



NOSE III - IV

Objectives:

- Know the causes, clinical manifestations and management of acute and chronic sinusitis.
- Know about fungal sinusitis in brief.
- Know the classification and management of sinusitis complications, in addition to the investigation and general management of orbital complications.
- Be familiar with the role of radiology in sinusitis.

[Color index : **Important** | **Notes** | Extra]

Resources: Slides+Notes+Lecture notes of ENT+433 team.

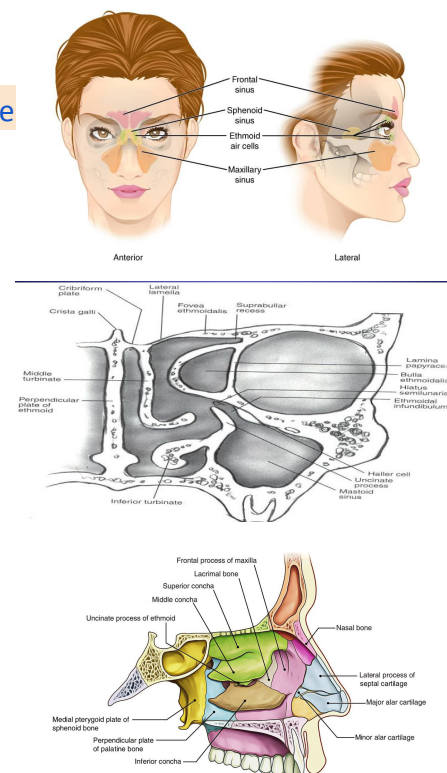
Done by: Saleh Alshawi, Suha alenazi, Razan AlSabti

Edited by: Sara Alkhalifah

Revised by: Adel Al Shihri, Lina Alshehri.

Anatomy:

- four pairs of paranasal sinuses: sometimes it's 3 pairs because the frontal sinus is anaplastic or hypoplastic
- lateral nasal wall: ostiomeatal complex:
- The importance of the nasal wall that all sinuses drain through it except sphenoid.
- All sinuses drain in the middle meatus "ostiomeatal complex" except sphenoid (sphenoethmoid recess area) and posterior ethmoid (superior meatus)
- Nasolacrimal duct in the inferior sinuses
- Histology: pseudostratified columnar ciliated epithelium



Introduction:

- Nasal infections are common cold (<7 days), acute sinusitis (10 days-3 months) and chronic sinusitis (> 3 months).
- All are an infection within the mucosa of the nasal cavity and paranasal sinuses, the difference between them lies in the **duration** and **some symptoms**.
- Generally they all present with same symptoms **PODS**:
 - Pain or facial pressure.
 - Obstruction.
 - Discharge (which is thick, purulent and sticky).
 - Smell.
 - Discharge can be either anterior rhinorrhea (from anterior nostril) or postnasal drip (expelled by the mouth or swallowed).
- These symptoms are different from Allergic rhinitis symptoms which are absent in common cold and sinusitis. (Sneezing, Itchiness and Runny nose "a term used to describe thin watery and frequent nasal discharge")

◆ Common cold:

- Very common, affects almost any person in life.
- Lasts for **less than 7 days**.
- Usually the cause is **viral** (Rhinovirus, Influenza A/B virus, parainfluenza virus, RSV).
- It gets better by time (worst symptoms are in first day then it gets much better by the last day), if it becomes better but then drop again (double peak or "worsening after initial

improvement"), it is considered as acute sinusitis even if less than 10 days.

- Why is this important? Because **management** will differ. Common cold is not managed by Antibiotics, rather you only advise the patient to rest, drink large amount of fluids and use analgesics and decongestant if needed.

Sinusitis:

- **Pathophysiology of Rhinosinusitis:**
 - Most important pathologic process in disease is **obstruction of natural ostia**.
 - Obstruction leads to hypooxygenation.
 - Hypooxygenation leads to **ciliary dysfunction and poor mucous quality**. **collection of secretion**
 - Ciliary dysfunction leads to retention of % Bacterial

Acute	the persistence of upper respiratory symptoms for greater than a 7--day course but lasts less than 3 weeks .
Subacute	nasal symptoms lasting 4 weeks to 12 weeks.
Chronic	persistence mucosal inflammation for > 12 consecutive weeks despite medical therapy or occurrence of more than 4 episodes a year. could be with or without nasal polyp

❖ Etiology:

Inflammatory most common	<ul style="list-style-type: none"> - URTI - Allergy
Mechanical	<ul style="list-style-type: none"> - Nasoseptal Deformity. - OMC Obstruction - Turbinate Hypertrophy - Polyps - Tumours - Large Adenoid - Foreign Bodies - Cleft Palate - Choanal Atresia Etiology
Systemic Disease	<ul style="list-style-type: none"> - Cystic Fibrosis - Immotile cilia Syndrome (Kartagener's Syndrome)
Miscellaneous	<ul style="list-style-type: none"> - Swimming - Flying - Diving

❖ Acute rhinosinusitis:

usually preceded by URTI

- **Inflammation of the mucosal lining** of the nasal cavity and paranasal sinuses that lasts for more than **10 days and less than 3 months**.
- It affects huge number of people worldwide and has an impact on their life.
- **Women** are affected more than men (Some studies accounted that women deal with children more than men and thus they are more exposed to microorganisms).

→ **Streptococcus pneumonia 20-30%**

→ **Haemophilus influenzae 15-20%**

→ **Moraxella catarrhalis. 16-20%**

- **Streptococcus Pyogenes 2-5 %**
- sterile 20-35%
- Anaerobes 2-5%
- Rare viruses (More in common cold), anaerobes, Staphylococcus
- Normal flora in the sinus -- controversy

Infection lasting less than three months with more severe symptoms. The most common cause of acute sinusitis is a **viral infection associated with the common cold**. Bacterial sinusitis occurs much less commonly, in only 0.5 to 2 percent of cases, usually as a complication of viral sinusitis. **Nasal sinus become infected whenever there is a blocked drainage introduced by allergy, infection, immunosuppression, or ciliary dysfunction**

→ Predisposing factors

1. **Nasal obstruction** by nasal polyps, tumors, mucous plug, edema, septal deviation or head trauma causing blockage of sinonasal pathway.
2. **Ciliary dysfunction** (Primary ciliary dyskinesia) like in Kartagener's syndrome.
 - ★ Both (obstruction & ciliary dysfunction) will result in stagnation of nasal secretions, creating a good environment for the bacteria to grow.
3. **Altered quantity or quality of the nasal mucous** (That's why patients with sinusitis are advised to drink large amounts of fluids to increase the quantity and to correct the quality of the mucous to be thin and excretable).
 - ★ This is commonly caused by dehydration (common in elderly) and cystic fibrosis (in which, mucous is thick and poorly discharged, almost 99% of cystic fibrosis patient will encounter an episode of sinusitis in their life).

→ History

- **Symptoms:** (PODS)
 - **Pain:** Ask about the site to know which sinuses are affected and to exclude other causes of upper facial pain and pressure e.g. Migraine)
 - **Obstruction:** Ask whether unilateral or bilateral (Each has a list of differentials).

- **Discharge:** Ask about thickness, consistency, color, amount, frequency Acute Sinusitis and if anterior or Posterior (post nasal discharge).
- **Decreased in smell sensation** (Anosmia "Complete" or Hyposmia "Partial").
- **Systemic symptoms:** **fever**, fatigue and muscle pain.
- **Ear symptoms:** patients with acute sinusitis may present with otitis media due to Eustachian tube dysfunction secondary to sinusitis.
- **Dental issues** (Especially if unilateral symptoms) (e.g. a patient presents with symptoms of acute sinusitis due to tooth extraction and spread of organisms "Usually anaerobes" from the tooth origin to maxillary sinus all the way to the other paranasal sinuses causing acute sinusitis). (In this case the Treatment is: Metronidazole or Clindamycin).
- **Visual and neurological symptoms:** symptoms of sinusitis complications (Will be discussed later).
- **Duration** (10 days – 3 months) Immune status (Be more aggressive in the treatment with immunocompromised patients).
- **it cause severe headache and aggravated by movement**

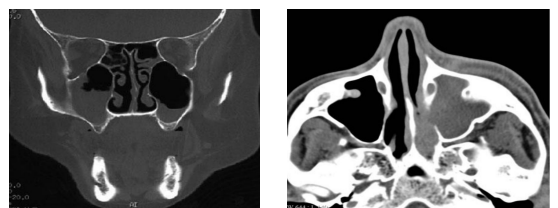
→ Examination

- Fever, facial edema, erythema and tenderness around the nose.
- Using a speculum to inspect the nose from inside or by a Rhinoscope: signs of inflammation (redness, swelling and discharge).
- Look at any cause of obstruction or deviated nasal septum.
- Sometimes, brief look at the oral cavity to see the teeth is important if you suspect dental origin of infection.

→ Investigations

- It depends on how bad the disease is, sometimes no investigations are required at all.
- If the patient is really sick, do: **CBC, ESR.**
- Culture: only done if the patient had been given antibiotics and didn't improve, or if you suspect uncommon microorganism.
- CT scan, when you suspect something serious (e.g. Meningitis, like when the patient reported photophobia).

- CT shows: Mucosal thickening, fluid filled sinuses and soft tissue density



→ Treatment

- The initial treatment aims to relieve the symptoms, since almost everyone **will improve within 7--10 days**. At this stage, antibiotics can only be used if there is clear evidence of severe bacterial infection.
- So as an initial treatment, we can give acetaminophen or ibuprofen for the pain, **flushing the nose and sinuses** with a saline solution to decrease pain associated with nasal

congestion, and **nasal decongestants** to temporarily treat congestion.

- **Nasal decongestants** → (No nasal adverse effects with Systemic nasal decongestant, but local might cause Physiological addiction).
- Antibiotics:
 - 1st line: Amoxicillin, if the patient is penicillin allergic, give Macrolides (Clarithromycin or Azithromycin). **penicillin is the 1st choice**
 - 2nd line (when 1st line treatment fails): Amoxicillin + Clavulanic acid, and if the patient is penicillin allergic, give Fluoroquinolones (Ciprofloxacin or Levofloxacin).
- Analgesics, Decongestant, High fluid intake and Sinus wash.
- Intranasal Corticosteroids. (Help to avoid the progression to chronic sinusitis).

❖ Chronic rhinosinusitis:

- Inflammation of the mucosal lining of the nasal cavity and paranasal sinuses that lasts **more than 3 months**.
- Those patients suffer a lot while nobody can feel or understand their problem, It's one of the diseases that severely affect the quality of life.

→ Predisposing factors

- Long standing nasal obstruction. **polyp , deviated nasal septum**
- Transnasal tube or NG tube that is left for a long time (e.g. in ICU).
- Atopic (Allergic) rhinitis.
- Primary ciliary dyskinesia.
- Poor quality of the mucous.
- Hormonal factors (chronic sinusitis is a common disease in puberty and pregnancy due to hormonal changes).
- Acid reflux (GERD).
- Immunodeficiency.
- Patients with hyper inflammatory status such as Wegener's disease (also called Granulomatosis polyangiitis).
- Dental procedures.
- Churg-Strauss syndrome.

→ Etiology

- Almost always a **bacterial** cause (Staphylococcus aureus, coagulase negative staphylococci and pseudomonas species and less commonly Bacteroides and other anaerobes).
- "Staph Aureus and some other bacteria are able to release what is called Superantigen; in which the immune system is activated aggressively in a nonfunctional way, this is done in order to distract the immune system from the site of infection and deviate it to other sites in the body. When this occurs, immune cells start proliferating to release huge amount of useless antibodies, those patients have Eosinophilia, Hyper IgE".

→ History

- Symptoms: Just like acute sinusitis (PODS).
- There are four cardinal signs/symptoms of CRS in adults:
 - Anterior and/or posterior nasal mucopurulent drainage.
 - Nasal obstruction/nasal blockage/congestion.
 - Facial pain, pressure, and/or fullness.
 - Reduction or loss of sense of smell.
- **No fever** (very important). However they may encounter other systemic symptoms (fatigue, tiredness and muscle pain).
- Ear symptoms.
- Halitosis.
- Dental issues.
- Visual and neurological symptoms.
- Immune status.
- **Duration: more than 3 months.**
- **It's important to ask about cough and exaggeration of asthma** (They are commonly associated with chronic sinusitis).
- It was noticed that when you deal with chronic sinusitis, asthma symptoms improve a lot and the need of asthma medications is reduced dramatically.

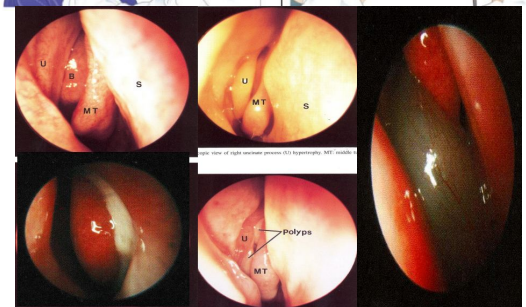
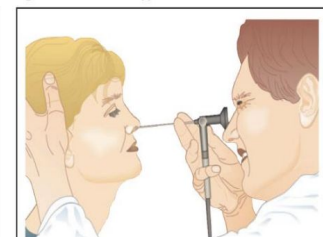
→ Examination

- Nasal exam:
 - Swelling and redness of nasal cavity using a Rhinoscope, you may also see nasal polyps as a predisposing factor to develop chronic sinusitis or as a complication of long standing chronic sinusitis.
 - Endoscopy **Flexible in children and mental retarded, or rigid** Finding:
 - Facial edema, erythema and tenderness around the nose.
 - **most of the time the polyp coming from middle meatus**
 - Brief dental exam.

Fig.2.1 Anterior rhinoscopy



Fig.2.2 Nasal endoscopy



→ Diagnosis of Chronic Rhinosinusitis:

◆ Major Factors:

Facial pain/pressure, Facial congestion/fullness, Nasal obstruction/blockage Nasal discharge/purulence/discolored, postnasal drainage, Hyposmia/anosmia Purulence in nasal cavity on examination, Fever

● Minor Factors:

headache **not a major symptoms only 10% of headache is due sinusitis**, fatigue, halitosis, dental pain, cough, ear pain\ pressure\fullness

→ Strong History of Sinusitis:

◆ One of the following situations:

- Two major factors.
- One major factor and two minor factors.
- Pus in the nose on examination

→ Investigations

- CBC (Eosinophilia, since many patients have chronic sinusitis due to allergic rhinitis).
- Culture (If the patient show no response to the treatment).
- **Radiography:**

- Identify which sinus involved and extent of the disease
- Roadmap for surgery

❖ Plain X Rays:

- Traditional views:
 - Water's: like when you in the water occipitomesital
 - Caldwell : occipitofrontal
 - Lateral
 - Submentovertex:

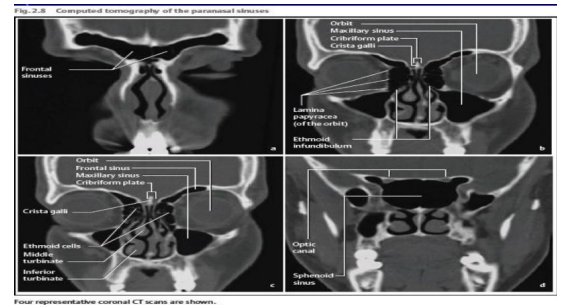
- ❖ CT (standard to be done in chronic sinusitis, to confirm the diagnosis and to assess the severity of the disease, also should be done pre-surgically). if you have alternative other than ct do not do that

➤ Study Type:

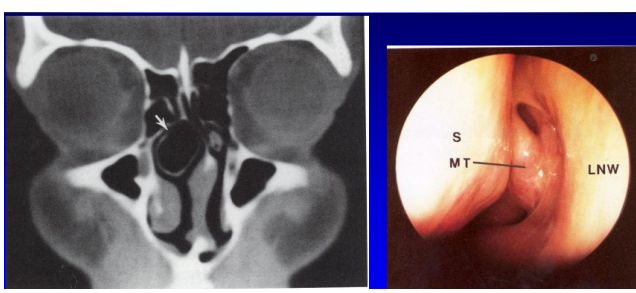
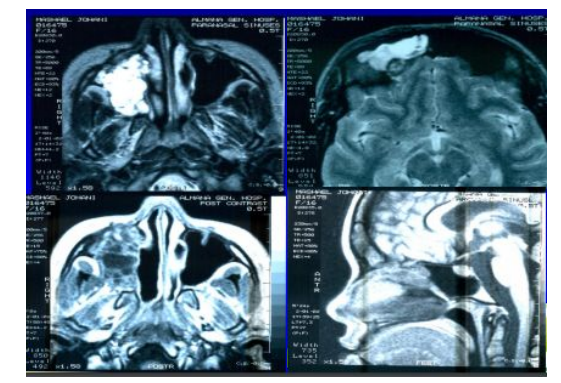
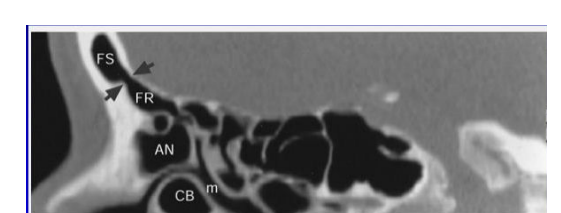
- Coronal perpendicular to the Hard Palate
- Axial Parallel to the Hard Palate
- Reformatted Sagittal
- Multiplanar CT Scan axial and reformatted other cuts

➤ Indications:

- Gold standard for CRS
- Planning surgery or failed medical management
- Clinical unresponsiveness to medical therapy
- Immunosuppressed patient
- Severe symptoms or signs
- Life threatening complications



Four representative coronal CT scans are shown.



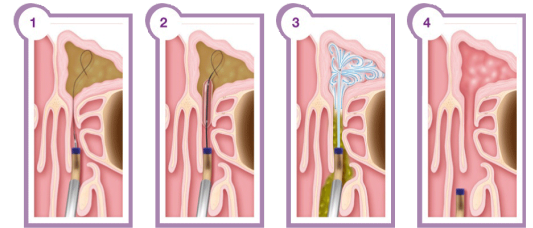
- concha bullosa: is when the middle turbinate is irritated
- paradoxical turbinate :which the turbinate is convex laterlay
- MRI (look for complications). Indicated for Disease Extension
- Others: IgE, ESR, Serology (in case of autoimmune diseases).

→ Treatment

- **Mainstay treatment is local intranasal corticosteroids.**
- Decongestant: topical or systematic. draw back of the topical decongestant: rebound (vasoconstriction, tolerance, rhinitis medicamentosa. so if you need to give it more than two weeks give systematic (Nose II lecture: 5 days)
- Systemic steroids orally (Only given in chronic sinusitis), Steroids may help decreasing polyps

size and improving olfaction.

- **Antibiotics** (same): given for 14 days.
 - 1st line: Amoxicillin, if the patient is penicillin allergic, give Macrolides (Clarithromycin or Azithromycin). from Doctor slides group F (**Penicillin 1st choice , Cephalo 2nd generation**)
 - 2nd line (when 1st line treatment fails): Amoxicillin + Clavulanic acid, and if the patient is penicillin allergic, give Fluoroquinolones (Ciprofloxacin or Levofloxacin).
- Next step is surgical treatment (**FESS¹**) **standard treatment for sinusitis, purpose is irrigate and drain the sinuses** + Steroids, given after the surgery to reduce the inflammatory changes (e.g. scarring) during the process of healing.
 - Excellent results: 71% normal at one year - Meta analysis 89% success - with 0.6% complications
- Computer Assisted Surgery
- Balloon Sinuplasty : **dilate and drain, only in chronic**
- Steam inhalation and nasal saline irrigation may help by **moistening dry secretions, reducing mucosal edema and mucus viscosity.**



Refractory Rhinosinusitis:

- persistent sinusitis in spite of proper medical and surgical treatment. Could be due to:
 - Allergy
 - Immunodeficiency
 - Cystic fibrosis
 - Ciliary dyskinesia disorders
 - **Gastroesophageal Reflux Disease**
 - Repeat treatment 2x or 3x over 2-3 Months
 - Obtain CT Scan
- pt come with rhinosinusitis : give 2 week antibiotics if not resolve check the antibiotic and try another course if no improvement do CT**

Complications or severe illness:

- ❖ IV Cefotaxime or Ceftriaxone ❖ Clindamycin

FESS: GOLD STANDARD FOR CHRONIC, ACUTE WHEN THERE IS COMPLICATION

Sinusitis Complication:

Three main categories:

- **Orbital**(60--75%)
- **Intracranial**(15--20)
- **Bony**(5--10%)

Radiography:

- Computed tomography (CT) best for orbit.

¹ Functional endoscopic sinus surgery

- Magnetic resonance imaging (MRI) best for intracranium.

❖ Orbital:

Orbital Complications: “Chandler Criteria”: Based on Eye Acute Infection and their anatomic location

❖ *Routes of spread: arterial - venous-lymphatic- direct.*

Five classifications:

- Preseptal cellulitis: lid edema otherwise normal
- Orbital cellulitis: diffuse edema
- Subperiosteal abscess: usually seen near lamina papyracea
- Orbital abscess: collection within orbit
- Cavernous sinus thrombosis: bilateral

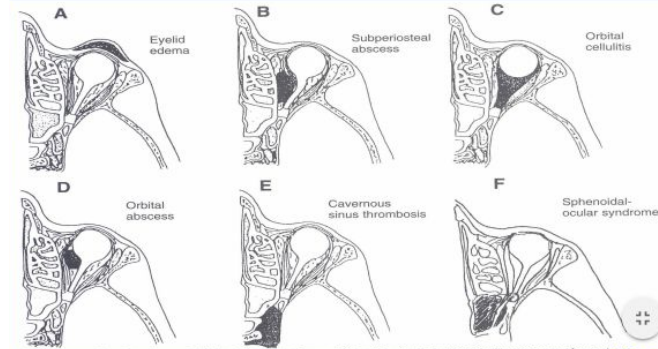


Fig. 4 Classifications of Orbital Complications of Sinusitis-A. Periorbital inflammatory edema (pre-

Grade	Presentation	Number	ARS	AFS	CRS
I Anatomical Disturbance	Proptosis	15(36%)	0	10	5
II Functional Involvement	Epiphoria Diplopia Ophthalmoplegia Ptosis	11(26%)	0	8	3
III Orbital Infection	Orbital cellulitis, Pre septal-cellulitis Orbital abscess Subperiosteal abscess	11(26%)	3	3	5
IV Visual Impairment	Visual Impairment, blindness	5(12%)	1	4	0

❖ Al Anazi & Al Dousary Classification:

- Clinical grading system that doesn't require Imaging
- Encompass Acute orbital infection and chronic Sinogenic pathology causing orbital manifestation.
- Radiologic findings does not correlate well with clinical severity.
- Chronic Paranasal sinus disease in (74 %) of the cases

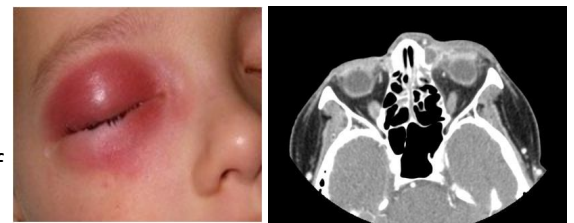
➔ Preseptal Cellulitis: stage 1

- Managed by antibiotics
- Periorbital inflammatory edema
- Obstruction of venous channels
- No vision loss
- No EOM limitation



➔ Orbital Cellulitis: stage 2

- Edema, chemosis, proptosis, pain
- No abscess
- Ophthalmoplegia may occur due to edema or spasm
- No visual loss
- Patients may complain of pain and diplopia and a history of recent orbital trauma or dental surgery.
- Diagnosis of sinusitis complications: based on the symptoms and the CT scan or MRI findings.



→ Subperiosteal Abscess: stage 3

- Globe displaced laterally or downward
- Orbital cellulitis present with decreased EOM
- Vision decreased
- Surgical drainage is indicated if there is worsening of visual acuity or extraocular movement, or in case of lack of improvement after 48 hours. **“Antibiotics then drainage”**
- Approaches :
 - External ethmoidectomy (Lynch incision) is most preferred.
 - Endoscopic ideal for medial abscesses.
 - Transcaruncular approach



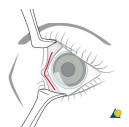
→ Orbital Abscess: stage 4

- Severe proptosis and chemosis
- Usually no globe displacement
- Ophthalmoplegia present
- Visual loss (13%) due to ischemia or neuritis
- Similar approaches as with subperiosteal abscess: **do fess+drainage**
 - Lynch incision.
 - Endoscopic.
- Cellulitis → treat it medically
- Abscess → drainage



→ Cavernous Sinus Thrombosis: stage 5

- Progressive symptoms
- Proptosis and fixation
- CN II, IV, VI
- Meningitis
- High mortality
- Symptoms of Cavernous Sinus Thrombosis :
 - Orbital pain, Proptosis, chemosis Ophthalmoplegia, **Symptoms in contralateral eye,** associated with sepsis and meningitis
 - Radiology: Better visualized on MRI.
- Mortality rate up to 30%. Needs surgical drainage and intravenous antibiotics.
- **Transcaruncular approach does not utilize a facial incision (picture)**



❖ Intracranial Complications :

Five types:

- **Meningitis “the most common”. in children**
- Epidural abscess.
- Subdural abscess.
- Intracerebral abscess.
- Cavernous sinus, venous sinus thrombosis

- Teenagers affected more because of developed frontal and sphenoid sinuses, and because they are more prone to URI's than adults.
- Thrombophlebitis originating in the mucosal veins progressively involves the emissary veins of the skull, the dural venous sinuses, the subdural veins, and, finally, the cerebral veins. By this mode, the subdural space may be selectively infected without contamination of the intermediary structure; a subdural empyema can exist without evidence of extradural infection or osteomyelitis.

❖ Mucocele:

- Mucoceles are chronic, cystic lesions of the sinuses lined by pseudostratified epithelium
- Expand slowly, often requiring many years
- Etiology is debated. Either due to obstruction of ostia or to simple obstruction of minor salivary gland.
- 30% are idiopathic

❖ Bony Complications:

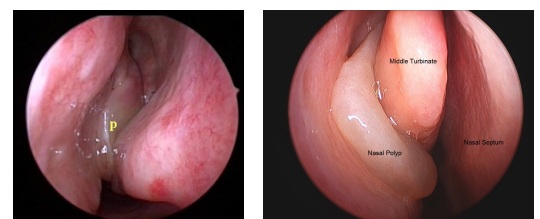
→ Pott's puffy tumor

- Frontal sinusitis with acute osteomyelitis.
- Subperiosteal pus collection leads to “puffy” fluctuance.
- Rare complication.
- High risk of intracranial extension; Rx: parenteral antibiotics, trephination, may require surgical debridement
- Sir Percivall Pott described Pott's Puffy tumor in 1768 as a local subperiosteal abscess due to frontal bone suppuration resulting from trauma. Pott reported another case due to frontal sinusitis.



- Other complications:

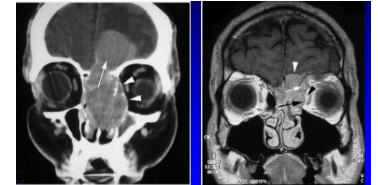
- Osteitis: diagnose initially with technetium bone scan (osteoblastic activity) and gallium bone scan (inflammation), follow with gallium scans; Rx: parenteral antibiotics, surgical debridement, sinus surgery.
- Superior Orbital Fissure Syndrome: fixed globe, dilated pupil (CN III, IV, VI), ptosis, hypesthesia of upper eyelid (CN V1); Rx: urgent surgical decompression
- Orbital Apex Syndrome: similar to Superior Orbital Fissure Syndrome with added involvement of optic nerve (papilledema, vision changes)
- Sinocutaneous Fistula: usually begins as a frontal osteomyelitis
- osteomyelitis (involvement of bone marrow) and Osteitis (no bone marrow)



Fungal sinusitis:

- Fungal rhinosinusitis is a fungal infection of the paranasal sinuses. Fungal colonization of the upper and lower airways is a common condition, since fungal spores are constantly inhaled into the sinuses and lungs.
- ❖ Invasive: involvement of basement membrane, immunocompromised
 - Presence of fungal hyphae within the mucosa, submucosa, bone, or blood vessels of the paranasal sinuses: patients in general are immunocompromised, usually due to diabetes, cancer, HIV, organ transplantation or using systemic or intranasal glucocorticoids.
 - Acute Invasive Fungal Sinusitis usually seen in immunocompromised patients and has a time course of days to few weeks
 - Chronic Invasive Fungal Sinusitis usually seen in patients who are less immunocompromised with a time course greater than 12 weeks.
 - Chronic Granulomatous Invasive Fungal Sinusitis
 - Mucormycosis is encountered in dust and soil and enters through the respiratory tract
 - Ketoacidosis predisposes to mucormycosis, as the fungus thrives in acidic environments
 - Initially seen as engorgement of turbinates, followed by ischemia and necrosis of the turbinates and adjacent nose
 - The fungus invades vascular channels and causes hemorrhagic ischemia and necrosis
 - Frequently fatal. 90% mortality in immunocompromised
 - Patients with acute invasive fungal sinusitis are usually hospitalized and are very sick with fever, cough, nasal discharge, headache, and mental status changes.
 - Signs and symptoms include **dark ulcers on the septum, turbinates, or palate. In the late stages, signs and symptoms of cavernous sinus thrombosis are present.**
 - Patients with chronic invasive fungal sinusitis present with symptoms of long-standing sinusitis. Symptoms are usually not acute, and fever and mental status changes are absent. Orbital apex syndrome, which is characterized by a decrease in vision and ocular immobility due to a mass in the superior portion of the orbit, is usually associated with this condition.
 - **Diagnosis:** early nasal endoscopy with biopsies of affected tissues. Cultures of the affected biopsy specimen are usually positive. Assessing the extent of infection should be done using CT scan or MRI.
 - **Treatment of acute invasive fungal sinusitis:** Initial systemic antifungal treatment after surgical debridement. High doses of **amphotericin B** (1--1.5 mg/kg/d) are recommended followed by oral itraconazole.
 - **Treatment of chronic invasive fungal sinusitis:** **Surgical treatment** is mandatory. Initiate medical treatment with systemic antifungals once invasion is diagnosed. **Amphotericin B** (2 g/d) is recommended; this can be replaced by **ketoconazole** or **itraconazole** once the disease is under control.
 - Examination:
 - ❖ Findings typically is broad
 - ❖ Intranasal inflammation and polyposis
 - ❖ Facial dysmorphism:
 - ❖ Proptosis
 - ❖ Telecanthus
 - ❖ Malar flattening
 - ❖ More often was seen in children than in adults (42% vs 10%)
 - ❖ Orbital Features
 - ❖ Proptosis usually occurs over long periods, no diplopia
 - ❖ Visual loss from AFS caused by compression of the ophthalmic nerve or inflammatory process
- ❖ Noninvasive: Absence of fungal hyphae within the mucosa and other structures of the paranasal sinuses:
 - Fungus Ball (fungus Mycetoma).
 - **Allergic Fungal Sinusitis: common**

- Involves a hypersensitivity response to colonizing fungi.
- Nasal obstruction
- Allergic rhinitis, or chronic sinusitis: Nasal congestion, Purulent rhinorrhea, Postnasal Drainage, or Headaches
- Patients with AFS are atopic, Unresponsive to antihistamines, Intranasal Corticosteroids, and prior immunotherapy
- Patients with AFS always are immunocompetent
- 5-10% of chronic rhinosinusitis patients actually cases of AFS
- Two thirds of patients report a history of allergic rhinitis
- 90% of patients demonstrate elevated specific IgE to one or more fungal antigens.
- 50% of patients in a series by Manning et al had asthma.
- No linkage to aspirin sensitivity has been established.
- pathological extension ->
- The treatment of choice:
 - **Endoscopic debridement (FESS)**
 - A perioperative short course of **steroids**.
 - Postoperative mold containing **immunotherapy** is a promising therapeutic advance in limiting recurrence.
 - The role of systemic **antifungal** therapy is inadequately studied.
 - **Itraconazole** orally is well tolerated and effective in vitro against common causes of AFS.
 - Amphotericin B
 - Treated with radical surgical debridement
- Unilateral Nasal Mass?
 - DDx: ❖ Allergic Fungal Sinusitis ❖ Antrochoanal Polyp ❖ Inverted Papilloma ❖ Carcinoma (**squamous cell carcinoma**)



Disease Of The Nasal Septum:

- The nasal septum is made up of bone and cartilage.
- It can be deviated, perforated, or collapsed.

❖ The Nasal Septum Development

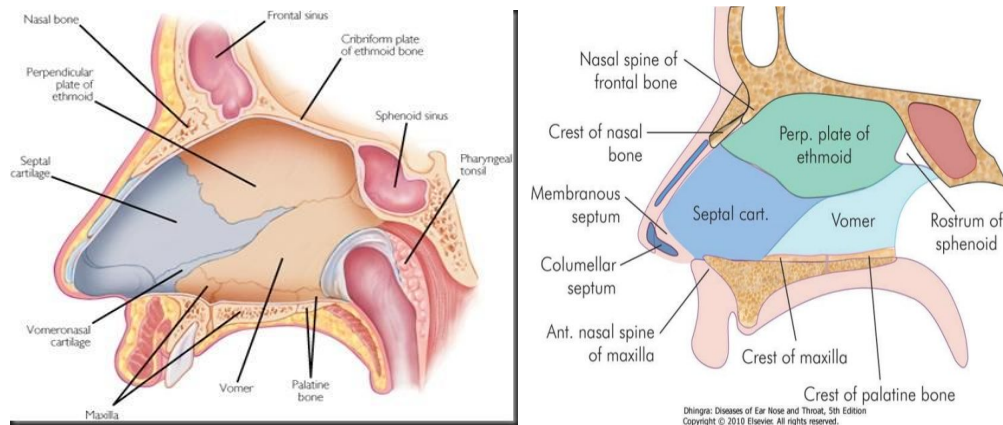
1-Cartilaginous Septum

- Septal (quadrilateral) cartilage
- The vomeronasal cartilages
- Medial crura of the alar (lower lateral) cartilages

2-The Membranous Septum (Mobile Septum)

- Anterior to the end of the septal cartilage. **lies behind the medial crura it gives us mobility and absorb shocks and trauma to the nose**
- It is formed by skin and subcutaneous tissue of the nasal columella.

- The nose is lined by pseudostratified columnar epithelium except the anterior 1cm which is the membranous septum is lined by squamous epithelium
- This membranous septum is lined by skin and hair so it will have skin disease rather than mucosal disease such as :hair follicles inflammation



3-Bony septum

- Composed of two major elements:

1-The Vomer:

- ◀ Develops from connective tissue membrane on each side of the septal cartilage.
- ◀ The intervening cartilage absorbed completed by mid adulthood.

2-The Perpendicular plate of the Ethmoid (Mesoethmoid):

- ◀ Ossification completed by 17th year of age.
- ◀ Replacement of cartilaginous septum with thin bone.
- ◀ At the nasal roof it articulates with the cribriform plate and extends as the crista galli.

◆ Inequality of Growth

- ◀ Creating **septal spur** → Elevations and ridgelike protuberances

◆ Deviated nasal septum

- ◀ Approximately 80 % of humans have DNS
- ◀ Any or all parts of the septum except for the **posterior free border at the choanae**.
- ◀ A common area of deflection is along the **articulation** between the vomer and the perpendicular plate of the ethmoid
- ◀ DNS to one side or S shape to both side
- ◀ The nasal septum is rarely exactly in the midline. Minor deviations are normal and cause no symptoms.

- ◀ **Marked** deviation will cause nasal airway **obstruction** and may contribute to sinonasal pathology by obstructing the normal sinus drainage pathways.
- ◀ Septal deviation can be corrected by surgery, with excellent results.
- ◀ Most cases of deviated nasal septum (DNS) result from **trauma**, either recent or long forgotten, perhaps during birth or childhood. ‘Buckling’ in children may become more pronounced as the septum grows.
- ◀ Maldevelopment → Congenital (considered in etiology in addition to trauma).
- ◀ Nasal surgery, including cosmetic surgery, can cause septal deviation.
- ◀ **Asymmetry of nasal septum can also be caused by:** spurs², crests³, dislocation of quadrangular septal cartilage⁴, buckling.
- ◀ **How to differentiate between DNS and septal spurs?**
 - ◆ **DNS there is concave part and the other part is convex**
 - ◆ **In septal spur there is only sharp projection no concave part is seen**

→ Effects

- Signs & Symptoms:
 - Nasal obstruction. **Not very common. Only in severe deviation.** may be unilateral or bilateral
 - **Nasal narrowing. More common.**
 - External deformity.
 - Crusting, epistaxis (due to dryness) (a sharp spur can be a focus for epistaxis (Fig. 17.2))
 - Recurrent sinus infection due to impairment of sinus ventilation by the displaced septum. **Doctor doesn’t agree with this point “dosen’t accept it”**
- The middle turbinate on the concave side of the septum may hypertrophy and interfere with sinus ventilation.
- Severe deviation is apparent on looking at the nose and septal surgery is an important component of aesthetic nasal surgery (septorhinoplasty).
- Can cause facial pain but this is rare.
- Otitis media. DNS may impair the ability to equalize middle-ear pressure.
- Nosebleeds – a sharp spur can be a focus for epistaxis (Fig. 17.2).

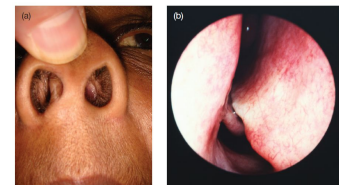
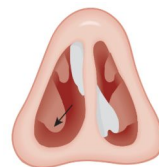


Figure 17.2 (a) Deviated nasal septum and (b) endoscopic view.

→ Diagnosis:

- The diagnosis is mostly clinical in deviated septum.



² Elevations and ridgelike protuberances

³ Maxillary crest is groove for septum to set sometimes we find this groove projecting little pit

⁴ As result of fault growth

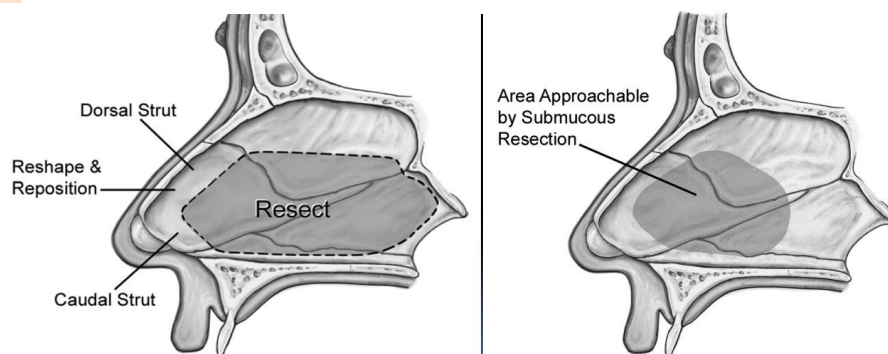
- Anterior rhinoscopy
- Sometimes we grading it into mild, moderate and severe according to the degree of deviation (comparing the two sides):
 - Mild: $\frac{1}{3}$ of the nose is obstructed
 - Moderate: more than $\frac{1}{3}$ of nose is obstructed
 - Severe: $\geq \frac{1}{2}$ of the nose is obstructed

→ Treatment

- If symptoms are minimal (**asymptomatic**) and there is only a minor degree of deviation, **no treatment** is needed.
- Septal deviations are often found in patients with allergic rhinitis. Treat the rhinitis rather than the septal deviation. Where symptoms are more severe correction of the septal deformity is justified (though never essential).

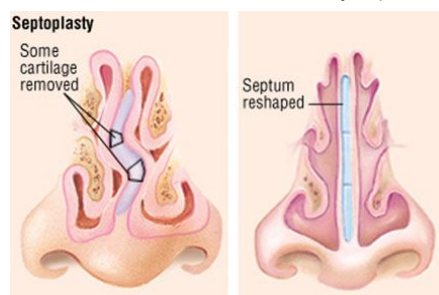
Surgical management⁵:

- **Submucous resection:** obstructing cartilaginous and bony portion. Removal of the deviated part



- **Septoplasty⁶**

- Surgery involves elevating mucosal flaps from the septal cartilage and resecting part of the deviated cartilage, straighten it and put it back in place (septoplasty, check the figure).
- Septal surgery should be undertaken with caution if at all in children as it may interfere with the growth of the mid-face.
- Nowadays we go in with certain techniques “we crush the deviated part with a specific tool for that” to repair the cartilage and put it back in place and also put splint inside “removable after 5 days”.



⁵ it is the only management for DNS in symptomatic patients

⁶ we have to preserve the L shape structure to support the nose

Indications of septoplasty:

1. Nasal obstruction (deviated nasal septum)
2. Epistaxis, chronic sinusitis (when septum is obstructing)
3. Access for transeptal sphenoidotomy
4. Headache from impacted spur
5. Septal neoplasia (rare)

➤ Complications of Septoplasty:

- Septal hematoma and abscess → due to infection
 - In septal hematoma, the cartilage of the septum receives its blood supply from the perichondrium, so if the hematoma was central it'll separate the perichondrium from the septum → Necrosis & deformity. And if you have a central hematoma → emergency/direct drainage.
- Septal perforation. when we are elevating the mucoperichondrium we have to make sure one part is intact to prevent perforation
- Saddle nose deformity (over resecting cartilage anteriorly)
- Synechia⁷ (Adhesions) → will lead to obstruction.
- Cribriform plate fracture
- Anosmia
- Bleeding

⁷ adhesion between the septum and the lateral nasal wall most of the time it happens at the level of turbulent, to prevent it put silastic

Emergencies in nasal obstruction:

Diagnosis	Emergency	Complications
Septal hematoma	Elevation of mucosal Septal cartilage necrosis, development perichondrium with cartilage devascularization	Septal cartilage necrosis, development of a saddle-nose deformity
Septal abscess	Intracranial extension of infection	Septal cartilage necrosis, development of a saddle-nose deformity, cavernous sinus thrombosis, intracranial infection
Mucormycosis	Tissue destruction	Extension to brain or orbit

❖ Hematoma of the septum

- How you differentiate hematoma from DNS?
- Use a probe if you feel something firm this is DNS if what you feel something soft it is hematoma

→ Etiology:

- Direct trauma.
- Operative trauma. "Septoplasty"
- Blood dyscrasias. "bleeding disorders"

→ Clinical features:

- Obstruction.
- Bleeding.
- Lacerations.
- Septal swelling

→ Complications:

- Cartilage necrosis, causing saddle nose deformity.
- Septal abscess.
- Cavernous sinus thrombosis
- Permanent thickening of the septum. At the site where the hematoma has developed → very common in patients who have gotten a Septoplasty due to trauma "it'll appear as a mass"

→ Treatment:

- Immediate Incision and drainage. Emergency
- Systemic Antibiotics. As a prophylactic



❖ Perforation of the septum

→ Aetiology:

- Perforation of the nasal septum may result from the following conditions:
 - Nasal surgery.
 - Trauma including repeated nose-picking.
 - Chronic inflammation, e.g. nasal granulomatosis, syphilis.
 - Inhalation of fumes, e.g. chrome salts.
 - Cocaine.
 - Carcinoma.

→ Effects:

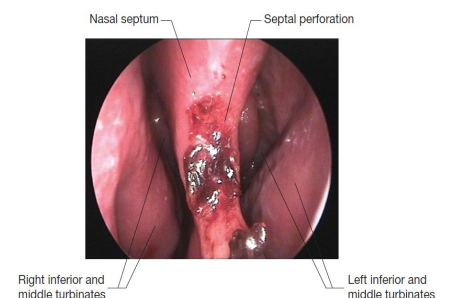
- Many septal perforations cause no trouble. They may give rise to epistaxis and crusting or rarely whistling on inspiration or expiration.
- A perforation is readily seen and often has unhealthy edges covered with large crusts.

→ Clinical features: “clinical features depend on the size and the site of the perforation”

- Asymptomatic.
- Crusting. Due to turbulence of air.
- Epistaxis.
- Whistling. “the smaller the size of the perforation the more the whistling” “And the bigger the perforation → the more obstruction → due to air instead of going back to the nasopharynx there’s going to be turbulence “

→ Treatment:

- No treatment. “in asymptomatic patients”
- Nasal wash.
- Surgical closure by silastic button or Consider sliding or rotating mucoperichondrial flaps with or without a fascial graft; contraindicated for large perforations (approximately >2 cm of vertical height)



→ Diagnosis

- Anterior rhinoscopy
- Biopsy of granulation tissue or abnormal mucosa

★ Functional Endoscopic Sinus Surgery⁸

the steps of FESS:

1. Medialized middle turbinate
2. Excise uncinata process
3. Anterior then posterior ethmoidectomies
4. Sphenoidotomy
5. Frontal recess sinusotomy
6. Create maxillary antrostomy

FESS Land Marks⁹ (CLOSE):

- | | |
|--------------------|---------------------|
| 1-Cribriform plate | 2- Lamina papyracea |
| 3-Orbit | 4- Sphenoid |
| 5-Ethmoid | |

FESS goals:

- Complete extirpation of all the disease
- Permanent drainage and ventilation of the affected sinuses
- Postoperative access to the previously diseased areas.

Indications for ESS:

- Chronic sinusitis
- complicated sinusitis
- recurrent acute sinusitis
- Failed medical management of acute sinusitis,
- fungal sinusitis
- Obstructive nasal polyposis
- Sinus mucoceles
- Remove foreign bodies
- Tumor excision
- Transsphenoidal hypophysectomy
- Orbital decompression,
- Dacryocystorhinostomy,
- Orbital nerve decompression
- Grave's ophthalmopathy
- Choanal atresia repair
- CSF leak repair
- Control epistaxis
- Septoplasty,
- Turbinectomy

⁸ minimally invasive surgical treatment which uses nasal endoscopes to enlarge the nasal drainage pathways of the paranasal sinuses to improve sinus ventilation. it has excellent results

⁹ it is important preoperatively to visualize these structures via CT scan and make sure they are intact

• Postoperative Care:

- Sinus Packing
- Oral Antibiotics for a minimum of 2 week
- Aggressive nasal hygiene to prevent adhesions (saline irrigations)
- Nasal steroids
- Nasal debridement at 1, 3, and 6 weeks

❖ Turbinate Hypertrophy

📖 causes:

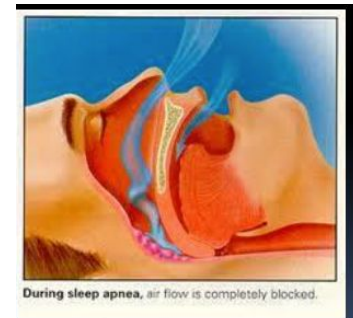
- infection
- compensation¹⁰
- dysfunctional
- allergies
- Rhinitis with chronic use of vasoconstrictors → rebound.
- Compensatory mechanism in septal deviation.

📖 manifestations

- Nasal obstruction
- mouth breathing

📖 treatment:

- Treat underlying cause
- surgical treatment: SMR, Turbinoplasty, SMD¹¹, somnoplasty RF, turbinectomy, ultrasonic reduction



★ Surgical reduction of the Inferior Turbinates

- Turbinate is another name for concha.
- Turbinate resection, Total “not done anymore because it’ll cause the loss of all the imp functions of the nose like ex: protection and conditioning” or partial.
- Out fracturing of the inferior turbinate. “to widen the airway, Dr said know the name of this procedure”
- Destructive procedures, including electrocautery, cryosurgery, laser surgery, and submucous resection.
- Nowadays we prefer going with the submucous resection due to less symptoms and less bleeding. But still the electrocautery is one of the best options but the problem with it is that it doesn’t provide permanent results (lasts for 3 years only) “temporary”

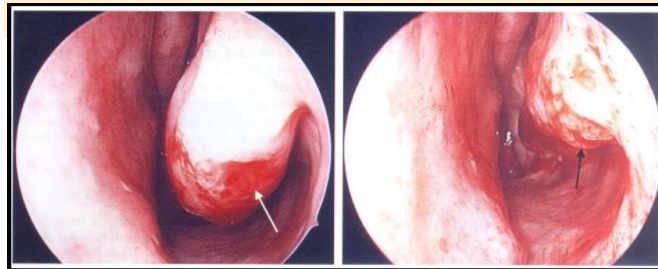


¹⁰ if i have septal deviation the turbinate will enlarge so the air will have turbulent flow if the air enters the nose it straight direction the patient will feel obstruction

¹¹ Submucous diathermy

TURBINATE REDUCTION GOALS:

- Mucosal preservation
- Controlled reduction
- Submucous scarring to reduce the erectile nature of the mucosa
- Bony reduction when necessary
- Minimal complications



Preoperative

postoperative

Epistaxis:

- Nosebleeds are common; they can be persistent, serious and life-threatening.
- One of the functions of the nose is to warm and humidify inspired air. The nasal mucosa has a very rich blood supply and undergoes constant variation in the state of engorgement of its blood vessels.
- Vessels from both the internal and external carotid artery contribute, i.e. the ethmoidal arteries from the internal carotid and the greater palatine, superior labial and sphenopalatine arteries from the external carotid.
- These vessels form a rich plexus on the anterior part of the septum – Little's area or 'Kiesselbach's plexus'.
- Nosebleeds in young patients usually settle quickly as the blood clots and the vessels go into spasm.
- In elderly patients the vessels are rigid and atheromatous.

→ Aetiology

- Some common causes are given in Table 34.1.
- Most nosebleeds are idiopathic.
- Spontaneous epistaxis is common in children and young adults; it usually arises from Little's area or from a prominent vein just below.
- It may be precipitated by infection or minor trauma, is easy to stop, but tends to recur.
- Nosebleeds in the elderly are far more difficult to treat.
- The bleeding site is often high up in the posterior part of the nose and on the lateral nasal wall.

Table 34.1 Causes of epistaxis

Local causes	General causes
Spontaneous	Cardiovascular conditions
Trauma	Hypertension, raised venous pressure
Tumours	Coagulation or vessel defects
Hereditary telangiectasia	Haemophilia
Nasal allergy	Leukaemia
	Anticoagulant therapy
	Thrombocytopaenia
	Fevers (rare)
	Influenza

- Local causes:

- Acute trauma, Chronic trauma.
- Deviated septum.
- Inflammation of the nose and sinuses.
- Tumors, Idiopathic.

- Systemic causes:

- Coagulation and bleeding diseases.
- Atherosclerosis.
- Familial hemorrhagic telangiectasia, “Autosomal dominant disease where they have no muscles around the blood vessels thus will present with bruises and GI bleeding”

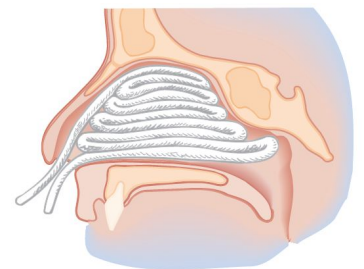
→ Treatment

- The treatment priorities are twofold: stop the bleeding and resuscitate the patient who has had a serious bleed.
- General measures. “ABC”
- Stop the bleeding.
- Prevent further bleeding.

★ Control the bleeding

- Digital pressure. +Leaning forward.
- Cautery. “With silver nitrate”.
- Anterior nasal packing, Postnasal packing.
- Arterial ligation : Maxillary, Ethmoidal, External carotid.
- Arterial embolization.

- In anterior nasal packing, it can be used for as long as needed. But usually we have to remove it before 24 hours or in left more than you need to give prophylactic Antibiotics → to prevent against infection “toxic shock syndrome”



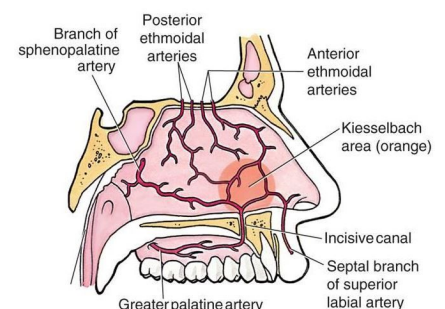
Anterior nasal packing.

→ Why bleeding from the nose?

- Vascular organ secondary to incredible heating/humidification requirements.
- Vasculature runs just under the mucosa. “Very rich in blood supply”.
- Arterial to venous anastomoses.
- ICA and ECA blood flow.

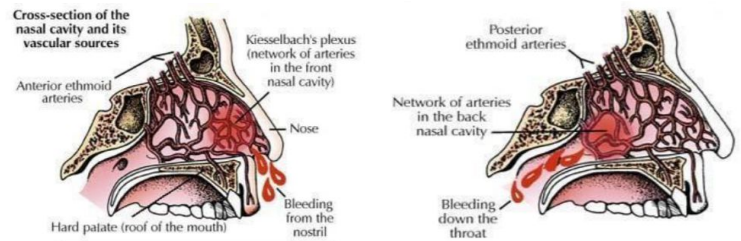
→ Sites:

- Anterior (Little’s area).
- Posterior (vicinity of sphenopalatine foramen)



★ **Kiesselbach's plexus/Little's area:**

1. Anterior Ethmoid (Ophth).
2. Superior Labial A (Facial).
3. Sphenopalatine A (IMAX).
4. Greater Palatine (IMAX)



★ **Woodruff's plexus:**

- Sphenopalatine A (IMAX).
- IMAX= Internal Maxillary Art.
- Anterior ethmoid art → came from ophthalmic → from internal carotid Superior labial Art → From facial → from external carotid artery Sphenopalatine → IMAX → From external carotid artery
- Greater palatine → Imax → External carotid artery.
- External carotid gives many branches in the neck, starting from: the superior thyroid, lingual, posterior auricular/occipital, internal maxillary, superficial temporal.
- Internal carotid branches in the neck → None, it moves upward into the brain and form the circle of wills then give the ophthalmic branch.

➤ **Angiofibroma**

- Juvenile nasopharyngeal.
- Benign.
- Adolescent Males "always males between 10-20 try to exclude angiofibroma first".
- Frequent chronic epistaxis.
- Nasal obstruction.
- Rhinorrhea.
- Conductive hearing loss.
- Diplopia.
- Otitis Media.
- Treatment: embolization & Surgery.

