

EPISTAXIS


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DEFINITION

- Acute hemorrhage from the nostril, nasal cavity, or nasopharynx.

Introduction

- One of the most common ENT emergency
- Male to female ratio 1.6:1
- Higher incidence in older patients
- Clinically- bleeding either from the lateral nasal wall or from the septum.

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- Lateral nasal wall bleeding is usually seen from the region of the sphenopalatine artery
 - Septal bleeding is usually from the anterior region.
 - Most cases are minor but can be life threatening e.g. in elderly

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- ❑ Minor epistaxis usually originates from the anterior nasal septum
 - ❑ Is often the result of minor trauma to the septal mucosa.
 - ❑ Children- A result of nose picking
 - ❑ Adults- A result of desiccation of the mucosa.

VASCULAR ANATOMY OF THE NOSE

- The nasal mucosa has a rich arborizing network of submucosal vessels.
- Arterial blood supply from internal and external carotid arteries.
- Confluence of the two systems occurs particularly at the caudal end of the septum.
- A number of arteries anastomose with each other- Little's area.

- The anterior septal plexus is termed Little's area or Kiesselbach's plexus
- Is a confluence of
 - Septal branch of sphenopalatine (Ext. Carotid artery)
 - Septal branch of superior labial artery (Ext. Carotid artery)
 - Greater palatine artery (Ext. Carotid artery)
 - Anterior ethmoidal (Int. Carotid artery)
- This is the site of most anterior epistaxis

- **Retrocullumellar vein** runs 2mm parallel and behind the collumellar
 - Is superficial
 - Is a common reason for venous bleeding in children
- Venous epistaxis from retrocolumelar vein tends to occur in subjects <35yrs
- Venous epistaxis usually short lived


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- **Woodruffs plexus** is a plexus of vessels lying inferior to the posterior end of the inferior turbinate.
 - ⊠ It is a frequent site of adult epistaxis.
 - ⊠ It causes a venous posterior bleed.
 - 70% of the bleeding occurs from the septum

Fig 1: Vascular supply of the nasal septum

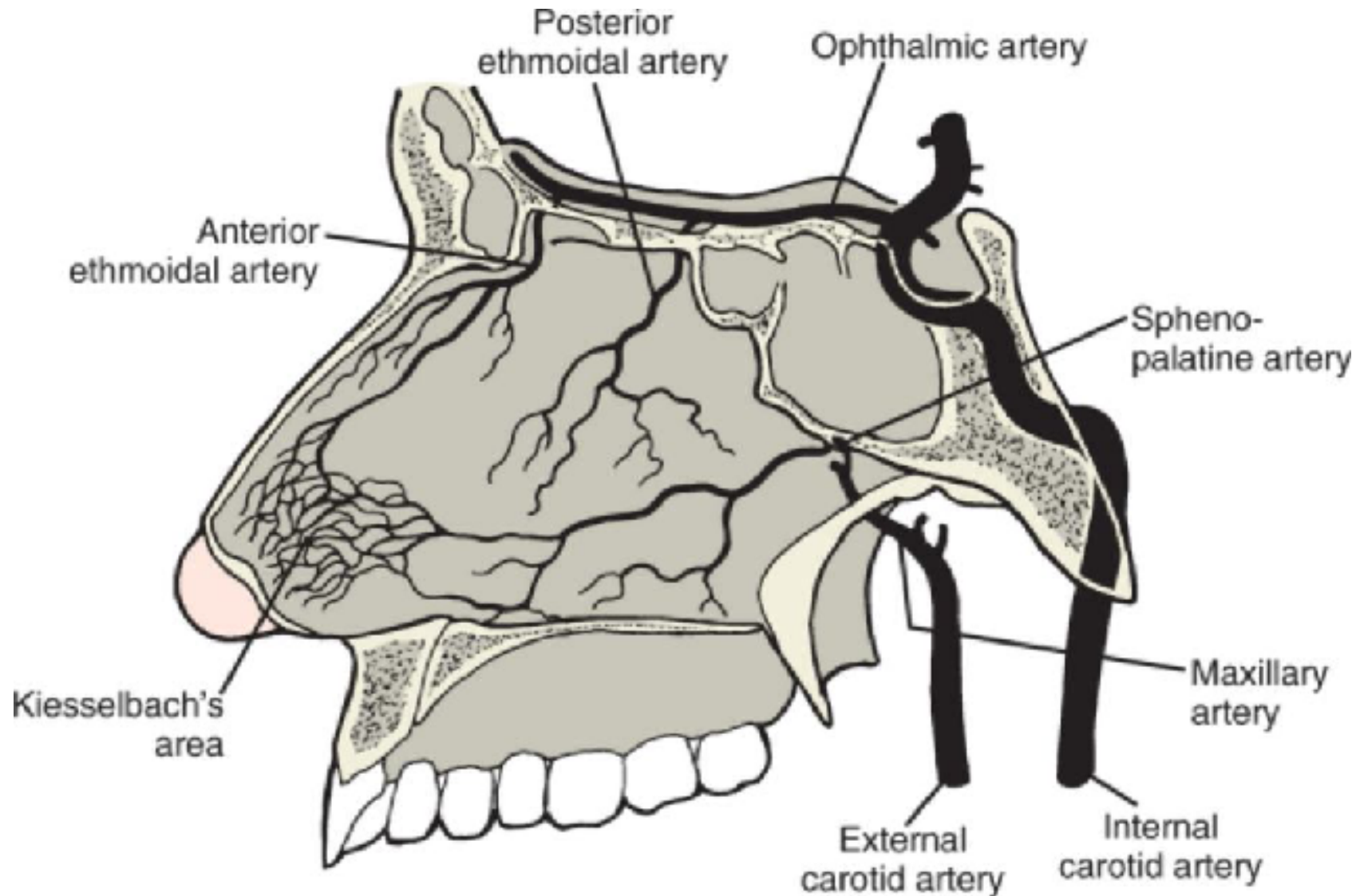
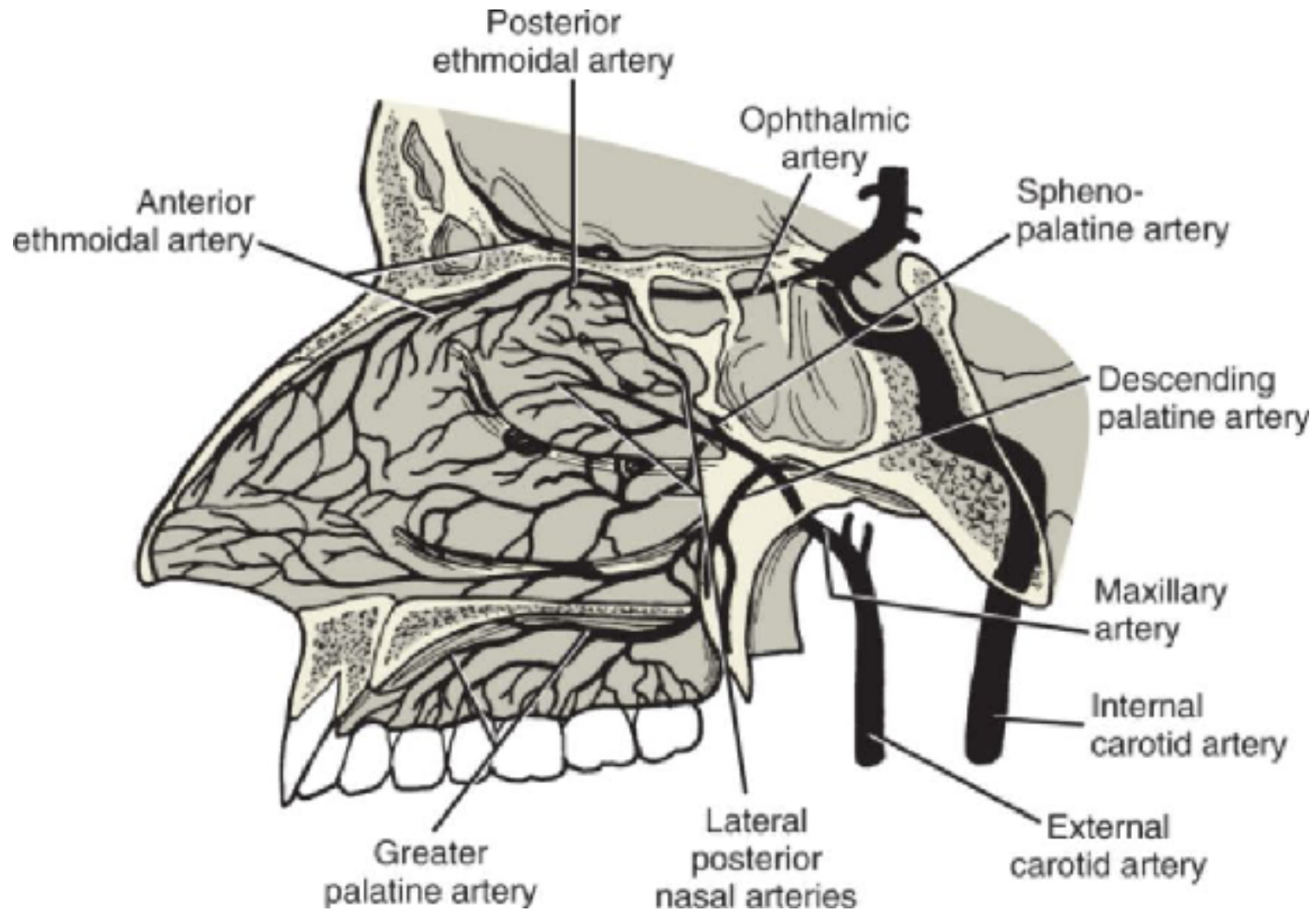


Fig 2: Vascular supply of the lateral nasal wall



AETIOLOGY

- Primary and Secondary epistaxis
- 70 - 80% of all cases are idiopathic : **Primary epistaxis.**
- Standardised description:
 - Anterior: Bleeding from a source anterior to the plane of the piriform aperture (anterior septum, vestibular skin, mucocutaneous junction.)
 - Posterior: Bleeding posterior to the piriform aperture
- May be multifactorial, with each factor playing a minor role.

LOCAL CAUSES

1. Trauma

Nose picking, facial trauma, RTA, fracture base of skull.

2. Idiopathic (from Little's area)

3. Inflammatory

Rhinitis (infective, allergic), Sinusitis, Specific nasal infections (bacterial, fungal, TB)

4. Anatomical/ structural deformities of the nose

- congenital or acquired
- deviated nasal septum
- Nasal spur
- Hypertrophied or rotated turbinates (paradoxical) drying, crusting, bleeding

5. Neoplastic (Benign or malignant)

- in the nose or paranasal sinuses and postnasal space tumours
- Juvenile angiofibroma (exclusively in the adolescent males, recurrent and severe episodes of epistaxis. Never biopsy since patient will bleed excessively)
- Aneurysms of internal carotid artery

6. Enviromental;

- high altitude
- air conditioning
- toxic or chemical irritant
- Cold winter weather

7. Foreign bodies;

- Unilateral, purulent nasal discharge and bleeding.
- Usually in children or the mentally retarded

8. Iatrogenic;

- excessive prescription of intranasal topical steroids
- can lead to changes in mucosa and bleeding
- After nasal surgery. (septoplasty, FESS)

GENERAL CAUSES

9. Hypertension - associated with local factors

- ⊠ elderly; arteriosclerotic vessels do not contract well and the nasal mucosa becomes atrophic hence dries up and cracks easily and vessels may rupture especially during a hypertensive episodes

10. Blood dyscrasias

- ⊠ vary in ability to cause epistaxis
- ⊠ usually diagnosed in early life by excessive bleeding after minor trauma.
- ⊠ Deficiency of factors VIII (Hemophilia A), (Hemophilia B). Factor IX
- ⊠ Von willebrands factor VII impaired PLT adhesiveness.
- ⊠ Leukemia, lymphomas, Idiopathic thrombocytopenic purpura ITP, osler Rendeu weber syndrome.

11. Alcohol abuse - poor diet especially Vitamin C, K deficiency

12. Parenchymal liver damage (decreased fibrinogen and prothrombin)

13. Pregnancy especially folic acid deficiency (decreased platelets)
14. Drugs (anticoagulants, aspirin, Nsaids, CAF, carbenicillin).
15. Systemic toxic agents- phosphorous, mercury.
16. Infectious diseases (Typhoid, rheumatic fever, whooping cough)
17. Cardiovascular disorders (MS, CHD, CCF, COA)
18. Immuno-suppression. (HIV)
19. Allergic diseases
20. Malnutrition

CLINICAL PRESENTATION

- ❑ Sudden onset
- ❑ Occasionally preceding headache
- ❑ May be unilateral,
- ❑ Smell of blood in the throat, trickling in the throat
- ❑ Swallow and vomit fresh blood
- ❑ Anxiety (increased PR, BP) increased bleeding
- ❑ Elderly decompensate very fast (hypovoleamic shock)

MANAGEMENT



1. Medical history
2. Physical examination
3. Laboratory investigations
4. Radiological investigations
5. EUA/ Endoscopy

GENERAL MANAGEMENT


- ABC
 - Depends on the degree of haemorrhage,
 - site (ant, post),
 - Age of patient,
 - hx of precipitating factors.
- An accurate patient history (location, duration and frequency), trauma, nasal blockage, rhinorrhea.
- Family history, drug history, tobacco and alcohol usage
- History of prior bleeding is important, general state of the patient (eg shock)
- Blood for GXM, coagulopathy

Assessment of blood loss

- Class I 10-15% of total blood volume (minimal blood loss <700ml)
- Class II 15-30%
- Class III 30-40%
- Class IV >40% (>2000ml)

□ **Rules of fluid replacement:**

- Crystalloid fluid= 3:1
- Colloids fluids= 1:1
- The patient is evaluated in the seated position,
- Adequate light suction anaesthetic solution,
- Packing materials and cautery.
- Topical vasoconstrictor and anaesthetic agent.
(oxymetazoline and xylocaine)
- Most bleeding sites are anterior and accessible to local treatment.
- Bleeding sites that are not visible on anterior rhinoscopy most likely from posterior (sphenopalatine artery)

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- ❑ Trivial haemorrhage- first aid measures.
 - ❑ Mild-moderate (patient may develop shock)
 - ❑ Main aim is to stop haemorrhage.
 - ❑ Firm pressure to the nostrils 5-10 mins seated upright, head facing downwards.
 - ❑ Advice the patient to breath thru the mouth.
 - ❑ Arrange for good light, nasal cannula and suction machine.
 - ❑ anaesthetic agent + vasoconstrictor in solution

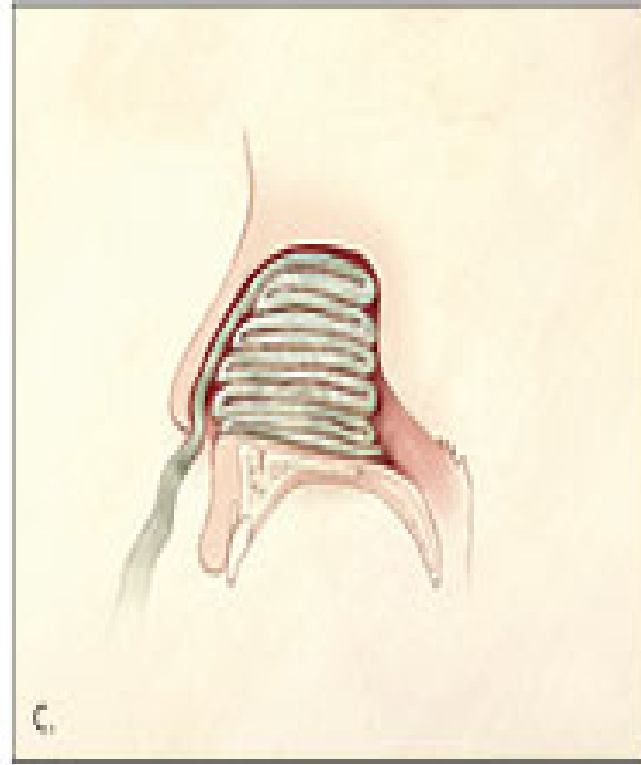
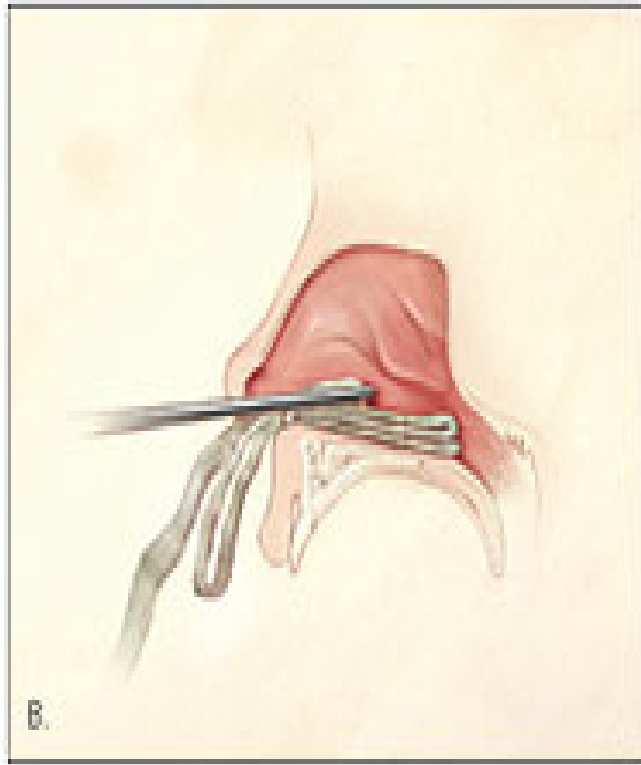
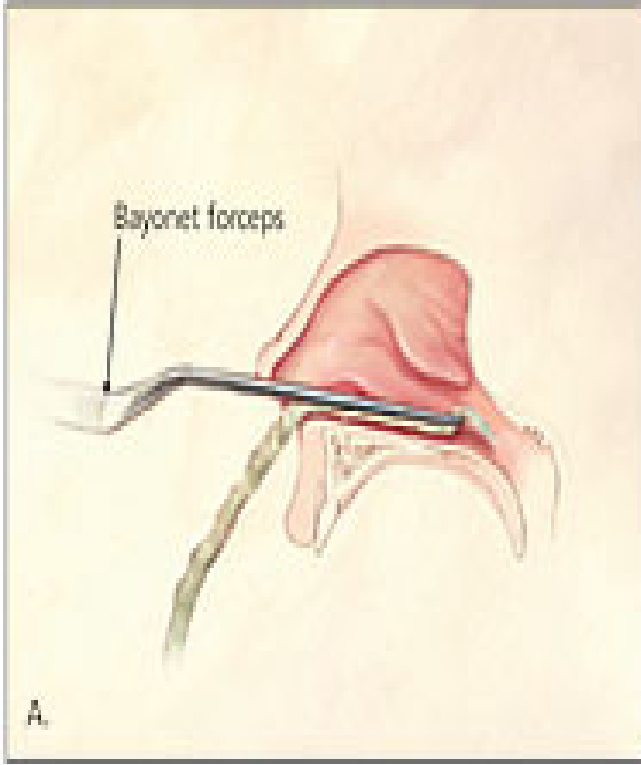
NON-SURGICAL MANAGEMENT

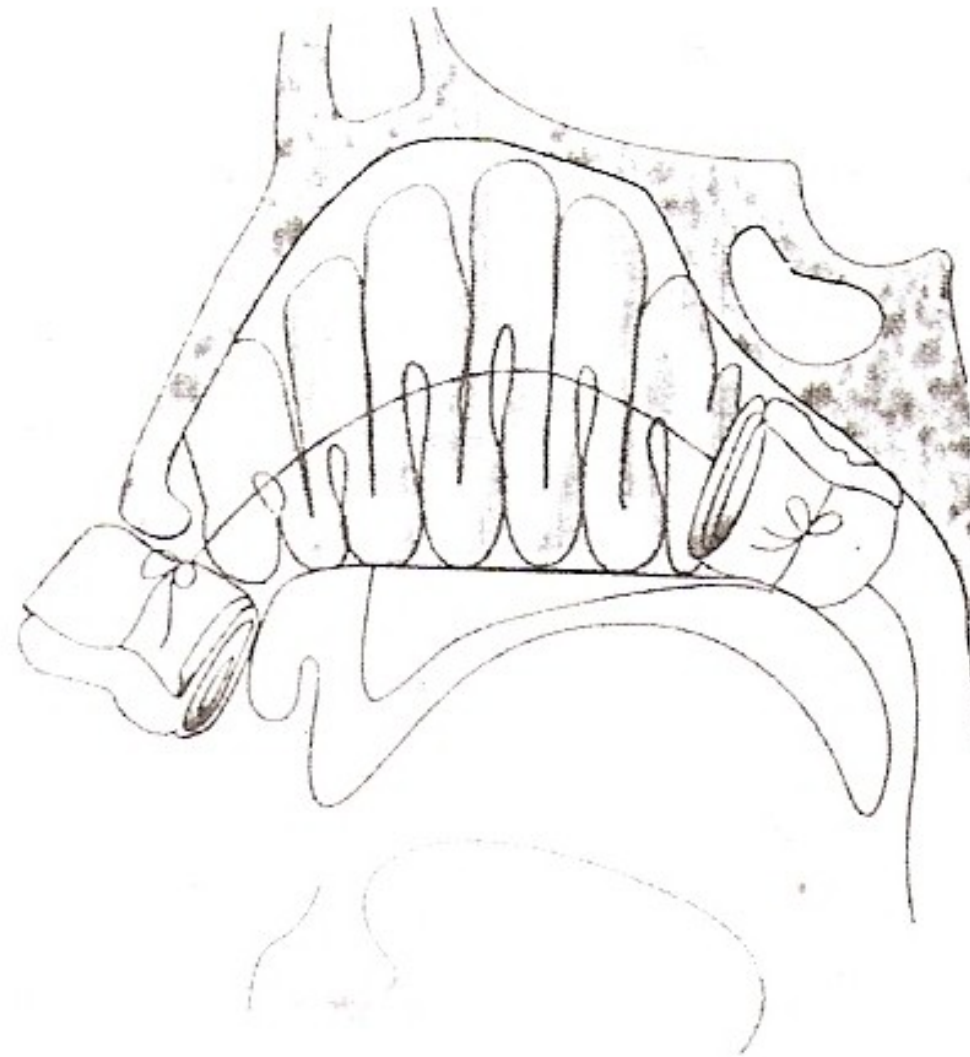
- Anterior nasal packing
- Posterior nasal packing
- Local cautery with silver nitrate
- Endoscopic guided cautery
- Posterior packing

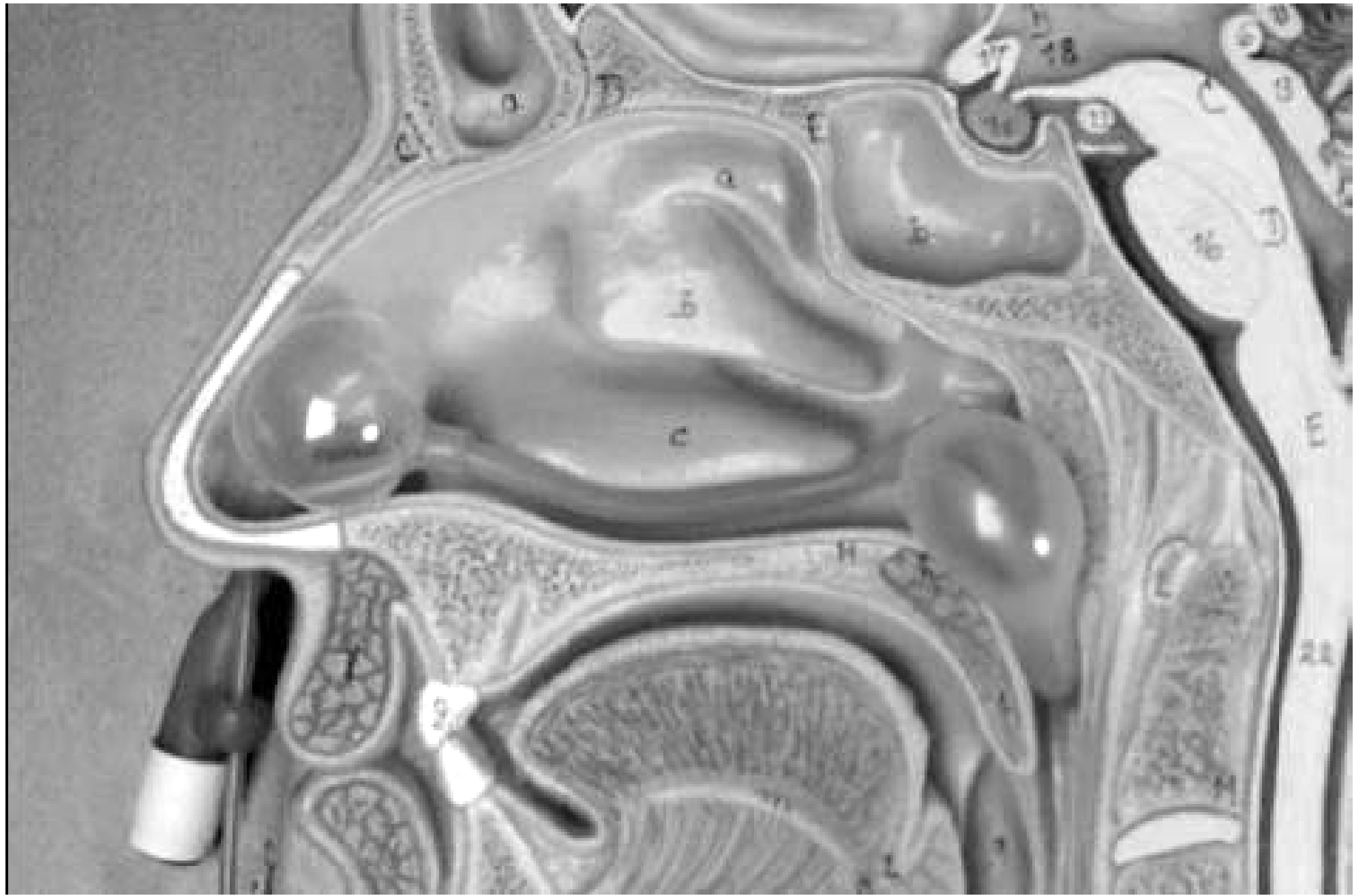
Nasal packing

- ⊠ Anterior- ribbon gauze impregnated with petroleum jelly or bismuth iodoform paraffin paste (BIPP).
- ⊠ Left in situ for 24 to 72 hrs.
- ⊠ Complications include sinusitis, septal perforation, alar necrosis and hypoxia.
- ⊠ There are special nasal tampons and ballon catheters.
- ⊠ Posterior- Under GA preferably. Can also use foleys catheter
- Hot water irrigation at 50 degrees (activates clotting system)
- Systemic medications- Tranexamic acid , inhibits fibrinolysis





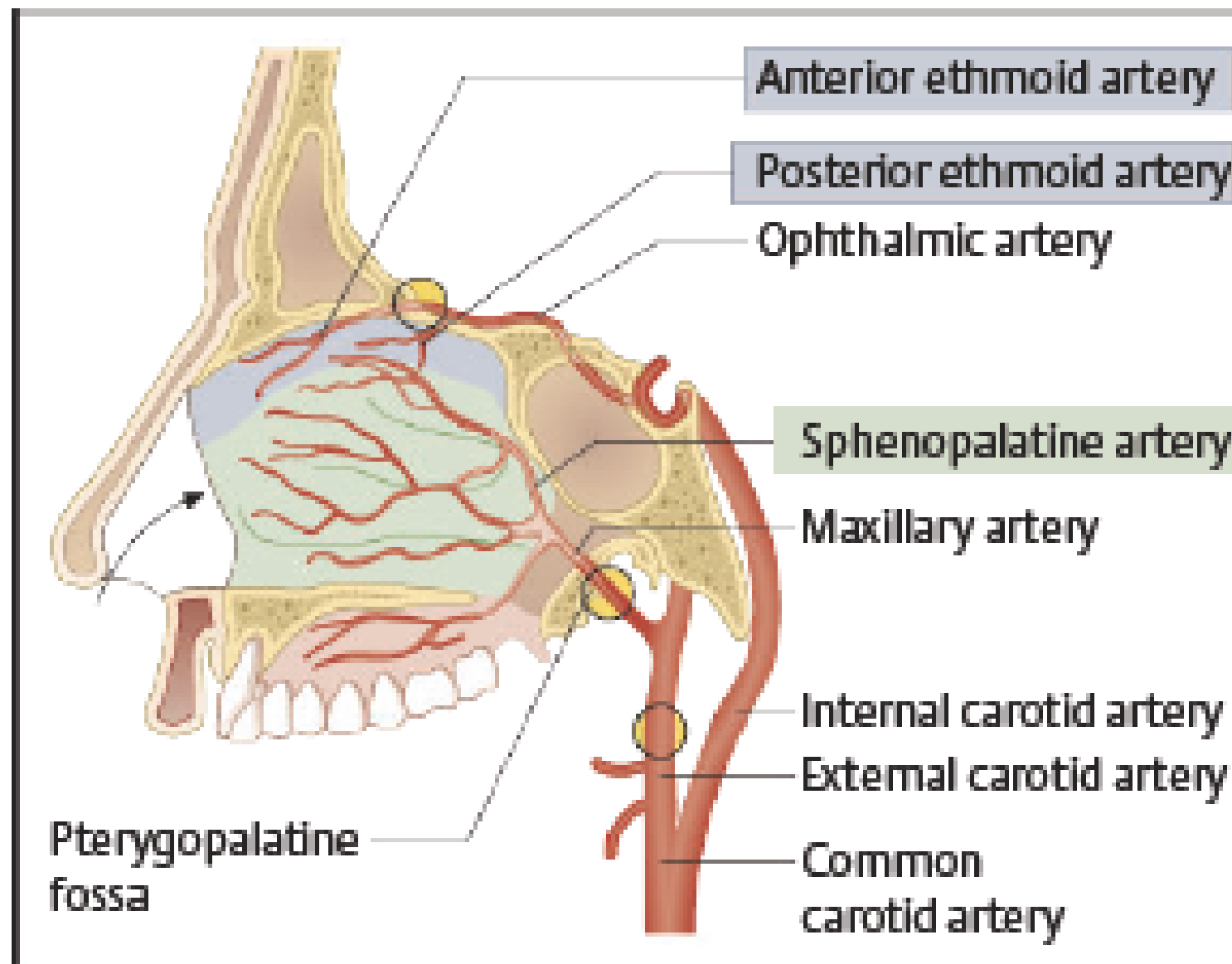




SURGICAL MANAGEMENT

- In cases of intractable bleeding ligation of arteries is performed
 - Sphenopalatine artery
 - Maxillary Artery
 - External carotid artery
 - Ant and Posterior ethmoids.
- Embolization with the use of polyvinyl alcohol foam

Fig. 3.8 Vascular ligation for severe epistaxis



Depending on the bleeding source, various vessels can be ligated through a cervical approach, by the transnasal endoscopic route, or by a transmaxillary route in the pterygopalatine fossa.

