias

Patients with abnormalities of the clotting system frequently experience epistaxis.¹⁴ Primary coagulopathies to consider include hemophilia, von Willebrand's disease, thrombocytopenia, and polycythemia vera. Secondary, or acquired, coagulopathies may be seen in patients with systemic diseases such as uremia, alcoholism, chronic liver disease, leukemia, myeloma, aplastic anemia, idiopathic thrombocytopenic purpura, or hypovitaminosis. Iatrogenic coagulopathies may stem from the use of warfarin (Coumadin), cyclooxygenase inhibitors (aspirin, nonsteroidal antiinflammatory drugs [NSAIDs]), or heparin. A careful history should be taken which includes prior evidence of a coagulation defect, such as easy bruising, a familial history of bleeding, or the use of products that might alter the clotting cascade.

iii. Hereditary hemorrhagic telangiectasia (HHT) (Osler-Weber-Rendu disease)

Hereditary hemorrhagic telangiectasia is an autosomal dominant vascular anomaly characterized by abnormal subepidermal vessels. In HHT there is a lack of elastic tissue in the capillary wall and a deficiency of smooth muscle in the feeding vessel which produces telangiectasias that bleed easily.¹⁵ Frequent epistaxis from nasal telangiectasias is the most common manifestation of HHT. Telangiectasias may be found in the skin, tongue, oral cavity, stomach, colon, and lung, and their presence confirms the diagnosis. The disease was initially noted by Suttén in 1864.¹⁶ In 1901, Osler¹⁷ provided the classic description of the disease which characteristically presents with epistaxis. Rendu,¹⁸ in 1896, distinguished this disorder from the hemophilias. Weber¹⁹ elaborated on the natural history of the disease, while Hanes²⁰ coined the term *hereditary hemorrhagic telangiectasia* and described the histologic findings. Many treatment modalities have been tried to control epistaxis, including the use of lasers,²¹⁻²⁵ estrogen,^{26,27} septal dermoplasty,^{28,29} various flaps,³⁰⁻³³ the use of human amniotic membrane,³⁴ and embolization,^{35,36} all with variable long-term success. Notably, the coagulation profile is usually normal in patients with HHT. It is not uncommon for these patients to undergo frequent transfusions in response to their recurrent bouts of nasal bleeding.

iv. Miscellaneous factors

It has been estimated that in as many as 10% of cases of epistaxis, no specific etiology or predisposing factor will be discovered.³⁷ This may reflect the fact that the nasal trauma may be so minor that the patient will be unable to recollect the incident. Persistent or recurrent epistaxis should lead the physician to a careful reassessment of the history and physical examination in conjunction with appropriate laboratory tests in an attempt to arrive at an accurate diagnosis.

INTERIM QUIZ 1
(Select the one best answer.)

1. Which one of the following arteries does not supply the internal nose?
a. sphenopalatine
b. anterior ethmoidal
c. posterior ethmoidal
d. superior labial
e. posterosuperior alveolar
2. The sphenopalatine is a branch of which artery?
a. internal carotid
b. superior labial
c. infraorbital
d. pharyngeal
e. internal maxillary
3. A common aberration observed during transantral ligation of the internal maxillary artery is the appearance of a double sphenopalatine artery. This is caused by
a. a vein occurring anterior to the sphenopalatine artery
b. the aberrant course of the middle septal artery
c. an anomalous branching of the nasopalatine artery
d. an aberrant sphenopalatine nerve
4. The approximate percentage of the population of the United States who have had epistaxis at least once in their lifetime is
a. 10%
b. 20%
c. 50%
d. 75%
e. 90%
5. An uncommon but more life-threatening cause of epistaxis is
a. aneurysm
b. hemophilia
c. arteriosclerosis
d. hemorrhagic telangiectasia
e. scarlet fever

6. A male adolescent has a history of unilateral nasal obstruction and bleeding. Of the following, the most likely diagnosis is
- foreign body
 - inverted papilloma
 - juvenile angiofibroma
 - septal perforation
 - vitamin deficiency
7. Posterior epistaxis is most commonly associated with
- arteriosclerosis
 - allergic rhinitis
 - atrophic rhinitis
 - upper respiratory tract infection
 - neoplasm
8. Which of the following is the most likely etiologic factor in epistaxis?
- age
 - season
 - trauma
 - barometric pressure
 - humidity
9. An underlying coagulopathy as the cause of epistaxis is suggested by all of the following except
- a family history of bleeding
 - easy bruisability
 - a coexisting hematologic malignancy
 - cutaneous telangiectasias
 - chronic aspirin ingestion

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INTERIM QUIZ 1 ANSWERS

1. e. This is one of the first branches of the third portion of the IMA; it exits the sphenopalatine fossa area quite laterally to supply the upper teeth, maxillary sinus, and buccal mucosa; as such, it is not a primary arterial feeder to the internal nose. Of the other choices in this question, the sphenopalatine is the major blood supply to the posterior and inferior portions of the nasal cavity; the ethmoidal arteries (branches to the internal carotid system) supply the superior portions of the nasal cavity; and superior labial artery, a branch of the facial artery, contributes to Kiesselbach's plexus.
2. e. The sphenopalatine artery is a branch of the IMA, part of the external carotid system.
3. c. The nasopalatine artery usually branches within the sphenopalatine foramen and is not accessible to the surgeon's view. If it branches directly from the IMA, it gives the appearance of a double sphenopalatine artery. Failure to recognize this anomaly for ligation of the sphenopalatine artery may well result in failure of the procedure because of nasopalatine artery supply to the bleeding point.
4. a. The text citation indicates that 11% of 6672 patients had at least one episode of epistaxis in their lifetime, although other clinicians have estimated that this could be as high as 60%. It must be kept in mind, however, that the vast majority do not require medical attention. Those requiring treatment are patients with recurrent bleeding or more severe posterior bleeding.
5. a. Posttraumatic aneurysms causing epistaxis result in a mortality rate of 50%. The aneurysm or arteriovenous fistula is often located in regions where surgical accessibility is difficult, such as the petrous portion of the temporal bone or in the cavernous sinus. Answer d is a good second choice for this question. Although it is uncommon and associated with large amounts of blood loss over a matter of years, the mortality rate is low. Similar observations can be made for hemophilia. Arteriosclerosis is commonly associated with posterior epistaxis. Not only are the effects of blood loss and posterior nasal packing in the elderly associated with a significant, although low, mortality, the severity of the basic underlying arteriosclerotic disease also has a poor prognosis. Infectious diseases associated with mucosal bleeding, such as scarlet fever, are rare.
6. c. Common experience indicates that while any of the choices (with the exception of the inverted papilloma) could be present in the male adolescent, the juvenile angiofibroma has such a striking predilection for this age group. In the presence of nasal obstruction and severe bleeding, this diagnosis must be considered. Imaging studies should precede biopsy. Intranasal examination may disclose a foreign body or septal perforation without difficulty. Epistaxis as the sole manifestation of a vitamin deficiency would be extremely unlikely. Inverted papilloma occurs more commonly in the 50- to 60-year age group and tends not to bleed as vigorously as an angiofibroma.
7. a. The findings of Shaheen¹² confirm the occurrence of arterial disease in the posterior vessels but do not indicate why the lesions are more prevalent in this location. Epistaxis from allergic rhinitis, atrophic rhinitis, and upper respiratory tract infection tends to be diffuse and is more likely to occur posteriorly than anteriorly.

Bleeding from a neoplasm depends upon the site of the neoplasm and its vascularity. Certainly, with the juvenile angiofibroma, posterior epistaxis is more common.

8. c. Nasal trauma sustained by external force and, more commonly, by digital manipulation of the nose is a clearly important and common cause of epistaxis even in the patient with an underlying disorder such as arteriosclerosis or von Willebrand's disease; vigorous nose blowing may be the precipitating event. The incidence of epistaxis varies with age, season of the year, and humidity. Changes in altitude (and, therefore, barometric pressure) are associated with epistaxis; however, changes in barometric pressure without change of altitude have no relationship to the incidence of epistaxis.
9. d. Both familial history of bleeding with minor trauma or surgery as well as easy bruisability are characteristics of inherited coagulation disorders such as hemophilia or von Willebrand's disease. Hematologic malignancies, such as leukemia or multiple myeloma, often contribute to bleeding because of thrombocytopenia or platelet dysfunction. Aspirin is a potent inhibitor of cyclooxygenase, a key enzyme in the platelet aggregation cascade. Chronic aspirin ingestion may result in a prolonged bleeding time. Multiple cutaneous telangiectasias are a feature of Osler-Weber-Rendu disease, which is an autosomal dominant inherited disorder of subepidermal vessels and not a coagulopathy per se.

B. Management of Epistaxis

1. General approach

The primary goal is to stop the bleeding. Once the bleeding is under control, an attempt should be made to identify the cause of the epistaxis. Questions included in the medical history should focus on the general medical condition of the patient in addition to characterizing the epistaxis (e.g., quantity, duration, periodicity, measures taken to stop the bleeding, etc.). Pertinent historical data should include a history of previous bleeding (either personal or familial), recent trauma to the head/neck or surgery, drug ingestion or exposure to toxins, a history of nasal obstruction (unilateral or bilateral), and recent upper respiratory tract infection.

Diagnostic efforts at this time may be incomplete because a thorough intranasal examination may not be possible in the setting of acute epistaxis. Imaging studies are not usually indicated in the acute setting, as the sinuses are normally filled with blood and the films are generally difficult to interpret.

During the physical examinations, the patient should be placed in a comfortable position with the head elevated. The availability of an assistant facilitates treatment. In addition to noting vital signs, it is important to estimate the volume of blood loss, the general medical condition of the patient, and the current hemodynamic status. One should note pallor of the conjunctivae and nail beds, hypotension, and tachycardia. Pulse oximetry, when available, also may be useful. In most instances the bleeding will be minimal and controlled easily. In cases of severe bleeding, however, one or more peripheral IV lines should be started and fluid resuscitation be initiated with crystalloid solution (e.g., Ringer's lactate). In all but the most severe of circumstances, colloid solutions and blood products are to be avoided.

Laboratory studies should be obtained when there has been significant or recurrent bleeding. A complete blood count (CBC) may identify anemia, although the hemoglobin and hematocrit may be normal in the acute setting and are not good predictors of actual blood loss. Thrombocytopenia will be diagnosed with a platelet count. All patients taking Coumadin should have a prothrombin time (PT) (and an international normalized ratio [INR]) to assess the extent of anticoagulation. A partial thromboplastin time (PTT) should be drawn on patients receiving heparin therapy. A prolonged bleeding time may reflect recent aspirin use and an elevated blood urea nitrogen (BUN), which inhibits platelet function, should be anticipated in patients with chronic renal failure.

2. Nonoperative treatment

a. Topical vasoconstriction and cauterization

To effectively control the bleeding with minimal discomfort to the patient one must identify the specific site of bleeding. This requires adequate lighting and appropriate instruments. Basic equipment includes a head mirror or an indirect light source, nasal speculum, bayonet forceps, and suction. In addition, topical anesthetic and vasoconstrictive agents, along with cautery and packing materials, should be readily available. A rigid nasal endoscope or flexible nasopharyngoscope should be used to identify sites of posterior bleeding and to examine the nasopharynx. Universal

precautions should be employed at all times. First, using the suction and bayonet forceps, the clots are removed from the nasal cavity. The nose may then be sprayed with topical anesthetic and vasoconstrictive agents (e.g., 4% Xylocaine and 2% Neo-Synephrine). Long cotton pledgets soaked with a local anesthetic (e.g., lidocaine, tetracaine) and a vasoconstrictor (e.g. epinephrine, Neo-Synephrine) are then placed in the nasal cavity along the floor and the septum using bayonet forceps. Topical cocaine serves as a potent vasoconstrictor and local anesthetic; however, its use is limited by its cardiac effects.³⁸ Once the nasal mucosa has been adequately anesthetized and decongested, a search for the bleeding source is undertaken. In most instances the bleeding site will be easily identified on the anterior nasal septum with the aid of the nasal speculum. However, in cases of posterior bleeding, nasal endoscopy with a rigid 0° or 30° telescope is used to identify the bleeding source.

If the site of bleeding is identified in the anterior aspect of the nose, chemical or thermal cautery can then be directly supplied. Silver nitrate applicator sticks are a simple and convenient method of cauterization. Excess silver nitrate should be removed with a cotton applicator to prevent any of the chemical from spreading beyond the intended area and irritating the nasal mucosa. The physician may then elect to cover the cauterized area with a small piece of antibiotic-impregnated (e.g., Bacitracin or Neosporin) oxidized cellulose (Surgicel) as an extra precaution against further bleeding. Avitene or Gelfoam may also be used. Indiscriminant cautery in the absence of a localized bleeding site should be avoided. Particular caution must be used when cauterizing the nasal septum since cauterizing both sides of the septal mucosa may result in an iatrogenic septal perforation.

Thermal cautery with or without suction has been shown to be very effective in controlling anterior as well as posterior epistaxis with a success rate approaching 90%.³⁹⁻⁴⁵ Thermal cautery of the anterior septum may be performed under local anesthesia in the office setting or in the emergency department. However, posterior epistaxis is usually more brisk, and successful control by thermal cautery is best achieved in the operating room under general anesthesia. Such a setting generally results in improved patient comfort, better ability to visualize the bleeding site, increased availability of instruments and assistance, and protection of the airway. Posterior endoscopic cauterization (PEC) has several advantages, including decreased hospital stay, reduced expenses, less discomfort, and lower overall morbidity compared with other methods of controlling posterior epistaxis, such as posterior packing and ligation of vessels. Disadvantages of PEC include the inability to localize the bleeding site when epistaxis is severe and ineffective cauterization because of anatomic considerations, such as septal deviation, septal spur, or turbinate hypertrophy. Potential complications of PEC include numbness of the palate, synechiae formation, and rarely, cataract formation.⁴⁵

b. Injection

The injection of a topical anesthetic (e.g., Xylocaine) with or without epinephrine either locally, at the site of bleeding, or in the greater palatine

canal (GPC) is an alternative strategy in the management of epistaxis. A greater palatine foramen block may be invaluable in treating posterior bleeding. The instillation of a vasoconstrictor into the pterygopalatine fossa via the GPC acts on the third part of the IMA as it divides into the branches that supply the posterior parts of the nose. The needle is inserted into the greater palatine foramen, which is located just anterior to the junction of the hard palate with the soft palate and approximately 0.75 cm medial to the junction of the second and third molars.⁴⁶ The pterygopalatine canal runs posterosuperiorly for 2.5 to 3.0 cm. The distance to the foramen rotundum is 3.5 to 3.6 cm and approximately 4.0 cm to the infraorbital fissure. Allowing for 0.5 cm for the oral mucosa, the sphenopalatine and descending palatine vessels are reached at a depth of 2.8 cm.⁴⁶ A 25-gauge 2.5-cm needle is optimal for this purpose. Usually, 3 mL of lidocaine/epinephrine is injected because of the anesthetic and vasoconstrictive qualities of the solution, although similar results may be obtained with the same quantity of saline alone. Injection of the GPC is a convenient, rapid means of achieving hemostasis, particularly in the patient whose bleeding site cannot be identified upon anterior inspection. However, it may be only a temporary solution, with rebleeding occurring in as many as 40% of the cases.⁴⁷

c. Lasers

Lasers may be used in controlling the recurrent and inconvenient bleeding experienced by people with HHT. Many types of lasers are available for this purpose, including Nd:YAG, Argon, KTP, and CO₂ devices.²¹⁻²⁵ Acute bleeding in patients with HHT is controlled using standard techniques while the laser is reserved to treat telangiectasias in the symptom-free interval. The expense and technical support needed to operate a laser make it impractical for routine use in the acute setting. In addition, repeated applications of this technique unfortunately often lead to septal perforations.

d. Miscellaneous treatment

Cryotherapy⁴⁸⁻⁵⁰ has been reported to stop epistaxis, although its use as a standard treatment modality has never gained acceptance. This method requires specialized equipment and results in rebleeding in approximately 20% of the cases.⁴⁹ Intranasal irrigation of hot water has also been described as a treatment for posterior epistaxis.⁵¹ Packing the nose with porcine strips⁵² and ham fat⁵³ have also been described. These methods are rarely used in the contemporary management of epistaxis.

e. Anterior nasal packing

If bleeding persists despite the above-mentioned steps, or if the bleeding is too brisk to accurately identify the source of bleeding, nasal packing may be required. Half-inch vaseline gauze (or plain gauze) impregnated with antibiotic ointment is usually used as a packing material. Bismuth iodoform paraffin paste (BIPP) on ribbon gauze is also a popular packing material. The pack exerts continuous pressure to stop the bleeding. The nasal cavity is packed in a layered fashion with both ends of the packing material being placed last in the vestibule to prevent a free end from

falling down into the oropharynx. By carefully layering the packing material in the nasal cavity, 72 inches of packing can easily be placed into each side. Proper technique eliminates trauma to the nasal mucosa, thereby avoiding further bleeding. It is advisable to pack the bleeding side only to avoid septal ulceration and necrosis from the increased compression of bilateral packing. Because this type of packing often leads to an unpleasant odor, and may occlude the sinus ostia, antibiotics are recommended for the duration of the packing.⁵⁴ Because collagen organization is complete by day five, the packing is traditionally left in place for 5 days to allow for formation of a mature thrombus.

Intranasal sponges (Merocel) are a popular, alternative form of nasal packing.⁵⁵ The compressed synthetic sponge will expand to many times its size and conforms to the nasal cavity upon the addition of saline. It controls bleeding by exerting gentle pressure on the bleeding site. Nasal sponges are available in a variety of sizes and may easily be trimmed with scissors. They are also made in preformed varieties to better fill the nasal cavity and may be obtained with an indwelling tube to facilitate breathing while the pack is in place. To insert the device, the compressed sponge, which is rigid, is first coated with an antibiotic ointment. It is then placed in the nasal cavity and approximately 10 mL of saline or sterile water is slowly injected into the anterior aspect of the sponge to inflate it. Topical epinephrine with or without lidocaine may be used to inflate the sponge instead of saline or sterile water. The attached string is then taped to the patient's cheek to prevent the sponge from falling back into the naso- and oropharynx creating an aspiration hazard. As with traditional gauze packing, the sponge is left in place for 3 to 5 days and the patient should remain on antibiotics during this time. These devices are effective and have the distinct advantage of rapid insertion when compared with traditional gauze packing. Pringle et al⁵⁶ have demonstrated that nasal sponges are better tolerated than traditional packing materials and are less painful when removed. The long (10 cm) nasal sponges are also effective for the management of most cases of bleeding from the posterior septum.

Epistaxis in patients who are anticoagulated or in the setting of a coagulopathy poses a challenge. The bleeding is manageable with packing in most cases but may recur when the packing is removed. To overcome this problem, the gauze or nasal sponge is placed in a finger cot (coated with antibiotic ointment) before insertion into the nose.⁵⁷ This method helps to reduce the inflammatory reaction to the packing material and facilitates removal. A suture should be attached to the anterior aspect of the pack and taped to the patient's cheek to prevent the finger cot (and packing) from becoming dislodged posteriorly. Any coagulation defect should be corrected before packing removal when possible (i.e., discontinue heparin, administer fresh frozen plasma [FFP], desmopressin acetate [DDAVP], or platelets). Absorbable hemostatic packing is another option in the patient with a bleeding tendency. Oxidized cellulose (e.g., Oxycel, Surgicel), microfibrillar collagen (Avitene), or gelatin (Gelfoam) can be used as packing material and all share the distinct advantage of being absorbable, thereby eliminating the need for removal. These materials are hemostatic and may be used to tamponade the bleeding site. Unfortunately, as many as one third of the patients initially treated with

this type of material still require more traditional nasal packing.⁵⁸ Telfa sponges may also serve as appropriate packing material. When doubly folded, these sponges exert gentle pressure to stop bleeding while their nonstick surface facilitates easy removal.

f. Posterior nasal packing

If nasal examination demonstrates the bleeding to be inaccessible to anterior techniques, a posterior nosebleed is diagnosed. A posterior nasal pack then becomes necessary for control. There are several methods available to control bleeding from the posterior aspect of the nasal cavity. Since most posterior bleeding is brisk and often occurs in the geriatric population whose hemoglobin and hematocrit may already be compromised, it is advisable to start an IV early and begin the administration of IV fluids. The patient should be placed in the head-up position to avoid aspiration. Additionally, as posterior packing can be very painful, a narcotic and sedative are given to relieve the patient's distress. The choice of which posterior pack to be used depends upon the physician's experience and expertise with the various packing materials. In all types of posterior packs, the packing serves as a semirigid posterior buttress against which the anterior packing can be placed to achieve tamponade.

Intranasal balloon tamponade (e.g., Postpac) is quick, effective, and is often used in the emergency department by medical personnel who are not trained in the use of traditional nasal packs. A 12F Foley catheter may also suffice for this purpose. The catheter is placed uninflated in the nose until its tip is visualized in the oropharynx. The catheter is slowly withdrawn as the balloon is filled with either water or air according to the manufacturer's instructions. The balloon is pulled into the posterior choanae and the bleeding is diverted anteriorly. Anterior packing is then placed to stop the bleeding. The catheter must be fixed at the nasal ala to prevent it from slipping posteriorly. This can be accomplished with a surgical clamp or tying it over a dental roll with care taken to prevent alar necrosis.⁵⁹ There are several commercially available balloon packs on the market (e.g., Epistat, Nasostat). These devices are dual balloon catheters equipped with an anterior and posterior balloon, thus anterior and posterior bleeding can be controlled with a single device. All of these devices are usually left in place for approximately 5 days unless further treatment is planned. Balloon tamponade does not control all cases of posterior epistaxis most likely because the pressure of the inflated balloon applied to the internal nose is uneven; some areas receive greater pressure (nasal septum) than others.

A gauze pack is softer and may conform more easily to the nasopharyngeal vault and posterior choanae. A pack is selected that is large enough to occlude the choana but not so big that it will interfere with swallowing. The pack is firmly seated by pulling it into the posterior end of the nasal cavity by means of two heavy sutures attached to a catheter previously passed through the anterior nose and pulled out through the mouth. It is advantageous to hold the packing in a long curved clamp to avoid its being caught by the soft palate. The examiner's index finger must press the pack into the nasopharynx to properly seat it in the posterior choana. An ante-

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rior nasal pack is inserted in the manner previously described in order to tamponade the bleeding site. The strings from the posterior pack are then tied around a small gauze pad or dental roll in one nostril rather than around the columella, since the latter technique may result in necrosis of the columella or septum or both. The pack is left in place for approximately 5 days, after which time it is withdrawn through the mouth by applying traction on the attached suture dangling in the oropharynx. An alternative way of managing the suture used in removing the pack is to leave it long and tape it to the cheek.

Posterior packs are not without their disadvantages. They are uncomfortable to insert and remain so while in place. The stress of placing the pack may be great enough to cause myocardial ischemia and infarction. Once in place, the pack may cause dysphagia, obstruction of the eustachian tube, hypoxia, and exacerbate angina.

Hypoxemia has long been associated with posterior nasal packing. A nasopulmonary reflex has been postulated to account for the hypoxia associated with posterior packing. The hypoxemia is thought to predispose to arrhythmias and sudden death. A drop in partial pressure of arterial oxygen and/or a rise in partial pressure of arterial carbon dioxide has been documented and postulated to be induced by a nasopulmonary reflex change in airway resistance and compliance.⁶⁰⁻⁶⁶ However, other reports have failed to document an association between nasal packing and hypoxia.⁶⁷⁻⁷⁰ Despite the controversial data, it is recommended that patients requiring posterior packing be admitted to an appropriate hospital unit for continuous pulse oximetry and cardiac monitoring.

Other potential complications of nasal packing include potential injury to the nares, septum, mucosa, and palate, dislodgement and aspiration of packing materials, local (nose, sinuses, and ears) and systemic infection,⁷¹ and rarely, toxic shock syndrome.⁷²

INTERIM QUIZ 2

(Select the one best answer.)

1. Reduction of blood flow through the sphenopalatine artery may be achieved by injection of local anesthetic into the
 - a. foramen rotundum
 - b. lesser palatine foramen
 - c. greater palatine foramen
 - d. intraorbital foramen
 - e. incisive foramen

2. Failure to control bleeding by nasal packing is usually the result of
 - a. use of a posterior balloon tampon
 - b. improperly placed packing
 - c. an occult bleeding diathesis
 - d. a bleeding point inaccessible to the packing
 - e. vaseline gauze

3. A 30-year-old man with acute myelogenous leukemia has a moderate unilateral epistaxis that has not stopped with sedation and ice compresses. Proper local management would be
 - a. pack the nose with oxidized cellulose
 - b. ligate both the ethmoidal and internal maxillary arteries
 - c. pack the nose both anteriorly and posteriorly with antibiotic-impregnated gauze
 - d. inject 3 mL of 2% Xylocaine in the pterygopalatine fossa
 - e. insert and inflate nasal balloon tampon

4. A 45-year-old man has had six episodes of bleeding from Kiesselbach's plexus on the left side in the past 6 months. These episodes stop with local pressure, but he desires some measure to prevent recurrence. A prominent vessel and overlying crust is noted along the mucocutaneous junction of the left nasal vestibule, but there is no active bleeding at the moment. Management should consist of
 - a. observation
 - b. anterior nasal packing
 - c. antibiotic ointment
 - d. electrocautery
 - e. septodermoplasty

5. A 25-year-old woman develops severe left-sided epistaxis that requires anterior packing for control. She has no prior history or family history of bleeding. Which test should be ordered to evaluate her condition?
- a. sinus radiographs
 - b. PT, PTT, bleeding time/CBC and platelets
 - c. bone marrow examination
 - d. carotid arteriogram
 - e. vitamin K assay

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INTERIM QUIZ 2 ANSWERS

1. c. Injection of a bolus of fluid into the greater palatine foramen will often compress the descending palatine and sphenopalatine arteries and reduce or abolish nasal epistaxis. In some cases, thrombosis of the vessel may occur so that the relief is permanent. However, in a large number of cases rebleeding occurs once the fluid has been absorbed. The greater palatine foramen is located on the posterolateral aspect of the hard palate just anterior to the junction of the hard and soft palate, opposite the interdental space between the second and third molar teeth. Location of the foramen is done in a blind manner since there are no visible external landmarks to guide the surgeon. Study of a dry skull is extremely important to determine the variability and the location of the canal and the feel of the needle as it enters the canal. Care must be taken not to overinject in order to avoid anesthetizing the optic nerve.
2. b. If the packing does not occlude the area around the bleeding point in all directions, then bleeding will continue. Therefore, it follows that if the packing does not properly surround the bleeding point it will be ineffective. The techniques in the outline in the text should be reviewed. Answer d is a good second choice answer to this question. In the normal nose, however, all areas are accessible to proper packing. A severely deflected septum, for example, will prevent meticulous packing. Vaseline gauze packs may shift their location and permit bleeding to recur but, if properly layered in place, there is little opportunity for such shifting to happen. Similarly, a properly placed posterior balloon tampon will serve as well as the posterior gauze pack in most cases. Answer c is a logical response to the question except that it is an extremely uncommon circumstance. Tamponade of bleeding will occur with the use of an anterior nasal pack even if vessel thrombosis does not happen. The pressure of the pack will permit only an ooze through its substance, and severe hemorrhage is generally not possible with a pack in place.
3. a. Generally, one avoids packing the nose under pressure in the presence of a bleeding diathesis, since the ulcerations that occur from the packing may bleed as vigorously as the original bleeding source. Therefore, hemostatic materials such as oxidized cellulose, microfibrillar collagen, or gelatin soaked in topical thrombin are the simplest and most effective local measures to use in this instance. Correction of the coagulation deficiency is the primary therapeutic approach. A nasal balloon tampon might be used for short periods of time to minimize severe hemorrhage until medical treatment of the disorder is carried out. Similarly, injection of Xylocaine into the pterygopalatine fossa may be an acceptable temporary alternative, but one worries about bleeding from the injection site as well. If one were to use gauze packing, it should be placed in a finger cot to reduce bleeding upon removal. Arterial ligation usually has no place in the management of patients with blood dyscrasias.
4. d. For patients with an enlarged vessel in Kiesselbach's plexus, electrocautery allows permanent thrombosis and obliteration of the offending vessel with a minimum of difficulty. Continued observation is generally not useful. Antibiotic ointment may allow the area to heal, but it is common experience to have bleeding recur at a later date. Anterior nasal packing is generally not employed in the absence of active bleeding. The septal dermoplasty is not indicated for this disorder.

der. A further medical evaluation is unnecessary if the bleeding is from a local problem such as outlined in this question. If electrocoagulation has failed or if the patient is bleeding from more than one point, then evaluation of the coagulation status would be indicated.

5. b. Radiographs are not helpful in the routine evaluation of acute epistaxis, since blood in the sinuses may obscure a soft tissue abnormality. A fracture or bone destruction from a neoplasm are the only significant findings that could be observed, and these should be suggested by the patient's history or noted on nasal endoscopy. Study of the patient's coagulation system is indicated in the presence of diffuse bleeding; one would be concerned about an acute acquired disorder. A CBC with a platelet count may demonstrate an underlying disorder, such as an occult malignancy of the hematopoietic system. Assay of vitamin levels is generally an impractical examination; liver function studies would be more appropriate here if the PT were greatly elevated. Bone marrow examination would not be carried out to evaluate epistaxis; it would only be indicated to evaluate, where appropriate, those abnormalities detected on medical evaluation.

C. Operative Treatment

Many surgical procedures have been described to treat epistaxis. Surgical intervention has generally been reserved for patients whose bleeding cannot be controlled with more conservative packing techniques. However, some surgeons now prefer operative intervention early in the treatment of epistaxis in an attempt to reduce morbidity, decrease costs, and shorten the hospital stay.

1. Arterial ligation

The decision to perform ligation of vessels is based on visualization of the bleeding point and a thorough knowledge of the arterial anatomy. In general, ligation as close as possible to the site of bleeding is preferable to more proximal occlusions that do not control the collateral flow and promote backbleeding. The use of improved clip applicators⁷³ has increased the ability to ligate vessels in confined spaces and the success rate for arterial ligation is now reported to be as high as 90%.⁷⁴ The primary vessels to consider for ligation, depending upon the site of bleeding, include the anterior and posterior ethmoid arteries and the IMA with its distal branches.

a. Ligation of the ethmoid artery

The ethmoid foramina through which the ethmoid arteries pass are situated in the frontoethmoid suture line between the orbital plate of the frontal bone and the lamina papyracea. The anterior ethmoid foramen is approximately 24 mm posterior to the anterior lacrimal crest, with the posterior ethmoid foramen located approximately 10 to 12 mm more posteriorly (Fig 4B). It should be noted that the posterior ethmoid artery is 4 to 6 mm anterior to the optic nerve.

A vertical incision is made halfway between the medial canthus of the eye and the midline of the nose, extending approximately 1 cm above and below the canthus. This incision is carried through the skin, subcutaneous tissue, and periosteum; care must be taken to identify and ligate the angular vein to prevent bleeding. The periosteum, including the medial canthal ligament, is elevated off the ascending process of the maxillary bone and the anterior lacrimal crest. The lacrimal sac and the periosteum of the orbit are retracted laterally. At the superior aspect of the lacrimal bone, the frontoethmoid suture is identified. Following the frontoethmoid suture line posteriorly, the anterior ethmoid artery is identified. This is doubly ligated with a surgical clip and may be divided as it enters the anterior ethmoid foramen. The use of a 0° nasal endoscope facilitates visualization of the artery during the clipping. Some surgeons prefer to use bipolar cautery instead of clipping the vessel. If one chooses to ligate the posterior ethmoid artery, the frontoethmoid suture line is followed more posteriorly until the vessel is identified and doubly clipped. Cautery is to be avoided in this area to prevent injury to the optic nerve. After skin closure, a moderate pressure dressing is applied to the incision for 24 hours to prevent postoperative edema and ecchymosis about the eye. Some surgeons prefer to place a passive drain for 24 hours. The potential for complications from this procedure include failure to identify the arteries, avulsion of the arteries, blindness, and pseudohypertelorism.

b. Ligation of the internal maxillary artery

Ligation of the IMA through a transantral approach has been considered the gold standard for the control of posterior epistaxis. This is because of the ease of the procedure as well as its high rate of success. When properly performed and combined with anterior ethmoid artery ligation, it is invariably successful.⁷⁵ Ligation of both blood supplies is particularly useful in patients who have undergone several packings resulting in macerated mucosa which prevents localization of the initial bleeding site. Ligation of the IMA through the transantral approach was first described by Seiffert⁷⁶ in 1928 and made popular by Chandler and Serrins.⁷⁷ Potential complications of the transantral approach include numbness of the teeth, lip, and cheek, oral-antral fistula, and rarely, blindness.^{75,78} Failure of the surgeon to identify the IMA or all of its branches is the most common cause of surgical failure using the transantral approach.⁷⁹ Three essential places to ligate the vessel in the pterygomaxillary fossa include the IMA just proximal to the descending palatine artery, the descending palatine artery, and on the IMA as distally as possible.⁸⁰ In practice, all vessels that are identified should be ligated.

A classic Caldwell-Luc approach (maxillary antrostomy) is used (Fig 5, A through D). After an incision is made in the gingivobuccal sulcus, the periosteum and soft tissues are elevated from the anterior face of the maxilla to expose the canine fossa. The maxillary antrum is entered and the opening is enlarged to a diameter of about 2.0 cm. The infraorbital nerve is identified and retracted superiorly. The posterior wall of the antrum is then stripped of its mucosa, and an opening is made in the inferior lateral portion of the posterior wall where the bone is thin. A Kerrison rongeur can be used to enlarge the opening in the superior and medial direction until there is an opening of 1.5 cm in diameter. The operating microscope is used to help in identifying the artery after the periosteum is incised. A variable amount of dissection in the fibroadipose connective tissue is necessary to identify the artery. Ordinarily, the vessels are immediately apparent. Blunt right-angle hooks are helpful in exposing the vessels. Each branch is elevated using the blunt right-angle hook and then clipped with one or more clips. Once one vessel is identified and clipped, the vessel is traced in both directions to identify other branches. The vessels need not be divided. Care is taken to identify and preserve any nerves posterior to the vessels.

Following ligation, nasal packing may be removed to evaluate the adequacy of the procedure. Persistent brisk bleeding may indicate either incomplete ligation of branches of the sphenopalatine artery or a misdiagnosis of the source of bleeding (e.g., the ethmoid vessels). Prior to closing the wound, a nasoantral window is created to facilitate postoperative sinus drainage. If the antrostomy is made before an intranasal examination, the surgeon may induce bleeding and have difficulty evaluating the efficacy of the procedure. The antrum need not be packed if adequate hemostasis can be obtained. However, a single layer of Gelfoam abutting the mucosa of the posterior antrum helps to prevent bleeding from this site. The anterior aspect of the nasal cavity can be packed lightly to control minor bleeding from excoriated mucosa. Postoperative care includes placing the

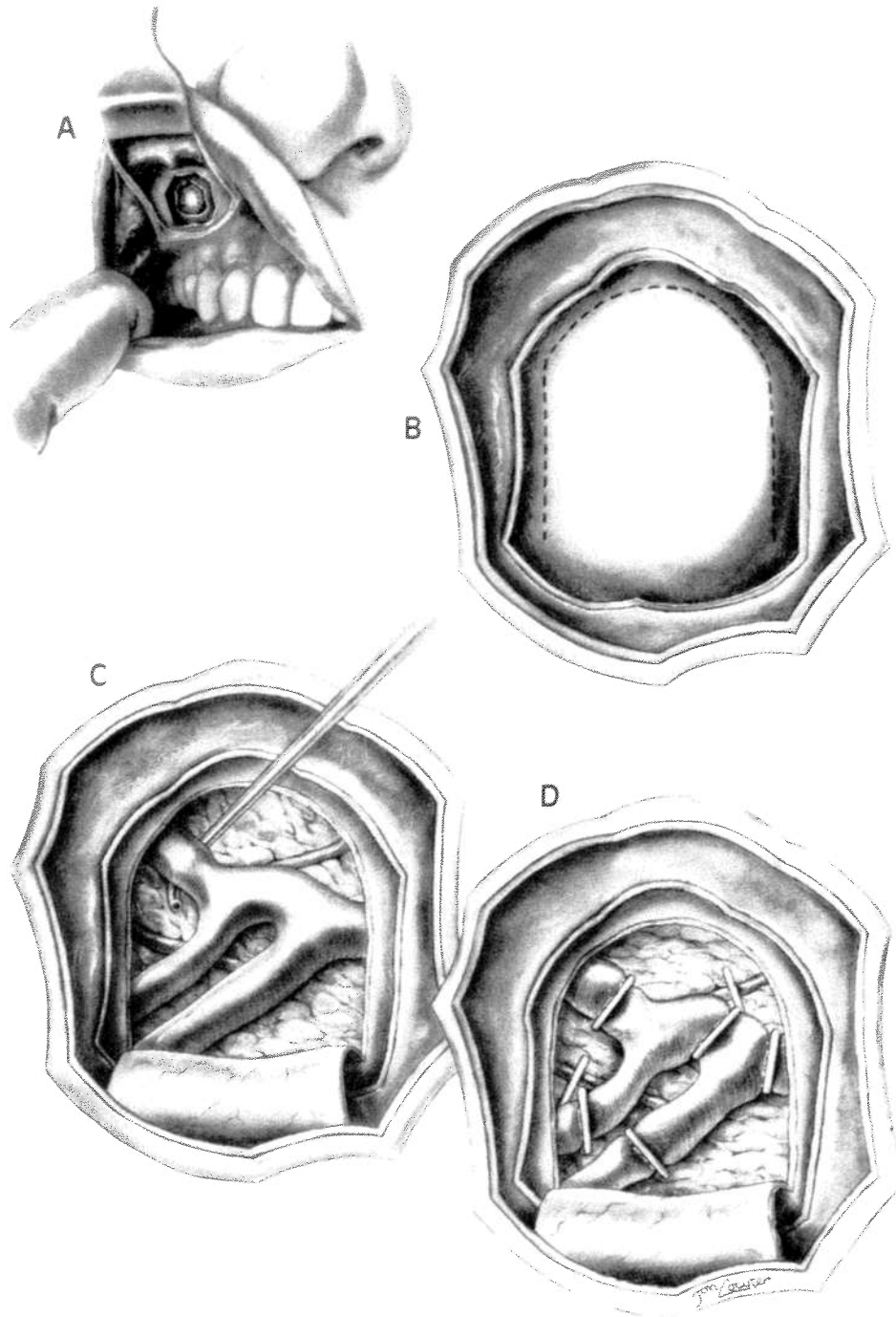


Fig 5.—A, View into maxillary sinus through Caldwell-Luc incision. B, Outline of periosteal flap to be reflected to expose pterygopalatine fossa. C, One view of arterial anatomy of pterygopalatine fossa as determined by anatomic dissection. D, Self-locking neurosurgical clips placed on all arteries identified.

patient in a semi-upright position with the application of an ice pack over the patient's face to prevent edema and ecchymosis. Nasal packing can be removed 12 to 24 hours after surgery.

Because of the potential complications related to the transantral approach, other techniques to ligate the IMA have been devised. Intraoral approaches to the IMA have been described.^{81,82} Although a high success rate is reported with these approaches, the IMA is more variable in its position and is clipped in a more proximal segment using these procedures.

c. Sphenopalatine artery ligation

In an effort to ligate the IMA at its most distal point (i.e., the sphenopalatine artery [SPA]) several investigators have devised methods to approach the distal IMA. Prades⁸³⁻⁸⁵ is credited as being the first to describe microsurgical ligation of the SPA in the 1970s. Simpson et al⁸⁶ selectively ligated the SPA using a transantral approach. Winstead⁸⁷ modified the procedure by removing the orbital process of the palatine bone at the upper medial corner of the posterior antral wall to identify the sphenopalatine foramen. Stamm and associates^{88,89} adopted a transnasal microsurgical approach to the SPA, utilizing an incision 1 cm anterior to the caudal border of the middle turbinate. A subperiosteal flap is then elevated and the SPA is identified at the sphenopalatine foramen. The transnasal approach to the SPA with the use of the nasal endoscope was not reported until 1992.⁹⁰ Other clinicians have subsequently reported endoscopic ligation of the sphenopalatine artery (ELSA).⁹¹

Endoscopic ligation of the sphenopalatine artery can be performed under local anesthesia, but general anesthesia is preferred in order to protect the airway in the event of posterior bleeding. Ligation of the ethmoid vessels should be performed first if done concurrently. Any anterior packing is then removed. Removal of the posterior pack or balloon is not necessary but facilitates visualization by allowing posterior drainage of blood into the nasopharynx. The nasal cavity is then decongested with topical anesthetic and vasoconstrictive agents on cotton pledgets. Bleeding when the pack is removed is unusual but can be controlled with the injection of lidocaine and epinephrine, by applying direct pressure from a pledget, or the area can be cauterized with bayonet bipolar or suction cautery. The nose is then examined with a 0° or 30° 4-mm endoscope. A large maxillary antrostomy is made that extends to the posterior wall of the antrum, so that the posterior edge of the antrostomy meets the posterior wall of the antrum. Added exposure of the middle meatus can be achieved by partial resection of the anterior end of the middle turbinate. Similarly, if a septal deviation or spur prevents adequate visualization, a septoplasty is performed. The bulla ethmoidalis and anterior ethmoid air cells are opened to improve exposure and to prevent postoperative ethmoid sinus obstruction. An elevator is then used to create a subperiosteal flap beginning at the posterior edge of the antrostomy. Working in an inferior to superior direction, the SPA is identified in the sphenopalatine foramen at the most superomedial aspect of the maxillary sinus. The SPA is then carefully followed laterally into the pterygopalatine space using a small Kerrison rongeur. Once the identity of the SPA is confirmed, hemoclips are applied to the vessel just

lateral to the sphenopalatine foramen. Other clips may be placed on distal branches of the SPA to protect against collateral circulation or a dislodged hemoclip. It may be necessary in some instances to introduce a trocar through the anterior maxillary wall to improve visualization. By placing the endoscope through the trocar, more room is created to work transnasally. A piece of Gelfoam is then placed over the exposed vessel and a Silastic nasal splint is left in place for 5 to 7 days to prevent synechiae. Intranasal packing is rarely needed and was found to be a cause of one of the reported failures by Snyderman and Carrau,⁹² as packing removal dislodged the clip from the vessel. If intranasal packing is required, it should be placed under endoscopic guidance to avoid inadvertent trauma to the hemoclips. The patients may then be discharged within 24 hours after this procedure (Figs 6 to 10).

Transnasal ELSA avoids the complications associated with traditional IMA ligation. There is also a theoretical advantage in that the vessel is ligated closer to the bleeding site reducing the chances for rebleeding through collateral circulation.

d. External carotid artery ligation

External carotid artery ligation has been used by some otolaryngologists with success in controlling epistaxis.⁹³ However, ligation of the ECA has been observed to have a high failure rate, presumably because such proximal ligation allows for collateral circulation and backbleeding.⁹⁴ Most otolaryngologists now advocate selective ligation of the terminal nasal branches of the ECA.

2. Endoscopic cauterization

Endoscopic cautery of anterior or posterior bleeding can be performed in the office setting or operating room. This method of controlling bleeding is attractive and has proven to be efficacious and expedient.³⁹⁻⁴⁵ Wurman and colleagues⁴² reported a greater than 90% success rate for posterior endoscopic cauterization (PEC). After decongesting the nasal mucosa, the nasal endoscope is used to identify the source of bleeding. Bipolar or suction cautery is then used to control the bleeding. If the patient has failed several previous attempts at packing, the nasal mucosa sometimes becomes so macerated that the original site of bleeding may not be readily apparent. In such an instance, it may be more practical to perform vessel ligation than to cauterize all identified sites of bleeding.

3. Miscellaneous procedures

Other surgical procedures have been proposed for treating epistaxis although they are not widely accepted. Septoplasty has been advocated to treat recurrent minor episodes of anterior bleeding. The anterior nasal vessels lie in a supraperichondrial plane and dissection there induces scarring that might lead to reduced bleeding.⁹⁵ Additionally, correction of a nasal deflection will theoretically reduce the formation of eddy currents that are implicated as the cause of bleeding in some patients. Surgical closure of the nostrils (Young's procedure) has been used in severe cases of HHT.^{96,97}



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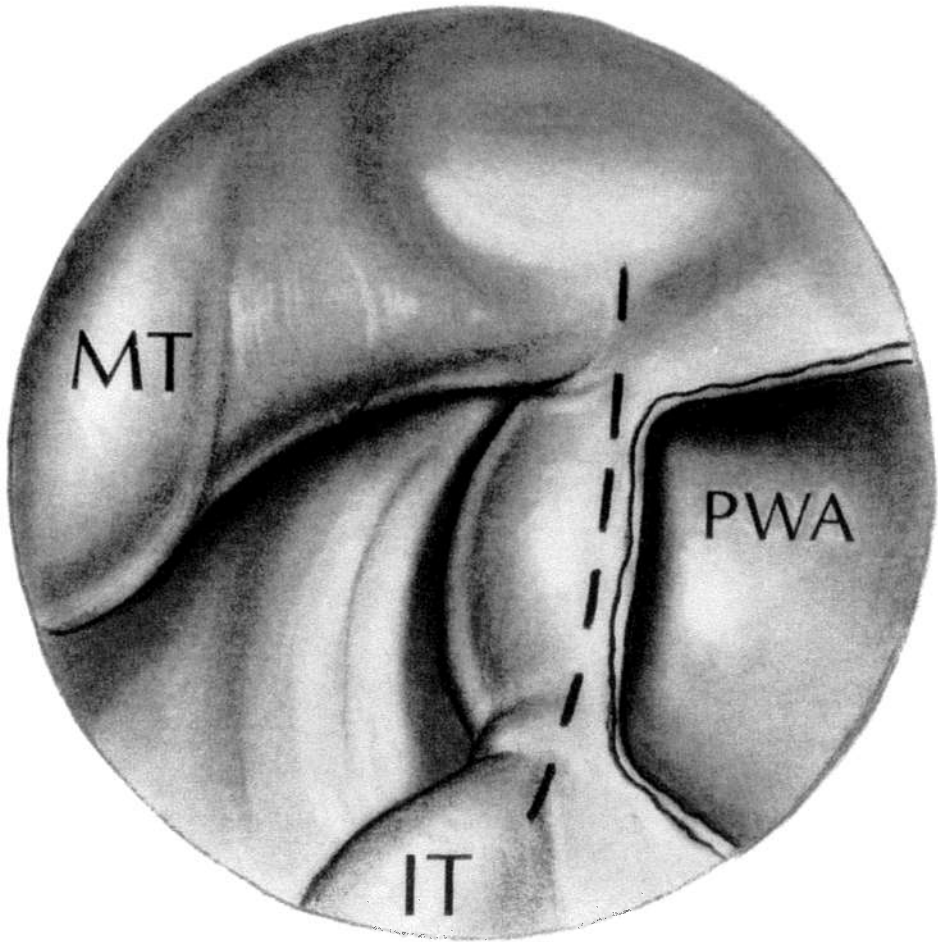


Fig 6.—A large middle meatal antrostomy is created. The mucosa is elevated in a subperiosteal plane at the posterior margin of the antrostomy (dotted line). MT, middle turbinate; IT, inferior turbinate; PWA, posterior wall of antrum.

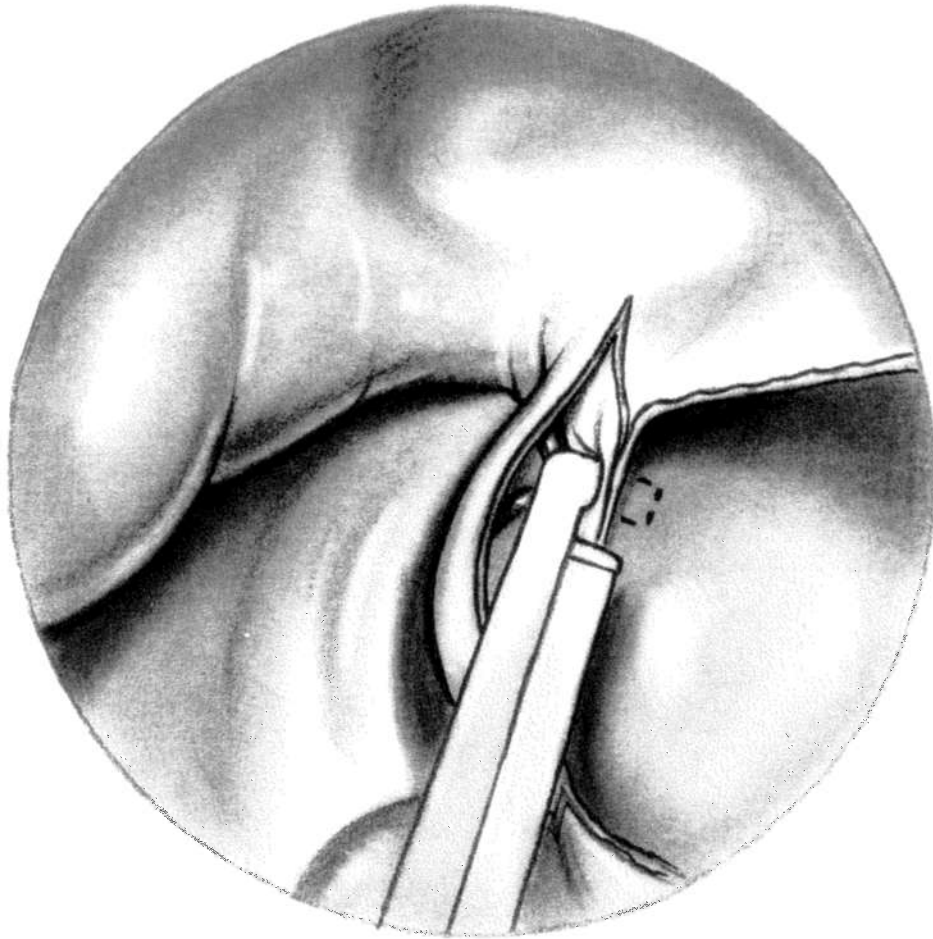


Fig 7.—Once the sphenopalatine artery is identified where it exits the foramen, a fine-tip rongeur is used to trace the artery laterally into the pterygopalatine fossa.

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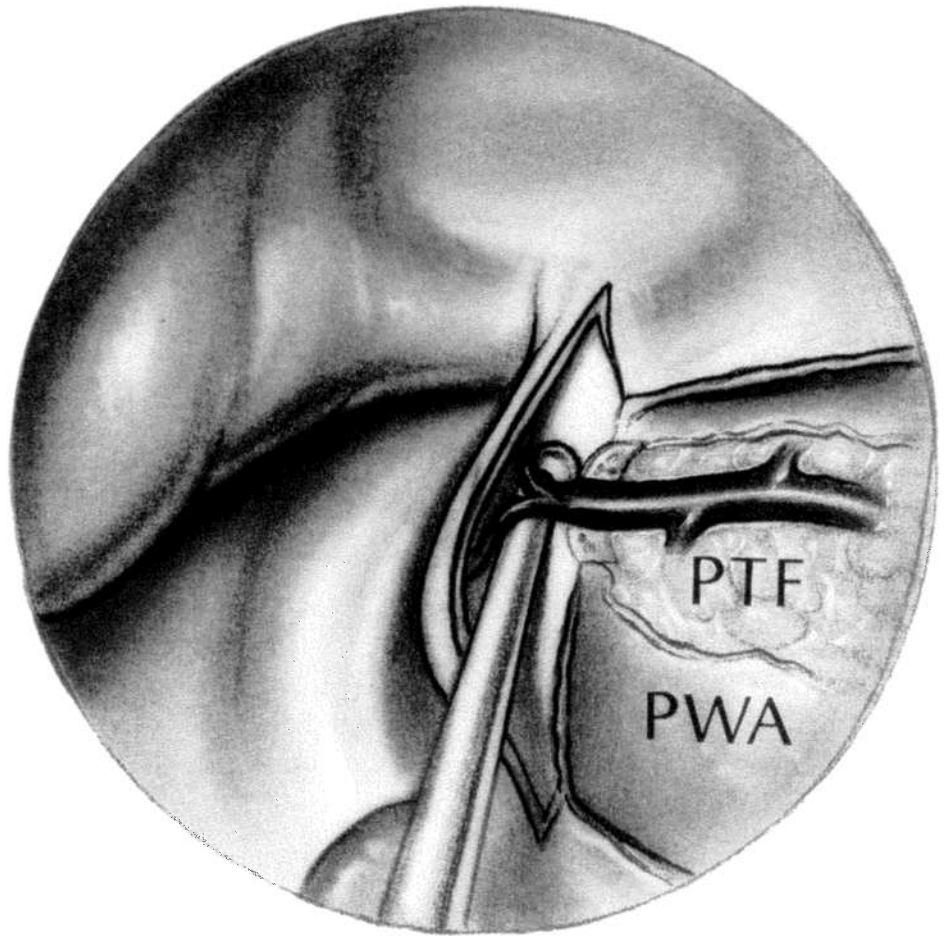
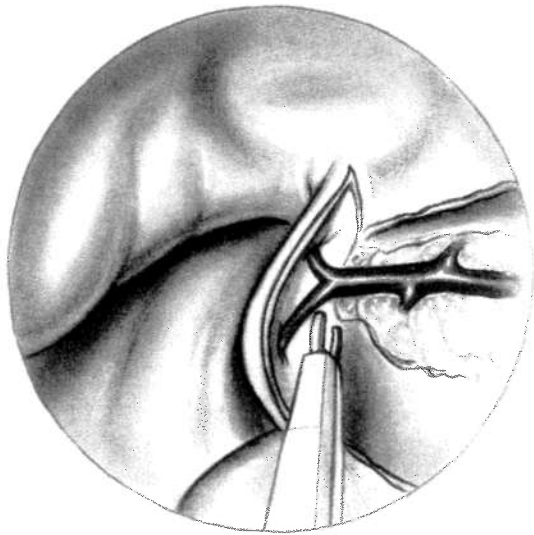
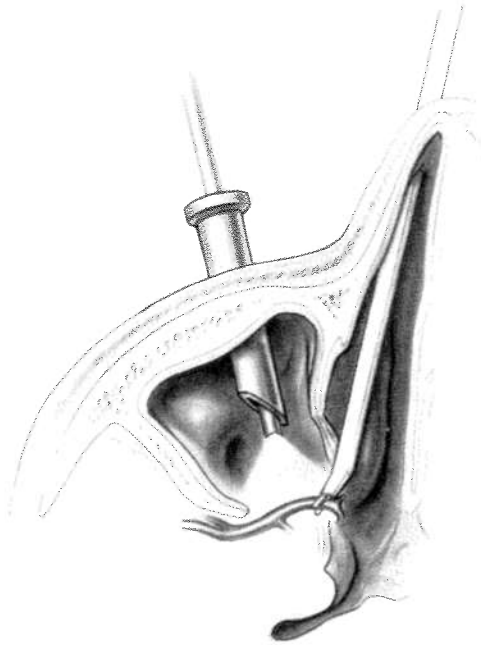


Fig 8.—After removal of bone from the posterior wall of the antrum (PWA), the course of the sphenopalatine artery in the pterygopalatine fossa (PTF) can be observed. The distal portion of the vessel is carefully dissected free from the adipose tissue using a blunt hook and suction.



A



B

Fig 9.—A. If there is adequate space for instrumentation, hemoclips may be applied to the sphenopalatine artery under endoscopic guidance through the middle meatus (B). If there is too much bleeding or insufficient space, it is helpful to use a transantral approach for the endoscope. This allows the introduction of hook or suction intranasally in addition to hemoclip applicator.

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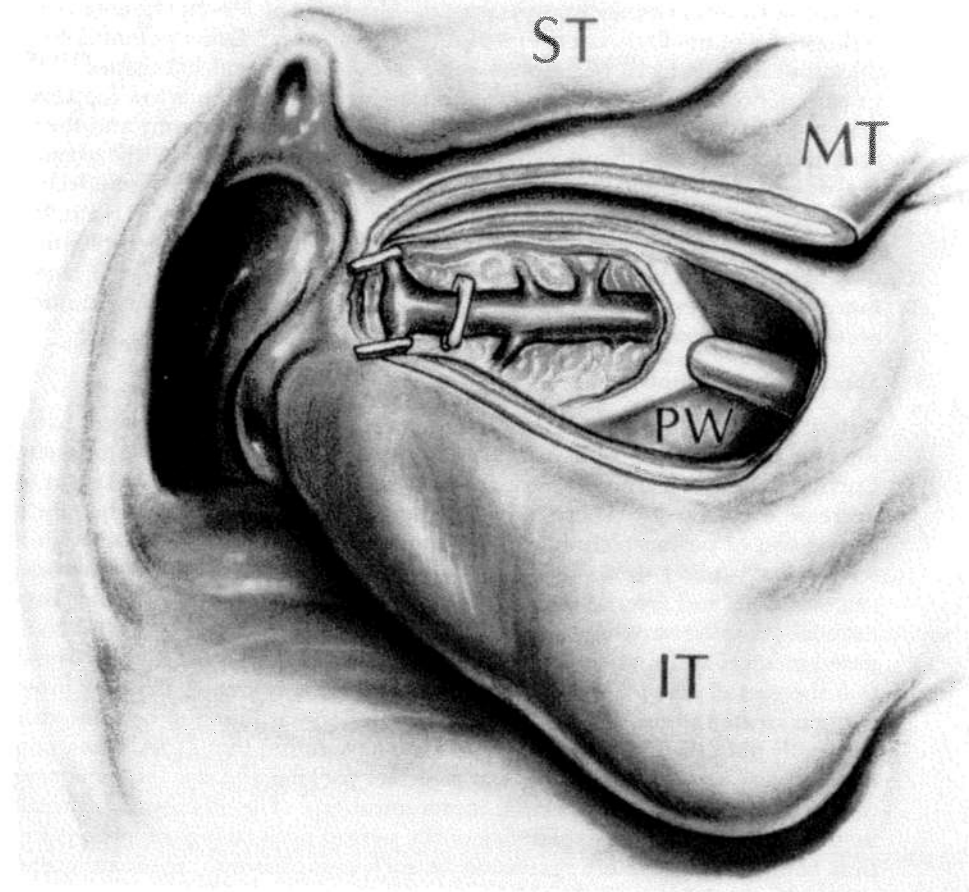


Fig 10.—After completion of the arterial ligation, hemoclips have been placed on the sphenopalatine artery lateral to the sphenopalatine foramen and its distal anterior and posterior branches. ST, superior turbinate.

4. Embolization/arteriography

Selective carotid arteriography with embolization can be useful in certain cases of epistaxis. Patients unable to undergo general anesthesia, with high comorbidities, or who have previously failed surgical ligation are all candidates for embolization.⁹⁸ Sokoloff et al⁹⁷ first reported the successful use of percutaneous embolization (PE) for the treatment of epistaxis in 1974. Many subsequent reports have confirmed the efficacy of embolization in controlling epistaxis.^{40,99-109} Arteriography will often fail to identify the site of bleeding unless the rate of blood loss is at least 0.5 mL/min.⁹⁹ The ipsilateral IMA and its distal branches are usually targeted. The ethmoid arteries are usually not candidates for embolization because of the risk of blindness or stroke. However, in

certain circumstances, such as preexisting blindness or life-threatening hemorrhage, the ophthalmic artery may also be embolized.¹¹⁰ Other potential complications include facial nerve palsy and necrosis of skin and soft tissues.¹¹¹⁻¹¹³ In experienced hands, the complication rate for embolization is low (approximately 1%). PE requires expertise in superselective arteriography and therefore may not be a treatment alternative in some medical centers if the personnel are not properly trained in these techniques. Embolization is also effective in reducing blood loss in patients with vascular sinonasal tumors and is a viable option in patients with HHT.^{100,113} One potential limitation of embolization may be prior vascular ligation. This is especially a problem when the larger more proximal vessels (e.g., external carotid) have been ligated, thus limiting access to the offending vessel during attempted embolization.

5. Special considerations

Anterior epistaxis is usually minor and easily controlled on an outpatient basis, whereas most patients with posterior bleeding are admitted to the hospital. Currently there is a trend to reduce the length of hospital admissions and to decrease overall costs. There have been several reports comparing the efficacy and cost of various techniques used to treat epistaxis.^{109,114,115} Barlow and co-workers⁴⁰ found similar failure rates for transantral ligation, endoscopic cauterization, and arterial embolization. They concluded that cost and institutional expertise should guide individual treatment. Schaitkin et al¹¹⁴ compared medical and surgical strategies for the treatment of epistaxis and found that the overall cost for treating patients surgically was greater than for those patients treated nonsurgically. Transantral ligation was found to be somewhat less costly than PE. Snyderman and Carrau have found that ELSA, when performed early, was less expensive than posterior packing and also less expensive than transantral ligation (personal communication). The efficacy of any treatment depends upon the experience of the person performing the procedure, thus confounding the interpretation of such comparisons. In treating epistaxis, the modality used should be the one that is most efficacious and cost effective in light of the patient's needs.

INTERIM QUIZ 3

(Select the one best answer.)

1. The initial bone removal of the posterior wall of the maxillary antrum for exposure of the pterygopalatine fossa is most safely and efficiently done in which quadrant?
 - a. superomedial
 - b. superolateral
 - c. inferomedial
 - d. inferolateral
 - e. in the center
2. The advantage of ELSA over IMA ligation is
 - a. patient comfort
 - b. reduced hospital stay
 - c. early ambulation
 - d. ligated vessel closer to bleeding site
 - e. infraorbital nerve anesthesia
3. When elevating the periosteum from the anterior antral wall, which structure should be identified and avoided?
 - a. infraorbital nerve
 - b. superior alveolar nerve
 - c. infraorbital artery
 - d. superior alveolar artery
4. Which is the most expeditious route for the ligation of the anterior ethmoid artery?
 - a. transorbital
 - b. transantral
 - c. intranasal
 - d. transcranial
 - e. percutaneous

INTERIM QUIZ 3 ANSWERS

1. d. An inferior lateral window through the posterior antral wall provides adequate exposure of the pterygopalatine fossa. This area of the wall is also paper thin, permitting easy resection. Additional bone removal in a superior and medial direction will allow the surgeon to follow the vessels medially to their entrance into the nasal cavity. The bone here is much thicker.
2. d. Ligating the vessel more distally has the theoretical advantage of avoiding collateral circulation. By not elevating a sublabial flap, the infraorbital nerve is not traumatized. Comfort, hospital stay, and ambulation are similar, although patients may have less pain and swelling since there is less dissection.
3. a. The infraorbital nerve is an important landmark in the determination of the superior extent of the antral window. Since injury to the nerve may result in numbness of the cheek, it is important to identify/preserve this structure.
4. a. The transorbital route allows easy access to the anterior ethmoid artery with a minimum of complications. The artery can be ligated safely from above using the transcranial route, but that is rarely indicated for treatment of epistaxis. The artery can be exposed by the transantral or intranasal approach; both approaches provide a narrow and poorly controlled access, and neither is considered safe.

Case

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VI. POSTTEST

Case 1

1. A 73-year-old, 140-pound man who is 5'10" tall enters the emergency room with a history of nasal bleeding for hours. He has a blood-saturated bath towel pressed to his nose. The initial step is to
 - a. obtain a complete history
 - b. record his vital signs
 - c. obtain a PT/PTT result
 - d. obtain hematocrit results
 - e. order a chest roentgenogram
2. He has a long history of hypertension controlled with methyldopa (Aldomet) and trichlormethiazide and a history of myocardial infarction 10 years previously for which he has been using digoxin, 0.25 mg, and aspirin, 1200 mg/d. There is no prior history of nasal bleeding. The blood pressure is 110/90 supine and 90/70 when sitting up, and the pulse is 140 and irregular. The nose is still bleeding, although at a slower rate. The next step is to
 - a. ambulate him and perform a complete neurological examination
 - b. administer morphine, 12 mg
 - c. start IV fluids with salt-poor volume expander
 - d. obtain PT, PTT results
 - e. order a chest roentgenogram
3. His general appearance is improved. After controlling the bleeding site with posterior and bilateral anterior nasal packs, the following laboratory values obtained from blood drawn when the IV was started are returned: HCT 30%, N_a 142, K 4.0, Cl 100, CO_2 20. The next step(s) include all of the following except
 - a. transfuse 2 units of whole blood
 - b. administer 40% oxygen
 - c. continue IV fluids
 - d. admit to the hospital
 - e. obtain an electrocardiogram

Case 2

1. A 55-year-old patient comes into the emergency room with nasal bleeding. On questioning, he complains of malaise, bone pain, and easy bruisability. The physical examination, in addition to bleeding from several anterior areas in the nose, reveals tenderness over the sternum and an enlarged spleen and liver. The initial screening test which is most informative is which of the following?
 - a. PT, PTT results
 - b. CBC, differential and platelets
 - c. arterial blood gases
 - d. liver scan
 - e. bone scan
2. Laboratory results are as follows: PT 17, PTT 34, hemoglobin 10, and platelets 100,000. Which test is most likely to be diagnostic?
 - a. factor VII assay
 - b. bone marrow biopsy
 - c. liver biopsy
 - d. skin biopsy
3. The proper management of this patient's bleeding would be to correct the underlying disorder, if possible, and to carry out which of the following procedures?
 - a. inject 3 mL of 2% Xylocaine into the pterygopalatine fossa
 - b. pack the nose both anteriorly and posteriorly with plain gauze
 - c. insert absorbable hemostatic packing in the nasal cavity
 - d. sedate the patient, place in semi-Fowler position, and spray the nasal cavity with cocaine 5% and Neo-Synephrine 1/4% every 3 to 4 hours
 - e. embolize the internal maxillary artery with Gelfoam beads
4. The patient continues to bleed from the nose in spite of excellent local measures. Proper treatment includes the administration of
 - a. salt-poor albumin
 - b. plasma cryoprecipitate
 - c. fresh-frozen plasma and vitamin K
 - d. platelets
 - e. packed red blood cells

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Case 3

1. A 14-year-old boy has a 3-month history of recurrent episodes of left-sided epistaxis that stop spontaneously and usually are about one-half cup in volume. His general health has been good and he denies any generalized symptoms. He is not bleeding at the present time. Initial steps include which of the following?
 - a. obtain additional history and examine the nose
 - b. administer Neo-Synephrine nasal spray
 - c. order CT scan of the head and neck
 - d. obtain a hematocrit
 - e. order coagulation studies
2. In addition to the epistaxis, he admits to recent headaches and left-sided nasal obstruction. Examination reveals a fleshy mass filling the superior part of the posterior one half of the left nasal cavity. The nasopharynx is inadequately visualized because of gagging. The next step is to
 - a. perform a biopsy of the mass
 - b. obtain sinus x-rays
 - c. order an enhanced CT scan
 - d. order an arteriogram
 - e. obtain a CBC
3. The CT scan confirms a vascular mass in the nasal cavity. While awaiting further therapy, the patient begins to bleed. Appropriate treatment at this stage includes
 - a. cautery
 - b. sedation
 - c. ethmoid artery ligation
 - d. vascular ligation
 - e. arteriogram and selective embolization
4. The arteriogram shows a vascular mass in the nose, left ethmoid labyrinth, and nasopharynx with a blood supply that is predominantly from branches of the ophthalmic artery. The most likely diagnosis is
 - a. juvenile nasopharyngeal angiofibroma
 - b. antral-choanal polyp
 - c. esthesioneuroblastoma
 - d. adenocarcinoma
 - e. mesenchymal neoplasm

Case 4

1. A 21-year-old woman seen in the emergency room is actively bleeding from the right side of the nose into a blood-soaked towel. She states that she has been bleeding heavily, but intermittently, for the past 3 days since her husband punched her. Attempts to visualize the bleeding point are difficult because of the profuse hemorrhage, but it appears to be posterior in origin. The next step is to
 - a. obtain sinus x-rays
 - b. insert a balloon tampon
 - c. start IV fluids and insert cotton pledgets saturated with a local anesthetic and a topical vasoconstrictor
 - d. reduce the nasal fracture
 - e. order an arteriogram
2. The above measures fail to control the bleeding which continues to ooze into her pharynx posteriorly. CT imaging fails to reveal a fracture. The next step is
 - a. posterior endoscopic cauterization
 - b. insert anterior/posterior packs
 - c. perform a septoplasty and packing
 - d. ligate the anterior ethmoid artery
 - e. ligate the internal maxillary artery
3. The nose is packed, but the patient continues to bleed. An internal maxillary artery ligation is carried out, but the posterior hemorrhage continues, although at a slower pace. The next step is to ligate the
 - a. right anterior/posterior ethmoid arteries
 - b. opposite internal maxillary artery
 - c. ipsilateral external carotid artery
 - d. opposite ethmoid arteries
4. Two days postoperatively the patient begins to hemorrhage again from the posterior nose, primarily on the right side. The next step is to
 - a. pack the nose
 - b. obtain an arteriogram
 - c. ligate the external carotid artery
 - d. ligate the right common carotid artery
 - e. ligate the left external carotid artery
5. No vascular anomalies are identified. Bleeding is right-sided through a persistent terminal branch of the sphenopalatine artery. The patient continues to bleed in spite of packing and ligation. The next step is to
 - a. re-explore the right pterygomaxillary fossa
 - b. ligate the left internal maxillary artery
 - c. ligate the right external carotid artery
 - d. perform selective embolization
 - e. repack the nose

Case 5

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Case 5

1. A 47-year-old man has been having intermittent increasingly severe left-sided epistaxis. He has no prior history of bleeding. His general health is good. When seen in the emergency room, his pulse is 100 and BP 170/95. Inspection of the nasal cavity reveals a bleeding point in the posterior left nasal cavity. A decision is reached to use anterior/posterior nasal packs. The most appropriate anesthetic technique is obtained by use of
 - a. topical 5% cocaine
 - b. topical lidocaine 1% with 1:100,000 epinephrine
 - c. greater palatine canal injection
 - d. topical cocaine plus injection of greater palatine canal
 - e. general anesthesia
2. Once inserted, the packs should ordinarily stay for what period of time?
 - a. until the bleeding stops
 - b. 6 to 12 hours
 - c. 2 to 3 days
 - d. 5 days
 - e. 1 week
3. Theoretically, the most effective antimicrobial agent to be used while the packs are in place is
 - a. amoxicillin
 - b. penicillin
 - c. gentamicin
 - d. amoxicillin plus clavulonic acid
 - e. clindamycin
4. The indication for arterial ligation in this patient is
 - a. development of sinusitis
 - b. severe pain
 - c. continued bleeding
 - d. hypoxia
 - e. suspected maxillary tumor

CREDIT REPORTING FORM

Self-Instructional Package (SIPac) 77399:

The Management of Epistaxis

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ANSWER SHEET FOR SIPac 77399

Case 1	Case 2	Case 3	Case 4	Case 5
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2.	2.	2.	2.	2.
3.	3.	3.	3.	3.
	4.	4.	4.	4.
			5.	

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Self-Instructional Package (SIPac) 77399. *The Management of Epistaxis*,
by Grandis et al.

POSTTEST ANSWERS

Case 1	Case 2	Case 3	Case 4	Case 5
1. b.	1. b.	1. a.	1. c.	1. d.
2. c.	2. b.	2. c.	2. a.	2. d.
3. a.	3. c.	3. e.	3. a.	3. d.
	4. c.	4. a.	4. b.	4. c.
		5. d.		

VII. REFERENCES

1. Petruson B: Epistaxis: A clinical study with special reference to fibrinolysis. *Acta Otolaryngol* 1974;S317:3-73.
2. Small M, Murray T, Maran A: A study of patients with epistaxis requiring admission to the hospital. *Health Bull (Edinb)* 1982;40:20-29.
3. Paff GH: *Anatomy of the Head and Neck*. Philadelphia, WB Saunders Co, 1973.
4. Weiss NS: Relation of high blood pressure to headache, epistaxis, and selected other symptoms. *N Engl J Med* 1972;287:631-633.
5. Pierce DL, Chasin WD: Treatment of epistaxis (Current Concepts in Therapy). *N Engl J Med* 1962;267:660-661, 748, 771.
6. O'Reilley BJ, Simpson DC, Dharmeratnam R: Recurrent epistaxis and nasal septal deviation in young adults. *Clin Otolaryngol* 1996;21:12-14.
7. Saunders WH: Septal dermoplasty: Its several uses. *Laryngoscope* 1970;80:1342-1346.
8. Stern SJ, Hanna E: Cancer of the nasal cavity and paranasal sinuses, in Myers EN, Suen JY (eds): *Cancer of the Head and Neck*, 3rd ed. Philadelphia, WB Saunders Co, 1996, pp 205-233.
9. Handa J, Handa H: Severe epistaxis caused by traumatic aneurysm of the cavernous carotid artery. *Surg Neurol* 1976;5:241-243.
10. Krempf GA, Noorily AD: Pseudoaneurysm of the descending palatine artery presenting as epistaxis. *Otolaryngol Head Neck Surg* 1996;114:453-456.
11. Millman B, Giddings NA: Traumatic carotid-cavernous sinus fistula with delayed epistaxis. *Ear Nose Throat J* 1994;73:408-411.
12. Shaheen OH: Arterial epistaxis. *J Laryngol Otol* 1975;89:17-34.
13. Jackson KR, Jackson RT: Factors associated with active, refractory epistaxis. *Arch Otolaryngol Head Neck Surg* 1988;114:862-865.
14. Beran M, Stigendal L, Petruson B: Haemostatic disorders in habitual nosebleeders. *J Laryngol Otol* 1987;101:1020-1028.
15. Jhanke V: Ultrastructure of hereditary hemorrhagic telangiectasia. *Arch Otolaryngol* 1970;91:262-265.
16. Suttén HG: Epistaxis as an indication of impaired nutrition and degeneration of the vascular system. *Med Mirror* 1864;1:769-772.
17. Osler W: On a family form of recurring epistaxis, associated with multiple telangiectasias of the skin and mucous membranes. *Bull Johns Hopkins Hosp* 1901;12:333-337.
18. Rendu M: Epistaxis répétées chez un sujet porteur de petits angiomes cutanés et mugeux. *Bull Mem Soc Med Hôp Paris* 1896;13:731-733.
19. Weber FP: Multiple hereditary developmental angiomas (telangiectasias) of the skin and mucous membranes associated with recurrent hemorrhages. *Lancet* 1967;2:160-162.

20. Hanes FM: Multiple hereditary telangiectasias causing hemorrhage (HHT). *Bull Johns Hopkins Hosp* 1909;20:63-73.

21. Vickery CL, Kuhn FA: Using the KTP/532 laser to control epistaxis in patients with hereditary hemorrhagic telangiectasia. *South Med J* 1996;89:78-80.

22. Siegel MB, Keane WM, Atkins JF, et al: Control of epistaxis in patients with hereditary hemorrhagic telangiectasia. *Otolaryngol Head Neck Surg* 1991;105:675-679.

23. Haye R, Austed J: Hereditary hemorrhagic telangiectasia—Argon laser. *Rhinology* 1991;29:5-9.

24. Kluger PB, Shapshay SM, Hybels RL, et al: Neodymium:YAG laser intranasal photocoagulation in hereditary hemorrhagic telangiectasia; an update report. *Laryngoscope* 1987;97:1397-1401.

25. Godberson GS: Indications, risks, and results of laser therapy for recurrent epistaxis, in Rudert H, Werner JA (eds): *Advances in Otorhinolaryngology*. Kiel, Germany, Christian Albrechts Universitat, 1995, pp 109-113.

26. Harrison DF: Use of estrogen in treatment of familial hemorrhagic telangiectasia. *Laryngoscope* 1982;92:314-320.

27. Vase P: Estrogen treatment of hereditary hemorrhagic telangiectasia: A double-blind controlled clinical trial. *Acta Med Scand* 1981;209:393-396.

28. Saunders WH: Septal dermoplasty for control of nosebleeds caused by hereditary hemorrhagic telangiectasia or septal perforations. *Trans Am Acad Ophthalmol Otolaryngol* 1960;64:500-506.

29. Ulso C, Vase P, Stoksted P: Long-term results of dermoplasty in the treatment of hereditary hemorrhagic telangiectasia. *J Laryngol Otol* 1983;97:223-226.

30. Strauss M, Zohar Y, Laurian N: The management of severe recurrent epistaxis due to hereditary hemorrhagic telangiectasia using regional facial cutaneous flaps. *J Laryngol Otol* 1985;99:373-377.

31. Bridger GP, Baldwin M: Microvascular free flap in hereditary hemorrhagic telangiectasia. *Arch Otolaryngol Head Neck Surg* 1990;116:85-87.

32. Benassayag C, Boudard P, Portmann D: Traitement d'une epistaxis par lambeau naso-genien bilateral au cours d'une maladie de Rendu-Osler. *Rev Laryngol Otol Rhinol (Bord)* 1989;110:305-307.

33. Zohar Y, Sadvor R, Shvili Y, et al: Surgical management of epistaxis in hereditary hemorrhagic telangiectasia. *Arch Otolaryngol Head Neck Surg* 1987;113:754-757.

34. Zohar Y, Talmi YP, Finkelstein Y, et al: Use of human amniotic membrane in otolaryngologic practice. *Laryngoscope* 1987;97:978-980.

35. Kendall BE, Joyner M, Grant H: Hereditary hemorrhagic telangiectasia-microembolization in the management of epistaxis. *Clin Otolaryngol* 1977;2:249-261.

36. Strutz J, Schumacher M: Uncontrollable epistaxis: Angiographic localization and embolization. *Arch Otolaryngol Head Neck Surg* 1990;116:697-699.

37. Hara HJ: Severe epistaxis. *Arch Otolaryngol* 1962;75:258-269.

38. Ross GS, Bell J: Myocardial infarction associated with inappropriate use of topical cocaine as treatment for epistaxis. *Am J Emerg Med* 1992;10:219-222.

39. Elwany S, Abdel-Fatah H: Endoscopic control of posterior epistaxis. *J Laryngol Otol* 1996;110:432-434. 59. 100:1
40. Barlow DW, Deleyiannis FWB, Pinczower EF: Effectiveness of surgical management of epistaxis at a tertiary care center. *Laryngoscope* 1997;107:21-24. 60. mona repor
41. O'Leary-Stickney K, Makielski K, Weymuller EA: Rigid endoscopy for control of epistaxis. *Arch Otolaryngol Head Neck Surg* 1992;118:966-967. 61. Rhinc
42. Wurman LH, Sack JG, Flannery JV: Selective endoscopic cautery for posterior epistaxis. *Laryngoscope* 1988;98:1348. 62. way, a
43. McGarry GW: Nasal endoscopes in posterior epistaxis: A preliminary evaluation. *J Laryngol Otol* 1991;105:428-431. 63. nasal
44. Bingham B, Dingle AF: Endoscopic management of severe epistaxis. *J Otolaryngol* 1991;20:442-443. 64. tients
45. Wurman LH, Sack JG, Flannery JV, et al: The management of epistaxis. *Am J Otolaryngol* 1992;13:193-209. 65. duce
46. Padrnos RE: A method of control of posterior nasal hemorrhage. *Arch Otolaryngol* 1968;87:181-184. 66. Clin (
47. Bharadwaj VK, Novotny GM: Greater palatine canal injections: An alternative to the posterior nasal packing and arterial ligation in epistaxis. *J Otolaryngol* 1986;15:94-100. 67. Laryn
48. Bluestone CD: Intranasal freezing for severe epistaxis. *Arch Otolaryngol* 1967;85:445-447. 68. reflex
49. Hicks JN, Norvis JW: Office treatment by cryotherapy for severe posterior nasal epistaxis: Update. *Laryngoscope* 1983;93:876-879. 69. nary 504-5
50. Bluestone CD: Intranasal freezing for severe epistaxis: A preliminary report of an experimental and clinical study. *Trans Am Acad Ophthalmol Otolaryngol* 1965;69:310-317. 70. reflex
51. Stangerup SE, Dommerby H, Lau T: Hot-water irrigation as a treatment of posterior epistaxis. *Rhinology* 1996;34:18-20. 71. 1971;
52. Heywood BB, Davis RB, Yorkers AJ: Treatment of epistaxis with porcine stripped packing. *Trans Am Acad Ophthalmol Otolaryngol* 1976;82:255-260. 72. ing. .
53. Gleason EB: The control of nasal hemorrhage. *Laryngoscope* 1898;4:143-146. 73. ligatic
54. Derkay CS, Hirsch BE, Johnson JT, et al: Posterior packing: Are intravenous antibiotics really necessary? *Arch Otolaryngol Head Neck Surg* 1989;115:439-441. 74. come
55. Doyle DE: Anterior epistaxis: A new nasal tampon for fast, effective control. *Laryngoscope* 1986;96:279-281. 75. the tr
56. Pringle MB, Beasley P, Brightwell AP: The use of Merocel nasal packs in the treatment of epistaxis. *J Laryngol Otol* 1996;110:543-546. 76. 1928;
57. Joseph MP, Kelly JH, Fried MP, et al: Alternatives to anterior-posterior packs for epistaxis. *Plast Reconstr Surg* 1981;67:530-533. 77. epista
58. Taylor MT: Avitene: Its value in the control of anterior epistaxis. *J Otolaryngol* 1980;9:468-471.

59. Kersch R, Wolff AP: Severe epistaxis: Protecting the nasal ala. *Laryngoscope* 1990;100:1348.
60. Cassisi NJ, Biller HF, Ogura JH: Changes in arterial oxygen tension and pulmonary mechanics with the use of posterior nasal packing in epistaxis; a preliminary report. *Laryngoscope* 1971;81:1261-1266.
61. Whicker JH, Kern EB: The nasopulmonary reflex in the awake animal. *Ann Otol Rhinol Otolaryngol* 1973;82:355-358.
62. Unno T, Nelson JR, Ogura JH: The effect of nasal obstruction on pulmonary, airway, and tissue resistance. *Laryngoscope* 1968;78:1119-1139.
63. Lin YT, Orkin LR: Arterial hypoxemia in patients with anterior and posterior nasal packing. *Laryngoscope* 1979;89:140-144.
64. Slocum CW, Maisel RH, Cantrell RW: Arterial blood gas determination in patients with anterior packing. *Laryngoscope* 1976;86:869-873.
65. Cook TA, Komorn RM: Statistical analysis of the alterations of blood gases produced by nasal packing. *Laryngoscope* 1973;83:1802-1809.
66. Jensen PF, Kristensen S, Juul A, et al: Episodic nocturnal hypoxia and nasal packs. *Clin Otolaryngol* 1991;16:433-435.
67. Larsen K, Juul A: Arterial blood gases and pneumatic nasal packing in epistaxis. *Laryngoscope* 1982;92:586-588.
68. Jacobs JR, Levine LA, Davis H, et al: Posterior packs and the nasopulmonary reflex. *Laryngoscope* 1981;91:279-284.
69. Morris HD, Doyle PJ, Riding KH, et al: The effects of posterior packing on pulmonary function in posterior epistaxis. *Trans Am Acad Ophthalmol Otolaryngol* 1976;82:504-508.
70. Loftus BC, Blitzer A, Cozine K: Epistaxis, medical history, and the nasopulmonary reflex: What is clinically relevant? *Otolaryngol Head Neck Surg* 1994;110:363-369.
71. Herzon FS: Bacteremia and local infections with nasal packing. *Arch Otolaryngol* 1971;94:317-320.
72. Hull HF, Mann JM, Sands CJ, et al: Toxic shock syndrome related to nasal packing. *Arch Otolaryngol* 1983;109:624-626.
73. Busch RF: A new vascular clip applicator for internal maxillary and ethmoidal artery ligations. *Otolaryngol Head Neck Surg* 1992;107:129-130.
74. Spafford P, Durham JS: Epistaxis: Efficacy of arterial ligation and long-term outcome. *J Otolaryngol* 1992;21:252-256.
75. Singh B: Combined internal maxillary and anterior ethmoidal arterial occlusion; the treatment of choice in intractable epistaxis. *J Laryngol Otol* 1992;106:507-510.
76. Seiffert A: Unterbindung der arteria maxillaris interna. *Z Hals Nasen Ohrenheilk* 1928;22:323-325.
77. Chandler JR, Serrins AJ: Transantral ligation of the internal maxillary artery for epistaxis. *Laryngoscope* 1965;75:1151-1159.

78. Beall J, Scholl P, Jafek B: Total ophthalmoplegia after internal maxillary artery ligation. *Arch Otolaryngol* 1985;111:696-698.
79. Metson R, Lane R: Internal maxillary artery ligation for epistaxis: An analysis of failures. *Laryngoscope* 1988;98:760-764.
80. Pearson BW, MacKenzie RG, Goodman WS: The anatomical basis of transantral ligation of the maxillary artery in severe epistaxis. *Laryngoscope* 1969;79:969-984.
81. Maceri DR, Makielsk KH: Intraoral ligation of the maxillary artery for posterior epistaxis. *Laryngoscope* 1984;94:737-741.
82. Stepnick DW, Maniglia AJ, Bold EL, et al: Intraoral-extramaxillary sinus approach for ligation of the maxillary artery: An anatomic study with clinical correlates. *Laryngoscope* 1990;100:1166-1170.
83. Prades J: Abord endonasal de la fosse pterygo-maxillaire. *LXXIII Cong Franc Compt Rendus Seanc* 1976:290-296.
84. Prades J, Bosch J, Tolosa A: *Microcirugia Endonasal*. Madrid, Garsi Edit, 1977.
85. Prades J: *Microcirugia Endonasal de la Fosa Pterigomaxillary del Meato Medio*. Barcelona, Salvat Edit, 1980.
86. Simpson GT, Janfaza P, Becker GD: Transantral sphenopalatine artery ligation. *Laryngoscope* 1982;92:1001-1005.
87. Winstead W: Sphenopalatine artery ligation: An alternative to internal maxillary artery ligation for intractable epistaxis. *Laryngoscope* 1996;106:667-669.
88. Stamm WK: Ein mikrochirurgische methode zur koagulation der a sphenopalatina als therapie der hinteren epistaxis. *Aktuelle Probl ORL* 1982;55:265.
89. Stamm AC, Pinto JA, Neto AF, et al: Microsurgery in severe posterior epistaxis. *Rhinology* 1985;23:321-325.
90. Budrovich R, Saetti R: Microscopic and endoscopic ligation of the sphenopalatine artery. *Laryngoscope* 1992;102:1390-1394.
91. White PS: Endoscopic ligation of the sphenopalatine artery (ELSA): A preliminary description. *J Laryngol Otol* 1996;110:27-30.
92. Snyderman CH, Carrau RL: Endoscopic ligation of the sphenopalatine artery for epistaxis. *Oper Tech Otolaryngol Head Neck Surg* 1997;8:85-89.
93. Waldron J, Stafford N: Ligation of the external carotid artery for severe epistaxis. *J Otolaryngol* 1992;21:249-251.
94. Bernstein L: Submucous operations on the nasal septum. *Otolaryngol Clin North Am* 1973;6:680.
95. Hosni AA, Innes AJ: Hereditary hemorrhagic telangiectasia: Young's procedure in the management of epistaxis. *J Laryngol Otol* 1994;104:754-757.
96. Gluckman JL, Portugal LG: Modified Young's procedure for refractory epistaxis due to hereditary hemorrhagic telangiectasia. *Laryngoscope* 1994;104:1174-1177.
97. Sokoloff J, Wickhom I, McDonald D, et al: Therapeutic percutaneous embolization in intractable epistaxis. *Radiology* 1974;111:285-286.

ry artery li-
analysis of
transantral
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Laryngoscope
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ry ligation.
al maxillary
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or epistaxis.
phenopala-
A prelimi-
e artery for
e epistaxis.
l Clin North
procedure
ry epistaxis
177.
s emboliza-

98. Rosnagle RS, Allen WE, Kier EL, et al: Use of selective arteriography in the treatment of epistaxis. *Arch Otolaryngol* 1980;106:137-142.
99. Parnes LS, Hennerman H, Vineula F: Percutaneous embolization for control of nasal blood circulation. *Laryngoscope* 1987;97:1312-1315.
100. Wills PI, Russell RD: Percutaneous embolization to control intractable epistaxis. *Laryngoscope* 1979;89:1385-1388.
101. Roberson GP, Reardon EJ: Angiography and embolization of the internal maxillary artery for posterior epistaxis. *Arch Otolaryngol* 1979;105:333-337.
102. Hicks JN, Vitek G: Transarterial embolization to control posterior epistaxis. *Laryngoscope* 1989;99:1027-1029.
103. Breda SD, Choi IS, Persky MS, et al: Embolization in the treatment of epistaxis after failure of internal maxillary artery ligation. *Laryngoscope* 1989;99:809-813.
104. Strutz J, Schumacher M: Uncontrollable epistaxis: Angiographic localization and embolization. *Arch Otolaryngol Head Neck Surg* 1990;116:697-699.
105. Vitek JJ: Idiopathic intractable epistaxis: Endovascular therapy. *Radiology* 1991;181:113-116.
106. Siniluoto TM, Leinonen AS, Karttunen AI, et al: Embolization for the treatment of posterior epistaxis. *Arch Otolaryngol Head Neck Surg* 1993;119:837-841.
107. Elden L, Montanera W, Terbrugge K, et al: Angiographic embolization for the treatment of epistaxis: A review of 108 cases. *Otolaryngol Head Neck Surg* 1994;111:44-50.
108. Elahi MM, Parnes LS, Fox AJ, et al: Therapeutic embolization in the treatment of intractable epistaxis. *Arch Otolaryngol Head Neck Surg* 1995;121:65-69.
109. Moser FG, Rosenblatt M, de la Cruz F, et al: Embolization of the ophthalmic artery for control of epistaxis: Report of two cases. *Head Neck* 1992;14:308-311.
110. Metson R, Hanson DG: Bilateral facial nerve paralysis following arterial embolization for epistaxis. *Otolaryngol Head Neck Surg* 1983;91:299-303.
111. de Vries N, Versluis RJJ, Valk J, et al: Facial nerve paralysis following embolization for severe epistaxis. *J Laryngol Otol* 1986;100:207-210.
112. Davis KR: Embolization of epistaxis and juvenile nasopharyngeal angiofibromas. *AJR Am J Roentgenol* 1987;148:209-218.
113. Shaw CB, Wax MK, Wetmore SJ: Epistaxis: A comparison of treatment. *Otolaryngol Head Neck Surg* 1993;109:60-65.
114. Schaitkin B, Strauss M, Houck JR: Epistaxis: Medical versus surgical therapy: A comparison of efficacy, complications, and economic considerations. *Laryngoscope* 1987;97:1392-1396.
115. Strong EB, Bell DA, Johnson LP, et al: Intractable epistaxis: Transantral ligation vs. embolization: Efficacy review and cost analysis. *Otolaryngol Head Neck Surg* 1995;113:674-678.

**OTOLARYNGOLOGY
SELF-INSTRUCTIONAL PACKAGE**

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