

Guidelines to the management of epistaxis

B. Bertrand*, Ph. Eloy*, Ph. Rombaux*, C. Lamarque**, J. B. Watelet***, S. Collet*.

*ENT and HNS Department, Catholic University of Louvain, Cliniques Universitaires UCL de Mont-Godinne, Yvoir, Cliniques Universitaires Saint-Luc, Brussels; ** ORL Department, Cliniques Saint-Luc, Bouge; *** ENT and HNS Department, University of Ghent, UZ Gent, Ghent

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Abstract. This article is a review of the literature on epistaxis and its treatment. Data were collected from MedLine until mid September 2005, and from others readings and books. Its first goal was to present to the Belgian ENT practitioners an overview, as complete as possible, of the modern concepts in etiologies, medical, conservative, surgical (including embolization), and adjunctive therapies of epistaxis. All these topics are discussed and commented, from a medico-surgical point of view, and also from a cost effectiveness one.

Foreword

First of all, it has to be stressed on that this topic on epistaxis – or nose bleed – is presented to you as a guideline only and no more.

The elements of history, discussion of pathophysiology, and details of manoeuvres are presented to help the physician best assess which patients are likely to benefit from this guideline, and which are most likely to need surgical therapy.

The suggested studies, physical examination – of the nasal cavities and of the total body –, medical material including rigid endoscope and fiberscope, plain X-ray, CT, MRI, lab exams, and others, are intended to maximize the adherence process to these guideline, they will not ensure successful treatments in every patient nor in every situation.

The present guideline should not be considered inclusive of all accepted methods of caring nose bleeds or exclusive of other cares

that can be assumed as reasonably aimed toward gaining the same results. The ultimate decision regarding the appropriateness of any specific procedure, therapy, or referral must be made by the physician him or herself in lights of – and depending on – all the circumstances presented by the patient as an individual and all surroundings.

Furthermore, medicine is a permanently changing science and not all therapies are clearly and definitely established. Obviously, new researches and discoveries are modifying medical and surgical treatments daily. That is why the reader should confirm the information in this guideline from other sources prior to use. In particular, all drug doses, indications, and contraindications should be confirmed in the package insert.

Background

Bleeding from the nose, or epistaxis – Greek for nosebleed that

means “which is leaking on, drop by drop” –, is a problem that has been a part of the human experience from earliest times. It was the subject for folklore and myth, and has been treated by physicians – and others – from the earliest times.

Centuries ago, Hippocrates reported that pinching the nose may help to stop bleeding. His technique is still in use today.

Writing magical words on the forehead with the patient's own blood, having the patient sniff his/her own dried blood into their nose, wearing amulets preferably tinted in red, have also been tried with more or less success obviously.

Carl Michel (1871), James Little (1879), and Wilhelm Kiesselbach were the first to identify the nasal septum's anterior plexus as a source of nasal bleeding.

Pilz was the first to treat epistaxis with ligation of the common carotid artery (1869). This was

followed by Seiffert who was doing the same with the internal maxillary artery via the maxillary sinus in 1928. Henry Goodyear performed the first anterior ethmoid artery ligation in the treatment of epistaxis.

The medical community's understanding of epistaxis has increased dramatically. Our treatment, though somewhat modified over the years, has continued to include techniques first noted several thousand years ago.

Epistaxis is very common in its frequency and varied in its presentation. The true incidence of epistaxis is not known in the literature because most episodes are self-treated and thus are not reported.

Most nose bleeds are mere nuisances, but some are quite frightening, and a few are even life threatening. The site of a patient covered in blood surrounded by a hoard of anxious family members can make even the most experienced emergency physician feel a sense of dread. When medical cares are needed, it is usually because of the problem is either recurrent or severe. Treatment depends on the clinical picture, the experience of the physician, the availability of ancillary services and for some cases, the hospital medical equipment and the expertise and skill of members of the staff.¹

In children, nosebleeds occurs also very commonly, especially in those between the ages of 2 and 10 years old. In most cases, they are secondary to local trauma and can be cared for by primary care physicians. In rare instances, however, a nosebleed may be difficult to control or may be a manifestation of a serious systemic illness. Referral to an otorhinolaryngologist or hematologist/oncologist is

usually not required except in these situations, and hospitalization is generally unnecessary.

Pathophysiology

The blood supply of the nose is complex and particularly rich with contribution from both internal and external carotid arteries.

The external carotid system supplies blood to the nose via the facial and internal maxillary arteries. The superior labial artery - one of the terminal branches of the facial artery - contributes to the blood supply of the anterior part of the nasal floor and of the septum through a septal branch. The internal maxillary artery enters the pterygomaxillary fossa and divides into 6 branches: posterior superior alveolar, descending palatine, infraorbital, sphenopalatine, pterygoid canal, and pharyngeal. The descending palatine arteries are running down through the greater palatine canals and supply the lateral walls of the nasal cavities. They go back to the nose via a branch in the incisive foramen to irrigate the anterior septum. The sphenopalatine arteries enter the nose through the sphenopalatine foramen which is situated approximately 10 mm dorsally to a theoretical line drawn between both the posterior attachments of the middle and lower turbinates to supply the lateral nasal walls. It also gives a branch to the septum. The internal carotid artery irrigates the nose through the ophthalmic artery that enters the bony orbit via the superior orbital fissure and divides into several branches.

One of which - the posterior ethmoid artery - exits the orbit for the nose through the posterior ethmoid foramen, located 2 mm to 9

mm anterior to the optic canal. The anterior ethmoid artery exits the orbit through the anterior ethmoid foramen. Both of the vessels cross the ethmoid roof to enter the anterior cranial fossa and then descend into the nasal cavity wherein they divide into lateral and septal branches to supply the lateral wall of the nose and the nasal septum. The Kiesselbach plexus, or Little area, is located on the lower third of the septal cartilage and is a very common spring for most anterior epistaxis. Many of the arteries supplying the septum have anastomotic connections in this plexus. The mucosa covering the area is thin and friable and the small vessels supplying the nasal mucous membrane have little structural support. Congestion of the vessels caused by conditions such as a URI or drying of the mucosa from low environmental humidity makes this area susceptible to bleeding. Sensory nerves follow the anatomical general pattern of the vessels. Trigeminal cranial nerves V1 and V2 supply the interior nose (anterior ethmoidal nerve and nasopalatine nerve respectively).

Clinical presentation

Nosebleeds may be either anterior or posterior in origin. Of the two categories of epistaxis, mundane and severe, the mundane, usually anterior epistaxis is the more common.

All individuals suffer occasional nose bleeds, of which the great majority are minor and occur on the anterior septum where they are easily managed.

Occasionally patients present with severe, life-threatening epistaxis arising from the larger vessels in the posterior and superior

nasal cavity. Such bleeds occur primarily in older patients, often with significant comorbidities. Then the bleeding compromises the patient's airway and results in hemodynamic instability. Management can be complex and severely tax the health care provider especially if he is not an otolaryngologist.

With anterior nosebleeds blood exits almost entirely from the anterior portion of the nose. With posterior nosebleeds, most of the bleeding occurs in the nasopharynx and mouth, although some blood exits through the anterior nose as well. Posterior epistaxis are often more severe and more difficult to control, and patients may present in a hemodynamically unstable condition as written before.

Patients with bleeding disorders may have recurrent nosebleeds and a history of prolonged bleeding, easy bruisability, or multiple bruises in unlikely locations. Alternatively, some patients who present with hematemesis have vomited swallowed nasal blood.

In children, most have a history of bleeding at home and minimal or no bleeding at all at the time of presentation. Parents and children, who are often frightened by nosebleeds, frequently overestimate the amount of blood lost.

Understanding and reassurance are important in dealing with patient anxiety.²

Epidemiology

Epistaxis is the second most common cause of spontaneous bleedings. Sixty per cent of patients may experience at least one episode of epistaxis during their lifetime. Eighty per cent of epis-

taxis occurs in Kiesselbach's plexus.³

In children, one can estimate that thirty percent have one nosebleed by the time they are 5 years of age. In children between 6 and 10 years old, frequency increases to 56%. Nosebleeds are rare in early infancy and infrequent after puberty. They occur much more frequently in the late fall and winter months, when URIs are common, environmental humidity is relatively low, and the use of heating systems results in dryness. Nosebleeds are also more common in children who live in dry climates, especially if they have a URI or allergic rhinitis.

Frequency

The true frequency of epistaxis is quite impossible to determine because most episodes resolve either spontaneously or with self-treatment, and therefore, are not reported.

Age

The general distribution is bimodal, with peaks in young children and elderly individuals.

Sex ratio

Incidence of epistaxis is a little bit higher in males than in females.

Mortality/Morbidity

For most people, epistaxis is a kind of a nuisance only. However, the problem can be life threatening, especially in elderly patients and among those with underlying additional medical problems.

Etiology

A sum up of etiologies can be displayed as follows below; they have been divided in local and systemic causes for more clarity.

Most etiologies are evident and commonly accepted. Some are not and/or are matter of discussions in the literature. That is why immediately after this synthetic paragraph a special discussion is done of some particular etiologies on which there is no definite consensus or clues. Some others are simply requesting further explanation and comments.

1. Local causes

1. Trauma (most common)

- Fracture(s): facial and nasal, of bone(s) and / or cartilage(s)
- Self-induced digital trauma, foreign body
- Iatrogenic: nasal / sinus / orbital / skull base surgery

2. Barometric changes

3. Nasal dryness - combination of dry air, septal deformities

4. Septal perforation

5. Chemical

- Cocaine abuse
- Nasal sprays (both steroids and decongestants)
- Ammonia
- Others: gasoline, phosphorus, chromium salts, sulfuric acid, etc.

6. Tumours

- Benign: polyps, inverting papilloma, juvenile nasal angiofibroma, septal angioma
- Malignant: squamous cell carcinoma, esthesioneuroblastoma

7. Inflammation

- Rhinitis: allergic, non allergic
- Sinusitis
- Infections: bacterial, viral, fungal

2. Systemic causes

1. Coagulopathies

- Anticoagulant use: coumadin,

heparin

- NSAIDs, aspirin
- Hemophilia
- Von Willebrandt disease
- Platelet defects
- Catamenial and pregnancy
- Hepatic insufficiency and alcohol⁴
- Scurvy
- Hemorrhagic fever (Dengue, Ebola, ...)^{5,6}

2. Granulomatous disorders

- Wegener's disease
- Mid face granuloma
- Sarcoidosis
- Syphilis
- Tuberculosis
- Rhinoscleroma
- Systemic lupus erythematosus
- Periarteritis nodosa

3. Intoxications: cobalt, phosphorus, arsenic, lead

4. Vascular

- Hypertension
- Circadian onset
- Atherosclerosis
- Osler-Weber-Rendu disease or hereditary hemorrhagic telangiectasia (HHT)

3. Idiopathic causes

4. Discussion of some etiologies

1. Trauma

Self-induced trauma from repeated nasal picking can cause anterior septal mucosal ulceration and bleeding. This scenario frequently is observed in young children. Acute facial and nasal trauma commonly leads to epistaxis. If the bleeding is from minor mucosal laceration, it is usually limited. However, extensive facial trauma can result in severe bleeding requiring nasal packing. In these patients, delayed epistaxis may signal the presence of a traumatic aneurysm.

Patients undergoing nasal surgery should be warned of the potential for epistaxis. As with nasal trauma, bleeding can range from minor (due to mucosal laceration) to severe (due to transection of a major vessel).

2. Septal deformities and perforations

Septal deviations and spurs may disrupt the normal nasal airflow, leading to dryness and epistaxis. The bleeding sites usually are located anterior to the spurs in most patients. The edges of septal perforations frequently harbor crusting and are common sources of epistaxis.

3. Inflammatory diseases

Bacterial, viral, fungal and allergic rhinosinusitis cause mucosal inflammation and may lead to epistaxis. Bleeding in these cases is usually minor and frequently manifests as blood-streaked nasal discharge. Granulomatous diseases such as sarcoidosis, Wegener granulomatosis, tuberculosis, syphilis, and rhinoscleroma often lead to crusting and friable mucosa and may be a cause of recurrent epistaxis.

4. Blood dyscrasias

There is a caveat for epistaxis. While most are simple, straightforward problems, they also can be the heralding sign of underlying coagulopathy. Hemophilia, von Willebrandt disease, thrombocytopenia, all may have epistaxis as their initial symptom.

Congenital coagulopathies should be suspected in individuals with a positive family history, easy bruising, or prolonged bleeding from minor

trauma or surgery. Acquired coagulopathies can be primary or secondary. Among the more common acquired coagulopathies are thrombocytopenia and liver disease with its consequential reduction in coagulation factors. Even in the absence of liver disease, alcoholism also has been associated with coagulopathy and epistaxis.

For those patients on anticoagulants, the altered coagulation should be carefully checked and monitored. For those on aspirin as a general prophylaxis 80 mg/d of aspirin is sufficient. Most use 300 mg daily or even more, as the adult aspirin tablets are easier to find and cheaper to purchase; 80 mg P.O. daily are enough to protect the heart and free the nose from an increased epistaxis risk.

In the *pediatric age group*, about one third of children with recurrent epistaxis have a diagnosable coagulopathy. In those cases, together with a positive family history, a prothrombin time (PT) and a partial thromboplastin time (PTT) are giving the most useful and cost-effective data.⁷

5. Scurvy

In western countries the incidence of scurvy appears to be on the rise. Populations at risk include the elderly, chronic alcoholics, diet faddists, the mentally ill, and patients with cancer, malabsorption, or who are on renal dialysis. The symptoms of scurvy are weakness, lassitude, depression, arthralgias, petechiae, perifollicular hemorrhage, follicular hyperkeratosis, corkscrew hairs, purpura, ecchymoses, gingival

swelling, hemorrhage, halitosis, poor wound healing, and loss of teeth.

Adults should receive 100 mg of vitamin C 3-5 x a day up to 4 gr followed by 100 mg/day. Infants and children should receive 10-25 mg 3 times a day. Symptoms disappear within 3-5 days.

6. Hypertension

Beran *et al.*⁸ reported that the distribution of blood pressure of habitual nose bleeders did not differ from that of the control group. This is in concordance with the data of Lubianca Neto *et al.*⁹, who also could not establish a definite association between blood pressure and history of adult epistaxis in hypertensive patients, although they found a link to left ventricular hypertrophy. The evidence for an association of duration of hypertension and left ventricular hypertrophy with epistaxis suggests that epistaxis might be a consequence of long term hypertension. They observed also the presence of enlarged vessels at rhinoscopy in hypertensive patients with a history of epistaxis.

Herkner *et al.*¹⁰ found higher blood pressure values in bleeding patients compared with controls. Padgham *et al.*¹¹ found a positive correlation between hypertension and bleeding from the middle meatus, but not with the severity of bleeding. The discussion on blood pressure and epistaxis will continue.¹²

7. Circadian variation of onset

Several physiopathological phenomena show two peaks during the day. In epistaxis, the time between the two peaks is about 12 hours.

Globally, all circadian rhythms in the cardiovascular system and the blood parameters enhance blood coagulation and formation of thrombus in the morning, whereas blood coagulation decreases in the afternoon. In a clinical study, Manfredini *et al.*¹³ suggest that blood pressure might trigger or be conducive to epistaxis and that might show circadian variation. A circadian rhythm of epistaxis was significantly found with a primary peak in the morning (8:00) and a smaller secondary peak in the evening (20:00). This is quite similar with hypertension, subarachnoid hemorrhage and rupture of aortic aneurysms. This deserves further investigation.

8. Arteriosclerosis

Atherosclerotic vascular disease is considered a reason for the higher prevalence of epistaxis in elderly individuals.

9. Hereditary hemorrhagic telangiectasia (HHT)

Hereditary hemorrhagic telangiectasia (Osler-Rendu-Weber disease) is an autosomal dominant disease associated with recurrent bleeding from vascular anomalies. The condition can affect vessels ranging from capillaries to arteries, leading to the formation of telangiectasias and arteriovenous malformations.

Histopathologic examination of these lesions reveals a lack of elastic or muscular tissue in the vessel wall. As a result, epistaxis can occur from minor trauma and tends not to stop spontaneously. Various organ systems such as the respiratory, gastrointestinal, brain and genitourinary systems may be in-

involved. The epistaxis in these individuals is variable in severity but is almost universally recurrent.

10. Idiopathic causes

Approximately 10% of patients with epistaxis have no identifiable causes even after a thorough evaluation.

Management and work up

1. History

- Specific questions about the severity, frequency, duration, and laterality of the epistaxis have to be asked.
- Inquiries about precipitating and aggravating factors and methods previously used to stop the bleeding must be made.
- A head and neck history with an emphasis on nasal symptoms has to be obtained.
- In addition, a general medical history concerning relevant medical conditions (e.g., hypertension, arteriosclerosis, coagulopathies, liver disease), current medications (e.g., coumadin, nonsteroidal anti-inflammatory drugs), smoking and drinking habits must be elicited.

2. General Assessment

- Airway patency has to be controlled and restored when necessary.
- Vital signs, pulse, respiration have to be checked.
- Pressure on nose has to be maintained (patient pinches anterior nose and leans forward). A swimmer's nose clip¹⁴ can be a very useful adjunct when treating patients with spontaneous, anterior epistaxis. This clip is efficient at providing constant, local-

ized pressure over the bleeding vessel, and alleviates the need to pinch the nose.

- Assessment of blood loss and side of bleeding (ask patient to quantify blood loss).
- Important and relevant medical problems: cardiac, cancer, previous epistaxis, allergies to medications (i.e.: local anaesthetics).
See details on drug safety in Table 1.
- Intravenous access has to be set up in place if indicated.
- Communication with patient can obtain consent and cooperation by explaining what is going to be done.

3. Examination

- Preparation
The patient has to blow his or her nose and all packing have to be removed even if bleeding is not active.
- Hardware
Before evaluating a patient with epistaxis, all the necessary topical medications, cauterization and packing materials should be ready. A good light is mandatory as well as an appropriate suction device, in order to clean up the nasal fossa from any cloths.
- Head and neck examination
A thorough head and neck examination must be performed if the patient's condition permits as well as anterior rhinoscopy before and after topical administration of medication.
- Anaesthesia +/- vasoconstrictors
See Table 1 for details on use, doses, safety in both adult and pediatric groups.
A good local anaesthesia allows further examination and pot-

ential treatments to be done in cozy conditions both for patient and physician. A topical anaesthetic, such as 4% aqueous lidocaine, and a vasoconstrictor, such as 0.05% oxymetazoline or 0.0002% epinephrine or another decongestant if not contraindicated, may be used. They can be applied via aerosolizing spray or cotton pledgets inside the nose for 7-10 minutes. With sprays topical medication may not stay in place long enough to be effective due to the blood flow.

Some epistaxis will stop at this early step.

- Endoscopy

Finally, endoscopy has to be performed using a flexible or preferably a rigid endoscope to inspect the entire nasal cavity, including the nasopharynx. The rigid endoscope is generally preferred because of its superior optic resolution and its ability to allow endoscopic suction and cauterization.

4. Conservative Treatment

1. Anterior Epistaxis

1. With patient in a sitting up position to squeeze anterior (compressible cartilaginous part) nose for 10 minutes.

2. Nasal cream

In children an antiseptic nasal cream can be very effective and avoids further manipulations in most cases.¹⁵

3. Cautery

Bleeding from the Kiesselbach plexus often is treated with silver nitrate cauterization. Other cauterizing

agents are also used: chromic acid, acetic acid, etc. Silver nitrate should be preferred because of the absence of any demonstrated carcinogenesis. The vessels leading to the site have to be treated first before treating the actual bleeding site. Random and aggressive cautery and cautery on opposing surfaces of the septum which is potential for septum perforation must be avoided. Silver nitrate doesn't work very well in active bleeding. Thirty seconds of exposure allowed silver nitrate to penetrate to a depth of approximately 1 mm penetration in a study by Lloyd *et al.*¹⁶ Longer exposure resulted in no significant additional. There was also no direct evidence that silver nitrate damaged directly the septal cartilage. Sseptal perforations in patients treated by means of topical silver nitrate may be attributable to necrosis of the septal cartilage due to damage to the perichondrium, from which it receive its blood supply.

4. Electrocautery using an insulated suction cautery unit also can be used. This method usually is reserved for more severe bleedings and for bleedings which are more posteriorly located. It requires local anaesthesia.¹⁷ The effectiveness of both cauterization methods can be enhanced by using rigid endoscopes, especially in the case of more posteriorly located bleeding sites.¹⁸ After the bleeding has been controlled, the patient is

Table 1
Drugs and material of epistaxis management

Drug category	Proof Level	Name	Adult Dose	Pediatric Dose	Contraindications	Pregnancy	Interactions	Precautions
Vasoconstrictor ; Act on alpha-adrenergic receptors in the nasal mucosa	Ia	Oxymetazoline 0.05%	2-3 sprays in each nostril q12h	6-12 years : 1-2 sprays each nostril q12h	Hypersensitivity ; MAOIs	Safety not established	Guanethidine ; Phenothiazine ; see under (a)	Do not use topical decongestants for >3-5 d ; see under (b)
Anesthetics ; see under (c)	III	Lidocaine 2 or 4% ; Xylocaine 2 or 4% ; Pantocaine 2 or 4% ; Cocaine 2 or 4% : see under (d) (use not permitted in some countries)	Spray or cotton pledgets	Not established	Hypersensitivity ; Adams-Stokes syndrome ; Wolf-Parkinson-White syndrome	Usually safe	None reported	Mucous membrane use only
Antibiotic ointments ; prevent local infection ; local moisturization	Ib	Mupirocin ointment 2% (Bactroban®) ; Bacitracin ointment	0.5 g in each nostril bid for 5 d	Not formally established	Hypersensitivity	Usually safe	None reported	Prolonged use may result in growth of resistant organisms
Cauterizing agents ; coagulate cellular proteins ; antibacterial effects	Ia	Silver nitrate ; cauterizing sticks and pearls	Not formally established	Not formally established, but commonly used	Hypersensitivity	Not established	Decreases effects of sulfacetamide preparations	Not for oral use ; Cauterize vessels one side at a time
Packing ; mechanical effect	III	Gauze ; Surgicel® ; Meroce® ; Oxygel®	NA	NA	NA	Safe	None reported	Mouth breathing, hypoventilation
Thrombin ; as local hemostatic	Ib	Thrombase® ;	FloSeal®	Cotton pledgets Gel	Not formally established	Not formally established	Not formally established	None reported
Adrenochrome ; enhance capillary resistance	III	adrenochrome semicarbazone ; AC-17® ; Adona® ; Adrenosem® ; Adrenoxy® ; Emex® ; Ibioxy® ; carbazochrome salicylate ; carbazochrome sodium sulfonate ; adrenochrome semicarbazone sodium sulfonate	Per Os : 10 to 30 mgr Also IM and IV – check package insert	Per Os : 5 to 10 mgr Also IM and IV – check package insert	Hypersensitivity	Unknown	None reported	
Terlipressin	Ib	Glypressin® ; Glypressin®	Gel ; IV: 20 to 30 microgr/kg ; check package insert	Not formally established	Hypersensitivity, asthma, hypertension, renal insufficiency, epilepsy	Potential abortion effect before the 4 th month		Monitoring of blood pressure and electrocardiogram

Table 1 (continuation)

Drug category	Proof Level	Name	Adult Dose	Pediatric Dose	Contra-indications	Pregnancy	Interactions	Precautions
Tranexamic acid	III	Exacyl®	PO : 1 to 1.5 gr/×3/d ; IV : 0.5 to 1 gr/×2 to 4/d ; Nose gel	20 mgr/kg/d ; check package insert	Hypersensitivity ; history of thrombosis ; renal insufficiency	To be avoided	Hormonal contraception	Chronic renal insufficiency : check creatinin level
Vitamin K, Phytonadione (Vitamin K1) ; Treat vitamin K deficiency	III	Synkavit® ; Konaktion® ; Vitamon K®	PO, IM, IV	PO, IM, IV	Hypersensitivity	Unknown	Warfarin (Coumadin) ; Cholestyramin ; Aspirin ; NSAID	
Etamsylate ; Benzenesulfonate derivative used as a systemic hemostatic.	II	Cyclonamine ; Etamsylate ; Altodor® ; Dicynone®	2-3 amp(of 250mgr)/day IM or IV then 6 co/d for 10 days	Half of the adult dose	Acute porphyria.	Unknown	No interaction is known up to now	As a precaution should not be administered during the first trimester of pregnancy, whereas during the second and third trimesters, it should be administered only if the expected therapeutic benefit is judged as superior to the potential risk for the foetus.
Estrogen-progesterone ; Ethinil-estradiol ; Hormonotherapy of HTT	II	Prémarin®	IM or IV 20 mgr ; as for hormonal contraception	Not established	Check package insert	Check package insert	Check package insert	Check package insert
Snake venoms	II	Reptilase® 1 U Klobusitzky / amp IM ; Reptilase® -R, 20 BU®, 1 ml/amp	1 amp IM	1 amp IM	Check package insert	Check package insert	Check package insert	Check package insert

Table 1 comments.

- Hypotensive action of may be reversed; concurrent administration with methyldopa may increase vasopressor response; concurrent use of MAOIs and ephedrine may result in hypertensive crisis. Pressor sensitivity to mixed-acting agents such as ephedrine may be increased. Guanethidine potentiates epinephrine and inhibits ephedrine. may reverse action of nasal decongestants such as oxymetazoline. TCAs potentiate vasopressor response and may result in dysrhythmias.
- Caution in: hyperthyroidism, coronary artery and ischemic heart disease, diabetes mellitus, glaucoma and in patients with an increased intraocular pressure, and prostatic hypertrophy. Hypertensive patients may experience change in blood pressure.
- Together with vasoconstrictors and β -adrenergics, their effect is prolonged and the pain threshold is increased (Oxymetazoline 0.05%; Ephedrine, Epinephrine, Adrenaline 1/50.000 or 1/100.000).
- Pantocaine and cocaine have additional vasoconstrictive effects on the blood vessels, and a particularly high affinity for fat tissues.

instructed to use saline spray and antibiotic ointment in his/her nose and to avoid strenuous activities for 7-10 days. NSAIDs are not to be used if at all possible. Digital manipulation of the nose is to be avoided. A topical vasoconstrictor may be used if minor bleeding recurs when eschars dislodge.

5. Gelfoam or Surgicel

Local resorbable hemostatic agents can be placed against the bleeding site. Absorbable materials may be used in patients with coagulopathy to avoid trauma upon packing removal.

6. Thrombin locally applied

On pledgets in the nose or more recently under the form of a gel.

Floseal, a hemostatic sealant composed of collagen-derived particles and topical bovine-derived thrombin, has been used as a high-viscosity gel for hemostasis in anterior epistaxis and as an easy alternative to nasal packing in patients suffering from acute anterior epistaxis in a study by Mathiasen and Cruz.¹⁹ A former work was done with this compound in ESS by Chandra *et al.*²⁰

7. Conventional packing

Nasal packing can be used to treat epistaxis that is not responsive to cautery. Two types of packing, anterior and posterior, can be placed. In both cases, adequate anaesthesia and vasoconstriction are necessary.

For anterior packing, various packing materials are avail-

able. They could be coated with an antibiotic and can be moisturized with a topical vasoconstrictor.

Anterior packs stay in 2-5 days, and patients may require analgesics. They also should be given an antibiotic to prevent rhinosinusitis and possible toxic shock syndrome.

Here are going some examples of material:

1. Nasal catheter – Epistat® (Xomed)

- Anaesthetize nose then before inserting
- Place ports horizontally so patient can eat/talk
- Inflate balloons with saline

2. Nasal tampon/sponge

Merocel® sponges can be placed relatively easily and quickly but may not provide adequate pressure.

- The nose has to be anaesthetized and sponge lubricated before inserting.
- Once inserted, sterile saline is injected into the sponge.
- Rewet prior to removal, check that none has broken off after removal.

3. Vaseline gauze

Petroleum jelly gauze with or without an antibiotic ointment traditionally is used. Layer it tightly and far enough backwards to provide adequate pressure. Blind packing with loose gauze is to be avoided.

- Leave ends protruding from anterior nose and don't let them dangle down pharynx.

- Packing has to be made in layers from nasal floor up.

2. Pediatric group

Most pediatric epistaxis can be effectively managed with a single out-patient consultation (91%) using a topical chlorhexidine and neomycin cream in addition in some case with a chemical nasal cautery with silver nitrate.^{15,21}

3. Posterior Epistaxis

Epistaxis that cannot be controlled by anterior packing can be treated with a posterior pack. Classically, rolled gauzes are used, but medium tonsil sponges can be substituted. Inflatable balloon devices, such as 12F or 14F Foley catheters, or specially designed (eg, Storz Epistaxis Catheter®, Xomed Treace Nasal Post Pac®) have become popular because they are easier to place.

Avoiding overinflation of the balloon is important because it can cause pain and displacement of the soft palate inferiorly, interfering with swallowing. Regardless of the type of posterior pack, an anterior pack also should be placed.

Patients with posterior packing should be admitted for close monitoring of oxygenation, fluid status, and adequate pain control. Need of adequate pain control has to be balanced with the concern over hypoventilation in the patient with posterior pack. They also should be given an antibiotic to prevent rhinosinusitis and possible toxic shock syndrome.

In a double blind placebo controlled study about antibiotherapy and nasal packing, Derkay *et al.* demonstrated that the

packing in the placebo group were foul smelling and heavily colonized with gram – while those from the antibiotic group were odor-free and lightly colonized by gram +. But no infectious complications were noted in both groups.²²

5. Surgical cares

Among most patients, the bleeding responds to cautery and/or packing. For those who have recurrent or severe bleeding for which medical therapy has failed, various surgical options are available.

Packing failure can be caused by inadequate placement from either lack of cooperation by the patient (especially those in the pediatric age group) or from anatomic factors such as a deviated septum. In these patients, a careful endoscopic examination under general anaesthesia may be considered.

1. Examination under general anaesthesia and septoplasty
Bleeding sites can be cauterized under endoscopic guidance, a deviated septum can be straightened, spurs can be removed, and meticulous packing can be placed. In addition, arterial ligation may be performed during the same setting if these steps fail to control the bleeding.
2. Fibrin glue
Topical use of fibrin glue especially in coagulopathies has been advocated.²³
3. Arterial ligation
The specific vessel(s) to be ligated depend on the location of the epistaxis. In general, the closer the ligation is to the bleeding site, the more

effective the procedure tends to be.

1. External carotid artery ligation

Ligation of the external carotid artery can be performed with the patient under local or general anaesthesia. A horizontal skin incision is made between the hyoid bone and the superior border of the thyroid cartilage. Subplatysmal skin flaps are then raised, and the sternocleidomastoid muscle is retracted posteriorly. Next, the carotid sheath is opened, and its contents exposed. The external carotid artery is identified by following the internal carotid for a few centimeters and dissecting the external carotid beyond its first few branches. After the external carotid has been positively identified, it usually is ligated just distal to the superior thyroid artery. Continued bleeding after ligation may be from anastomoses with the opposite carotid system or the ipsilateral internal carotid artery.

2. Internal maxillary artery ligation

This procedure has a higher success rate than external carotid artery ligation because of the more distal site of intervention. Traditionally, the internal maxillary artery was accessed transantrally through a Caldwell-Luc approach.²⁴

The posterior sinus wall is removed, and the posterior

periosteum opened. The internal maxillary artery and 3 of its terminal branches (i.e., sphenopalatine, descending palatine, pharyngeal) are elevated and clipped. The posterior sinus wall is then packed with gelfoam, and the Caldwell-Luc incision is closed.

Transoral and transnasal endoscopic approaches have been described.

The transoral approach is useful in patients with midface trauma, hypoplastic antra, or maxillary tumours.²⁵

The buccinator space entered through a gingivobuccal incision. The buccal fat pad is removed, and the attachment of the temporalis muscle to the coronoid process is identified next. This process facilitates the identification of the internal maxillary artery which is clipped. This procedure has a higher failure rate because the site of ligation is more proximal than in the transantral approach. The endoscopic method requires high skills with endoscopes and endoscopic instruments. A large middle meatal antrostomy is made to expose the posterior sinus wall. The middle turbinate can be resected partially to ensure adequate exposure. The remaining steps are similar to the traditional transantral approach.

3. Endoscopic sphenopalatine artery ligation (ESAL)

or transnasal endoscopic sphenopalatine artery ligation (TESPAL)

Endoscopic technique also can be used to ligate the sphenopalatine artery at its exit from the sphenopalatine foramen, results are good and abundantly reported in the literature.²⁶⁻³⁰

An incision is made just dorsally to the posterior attachment of the middle turbinate. The mucosal flap is elevated to reveal the sphenopalatine artery, which is clipped and ligated.

A recurrent bleed rate of 3/10 was found, which is higher than previously published results, in a study published by Rockey and Anand.³¹

4. Ethmoid artery ligation

If bleedings occur high in the nasal vault, consider ligation of the anterior and/or posterior ethmoid arteries. These arteries are approached through an external ethmoidectomy incision. The anterior ethmoid artery usually is found 22 mm (range, 16-29 mm) from the anterior lacrimal crest. If clipping the artery does not stop the bleeding, then the posterior ethmoid artery may be ligated. This artery is found approximately 12 mm posterior to its anterior counterpart. It should be clipped, not cauterized, because it is only 4-7 mm anterior to the optic nerve. A description of an endoscopic, intranasal tech-

nique for ligation of the anterior ethmoid artery in a single case report was made by Woolford and Jones.³²

4. Embolization / Interventional radiology

Bleeding from the external carotid system may be treated with embolization, either as a primary modality in poor surgical candidates or as a second-line treatment in those for whom surgery has failed.³³⁻³⁵

Preembolization angiography is performed to check for the presence of any unsafe communications between the internal and external carotid systems. Selective embolization of the internal maxillary artery and sometimes the facial artery may be performed. Ligation of the internal maxillary artery and selective percutaneous embolization gave similar results in a study performed by Romagnoli *et al.* among 203 patients.³⁶

Superselective embolization with gelatin sponge for intractable epistaxis report no complication in 37 patients with a success rate of 94.6% after 7 days, remaining stable after 21 months.³⁷

Postprocedure angiography can be used to evaluate the degree of occlusion. The most common reason for failure is continued bleeding from the ethmoid arteries. As the anterior ethmoid arteries arise from the ophthalmic which itself comes from the internal carotid, they must not be embolized.

The disadvantages of arteriography and embolization are the expense and the small risk of a stroke (CVA is quoted as 1 in 100) even among skilled and long trained radiology teams. For the most part angiography and embolization are reserved for very sick individuals and for whom surgery is an unsafe option. Embolization works best in active bleeding as one can see which artery is involved.

5. Treatment of hereditary hemorrhagic telangiectasia (HHT)³⁸

Treatment of this disease is palliative because the underlying defect is not curable. Options include coagulation with potassium-titanyl-phosphate (KTP) or neodymium:yttrium-aluminum-garnet (Nd:YAG) lasers^{39,40}

septodermoplasty, embolization, nasal closure, and/or estrogen-progesterone therapy. Topical estrogens combined with argon plasma coagulation in the management of epistaxis in HHT significantly prolong the hemorrhage-free interval.⁴¹

We find also a single case report of an intractable epistaxis secondary to HHT successfully controlled by the application of fibrin glue. Further studies are needed.⁴²

In some very difficult cases, nasal obliteration (Young's procedure)⁴³

has been proposed, with more recently a variant through a nasal obturator.⁴⁴

Systemic estrogen-progesterone at doses used for oral contraception may eliminate

bleeding in symptomatic HHT. Tamoxifen has been successfully used in two cases.⁴⁵

6. Sequential management

The therapeutic escalation has been discussed by Simmen and Heinz in a review of 335 hospitalized patients. Their conclusions can be summarized as follows: i) first, localization of bleeding and bipolar cautery have to be performed, ii) if they not succeed, Merocel® pack, iii) if rebleeding, there is the time for operating theater and general anaesthesia for an endoscopic control and/or balloon and, iv) if failure again, embolization, ligation, endonasal obliteration can take place.⁴⁶

6. Compared cost-effectiveness

A retrospective review of Nationwide Inpatient Sample in the USA (1998-2000) was published recently by Goddard and Reiter.⁴⁷

A total of 9778 admissions with admitting diagnosis “epistaxis” were identified. Among admissions involving 1 treatment, 454 (9.6%) received arterial ligation, 94 (2.0%) embolization, and 4188 (88.4%) nasal packing. There were no differences in length of stay, transfusions, complications, or deaths between groups. Mean total hospital charges were USD 6282 for the packing group, USD 12805 for the ligation group, and USD 17517 for the embolization group; differences between ligation and packing groups, and embolization and packing groups are statistically significant. They concluded that nasal packing is associated

with lower hospital charges and similar complication rates as arterial ligation or embolization. And they conclude also that further studies are needed to quantify other outcome measures, such as recurrence rates and patient quality of life.

In the treatment of recurrent posterior epistaxis ESAL was compared to conventional packing by Moshaver *et al.*⁴⁸

The overall calculated cost of patients undergoing ESAL was CAD 5133 compared with CAD 12213 in the conservative group, resulting in an average saving of CAD 7080 per patient. There was overwhelming patient satisfaction with ESAL compared with nasal packings.

At comparing endoscopic ligation to embolization Miller *et al.*⁴⁹ demonstrated that the mean total charge was USD 14088 for embolization and USD 7561 for transnasal endoscopic sphenopalatine artery ligation (TESPAL).

7. Patient self cares

Nose picking is a contributing factor in epistaxis. Adults can usually be instructed to be more careful. Children pick their nose at night and this is best controlled by placing a glove or sock over their hands so that they can not pick their nose any longer.

Patients should avoid hot and spicy foods and drink plenty of fluids and strenuous activities, hot showers, and digital trauma. Nasal saline spray can be liberally used. Digital pressure and ice packs are to be used as needed for minor recurrences.

Dry climates predispose to

recurrent nose bleeds. Daily or twice daily application of vasolinated ointment provides substantial benefit.

8. Medications

Pharmacotherapy plays only a supportive role in treating the patient with epistaxis.

See Table 1 for details on use, doses, safety and level of proof. Let us cite:

Adrenochromazone

(Adrenoxy),⁵⁰⁻⁵²

Etamsylate (Dycinone),⁵³

Terlipressin (Glypressine,)

Vinayak et al found a statistically significant benefit compared to placebo with intravenous glypressin in acute epistaxis where no localized point was found and in place of a form of nasal packing.⁵⁴

In an other study, although 50% of epistaxis with not clearly localized lesions did stop bleeding, there was no significant difference in effect with placebo.⁵⁵

Tranexamic acid (Exacyl,)

Oral tranexamic acid is of no proven value as an adjunct in the treatment of epistaxis in patients requiring hospital admission concerning severe rebleeds. But the gel seemed to have a beneficial effect.^{56,57}

9. Complications of treatments

1. Cautery

Synechia, septal perforation

2. Anterior packing

Synechia, rhinosinusitis, toxic shock syndrome, eustachian tube dysfunction

3. Posterior packing

Synechia, rhinosinusitis, toxic shock syndrome, eustachian tube dysfunction, dysphagia, scarring of nasal ala and columella, hypoventilation, sudden death

4. Transantral internal maxillary artery ligation

Anaesthetic risks, rhinosinusitis, oroantral fistula, infraorbital numbness, dental injury

5. Transoral internal maxillary artery ligation

Anaesthetic risks, cheek numbness, trismus, tongue paresthesia

6. Anterior/posterior ethmoid artery ligation

Anaesthetic risks, rhinosinusitis, lacrimal duct injury, telecanthus

7. Embolization

Facial pain, trismus, facial paralysis, skin necrosis, stroke, hematoma

10. Prognosis

The prognosis is good but variable. With adequate supportive care and control of underlying medical problems, most patients may not experience any rebleeding. Others may have minor recurrences that resolve spontaneously or with minimal self-treatment. A small percentage of patients may require repacking or more aggressive treatments. Patient with Osler-Weber-Rendu disease tends to have multiple recurrences regardless of the treatment modality. Factors predicting rebleeding are age, prior hypertension, anticoagulant use, vital signs, type of posterior packing (gauze or balloon), prior severe posterior epistaxis and pack removal within 48 hours after admission.⁵⁸

At a first sight rebleeding is not driven by whether the treatment was surgical or not. The rebleeding rate was 33% after embolization, 33% after endoscopic cautery and 20%

after ligation in a study by Barlow *et al.*⁵⁹

11. Patient education

Use nasal saline spray
 Avoid hard nose blowing or sneezing
 Sneeze with the mouth open
 Do not use nasal digital manipulation
 Avoid hot and spicy foods
 Avoid taking hot showers
 Avoid aspirin and other NSAIDs, avoid local nasal sprays of corticosteroids for a while when possible
 Self treatment for minor epistaxis: firm digital pressure on the nasal pyramid for 5-10 minutes, use of an ice pack

12. Lab studies

Lab tests to evaluate the patient's condition and underlying medical problems may be ordered depending on the clinical picture at the time of presentation.

If the bleeding is minor and not recurrent, then a lab evaluation may not be needed.

If bleeding is recurrent or severe, studies to evaluate the fluid status, coagulation profiles, and relevant systemic diseases are needed. These may include a complete blood count (including platelet

Table 2

Short guide to interpret PT and PTT results

PT	PTT	Acquired Disorder	Congenital Disorder
Long	Normal	Liver disease or vitamin K deficiency	Factor VII deficiency
Normal	Long	Lupus anticoagulant Acquired factor VIII inhibitor	Factor VIII (hemophilia A), IX (hemophilia B), or XI deficiency
Long	Long	DIC, liver disease	Fibrinogen, prothrombin, factor V or X deficiency
Normal	Normal	Thrombocytopenia, qualitative platelet disorder	Mild factor deficiency, mild von Willebrandt disease, factor XIII deficiency

count), prothrombin time, activated partial thromboplastin time, and a chemistry panel (including liver function tests). Other more specialized studies, such as bleeding time and various assays for coagulation factors and platelet function, may be considered if warranted.

These tests are to be performed in the absence of anticoagulant therapy but when bleeding suggests a coagulopathy. When results are out of range it could be suggested to address to a hematologist for further advices, investigations and treatments (See Table 2).

a) Prothrombin time (PT)

When increased, deficiencies of the "extrinsic" and "common" factors prothrombin, fibrinogen, V, VII, or X are suspected.

b) Partial thromboplastin time (PTT)

When increased, deficiencies of the "intrinsic" and "common" factors prothrombin, fibrinogen, V, VIII, IX, X, or XI are suspected. Deficiencies of the "contact" factors XII, Fletcher, or Fitzgerald fac-

tors also prolong the PTT but are *not* associated with bleedings.

13. Imaging studies

CT scanning and/or MRI may be indicated to evaluate the surgical anatomy and to determine the presence and extent of rhinosinusitis, foreign bodies, benign tumours and neoplasms.

Medico-legal pitfalls

1. Failure to recognize the severity of the bleeding, especially in patients with posterior epistaxis in whom most of the blood may be swallowed.
2. Failure to diagnose serious etiologies (e.g., neoplasm, aneurysm, systemic coagulopathies).
3. Failure to prescribe antibiotic therapy to prevent rhinosinusitis and possibly toxic shock syndrome in patients with nasal packing either anterior or posterior or both.

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- Prof. Bernard BERTRAND
ENT and HNS Dept.
Cliniques universitaires UCL de Mont-Godinne
avenue Thérassé, 1, B-5530 Yvoir, Belgium.
E-mail: bernard.bertrand@orlo.ucl.ac.be

CME questions

1. The sphenopalatine artery enters the nose through the sphenopalatine foramen which is situated :
 - A - 10 mm dorsally to a theoretical line drawn between both the posterior attachments of the superior and middle turbinates
 - B - 10 mm dorsally to a theoretical line drawn between both the posterior attachments of the inferior and middle turbinates
 - C - 30 mm dorsally to a theoretical line drawn between both the posterior attachments of the inferior and middle turbinates
 - D - 2 mm – 9 mm anterior to the optic canal
 - E - on the lower third of the septal cartilage

2. Hereditary hemorrhagic telangiectasia is characterized by ... except one
 - A - autosomal dominant disease
 - B - formation of telangiectasia and arteriovenous malformation
 - C - high incidence of angiofibroma
 - D - a lack of elastic or muscular tissue in the vessel
 - E - multisystemic involvement

3. Endoscopic sphenopalatine artery ligation can be performed ... except one
 - A - for posterior epistaxis
 - B - to ligate sphenopalatine artery
 - C - to ligate ethmoid anterior artery
 - D - after a mucosal incision on a line just posterior to the posterior attachments of the middle and lower turbinates
 - E - under general anaesthesia

4. Nasal packings require
 - A - antihypertensive agents
 - B - thrombin
 - C - tranexamic acid
 - D - mupirocin
 - E - oral antibiotherapy

5. A 12 year old boy complains of nasal obstruction and epistaxis. The demographics and symptoms are most typical of :
 - A - Choanal polyp
 - B - Cystic fibrosis
 - C - Juvenile nasal angiofibroma
 - D - Nasopharyngeal sarcoma
 - E - Osler-Weber-Rendu disease

6. To determine which patients will have coagulopathy associated with epistaxis, the most complete and cost-effective method is :
 - A - CBC, platelet count, and bleeding time
 - B - platelet count
 - C - bleeding time

- D - history, prothrombin time and partial thromboplastin time
- E - careful history

7. Which one of the following arteries must not be embolized when treating epistaxis ?

- A - Anterior ethmoid artery
- B - Sphenopalatine artery
- C - Internal maxillary artery
- D - Facial artery
- E - Descending palatine artery

8. Which one of the followings signals the presence of a traumatic aneurysm ?

- A - Severe anterior epistaxis
- B - Facial trauma
- C - Fever and vomiting
- D - Delayed epistaxis
- E - Out of range results for PTT

9. In case of recurrent epistaxis PT normal and PTT long do not suggest one of the following coagulopathies

- A - Acquired factor VIII inhibitor
- B - Factor VIII deficiency (hemophilia A)
- C - Factor IX deficiency (hemophilia B)
- D - Factor XI deficiency
- E - Factor VII deficiency

10. Which one of these medical adjunctive therapies is granted of a Ia level of proof ?

- A - Adrenochromazone
- B - Etamsylate
- C - Terlipressin
- D - Tranexamic acid
- E - None of the above

Answers: 1B; 2C; 3C; 4E; 5C; 6D; 7A; 8D; 9E; 10E