ENT NOTES
LEVEL VI MBCHB
2019

COMPILED BY: NAILA KAMADI

OUTLINE

- History taking & examination – 5
- Adenotonsillar disease – 27
- Pharyngitis – 107
- Laryngitis – 162
- Allergic rhinitis & nasal polyposis – 172
- Acute Otitis Media (AOM) & Otitis Media with Effusion (OME) – 256

CONT.

- Chronic Otitis Media – 290
- Hearing Loss – 314
- Upper Airway Obstruction – 340
- Foreign Bodies in ENT – 359
- Tracheostomy – 399
- Epistaxis – 452
- Neck trauma – 487

CONT.

- Vertigo & tinnitus – 554
- Diseases of salivary glands (non neoplastic & neoplastic) – 618
- ENT manifestations of HIV – 736
- Management of Neck Masses – 787
- Cancer of the Larynx – 817
- Nasopharyngeal carcinoma – 864

HISTORY TAKING & EXAMINATION IN ENT 24/1/2019

BY: PROF. MUTHURE MACHARIA

WHAT IS ENT

- It exposes you to:
  - Pediatric patients
  - Adult patients
  - Medical problems
  - Surgical problems

INTRODUCTION

- History and clinical examination is the basis of diagnosis.
- A wholesome approach should be taken towards the patient.
- Maintain the patient's dignity during history taking & examination.
- Be sensitive to cost – effectiveness of investigations – what will be the value of investigations in determining management?

BASIC EQUIPMENT

- Good light
  - Head mirror
  - Head light
  - Torch
- Patient's comfort
- Aural instruments
- Nasal instruments
- Oropharyngeal instruments

History taking

- HPI
  - Duration, onset, progression, severity
  - Accompanying complaints
  - Systemic disease
  - Medications being taken or previously taken for illness
- History of past illness
- Personal history
- Family history

The ear: signs & symptoms

- Hearing loss
- Dizziness
- Pain
- Blockage
- Tinnitus: trauma, cerebellopontine angle lesion, loss of hearing
- Discharge
- Itching
- Swelling
- Deformity
- Fluid in the ear: ?lymphoid hyperplasia secondary to immunosuppression; nasopharyngeal carcinoma

Examination

- Physical exam
  - External ear
    - Pinna and surrounding areas
    - External auditory canal
  - Tympanic membrane: intact, normal color
  - Middle ear
  - Mastoid

The ear:

- Functional exam
- Auditory function
  - Voice tests
  - Tuning fork tests: rhinne's, weber's
- Vestibular functions
  - Nystagmus
  - Positional tests
  - Fistula tests

investigations

- Audiometric:
  - Pure tone audiometry
  - Tympanometry
- Biochemistry
  - Blood sugar: otitis externa maligna→ ?DM
  - urea/ electrolytes
- Serological/ immunological
- Radiological: plain X rays, CT scan, MRI, angiography

Nose & sinuses

- Discharge
- Swelling/ Deformity
- Obstruction
- Bleeding
- Headache (rhinogenic/ sinogenic)/ facial pains
- Pains
- Smell disturbance
- Snoring
- Sneezing
- Post – nasal drip

P/E of nose and sinuses

- External nose
- Vestibule
- Anterior rhinoscopy
- Posterior rhinoscopy
- Nasal endoscopy
- Sinuses

Nose and sinuses

- Functional examination
- Patency e.g. choanal atresia
  - Mirror/ spatula test: holding mirror in front of test and check for misting
  - Cotton test
  - Acoustic rhinomanometry
- Smell test

investigations

- Hematological
- Nasal smear
- Allergy tests
- Immunological
- Radiological
- Plain X rays, CT scans, MRI
- Renal function tests: Wegener's granulomatosis (nasal, pulmonary & renal symptoms)

Throat symptoms

- Pain
- Difficulty in swallowing
- Disturbance of taste
- Pain in swallowing
- swelling
- Sore throat
- Hawking
- Lump
- Dysphonia
- Voice changes
- Cough
- Halitosis
- Burning sensation
- DIB
- Ulcers/ sores

P/E

- Lips
- Buccal mucosa
- Gums/ teeth
- Hard palate
- Tongue: palpate the tongue for lumps
- Floor mouth: one hand on the floor of the mouth and the other on the inside…ballot
- Retro molar trigone

P/E

- Soft palate
- Tonsils
- Base of tongue
- Postpharyngeal wall
- Vallecular
- Hypopharynx
- oropharynx

Functional examination

- Taste
- Indirect laryngoscopy
- Flexible nasolaryngoscopy
- Rigid laryngoscopy
- videostroboscopy

Throat investigations

- Hematological
- Serological/ immunological
- Radiological: plain X rays, CT scans, MRI, contrast studies

Neck symptomatology

- Pain
- Swelling
- Discharge
- Deformity

P/E

- Levels of LN (I → IV)
- Descriptions of swellings/ masses
- Investigations
- Hematological
- Serological/ immunological
- FNAC
- Radiological:
  - Plain X ray, CT scans, MRI, angiography, U/S, sialography

Other systems

- Neurological
- Respiratory: one airway, one disease
- Cardiovascular
- GIT
- Others depending on presentation

Sub - specialties

- Pediatric otolaryngology
- Otology/ neurotology
- Rhinology
- Sleep medicine
- Facial plastic and reconstructive surgery
- Laryngology
- Allergy
- Head and neck surgery

ADENOTONSILLAR DISEASE LEVEL VI 10TH/JAN/2019

BY: DR. CATHERINE IRUNGU

TYPED BY NAILA KAMADI

Outline

- Adenotonsillar surgery (MC pediatric surgery)
- Recurrent infections
- Obstructive Sleep Apnea (OSA)
- Complications
- Evolution of the surgical technique

Anatomy

- There are 3 main groups of lymphatic tissue in the head & neck:
  1. **WALDEYER'S RING**
  2. **TRANSITIONAL LYMPHATICS**
  3. **CERVICAL LYMPH NODES**

1. Waldeyer's ring

- This is a **ring of mucosa associated lymphoid tissue (MALT)** that is integral to immunoglobulin immunity.
- It is composed of the following:
  1. Nasopharyngeal tonsils (Adenoids)
  2. Tubal/ eustachian tonsils
  3. Palatine tonsils
  4. Lingual tonsils
- A tonsil is any collection of lymphoid tissue with *no afferent vessels* but has efferent vessels, unlike a lymph node.

Cont.

- **Adenoids/ nasopharyngeal tonsils**: are found on the posterior wall & roof of the nasopharynx.
  - These **CANNOT** be visualized through the oral cavity (unless a laryngeal mirror is used, i.e., posterior rhinoscopy) since the palate covers them. They can only be visualized through the nasal cavity using endoscopy.
- **Tubal/ eustachian tonsils**: are found at the pharyngeal opening of the eustachian tube (ET)
- **Palatine tonsils**: are embedded in the lateral wall of the oropharynx on either side between the pillars (***palatoglossal & palatopharyngeal arches***) of the fauces.
- **Lingual tonsils**: lie at the base of the tongue just anterior to the epiglottis

Adenoids/ nasopharyngeal tonsils

- Grow from 0 – 5 years; Atrophy at 8 – 10 yrs.
- No afferent lymphatics.
- Efferents:
  1. Retropharyngeal
  2. Upper deep cervical nodes (levels I & II; they increase in size during infection)

Palatine tonsils

- Develop from **2nd internal branchial pouch..**
- Grow rapidly till 5 – 6 years
- Can be visualized through the oral cavity as they lie in the tonsillar fossa.
- They consist of a collection of lymphoid tissue covered by ***non – keratinizing squamous epithelium***, hence are easy to spot on examination.

Cont.

- Peak size in puberty; from late puberty onwards this lymphoid tissue undergoes progressive atrophy.
- No afferent lymphatics.
- Efferents:
  - Jugulodigastric (between IJV & digastric muscle)
    - The tonsillar/ jugulodigastric node is affected in tonsillitis (it is the main node draining the tonsil) & it is the MC lymph node in the body to undergo pathological enlargement.
  - Deep cervical nodes
  - Retropharyngeal nodes

Adenoids vs. Tonsils

Adenoids:

- Nasopharyngeal
- Have ciliated columnar epithelium
- Has furrows
- Most immunologically active at 2 – 5 yrs.
- Start atrophying at 8 – 10 yrs.
- Disappear at 20yrs.

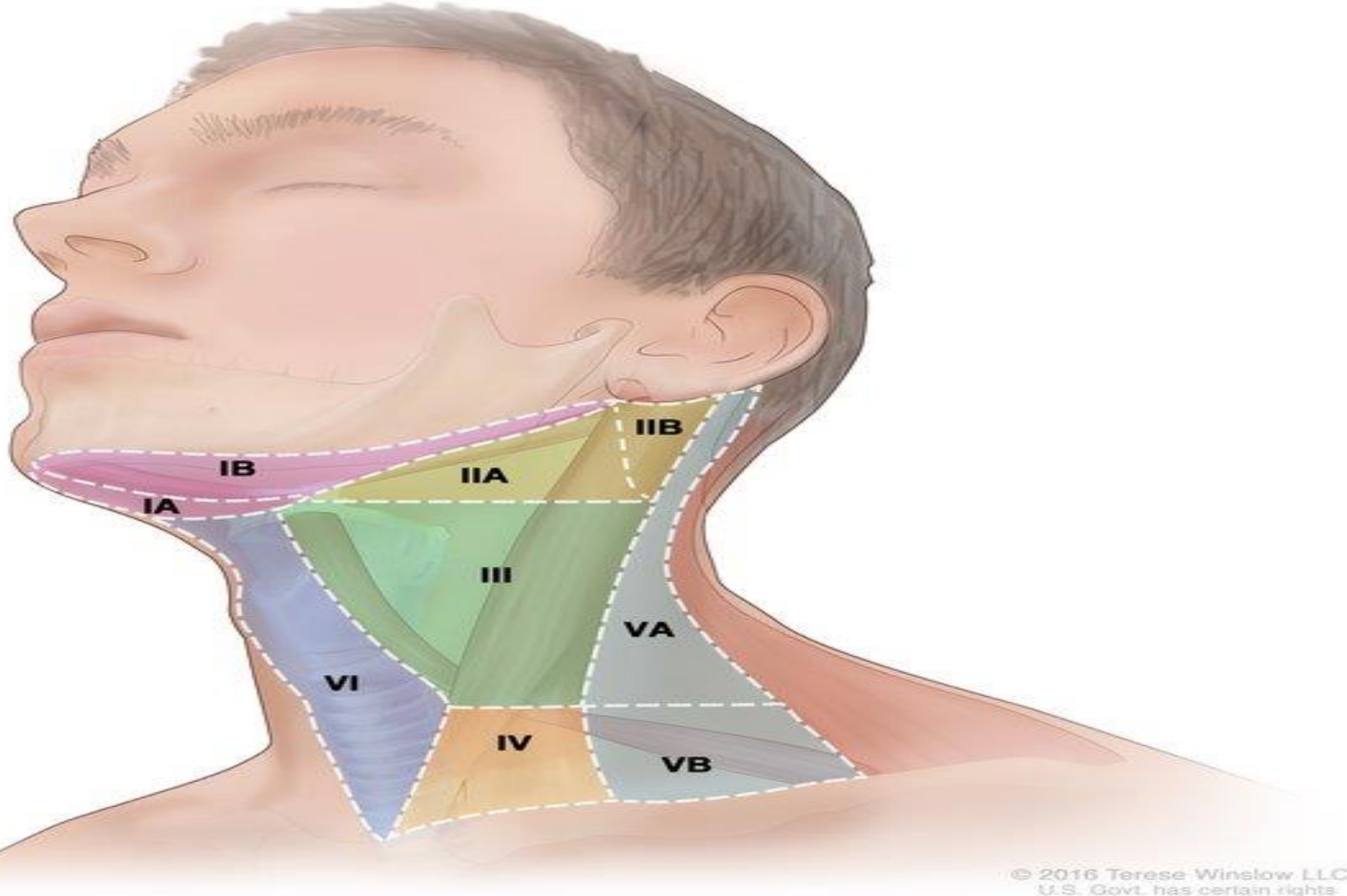
Tonsils

- Oropharyngeal
- Non – keratinizing squamous epithelium
- Has crypts
- Most immunologically active at 4 – 10 yrs.
- Start atrophying at 12 – 15 yrs.
- Regress partially at 18 yrs.

Cervical Lymph Nodes are grouped into levels I – VII:

Submandibular & submental: Level I
Upper jugular nodes: Level II
Middle jugular nodes: Level III
Lower jugular nodes: Level IV
Posterior triangle nodes: Level V
Anterior compartment nodes: Level VI
Superior mediastinal nodes: Level VII

Lymph Node Groups of the Neck



Introduction

- Tonsil & adenoid infection are very common in **childhood.**
- **Adenoid - Tonsil Surgery (ATS)** is the MC performed procedure in the history of surgery.
- For airborne antigens, the adenoids & tonsils are the *1st site of immunological contact*.

Organisms cultured from tonsils & adenoids.

- **Bacteria**
  - ***Aerobic***: ***Streptococcus spp.*** (A – G), ***Haemophilus spp.***, ***Moraxella spp.***, ***Staph. aureus***, *Neisseria spp.*, *Mycobacteria* spp.
    - Group A β – Hemolytic Streptococci (GABHS) i.e., *S. pyogenes,* is the most worrisome due to the autoimmune sequelae it is associated with.
    - Groups B, C, & G are usually seen ***before 6 months***
    - *S. pneumoniae* is associated with intracranial progression to meningitis.
  - ***Anaerobic***: *Bacteroides*, *Peptococcus*, *Peptostreptococcus*, *Actinomyces*

Cont.

- Viruses: EBV, adenovirus, influenza A & B, herpes simplex, RSV, parainfluenza
  - ***Predominantly, infections of the upper aerodigestive tract are viral***
- NB: If a patient is not responsive to β – lactam antibiotics, send them for M,C & S to screen for an atypical infection.

Disease Spectrum

- Acute adenoiditis (A_cA)
- Acute tonsillitis (A_cT)
- Acute adenotonsillitis (A_cAT)
- Recurrent & chronic adenoiditis
- Recurrent & chronic tonsillitis
- Recurrent & chronic adenotonsillitis

1. ACUTE ADENOIDITIS (A_cA)

Etiology:

- 85% viral, 5 – 30% bacterial
- MC bacterial etiological pathogens:
  - GABHS is an important pathogen due to the potential non – suppurative complications, i.e., Acute rheumatic fever (ARF), Acute glomerulonephritis (AGN)
  - Others: *H. influenza*, *S. aureus*, *S. pneumoniae*

Symptoms

- Snoring, purulent rhinorrhea, nasal blockage, fever, recurrent otitis media (ROM), rhinosinusitis.

1A. Recurrent A_cA

- This is defined as ≥ 4 episodes of AcA in 6 months.
- Symptoms: rhinorrhea, nasal blockage, ***mouth breathing***
- Recurrent Acute Rhinosinusitis (ARS) is similar to recurrent AcA & the 2 can be differentiated by a CT scan.
  - Symptomatology: ARS is characterized by **double sickening**, i.e., sick → recovered → sick again
- DDx: GERD induced adenoiditis in < 2 yrs.
  - Pathophysiology: Gastric acid & pepsin cause inflammation

1B. Chronic adenoiditis

Symptoms persisting for > 14 days:

- Persistent rhinorrhea, post nasal drip, ***malodorous breath/ halitosis***
- Associated with:
  - ***Otitis media (serous/ OME) if it persists for >3 months*** since as adenoids enlarge, the ET opening in the nasopharynx is blocked
  - Acute rhinosinusitis (ARS), Chronic rhinosinusitis (CRS)
  - ***Craniofacial growth abnormalities*** may develop due to chronic mouth breathing, i.e., adenoid facies.
- In unremitting disease: consider GERD as a differential.

2. ACUTE TONSILLITIS

- This is acute inflammation of the palatine tonsils usually due to ***Streptococcal*** or less commonly, viral infection.
- Symptoms include:
  - Fever
  - Sore throat
  - Pain that is:
    - Most marked when swallowing, i.e., dysphagia
    - Referred to the ears (tympanic branch of glossopharyngeal nerve)
  - Headache
  - Lethargy/malaise

Cont.

- Signs include:
  - Tender lymphadenopathy
  - Erythematous tonsils: if this is the only sign, the diagnosis is probably a simple pharyngitis.
  - Purulent exudates
  - White membrane: thin, non – confluent & confined to the tonsil; peels away without bleeding (compare with diphtheria)
  - Airway obstruction

Cont.

- Resolves in 3 – 4 days.
- ***Bacterial etiology***: > exudate, ↑ WBC with granulocytic shift & frank lymphocytosis
  - Treat for GABHS with **amoxicillin.**
  - Throat cultures/ rapid antigen test for GABHS in recurrent disease.
- ***Viral etiology***: ↓ grade fever, < exudate, ↓ WBC with a lymphocytic shift.

Criteria for diagnosis of bacterial tonsillitis

Fever > 38^0C

Anterior cervical lymphadenopathy

Pharyngeal tonsillar exudate

Absence of cough

Criteria for diagnosis of viral tonsillitis

- Coryza
- Conjunctivitis
- Cough
- Hoarseness
- Spontaneous resolution (normally)

2A. Recurrent acute tonsillitis – indication for tonsillectomy

- 4 – 7 separate episodes in the past 1 yr.
- ≥5 separate episodes/ yr. in 2 years
- ≥ 3 separate episodes/ yr. in 3 years

2B. Chronic tonsillitis

- Chronic sore throat; lasting ≥ 3 months
- Malodorous breath (halitosis)
- Presence of tonsilloliths (excessive tonsillar debris)
- Peri tonsillar erythema
- Persistent tender cervical lymphadenopathy
- Common in: GERD, allergic pharyngitis

Differentials

- **Infectious Mononucleosis** (Kissing disease) – EBV.
  - This is associated with micro – petechiae of the soft palate; atypical lymphocytes on a PBF & a positive monospot test.
- **Scarlet fever** (complication of GABHS; severe rush + sore throat; query in patient from a refugee camp)

Cont.

- **Diphtheria** (query in patient from non – vaccinated communities)
  - The membrane is dirty grey, thick and tough; it bleeds if peeled away;
- **Leukemia** (chronic odynophagia with redness & erythema)
- **Other malignancies**
- **Agranulocytosis**

ALWAYS SEND FOR FHG

Cont.

- **Vincent's angina/ Trench mouth**
  - Characterized by superficial, painful ulcers with erythematous borders
- **Syphilitic gumma**
- **TB**
- **Leprosy**
- **Fungi**
- **Crohn's disease**

Complications of adenotonsillar infection

Local:

- Laryngeal edema → UAO
- Abscesses:
  - **Peri – tonsillar abscess** (starts as cellulitis of the salivary glands): do an I & D ***(not that simple; watch out for the greater palatine artery; patient could bleed to death)***
  - Parapharyngeal
  - **Retropharyngeal abscess** (midline but slightly below the base of the tongue)

Cont.

- Suppurative adenitis
- AOM
- Chronic tonsillitis
- **Hemorrhagic tonsillitis**

Cont.

General:

- Septicemia (tonsils are a very vascular area)
- Febrile convulsions (absolute indication for surgery)
- Meningitis

Cont.

- Acute Rheumatic Fever
- Acute Glomerulonephritis
- Guttate psoriasis
- Lemierres syndrome (IJV thrombophlebitis): most often occurs when a bacterial throat infection (esp. by *Fusobacterium necrophorum*) progresses to the formation of a peri – tonsillar abscess. May progress to sepsis and septic embolism(usually pulmonary)

Assessment

- Clinical history : Snoring, recurrent URTI, sore throat
- Clinical examination:
  - External exam:
    - Face: Craniofacial anomalies, adenoid facies
      - Crowded teeth; small nose; stained teeth; white, flaky and dry lips
      - Stigmata of allergy: Dark circles around the eye, crease on nose etc.
    - Anterior rhinoscopy: Use a torch
    - Assess for patency
      - Use metal spatula to block either nostrils (younger children)
      - Ask older children to breathe heavily
    - Posterior rhinoscopy: Use endoscope

Cont.

- Oral cavity & oropharynx → tonsillitis, PTA
- Nose → rhinorrhea, HIT
- Ear → AOM, OME, retraction of tympanic membrane
- Neck → obvious masses, LN
- Chest → heart murmur, symptoms of RHD, congenital heart diseases
- Abdomen → Splenomegaly

Clinical features

- Pyrexia, malaise, headache, sore throat, dry throat, thirst.
- Voice change → hot potato, saliva accumulation
- Otalgia → referred pain, AOM, OME.
- Odynophagia, dysphagia
- Symptom duration 5 – 6 days

Investigation

- ***History*** & ***Physical examination***
- ***FBC***: Neutrophilia → Acute bacterial; Monocytosis → TB; Lymphocytosis → viral
- ***Throat swab*** → microscopy, culture & sensitivity
  - Throat swabs are frequently contaminated by oral flora therefore not routine.
- ***Serologic tests***: If an immuno - deficient syndrome is suspected e.g. Hypogammaglobulinemia.
- **NOTE: In SCD, adenotonsillar disease (ATD) should be treated as an emergency. This is because, infection can predispose the patient to dehydration, acidosis & hypoxia, all of which can precipitate a sickling crisis.**

Medical Management

- ***Penicillins*** – 1st line for GABHS
- In the event of acute Upper Airway Obstruction (UAO):
  - Secure & protect airway (ETT?); steroids, IV antibiotics, immediate tonsillectomy for poor response
  - ***When suspecting impending UAO always error on the side of caution. Refer urgently after starting on IV steroids (Dexamethasone or high dose prednisolone); don't give oral medications.***
- Recurrent tonsillitis – Augmentin (Amoxicillin/ clavulanic acid)
- Chronic tonsillitis or UAO → treat for 3 – 6 wks.

Peritonsillar abscess

- This is characterized by a swelling superior to the tonsil & accompanied by tender lymphadenopathy.
- The Peritonsillar area is a potential space & abscess formation is associated with smoking.
- Symptoms: Fever, drooling, trismus, sore throat/ odynophagia, hot potato voice, dysphagia
- Signs: Unilateral soft palate swelling, Uvula deviation

Treatment

- Supportive:
  - **Analgesics**
  - **Rehydration**
  - IV **Steroids** (*if there is edema*)
- Definitive:
  - Needle aspiration (if it is a frank abscess)
  - I & D (if recurrence occurs after needle aspiration)
  - IV ***antibiotics***

Prognosis

- There is a 20% chance of recurrence after 1st episode and a 50% chance after the 2nd episode
  - Interval tonsillectomy is indicated in repeat abscess.

Hemorrhagic tonsillitis

- **Recurrent bleeding** from prominent vessels in chronic tonsillitis
- It can be **diffuse parenchymal bleeding**
- Locally controlled in most patients
- Younger patients taken to theatre because of poor co - operation
- Tonsillectomy indicated if recurrent, anemic or not responsive to local control
- Rule out bleeding diathesis.

Sequelae

- UAO
- Paranasal sinus disease
- Craniofacial malformations
- Otological disease
- Cardiac disease

UAO

- Increased PCO_2 → Decreased PO_2 → Reflex VC in pulmonary circulation in response to relative hypoxia → Pulmonary HTN → **Cor pulmonale.**
- In children presenting for ATS, Pulmonary HTN is MC.

Treatment

- Medical
  - Rest, rehydrate, analgesics → sufficient in mild cases
  - Benzyl penicillin IV/ IM then continue with oral penicillin V OR:
  - Erythromycin, sulphonamides (for community acquired ATD)
  - Resistant cases → consider clindamycin, ciprofloxacin +/- metronidazole (anaerobic)
- Surgical (recurrent disease)
  - Tonsillectomy & Adenoidectomy (T & A)

Paranasal sinus disease

- Maxillary sinusitis can result.
- Plain X ray: (Water's view)
  - Maxillary sinusitis
    - Fluid level & a meniscus in the affected maxillary sinus
- CT scan
  - Enlarged turbinates
  - Opacification in the affected sinus

Craniofacial malformations

- Long, open – mouthed dull face
  - Hyper – somnolence
  - Chronic mouth breathing
- Under – developed nostrils: Since the child becomes an obligate mouth breather
- Micrognathia
- High arched palate
- Short upper lip
- Prominent crowded upper teeth

Otitis

- This is inflammation of the middle ear; Classification:
  - **Acute otitis media (AOM)**: Usually a bacterial infection accompanied by a viral URTI; rapid onset of signs & symptoms
  - **Recurrent AOM:** AOM for ≥ 3 months in 6 months or for ≥ 4 months in 1 year.
  - **Otitis Media with Effusion (OME):** Painless hearing loss & intermittent purulent ear drainage that follows AOM or arises without prior to AOM.
  - Chronic OME: Persistent Otorrhea present for > 6 weeks

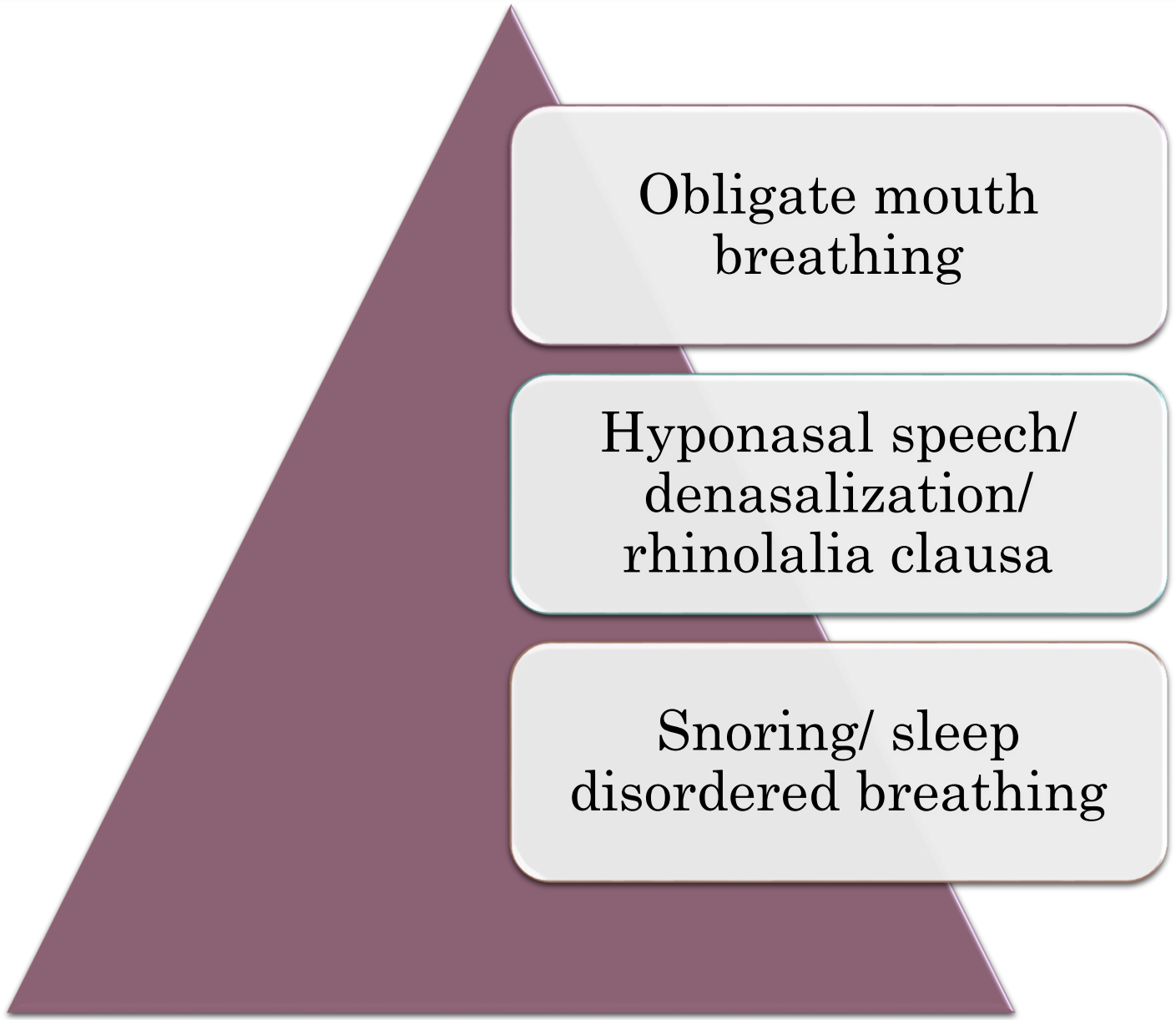
OME

- Mechanical obstruction of Eustachian Tube (ET) opening
- Chronic AOM causing effusion
- Eustachian Tube dysfunction
- Air bubble in the middle ear

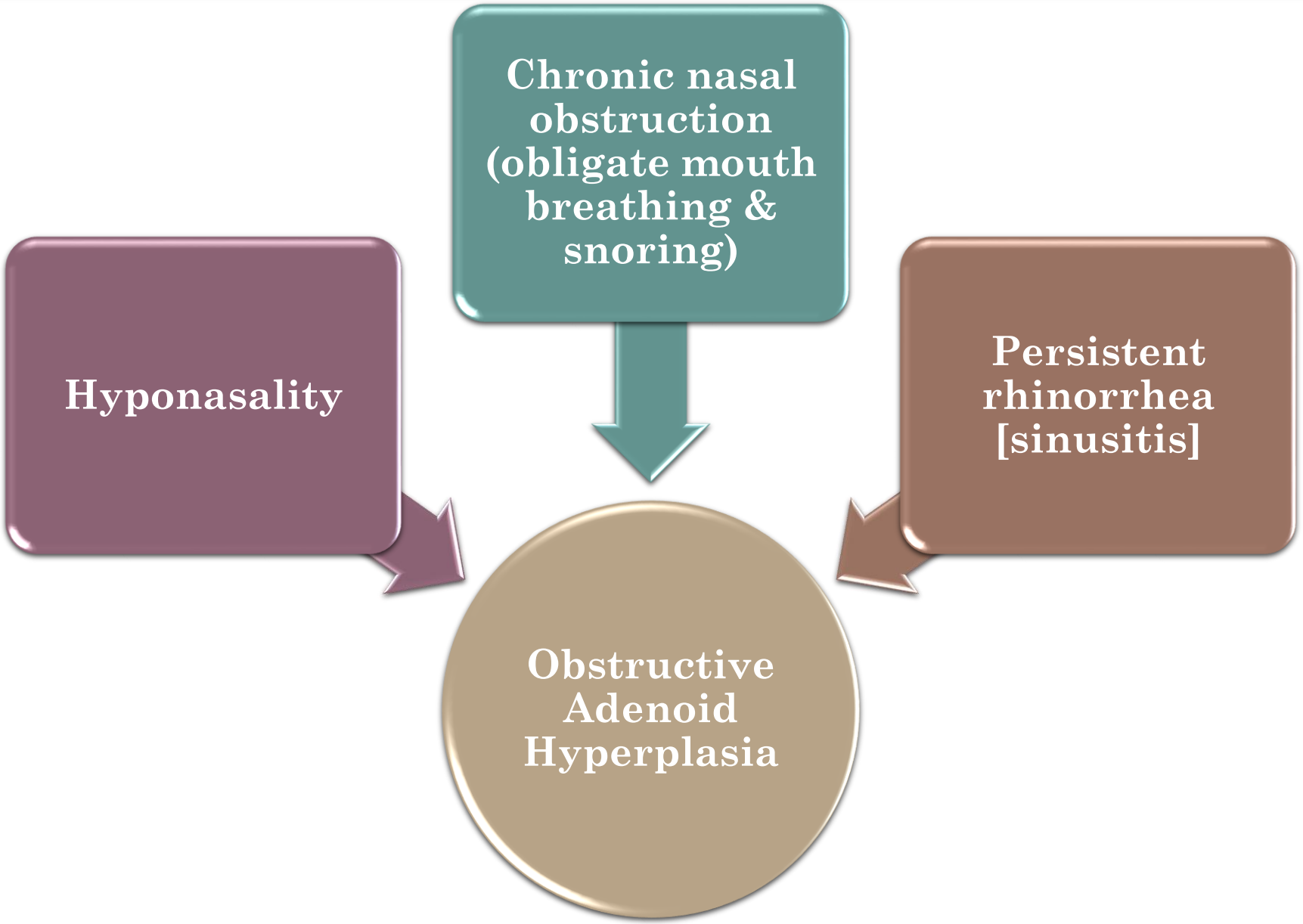
Cardiac disease: echocardiography

- Hypertrophy of right side of heart
- Pulmonary HTN

OBSTRUCTIVE AIRWAY DISEASE – Adenoid ± tonsillar hypertrophy



Obstructive adenoid hyperplasia (triad)



Pathological comorbidities of adenoid hypertrophy

- Obstructive sleep apnea
- Enuresis: Sleep too deeply when they get to sleep
- Poor school performance due to daytime hyper – somnolence in children:
  - NB: *unlike in adults, hyper – somnolence in children results in irritability & hyperactivity & thus children may be misdiagnosed with ADHD*)
- Craniofacial malformations
- Rhinorrhea (sinusitis)
- Pulmonary hypertension and cor pulmonale

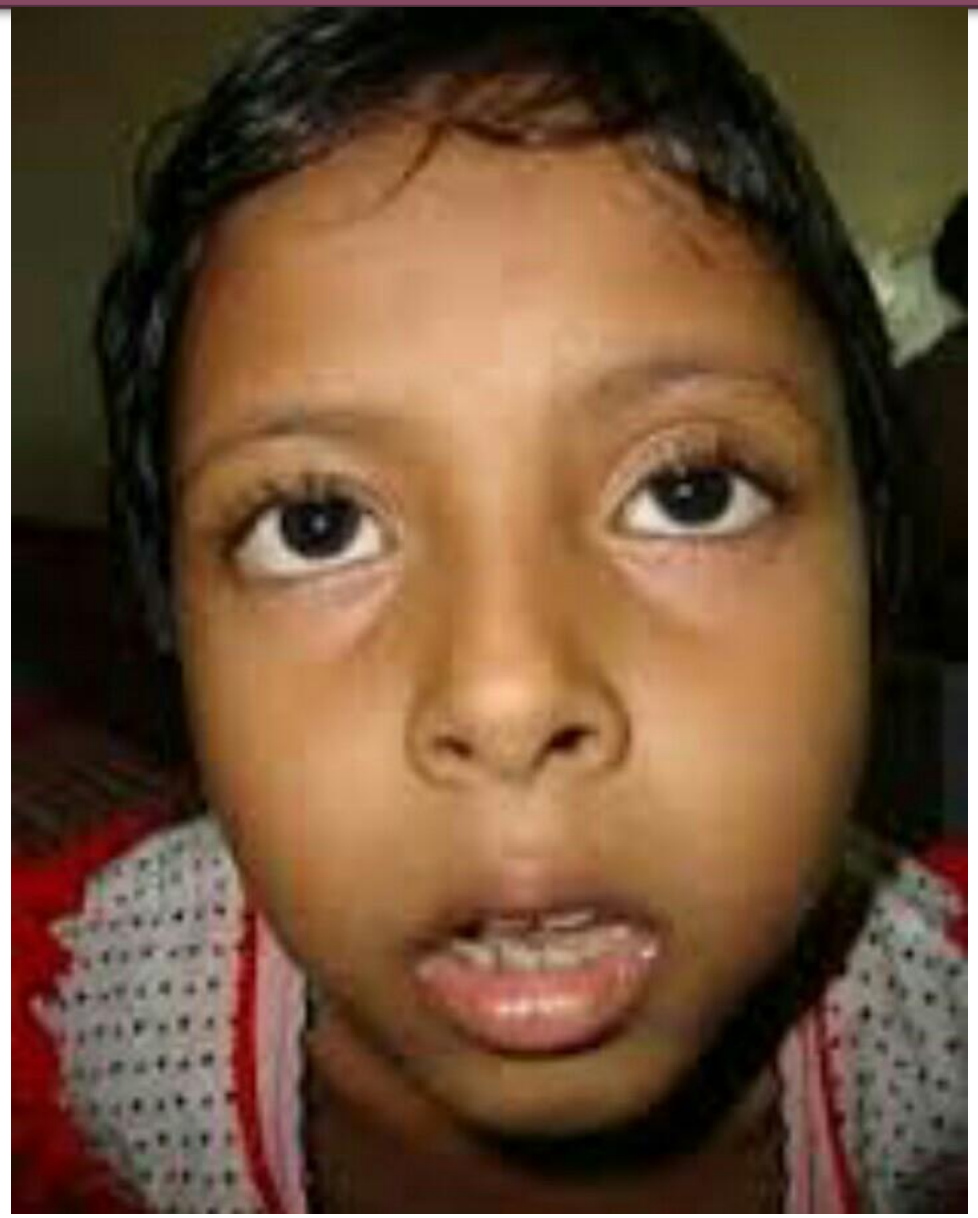
Cont.

- Recurrent otitis media
- OME: due to chronic blockage of ET & negative pressure in the middle ear (affects hearing)
- Recurrent URTIs (change in child's play habit)
- Behavioral disturbances
- Neurocognitive deficits
- Failure to thrive: Due to poor appetite and resulting delayed milestones

Cranio – facial growth abnormalities/ adenoid facies

- Mouth breathing
- Elongated face
- Hypoplastic maxilla
- Prominent incisors: hypoplastic maxilla but elongated teeth
- Short upper lip
- Elevated, pinched nostril
- High arched palate
- Constant rhinorrhea

Adenoid facies



- 'Dumb face'; easily confused with a neurocognitive congenital defect
- Send for ***lateral neck X – ray soft tissue window***

Unilateral tonsillar hypertrophy

- **Apparent vs. true enlargement**
  - Apparent: asymmetric anatomical positioning of 1 tonsil deeper in its pouch (common)

<u>Causes of true unilateral tonsillar hypertrophy:</u>

1. **Non – neoplastic**: Acute vs. chronic infective (*TB, syphilis, chronic actinomycosis, fungi*)
2. **Neoplastic**: R/O neoplasia if → fast enlargement, change in voice (*hot potato voice*), new onset snoring, dysphagia, neck lymphadenopathy
   a. ***Benign***: Lipoma, fibroma
   b. ***Malignant***: lymphoma (MC), papilloma, SCC, 2^{o} metastases (melanoma, lung Ca)
3. **Congenital**: hemangioma, cystic hygroma, lymphangioma, teratoma

Cont.

- Management
  - Excision biopsy (tonsillectomy): histology, culture for aerobic, anaerobic and fungal organisms
  - Imaging (before surgery): check for extension beyond tonsillar capsule

Obstructive tonsillar disease

- **CLINICAL FEATURES:** Muffled voice; Hyper – nasality; Dysphagia; Snoring / sleep disturbance; Changes in craniofacial skeleton

BRODSKY GRADING OF THE SIZE OF TONSILS ACCORDING TO OROPHARYNGEAL WIDTH OCCUPANCY:

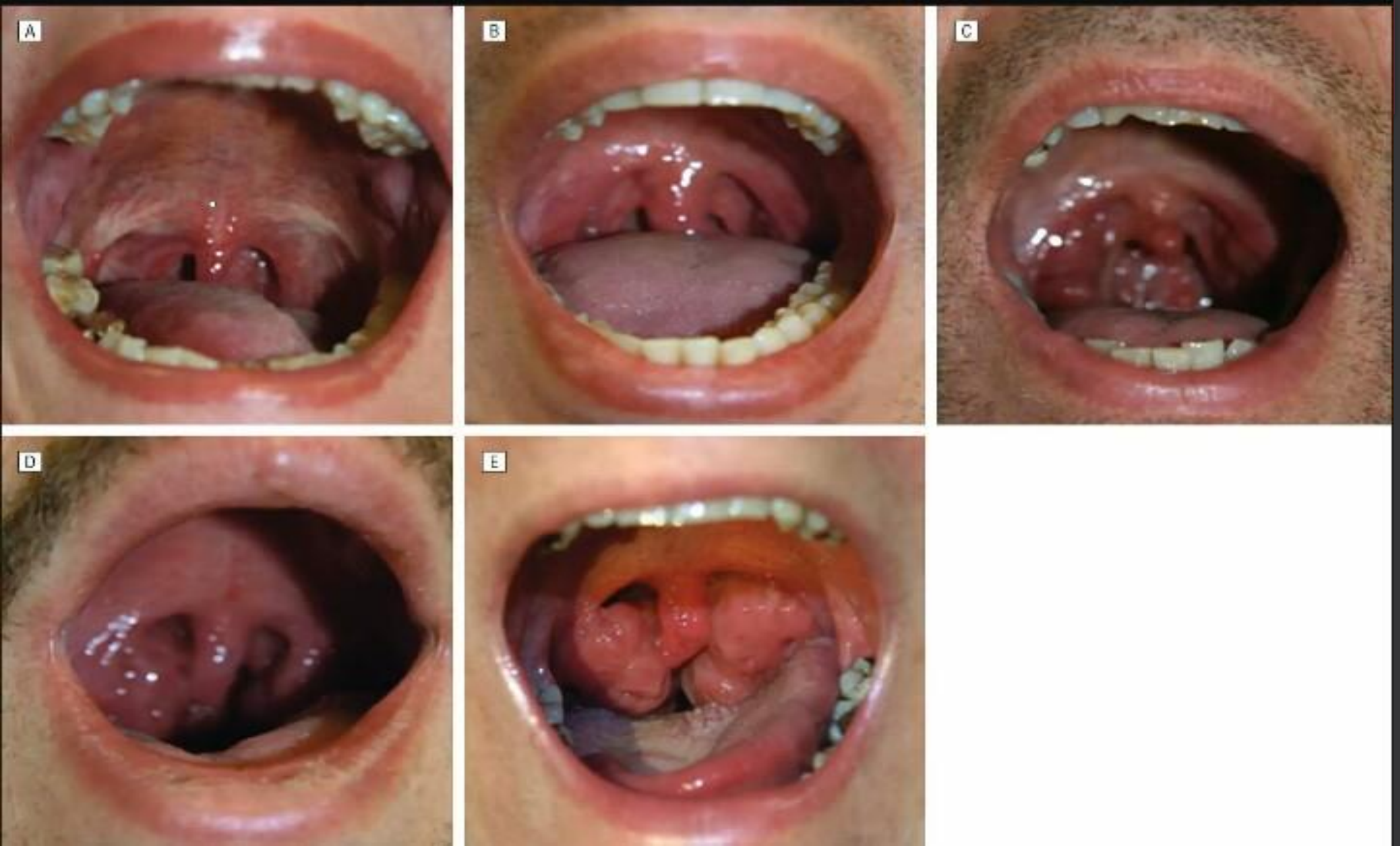
A. Tonsils within the tonsillar fossa (0)

B. Tonsils just outside the fossa & occupy ≤25% of the oropharyngeal width (+1)

C. Tonsils occupy 26 – 50% of the oropharyngeal width (+2)

D. Tonsils occupy 51 – 75% of the oropharyngeal width (+3)

E. Tonsils occupy 76 – 100% of the oropharyngeal width (+4) i.e. ***kissing tonsils***


A
B
C
D
E

Surgical management

- Adenoidectomy
- Tonsillectomy: (find out the methods of tonsillectomy)
  - Post – tonsillectomy one will see ***empty fauces*** (OSCES)
- Adenoid – tonsillectomy
- I & D
- Resection of malignancy

Indications for adenoidectomy

- ≥ 4 episodes of ***recurrent purulent rhinorrhea*** in 12 months in a child < 12 yrs.
- ***Persisting symptoms*** of adenoiditis after 2 courses of antibiotic therapy.
- ***Obstructive Sleep Apnea (OSA)*** with nasal airway obstruction >3 months.
- ***Rhinolalia clausa***
- ***OME*** > 3 months or a 2nd set of ventilation tubes

Cont.

- ***Dental malocclusion*** or orofacial growth disturbance
- Cardiopulmonary CP^0 – ***cor pulmonale***, PAH (Vasoconstriction in the pulmonary circulation due to chronic hypoxia), RVH with UAO, may lead to global heart failure
- Adenoid hypertrophy with ***chronic sinusitis***

Paradise Criteria for tonsillectomy in pediatric & adolescent patients.

Minimum sore throats in a year

- Atleast **7** in the previous year
- OR Atleast **5** in each of **two** previous years
- OR Atleast **3** in each of **three** previous years

Sore throat with *atleast one*

- Fever > 100.9°F (38.3°C)
- OR Tender Cervical Lympadenopathy of size > 2 cm
- OR Tonsillar exudate
- OR Culture positive for GABH (Group A β Hemolytic Streptococcus

ANTIBIOTIC

- With administration of *adequate dosing of Antibiotic* for proven or suspected GABH infection.

The American Academy of Otolaryngology – Head & Neck Surgery (AAO – HNS) Guidelines

Absolute indications for surgery:

- ***Enlarged tonsils*** that cause:
  - UAO; Dental malocclusion/ affecting orofacial growth
  - Severe dysphagia: Watch out for rebound over – feeding (& childhood obesity) post – tonsillectomy due to prior chronic starving. Advice caregiver on normal feeding.
  - Sleep disorders OR
  - Cardiopulmonary complications.

Cont.

- ***Peritonsillar abscess*** that is unresponsive to medical management & drainage documented by surgeon, unless surgery is performed during acute stage.
- Tonsillitis resulting in ***febrile convulsions***.
- Tonsils requiring ***biopsy*** to define tissue pathology.

The American Academy of Otolaryngology – Head & Neck Surgery (AAO – HNS) Guidelines

Relative indications for surgery:

- ≥ 3 tonsil infections/ yr. despite adequate medical therapy.
- Persistent foul taste or breath (halitosis) due to chronic tonsillitis that is not responsive to medical therapy.
- Chronic or recurrent contagious rhino – tonsillitis in a streptococcal carrier not responding to beta lactamase resistant antibiotics
- Unilateral tonsillar hypertrophy that is presumed to be neoplastic (unless proven otherwise by biopsy)
- Tonsillar cysts: may cause dysphagia or voice change

Contraindications

- Disorders of hemostasis (absolute) → the tonsils are highly vascularized

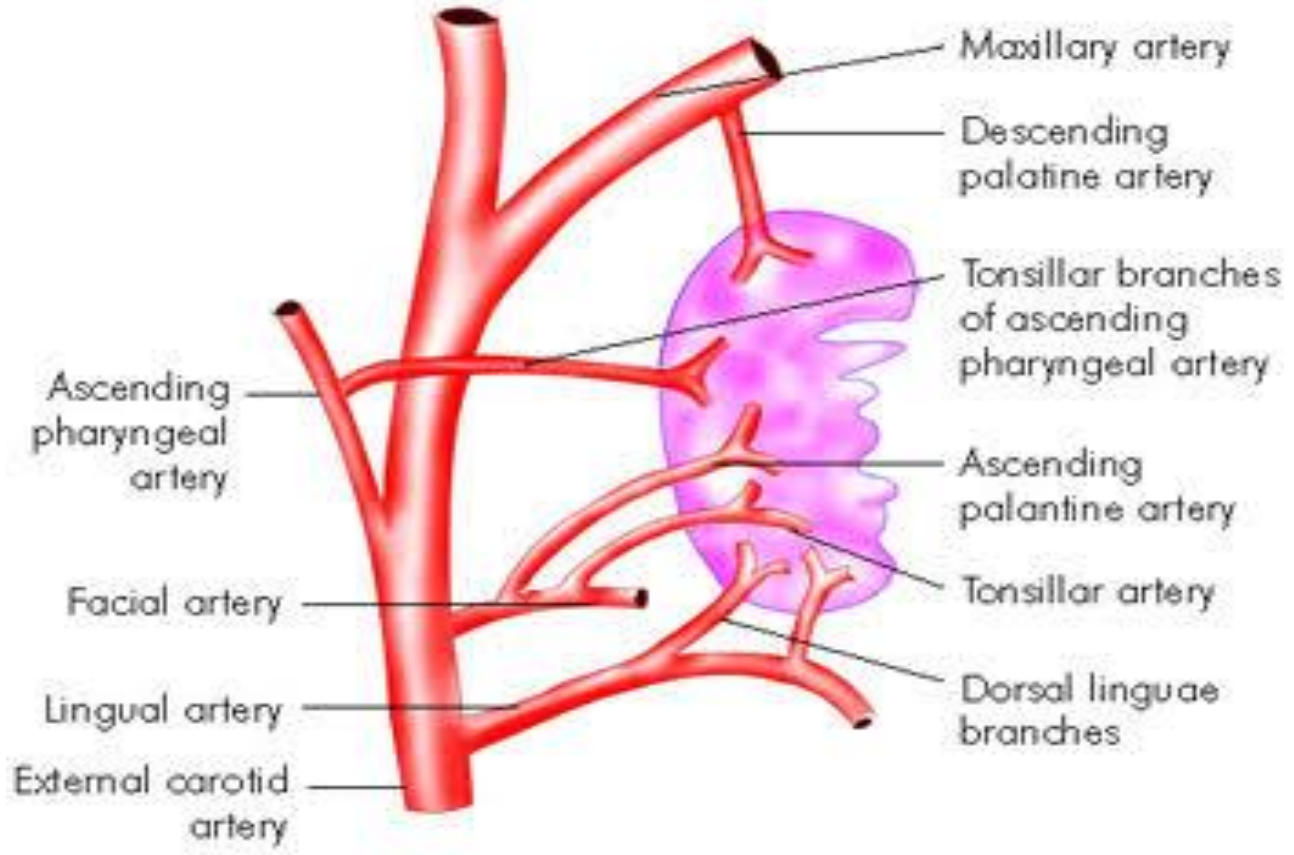


Fig. 50.3 Arterial supply of tonsil.

Cont.

- Acute infection
  - If the surgery is elective, post – pone until the acute infection resolves. This prevents super – infection of the surgical wound.
- Overt or sub mucous cleft palate (CP) – bifid uvula
  - Don't remove adenoids in a child with a cleft palate because of the risk of aggravating the **velo - pharyngeal incompetence** and causing **hyper - nasal speech** & **nasal regurgitation**

Cont.

- Velopharyngeal Insufficiency (VPI)
- Malignant hyperthermia: will react to anesthesia
- Neurologic or neuromuscular abnormality with impaired palatal function
- Anemia

Pre – operative evaluation

- History: bleeding disorder, comorbidities, speech development, dysphagia
- Physical examination: cleft palate, FTT, syndromic disease
- Laboratory investigations: FHG, bleeding time
- CXR (RVH), echo to see if they can withstand surgery.

Methods used

- ***Cold***
  - Dissection & snare
  - Guillotine method
  - Intracapsular (capsule preserving) tonsillectomy
  - Harmonic scalpel
  - Plasma – mediated ablation technique
  - Cryosurgical technique
- ***Hot***
  - Electrocautery (MC)
    - Mono – polar cautery
    - Bipolar cautery
  - Laser tonsillectomy (CO_2 or KTP)
  - Coblation tonsillectomy
  - Radiofrequency

Cold knife

- Gold standard
- Less post – operative pain
- Post – tonsillectomy hemorrhage less common than electro - cautery
- Least expensive
- More intra-operative blood loss
- Cause bacteremia
- Takes longer

Peri – operative management

- **Steroids**
- ***Bismuth/ Afrin***: Coagulant; Reduces likelihood of post-operative bleeding
  - Aspiration → death therefore not used in Kenya
- ***Post – operative antibiotics***
- ***Post – operative pain control***
  - Paracetamol
  - Narcotics
  - NSAIDs
  - Extremely severe in day 3 → day 7 therefore give tramadol.

Cont.

- Good hydration
- Move from liquid to semisolid to solid → as per patient's ability
- Avoid smoking → delays healing and predisposes to infections
- Avoid Valsalva maneuvers.

High risk groups after surgery

- < 3 years old
- Severe Obstructive Sleep Apnea (OSA)
- Bleeding disorders e.g. hemophilia, VWD, on warfarin
- ISS: High dose steroids, HIV, congenital
- Infections
- Fever
- Malnutrition < 80% of expected weight for age
- Cor – pulmonale: An episode or previous history

Immediate complications

- Hemorrhage (seen esp. in patients with mild bleeding disorders like VWD, thrombasthenia that go undiagnosed)
- UAO (laryngeal edema, clot)
- Pulmonary edema
- Dehydration (due to odynophagia post – op)
- Death – anesthetic complications
- Fever
- Vomiting

Cont.

- Grisel syndrome (common in down's syndrome; non – traumatic subluxation of the atlanto – axial joint caused by inflammatory ligamentous laxity; results in torticollis)
- Trauma (mostly due to instrumentation) – lips, TMJ, dental; nerve injury – CN IX (sits outside the tonsil capsule), taste affected; burns by cautery.
- Infection/ abscess

Late complications

- Nasopharyngeal stenosis
- VPI
- Regrowth of adenoid (20%)
- Pseudo aneurysm of ICA (sits just outside the tonsillar capsule),
- Eagles syndrome/ stylohyoid syndrome: (injury of styloid process)
- ET injury – patulous ET
- COME

TYPED BY NAILA KAMADI

I HEAR & I FORGET.

I SEE & I REMEMBER.

I DO & I UNDERSTAND.

- CONFUCIUS

JESUS IS SOVEREIGN

PHARYNGITIS

BY: DR. OMUTSANI

KNH

Definition

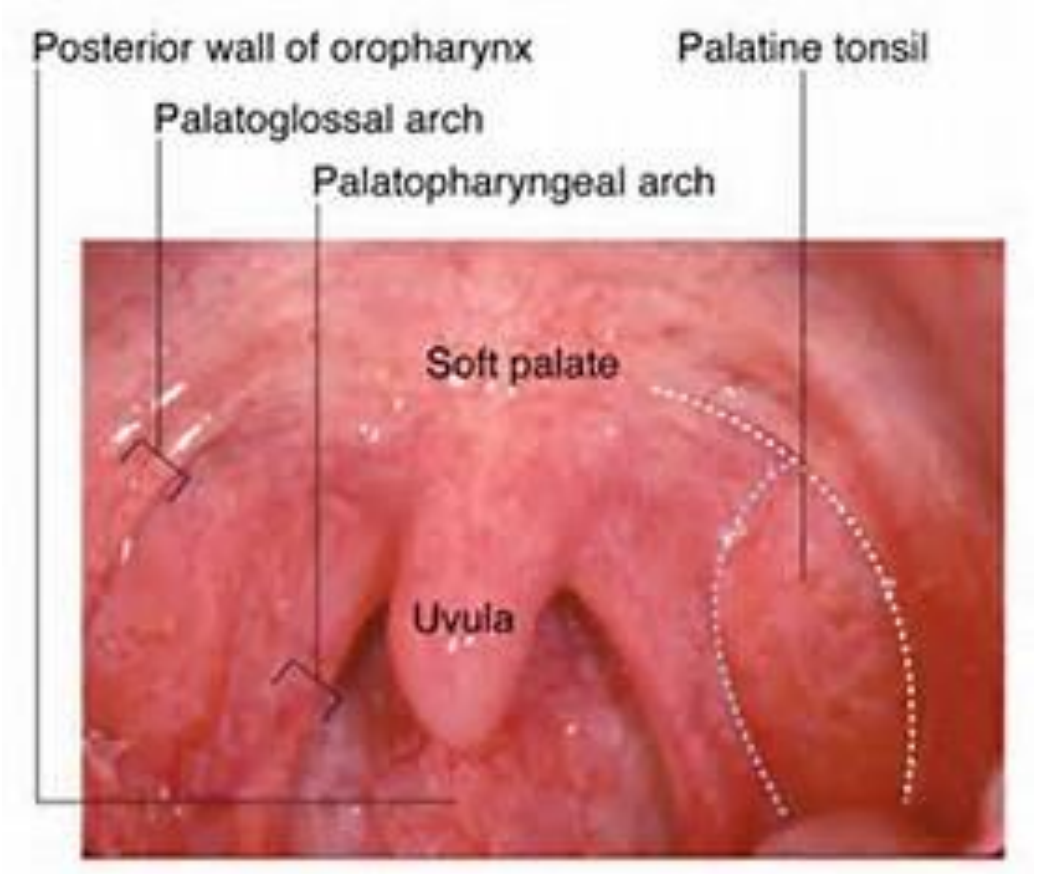
- Inflammation of the mucous membrane and submucosal structures of the pharynx

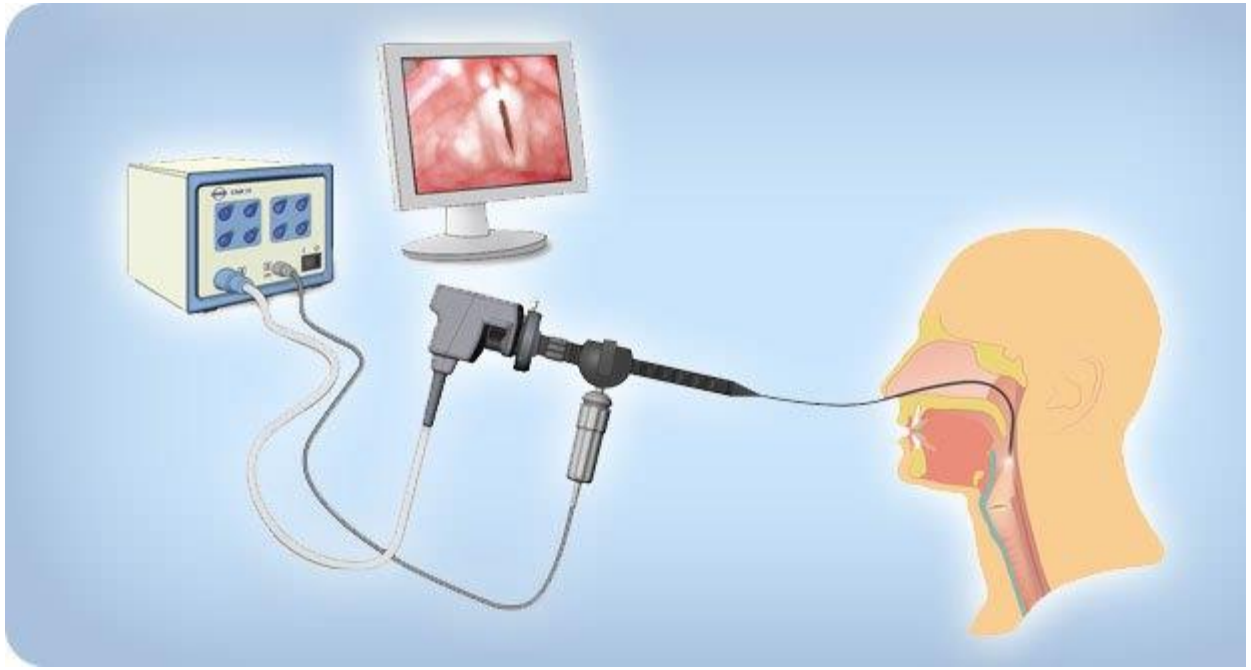
- Affects over 1.9 billion people world wide
- 15-25% children
- 8% adults
- Common consultation

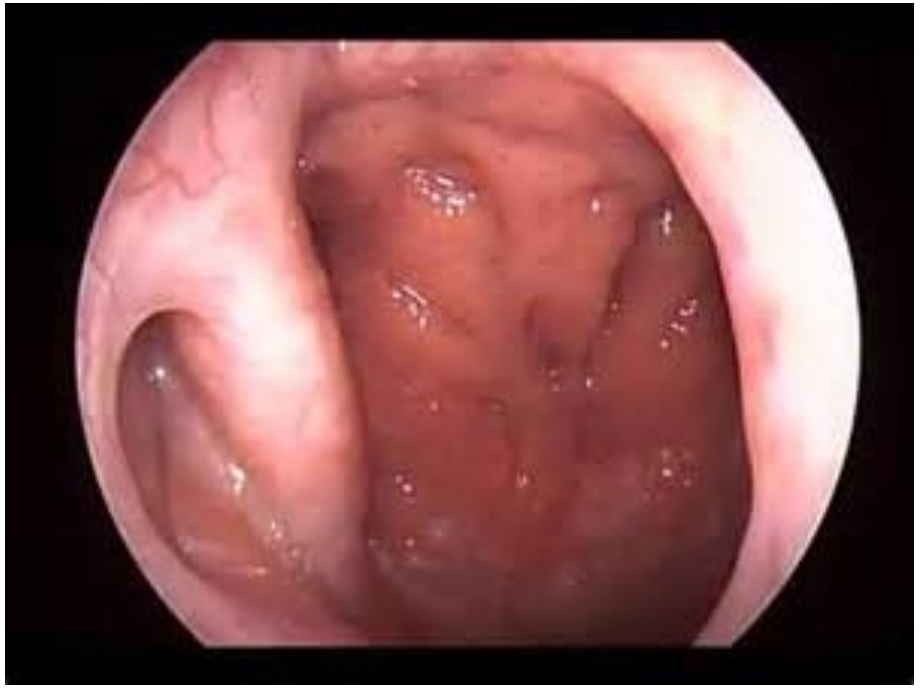
Anatomy of the pharynx

- Continuation of the digestive tract from the
- oral cavity
- • Funnel-shaped fibromuscular tube
- • Approximately 15 cm long
- • Common route for air and food

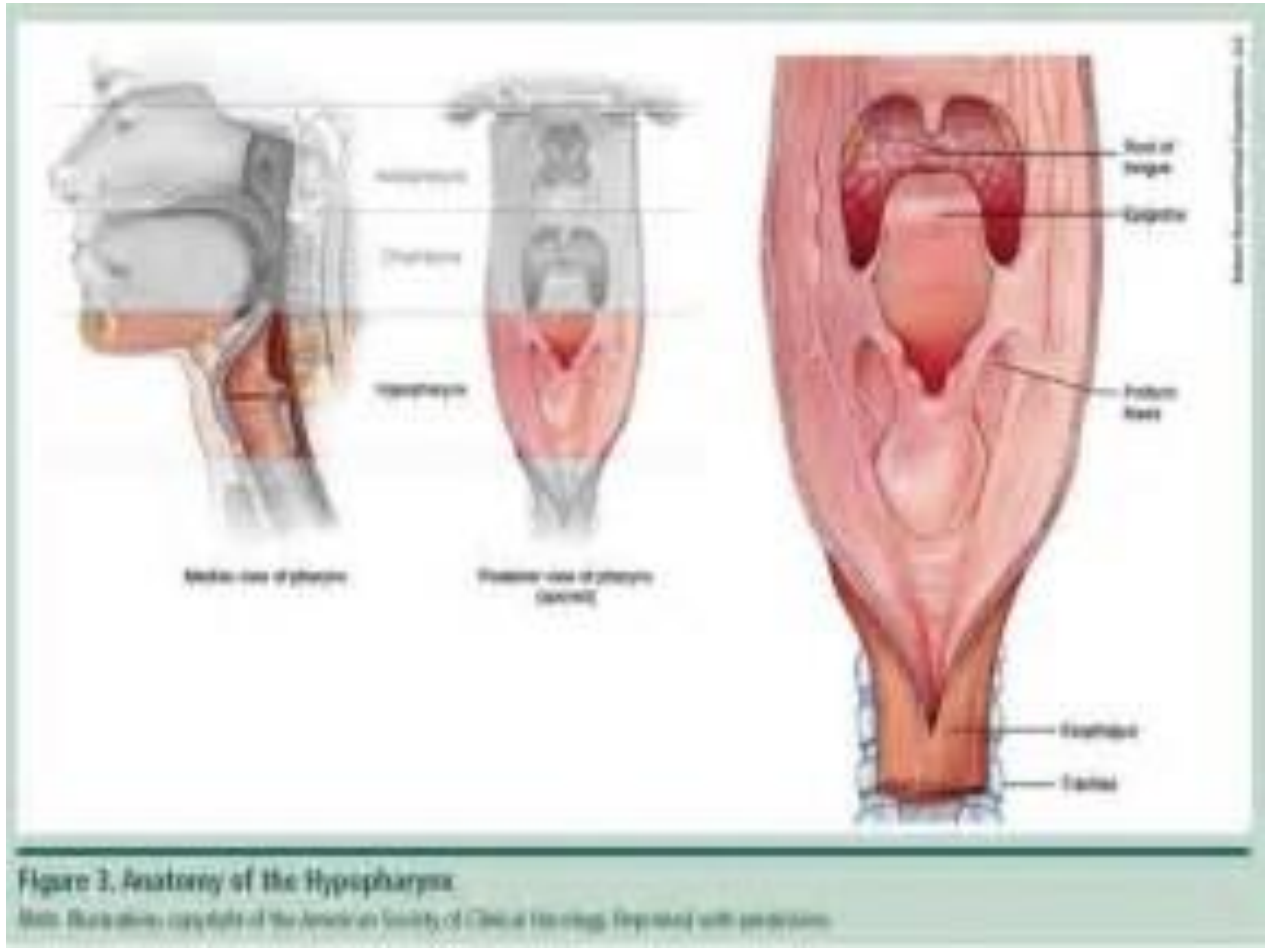
oropharynx

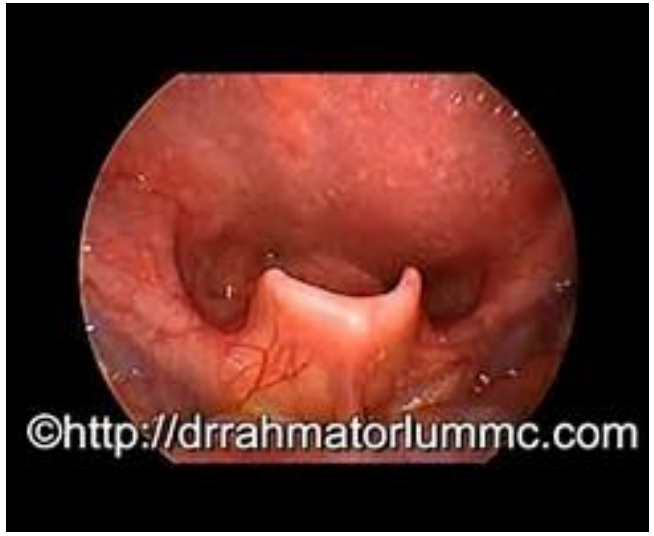

Posterior wall of oropharynx
Palatine tonsil
Palatoglossal arch
Palatopharyngeal arch
Soft palate
Uvula

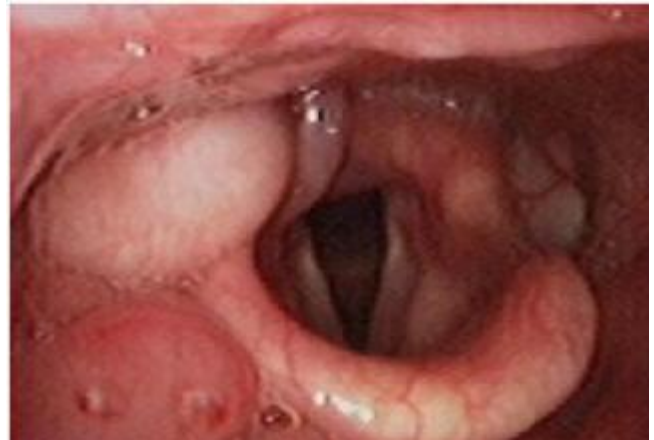




hypopharynx







Pharyngeal wall is composed of five layers

- Mucous membrane covered with pseudostratified
- ciliated epithelium superiorly and stratified squamous epithelium inferiorly
- submucosa
- fibrous layer forming pharyngobasilar fascia
- muscular layer (inner longitudinal and outer circular
- Loose connective tissue – the buccopharyngeal fascia

functions

- Airway
- Digestive
- Speech resonance
- Immune system

Classification

- Acute
- Chronic

- Acute recurrent
- Chronic with acute exacerbations

Pharyngeal mucosa exhibits an inflammatory response to many agents

- – infections
- – allergies
- - Laryngopharyngeal reflux
- – Environmental pollutants
- – Neoplasm
- – Granulomatous disease
- – Chemical and physical irritants
- Trauma (physical, burns)

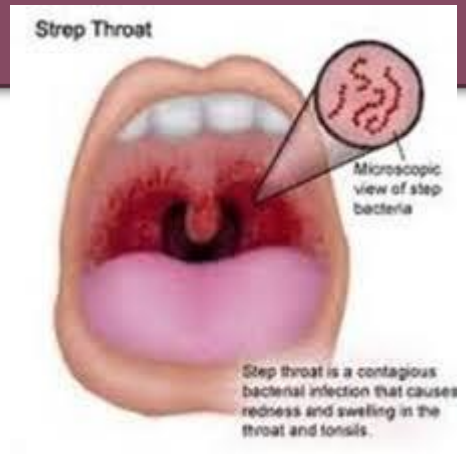
Pharyngitis-aetiology

- *Viral (42%)*- a) Adenovirus (most common 31%)
  b) Epstein –Barr virus(6%)
  c) Influenza virus(5%)
- *Bacterial* –Mixed infection common(48%)
  -beta-hemolytic streptococci(38%)
  -H. influenza
  -staphylococcus aureus
  -diphtheria
  -gonococcus
  -anaerobes remain uncertain.
- *Fungal* –Candida albicans.

viral

- Adenovirus –.
- Orthomyxoviridae which cause influenza –
- Infectious mononucleosis ("glandular fever") caused by the Epstein–Barr virus.
- Herpes simplex virus mouth ulcers.
- Measles
- Common cold: rhinovirus, coronavirus, respiratory syncytial virus, parainfluenza virus

bacterial



- Group A streptococcus
- *Streptococcus pneumoniae*
- *Haemophilus influenzae*
- ***M. cattaralis***
- *Bordetella pertussis*
- *Bacillus anthracis*
- *Corynebacterium diphtheriae*
- *Neisseria gonorrhoeae*
- *Chlamydophila pneumoniae*,
- *Mycoplasma pneumoniae*

fungal

- candida

- Diagnostic approach

history

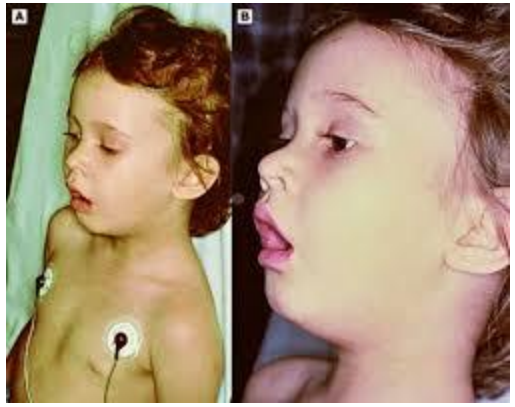
- Fever
- General malaise/body aches
- Headache
- Loss of appetite
- Cough
- Change in voice

history

- Sore throat
- Pain
- Odynophagia
- Dysphagia
- Cough
- Neck nodal swelling
- Difficulties in breathing

Physical exam

- Asses airway
- Temp
- Hydration status
- Head, ear, eyes, nose and throat
- Lymphadenopathy
- CVS
- Pulmonary assessment
- Abdominal exam
- skin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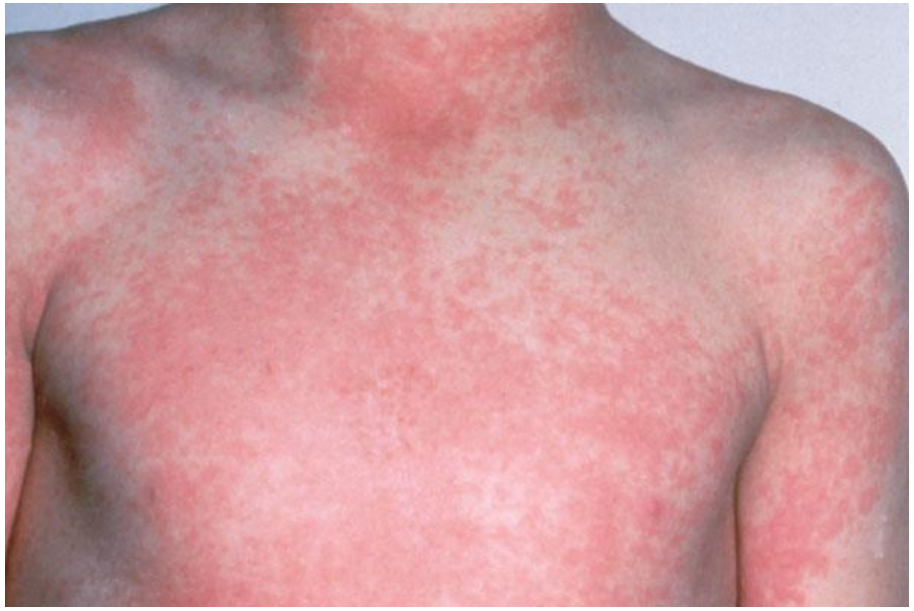

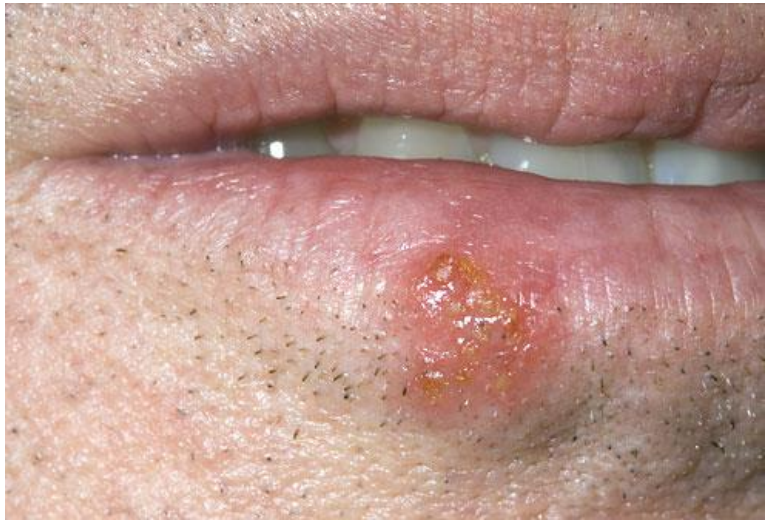

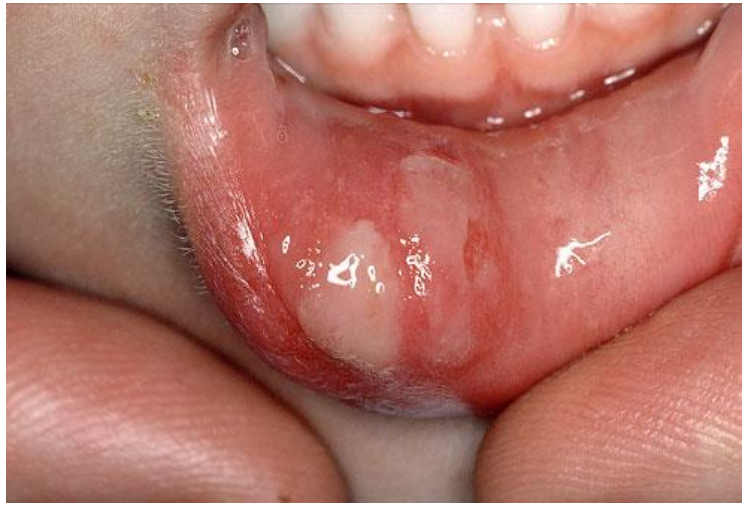

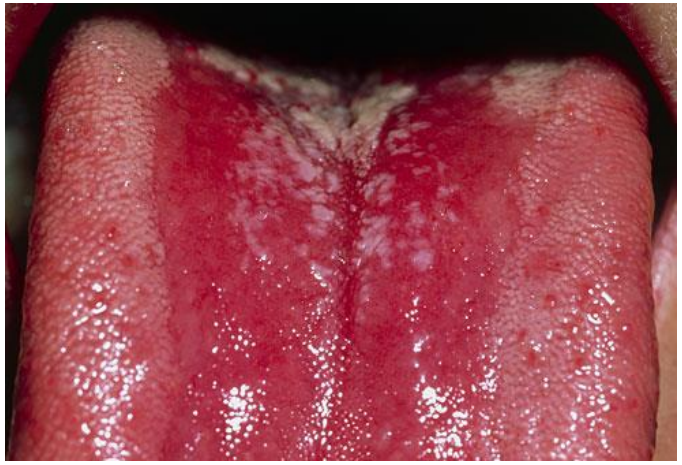

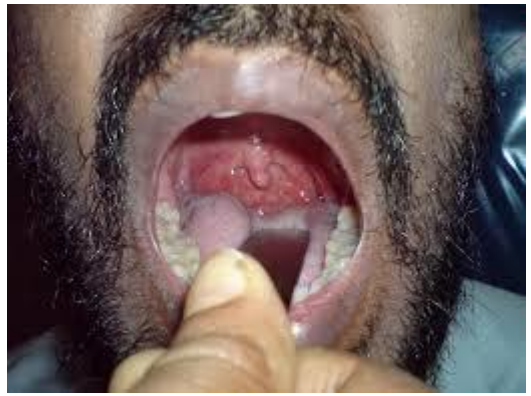

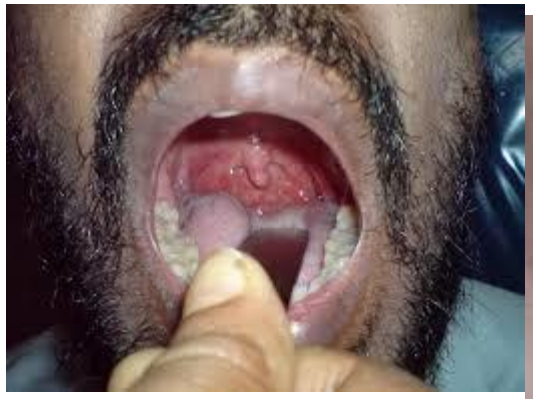

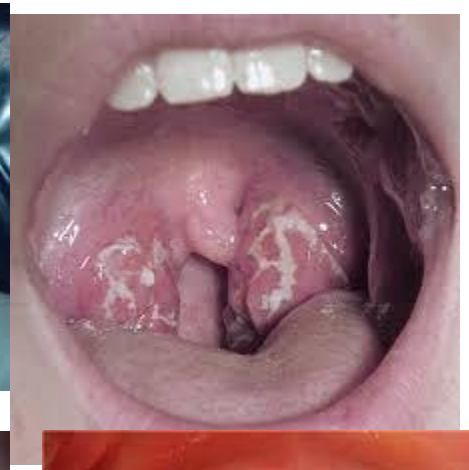

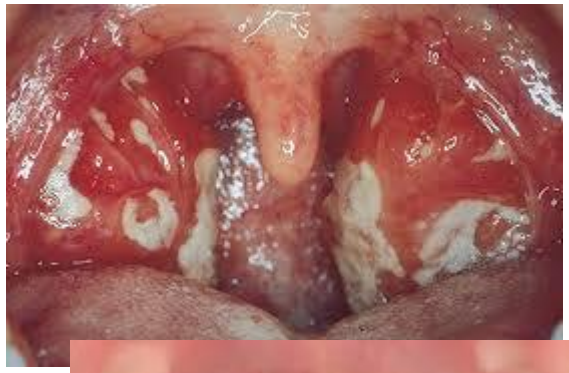


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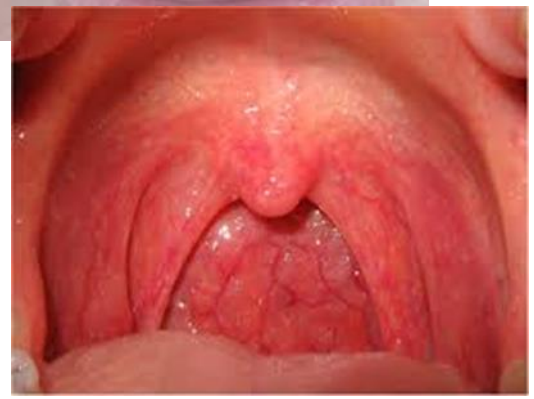
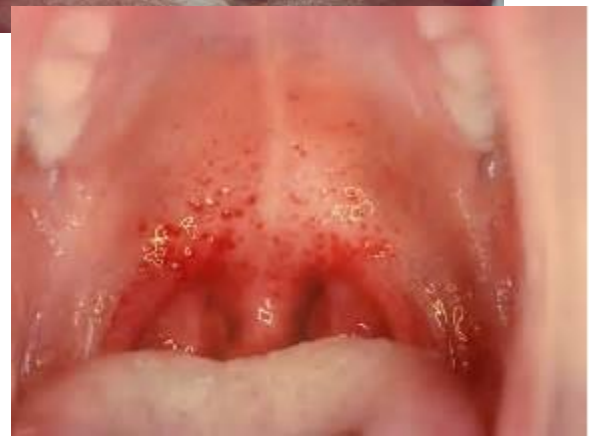
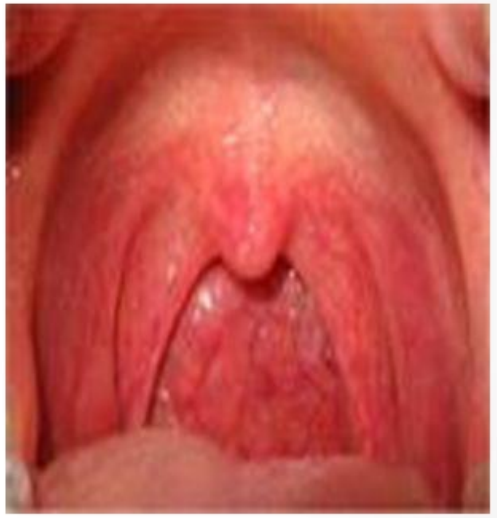
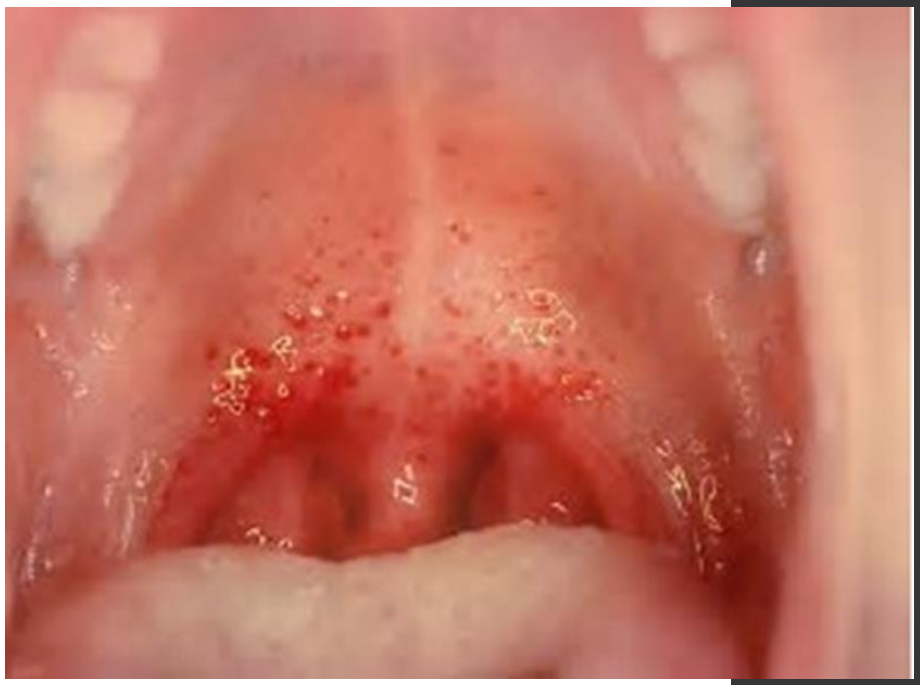




Viral Pharyngitis

- Gradual Sore throat
- Erythema, inflammation of pharynx and tonsils (may be slight)
- Vesicles or ulcers on tonsils
- Fever (usually low grade)
- Hoarseness, cough, rhinitis, conjunctivitis, malaise, anorexia
- Cervical lymph nodes may be enlarged, tender
- Usually lasts 3-4 days then resolves spontaneously





'Bull neck': a sign of diphtheria, due to enlarged lymph nodes in the neck

Lab diagnosis

- G A beta haemolytic streptococcal rapid antigen detection test (RADT)
- Throat culture
- Monospot (95% sensitive children less than 60% in infants)
- Peripheral smear
- G.C culture

imaging

- Not indicated unless

  lateral neck
- Suspected epiglotitis
- Air way concern
- Concern of neck abscess

management

Management Centor criteria

- Indication of probability of strept. Infection

	Score
Temperature ~>38°C	1
Absence of cough (as a cough is more likely to be associated with a viral infection)	1
Swollen tender anterior cervical lymph nodes	1
Tonsillar swelling or exudates	1
Age	
3–14 years	1
15–44 years	0
45 years or older	–1

management

- Analgesics
- Steroids
- Viscous lidocaine
- Antibiotics
- Oral analgesic solutions phenol, benzocain, menthol, cetylpyridinium
- Salt gaggles with bicarbonate

- 1st line

(penicillin)
  - amoxycillin
  - Erythromycin
  - macrolides

- 2nd line
  - augmented amoxycillin
- 3rd line
  - cephalosporins
- special

- Alternative
- Zinc
- Vitamin c
- Chicken soup
- Echinacea
- steam inhalation
- Pelargonium sidoides extract

- 2013 cochrane .no evidence
- Affects duration when taken early
- No effect on severity
- Side effects

- Vitamin C
- 1g/day no influence of common colds in a community
- 5 randomised double blind
  - Shortened
  - Decreased severity

echinacea

- Plant extract
- NO BENEFIT
- 2007 meta-analyses showed that it may reduce but studies are inconsistent
- 12

Chicken soup

- No evidence
- No medicinal value
- Encourages hydration

Steam inhalation

- 2006 systematic review there is insufficient support
- Burn accidents

- Singh M (2006). heated humidified air for common cold
- Akhavani (2006) steam inhalation treatment for children pead jounal of practice **55**(516):557


Google
GUIDELINES

- General hygienic practices

- Clean water
  - 50%

- Soap
  - 70%

- Antisceptic soap
  - 90%

Nose blowing

Prevention

Use of barrier

vaccination

- Diphtheria
- flu

- Prophylaxis
  - Penicillin
  - Sulfadiazine
  - macrolide

- The main concerns

  are more serious conditions like
  - Acute tonsillitis
  - Epiglotitis
  - Peritonsillar abscess
  - Retropharyngeal abscesses
  - Diphtheria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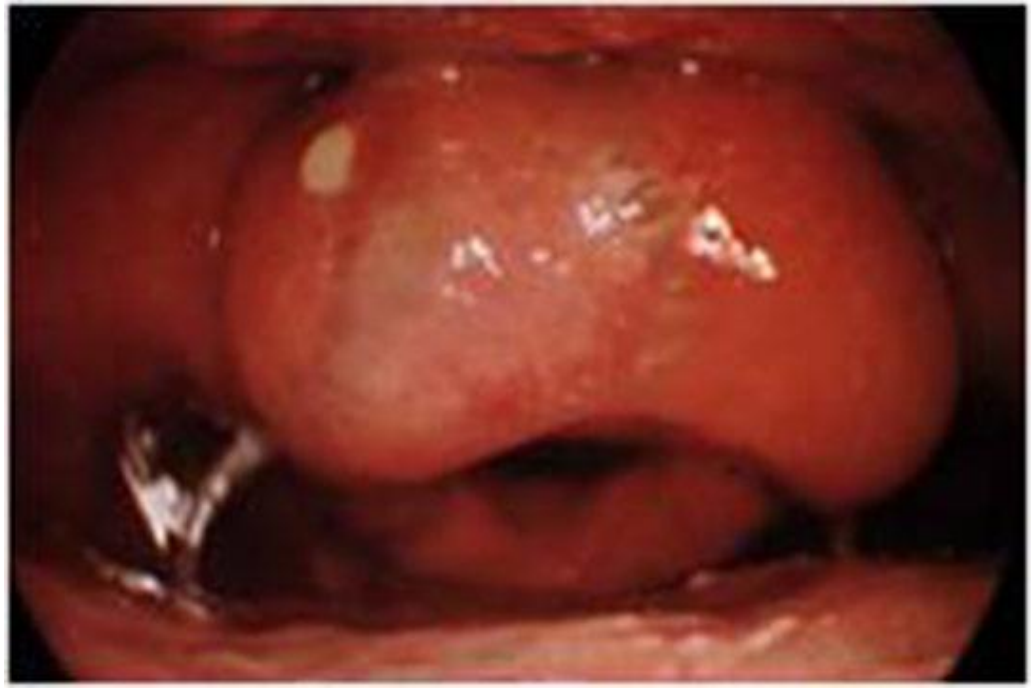

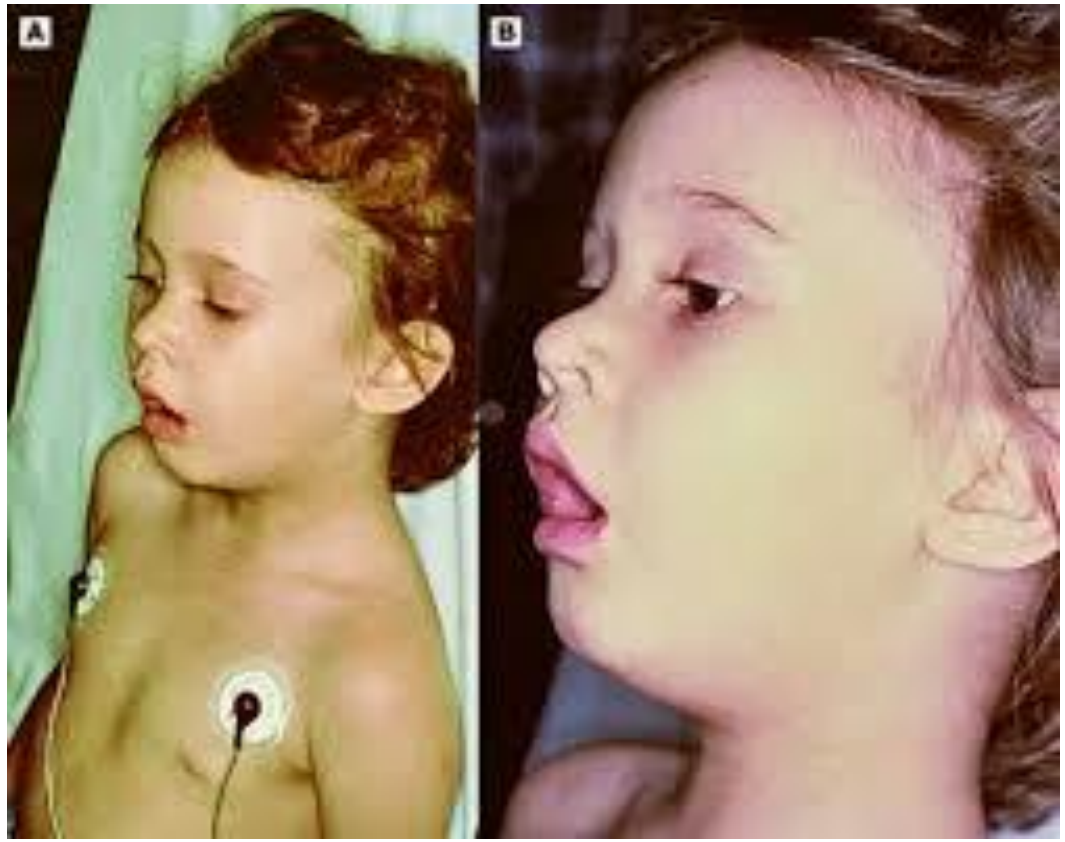
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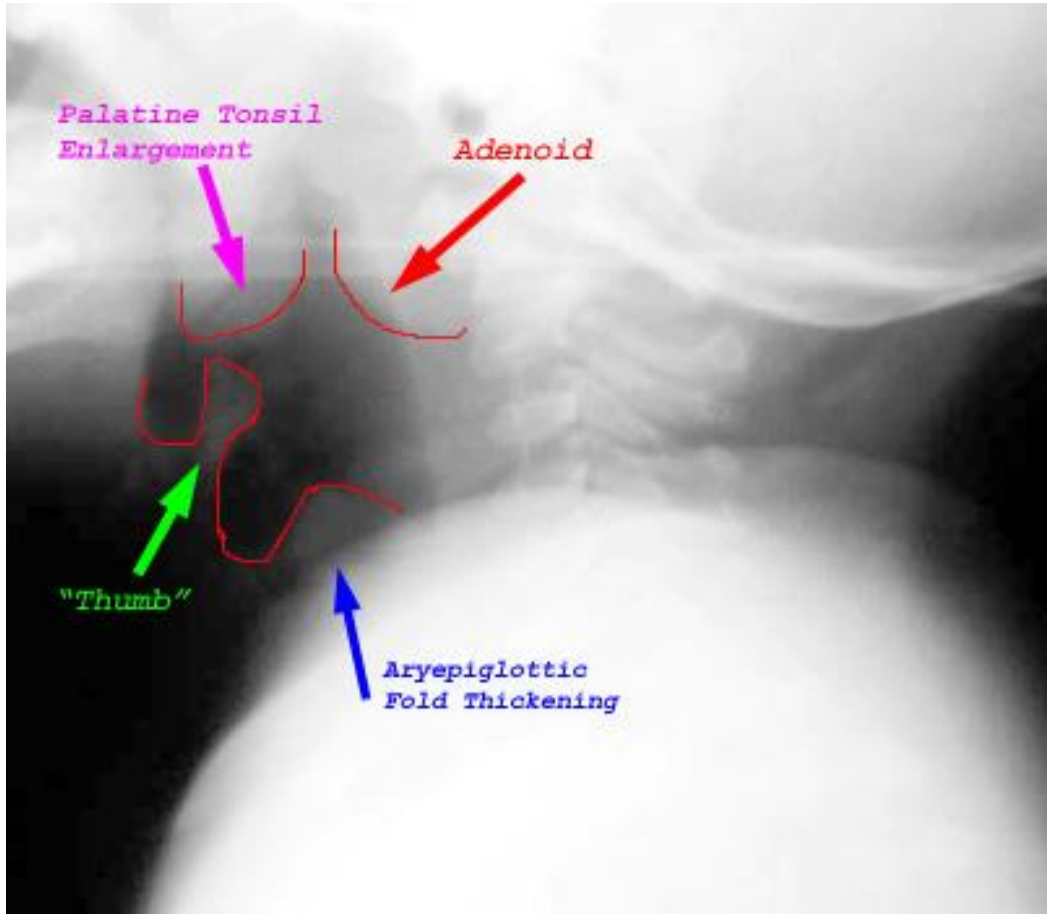
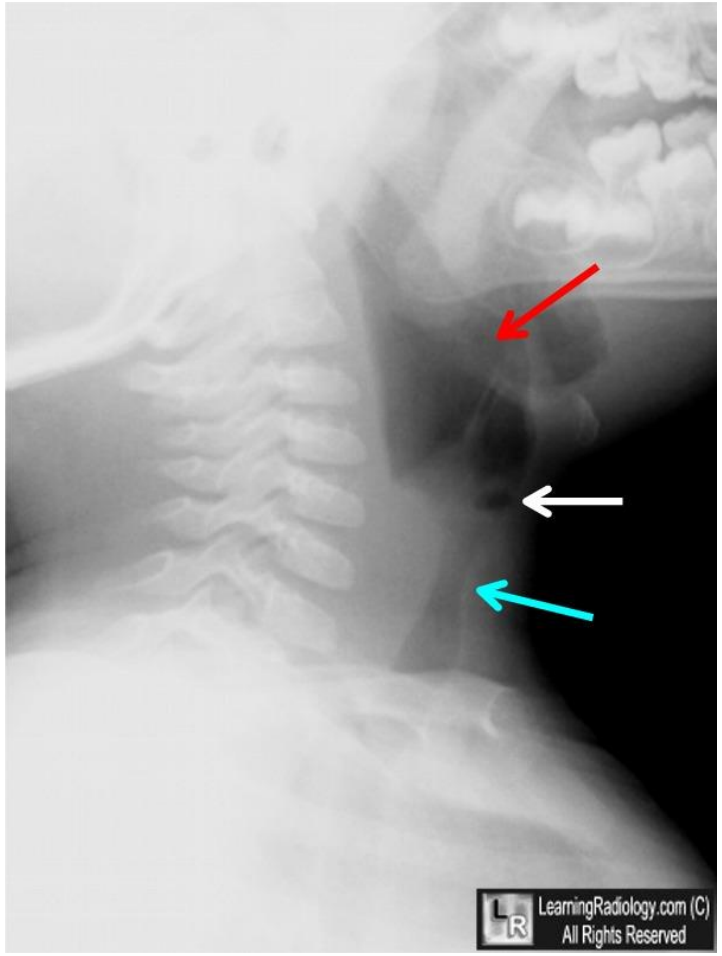

Palatine Tonsil
Enlargement
Adenoid
"Thumb"
Aryepiglottic
Fold Thickening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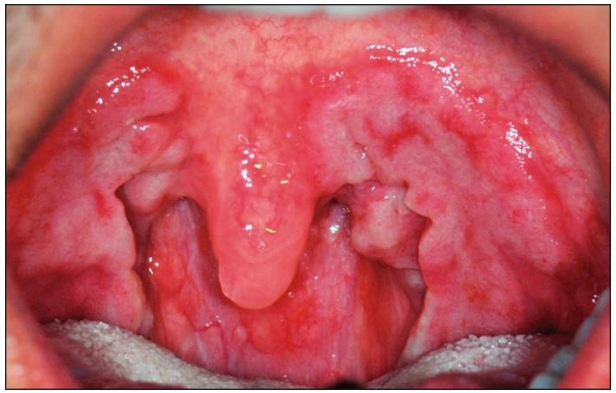
- Pts name Dr omutsani
- cough
- Throat irritation
- Temp 36.6 c
- Age 52yrs
- Feeding well



Strep Throat *(Streptococcal Pharyngitis)*

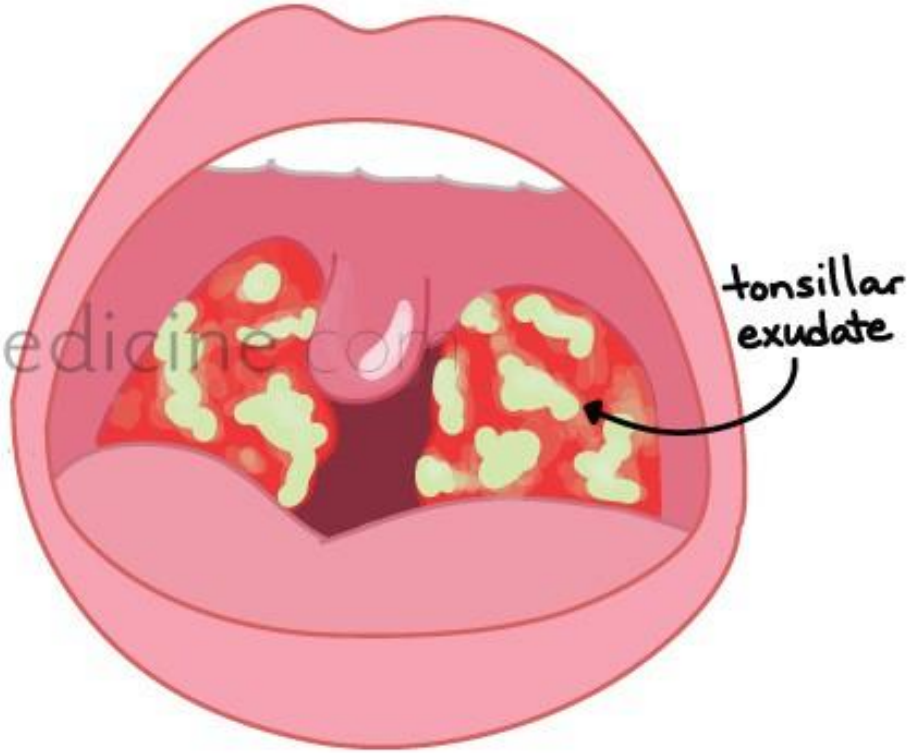
McIsaac Criteria

Centor Criteria

Add 1 point for each:

1. Fever (subjective or >38 C)
2. Ø cough
3. Tender lymphadenopathy (anterior cervical)
4. Tonsillar exudate

5. Age
   - 3-14 yrs: *Add 1 point*
   - 15-44 yrs: Ø
   - 45+ yrs: *Subtract 1 point*

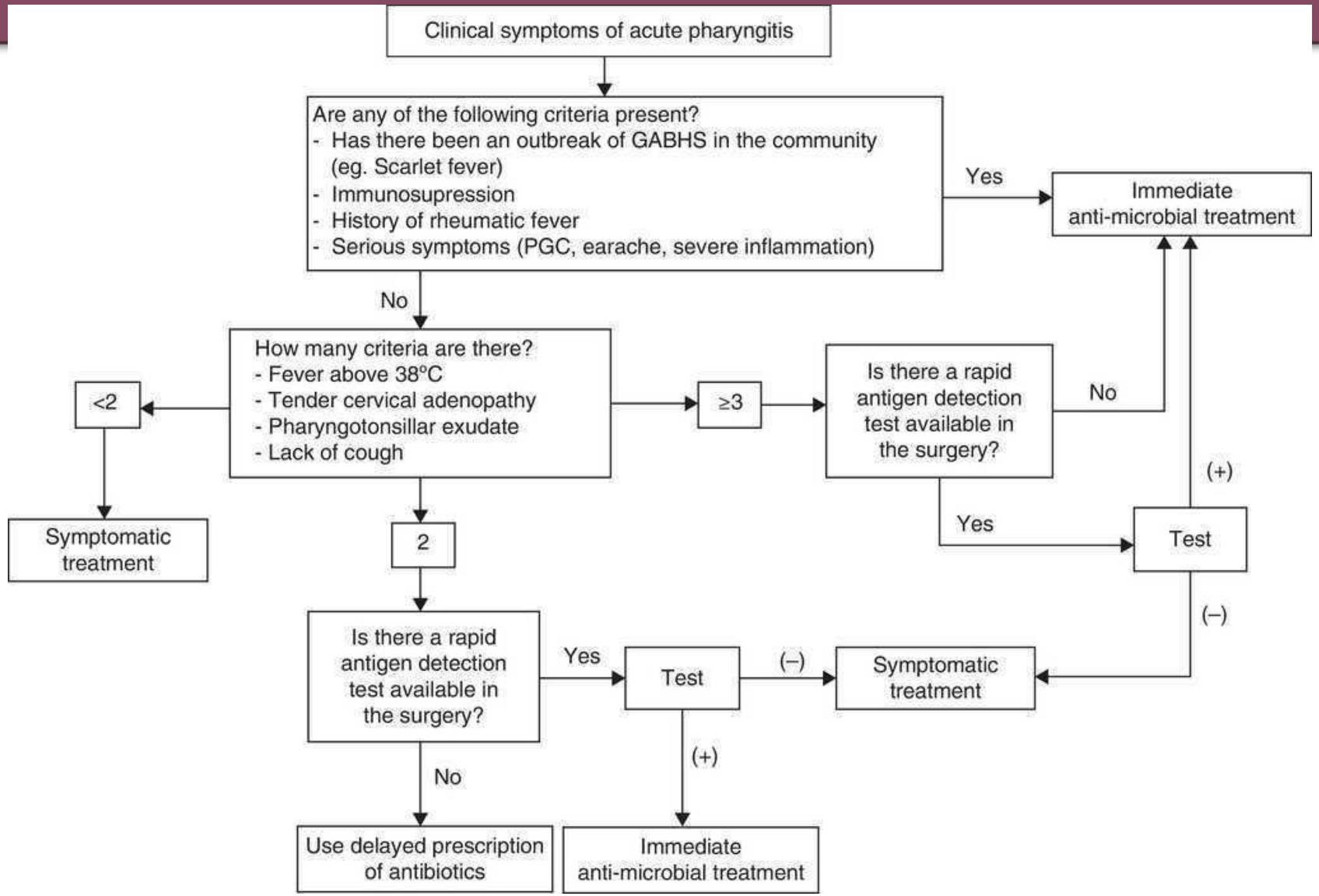


Total score

0-1 Unlikely strep pharyngitis

2+ Throat swab & culture or rapid antigen test

4+ Lab confirmation +/- empiric antibiotics


Clinical symptoms of acute pharyngitis
Are any of the following criteria present?
- Has there been an outbreak of GABHS in the community (eg. Scarlet fever)
- Immunosupression
- History of rheumatic fever
- Serious symptoms (PGC, earache, severe inflammation)
Yes
Immediate anti-microbial treatment
No
How many criteria are there?
- Fever above 38°C
- Tender cervical adenopathy
- Pharyngotonsillar exudate
- Lack of cough
<2
Symptomatic treatment
2
≥3
Is there a rapid antigen detection test available in the surgery?
No
Yes
Test
(+)
(–)
Symptomatic treatment
Is there a rapid antigen detection test available in the surgery?
Yes
Test
(–)
Symptomatic treatment
(+)
No
Use delayed prescription of antibiotics
Immediate anti-microbial treatment

LARYNGITIS

DR KIPINGOR M.K.
ENT SURGEON.

LARYNGITIS:

- Definition
- Aetiology
- Pathophysiology
- Diagnosis
- Work – up
- Treatment/management

Definition:

- Laryngitis – inflammation /swelling of the larynx
- Acute laryngitis – inflammation of abrupt onset/self limiting/symptoms < 3 weeks.
- Chronic laryngitis – symptoms > 3 weeks
- Associated with urti's

Aetiology:

- Voice abuse
- Infections – viral/bacterial
- Irritants – smoke/inhalers/alcohol
- Acid reflux
- Environmental pollutants
- allergy

Pathophysiology:

- Acute – oedema of vocal folds/hoarse voice/ aphonia.
- L/E – red swollen mucosal membranes/stiffened

Diagnosis:

- History:
- P/E : IL – swelling/hyperaemia/ exudate over vocal cords

Treatment:

- Steam inhalation/hydration
- Voice rest
- Avoidance of environmental factors – smoking
- Antibiotics
- Antacids
- mucolytics

Work – up

- CXR
- Laboratory tests
- IL/FLEXIBLE
- CULTURES

Chronic laryngitis:pathophysiology

- Irreversible alteration – laryngeal mucosa
- Persistent reactive/ repetitive process
- Changes to ciliated epithelium
- Mucus stasis - reactive cough
- laryngospasm

Chronic laryngitis:causes

- Smoking
- Alcohol
- GERD/LPR
- Infections – tb/viruses/bacteria/fungal
- Voice abuse
- Allergy
- Environmental factors – dust/fumes/chemicals
- Systemic disorders – autoimmune

ALLERGIC RHINITIS & NASAL POLYPOSIS
LEVEL VI MBCHB
2019

BY: DR. CATHERINE IRUNGU

TYPED BY NAILA KAMADI

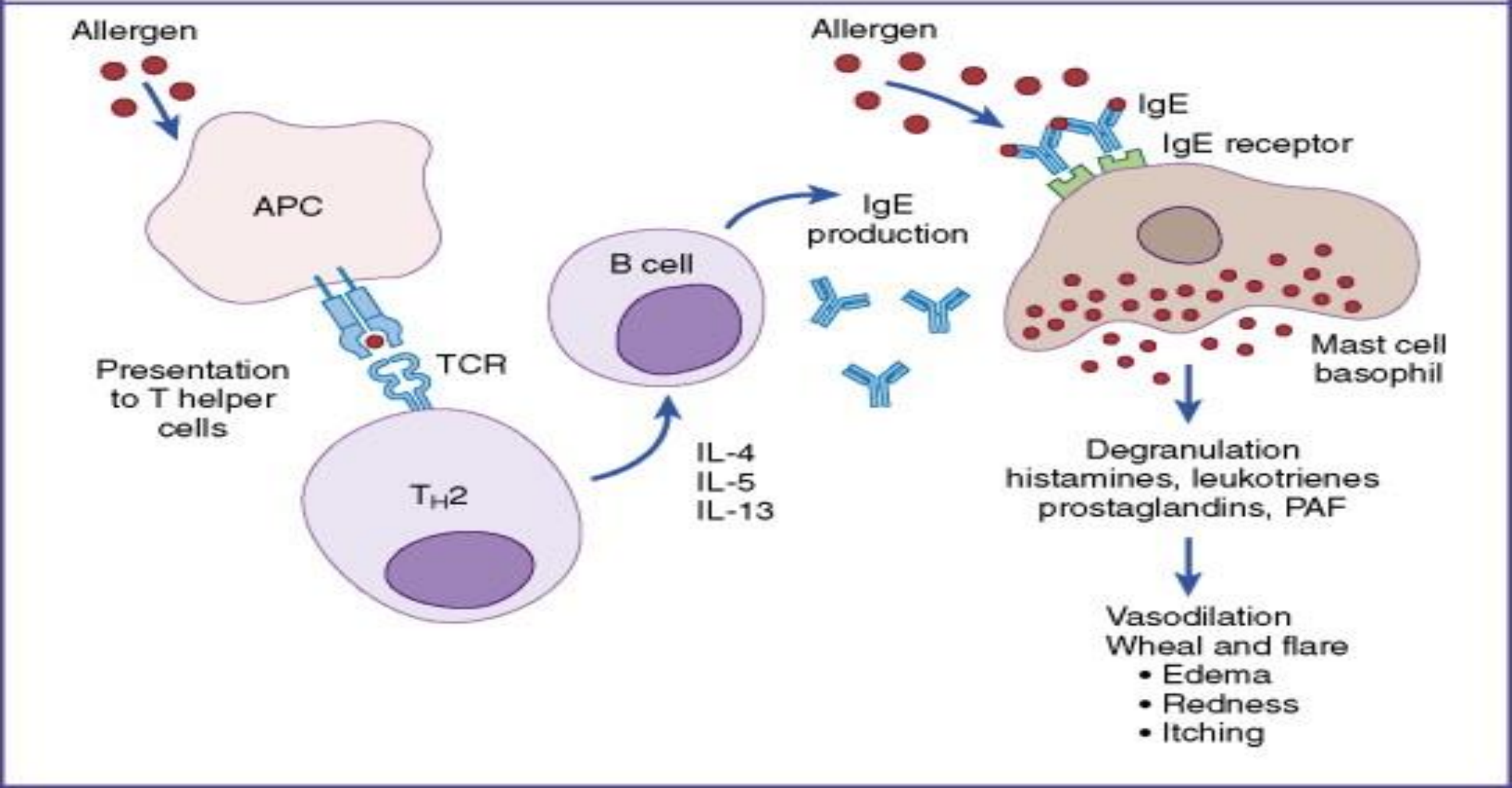
Definitions

- ***Hypersensitivity***: this is a disease state due to repeated exposure to antigen that entails an ***altered exaggerated/ excessive immune response***.
- ***Allergy***: altered state of reactivity due to contact with an organic substance

Types of hypersensitivity reactions

	Hyper – sensitivity reaction	Mediator	Examples
Type I	Immediate/ anaphylactic	IgE, Mast cell & Basophils	Allergic rhinitis
Type II	Cytotoxic	IgM and IgG	Drug induced hemolytic anemia
Type III	Immune complex	IgG	SLE, RA
Type IV	Delayed	T lymphocytes, monocytes	Mantoux reaction, TB, leprosy

Type I Anaphylactic Hypersensitivity (Acute)



Rhinitis

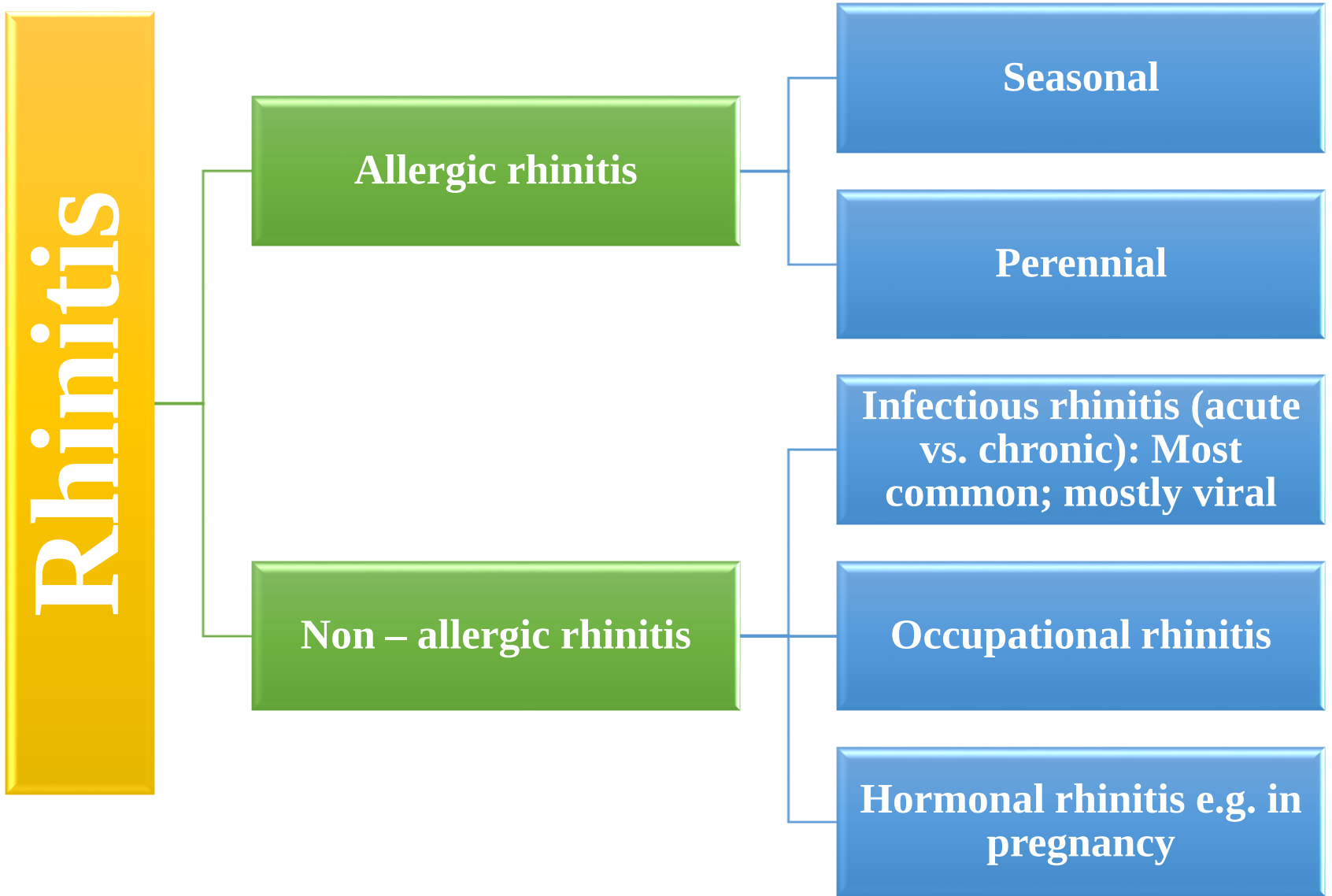
- This is inflammation of the mucus membranes of the nose.
- If the paranasal sinuses are involved → ***rhinosinusitis***
- Inflammation of the mucus membranes is characterized by interaction of inflammatory mediators.
- The ***eyes***; ***eustachian tubes*** & ***middle ear***; ***sinuses*** & ***pharynx*** may also be involved.

Allergic rhinitis

❖Triggered by ***IgE – mediated response*** to an extrinsic antigen causing:

- ✓Itching: nose, eyes, ears, plate, & sometimes the pharynx.
- ✓Sneezing.
- ✓Nasal congestion.
- ✓Rhinorrhea.

Classification of rhinitis



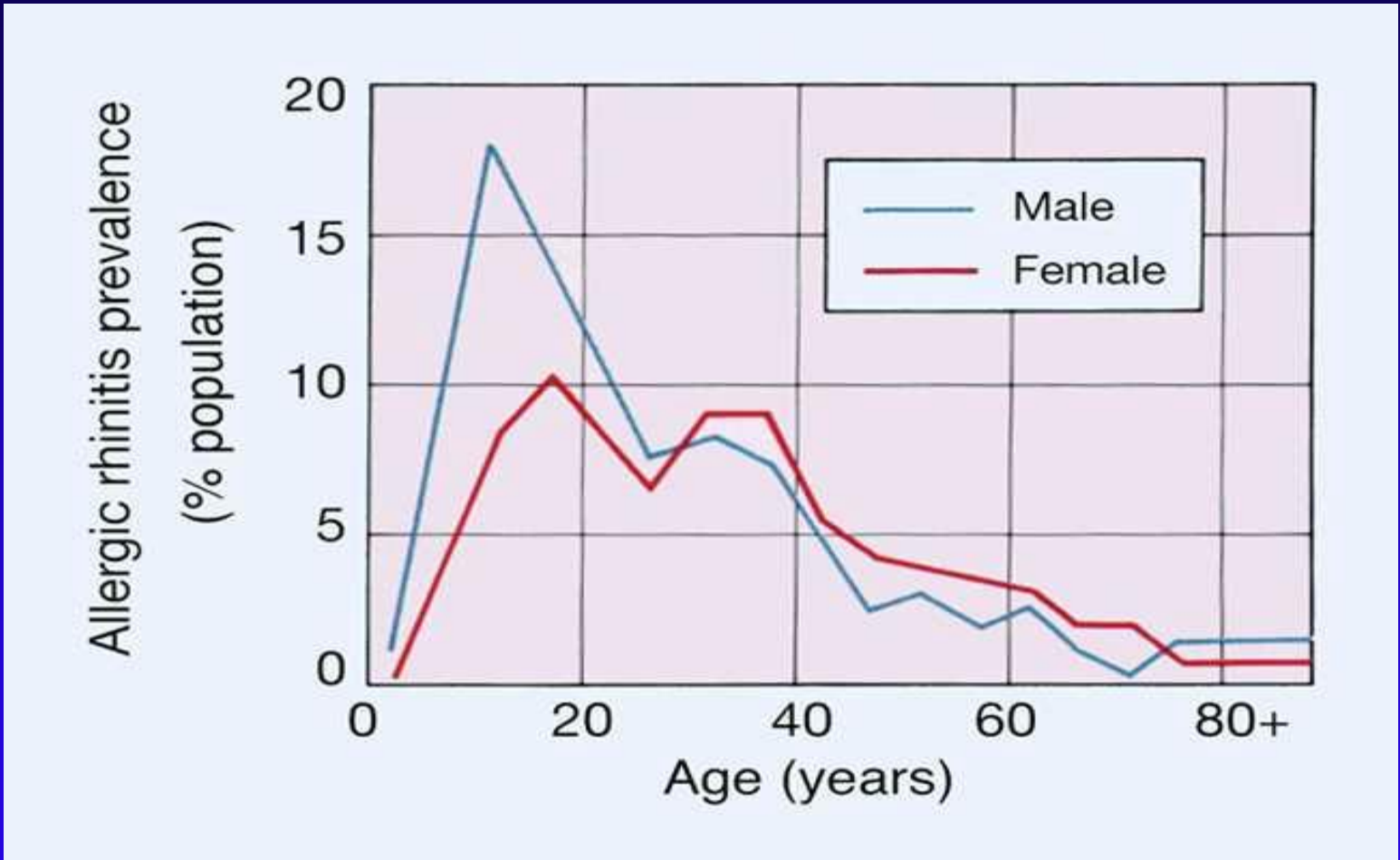
Cont.

- It is an inflammatory disorder driven by ***TH_2 lymphocytes*** that direct production of ***specific IgE*** by ***B lymphocytes*** via the production of ***interleukins 4, 5 & 13.***
- There is also an associated ***clonal expansion of eosinophils*** mediated by ***IL 5/ Eosinophil Differentiation Factor.***

Prevalence of allergic rhinitis by age

- More prevalent in the ***young***.
- Has a ***male preponderance*** but *as age increases prevalence in males drops but that in females remains high.*

Prevalence of Allergic Rhinitis by Age Group



Clinical assessment & classification

	SNEEZERS/ RUNNERS	BLOCKERS
Sneezing	Esp. paroxysmal	Little or none
Rhinorrhea	Watery – anterior & posterior	Thick mucus, more posterior
Itching	Yes	No
Blockage	variable	Often severe
Diurnally	Worse during the day; Improving at night	Constant; may worsen at night
Conjunctivitis	Often present	

Allergic Rhinitis & its Impact on Asthma (ARIA) Classification in untreated patients.

Intermittent
Symptoms
<4 days per week
or <4 consecutive weeks

Persistent
Symptoms
≥4 days per week
and ≥4 consecutive weeks

Mild
all of the following:
1) Normal sleep
2) No impairment of daily activities
3) No impairment of work/school
4) Symptoms present but not troublesome

Moderate – Severe
one or more of:
1) Disturbed sleep
2) Impairment of daily activities
3) Impairment of work/school
4) Troublesome Symptoms

Allergic rhinitis & asthma.

- Asthma & allergic rhinitis share a common inflammatory process.
- Up to 80% of asthmatic patients have allergic rhinitis.
- Allergic rhinitis may complicate asthma management.
- The treatment strategy should address both the upper and lower airway.

Determinants of allergy

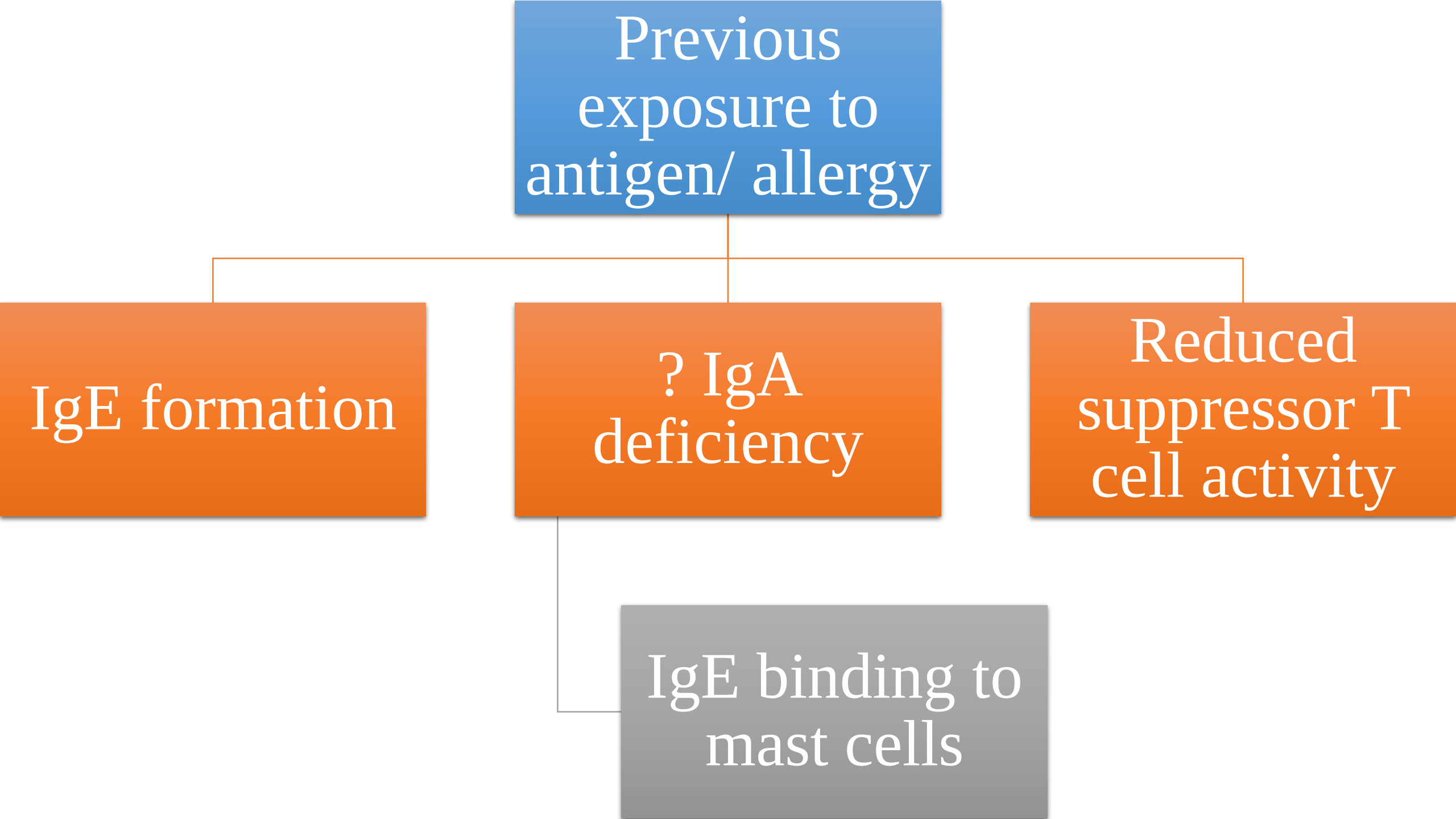
- Genetics
- Environmental factors.
  - ✓Lifestyle related
  - ✓Occupational factors.

Allergens

- Inhaled:
  - ✓Dust mite droppings
  - ✓Pollen
  - ✓Animal dander
  - ✓Fungal spores
  - ✓Wood
  - ✓Cockroach
- Ingested
  - ✓Eggs
  - ✓Poultry
  - ✓Milk
- Perennial
  - ✓Dust mites
  - ✓Pets
  - ✓Cockroaches
  - ✓Mice
- Seasonal: pollen

Risk factors for allergic rhinitis

- Positive family history
- Male gender
- Higher socioeconomic class (Hygiene hypothesis)
- 1st born
- Maternal asthma
- Serum total IgE > 100iU/l


Previous exposure to antigen/ allergy
IgE formation
? IgA deficiency
Reduced suppressor T cell activity
IgE binding to mast cells

Cont.

- Allergic rhinitis is an inflammatory disorder driven by TH_2 lymphocytes
  - ✓Directs production of specific IgE produce IL4, 5
  - ✓IgE production by B lymphocytes
  - ✓Clonal expansion of eosinophils

2 phases

- Sensitization phase
- Reactive phase
  - ✓Early
  - ✓Late

Sensitization phase

- Susceptible individual is repeatedly exposed to an environmental allergen over time.
- Allergen is processed & presented to TH_2 cells by ***dendritic cells***.
- TH_2 cells release mediators (IL4 & 5) which stimulate an allergic type response
  - ✓The key cytokine in this phase is IL4.
- Therapy: ***mast cell stabilizers***

Early reactive phase

- Allergen contacts IgE on the mast cell surface
- Results in degranulation & release of:
  - ✓Histamine
  - ✓Kinins
  - ✓Leukotrienes
  - ✓Prostaglandins
- Therapy: ***antihistamines & LT antagonists***

Mediators act on several sites in the nose including:

- Trigeminal nerve receptors: sneezing, pruritus.
- Vascular receptors: vasodilation, edema, nasal blockage.
- Mucus glands: rhinorrhea
- There is a role of NO.

Late reactive phase

- Occurs 3 – 8 hrs. later & only in 50% of patients.
- Cytokines, chemokines & LTs released from mast cells and T cells ***up – regulate adhesion molecules on vascular endothelial cells***.

Cont.

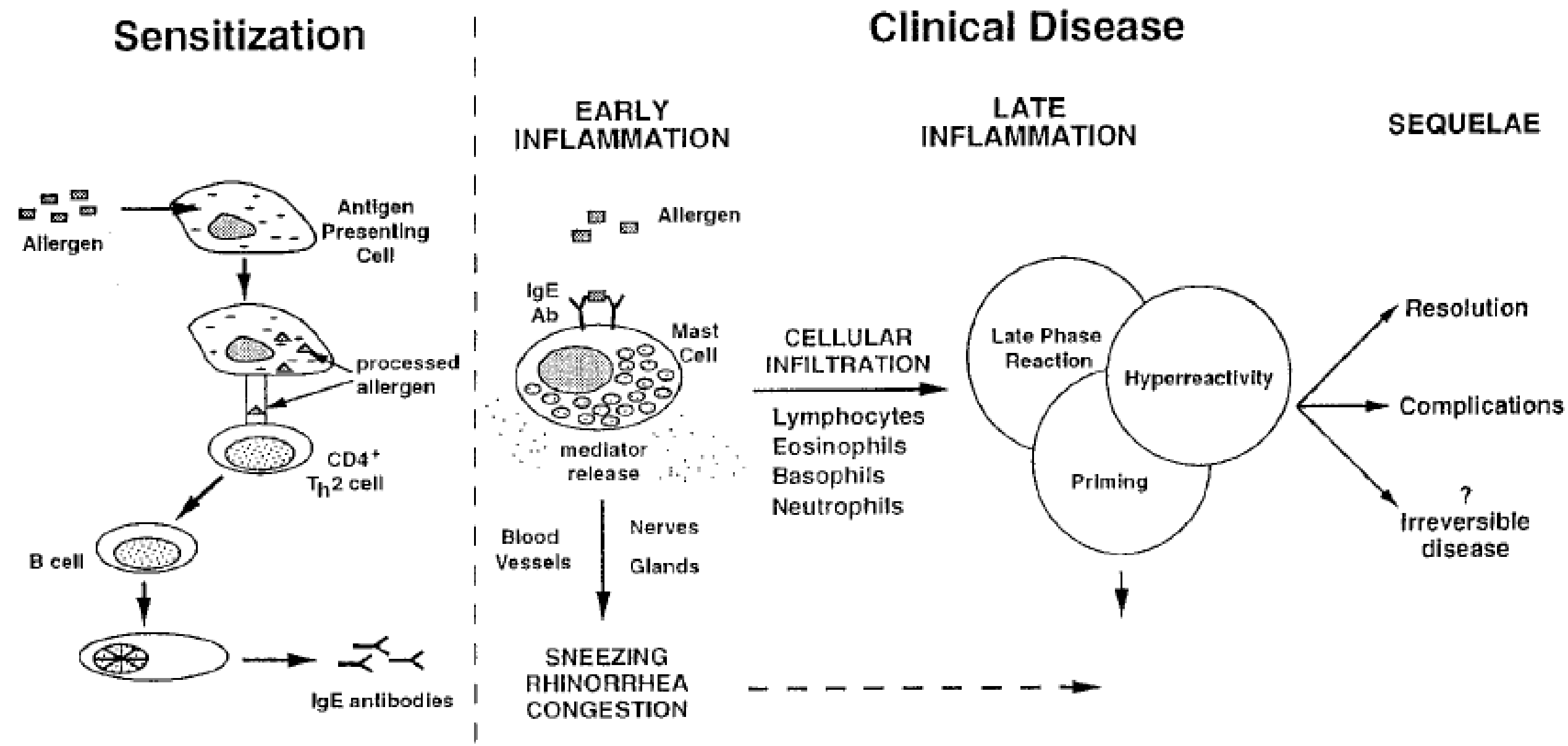
- Eosinophils, neutrophils, lymphocytes & basophils stick to epithelium & migrate into tissues.
- Cells release own mediators which increase sensitivity to smaller amounts of allergen and non – specific irritants.

Pathophysiology: "**RUNNERS**"

- Mediators of inflammation result in:
  - ✓Vasodilation
  - ✓Mucus production
  - ✓Edema
  - ✓Neural stimulation (trigeminal nerve receptors)
- Early acute inflammation results in:
  - ✓Profuse watery rhinorrhea
  - ✓Sneezing
  - ✓Itching

Pathophysiology: "**BLOCKERS**"

- Late phase mediators & ongoing inflammation results in:
  - ✓Persistent nasal congestion
  - ✓Posterior mucus discharge
  - ✓Obstruction of osteomeatal complex (OMC)
  - ✓Mucosal hypertrophy

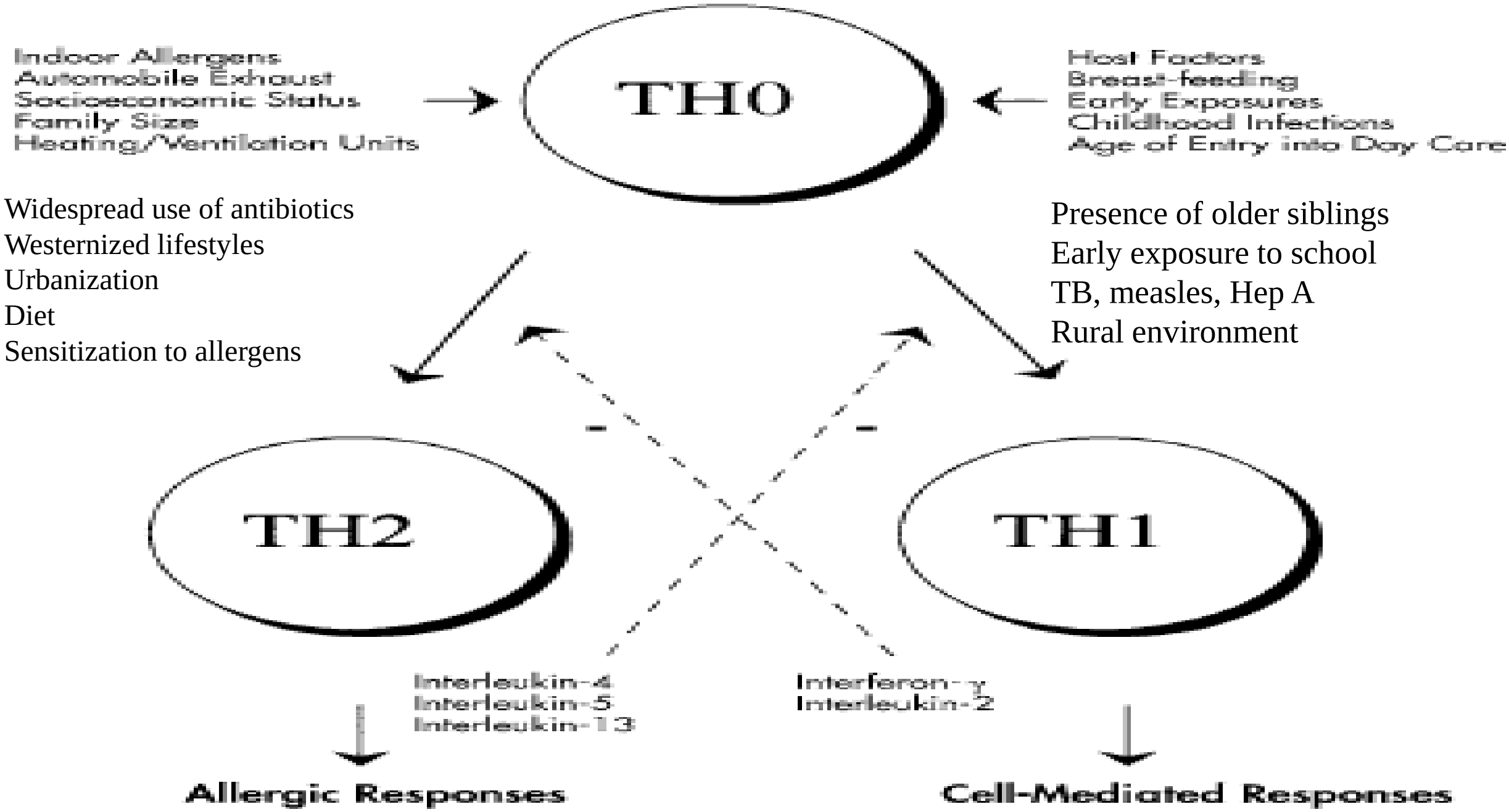

Sensitization
Clinical Disease
EARLY INFLAMMATION
LATE INFLAMMATION
SEQUELAE
Allergen
Antigen Presenting Cell
processed allergen
CD4+ Th2 cell
B cell
IgE antibodies
Allergen
IgE Ab
Mast Cell
mediator release
Blood Vessels
Nerves
Glands
SNEEZING
RHINORRHEA
CONGESTION
CELLULAR INFILTRATION
Lymphocytes
Eosinophils
Basophils
Neutrophils
Late Phase Reaction
Hyperreactivity
Priming
Resolution
Complications
?
Irreversible disease

The ‘one airway’ hypothesis

- Upper airway inflammation results in lower airway inflammation.
- Mediated in part by ***cholinergic stimulation of bronchoconstriction***
- Plays a role in exercise induced asthma
- Treatment of AR (allergic rhinitis) markedly improves asthma signs & symptoms

Allergy gene – IL4

- Key cytokine in allergic inflammation.
- Genetic abnormality in IL – 4 gene → ↑ TH_2 activity & ↑ IgE synthesis & secretion by B lymphocytes
- ↑ ratio of TH_2: TH_1 lymphocytes (cytokine priming)
- Up – regulates IgE receptors on:
  - ✓B lymphocytes
  - ✓Mononuclear phagocytes
  - ✓Mast cells (IL4 is a growth factor for mast cells)
  - ✓Basophils
- IgE dependent mast cell activation induced by IL – 4
- IL4 possibly works with IL5 as a growth factor for eosinophils.



Widespread use of antibiotics
Westernized lifestyles
Urbanization
Diet
Sensitization to allergens

Presence of older siblings
Early exposure to school
TB, measles, Hep A
Rural environment

Genetic factors associated with asthma

- Predisposition to develop IgE mediated responses to common allergens
  - ✓Genetic abnormality in the IL4 gene leading to increased TH2 activity and increased IgE synthesis
- Other genes:
  - ✓Cytokines, IL4 receptor, IFN γ, TARC genes, β adrenergic receptor, LT genes.

Hygiene hypothesis

- Chronic exposure to allergens in genetically susceptible individuals
- This allergen exposure may begin in utero via trans placental leakage of allergens.
- Lack of exposure to certain infections: *H. pylori*, endotoxin, TB, measles, toxoplasmosis, hepatitis shifts cytokine profile to an allergic one (TH_2)
- Similar correlates have been seen for other hypersensitivity (autoimmune) disorders such as MS, type I DM.

Factors favoring the Th1 phenotype

Presence of older siblings

Early exposure to day care

Tuberculosis, measles, or hepatitis A infection

Rural environment

Factors favoring the Th2 phenotype

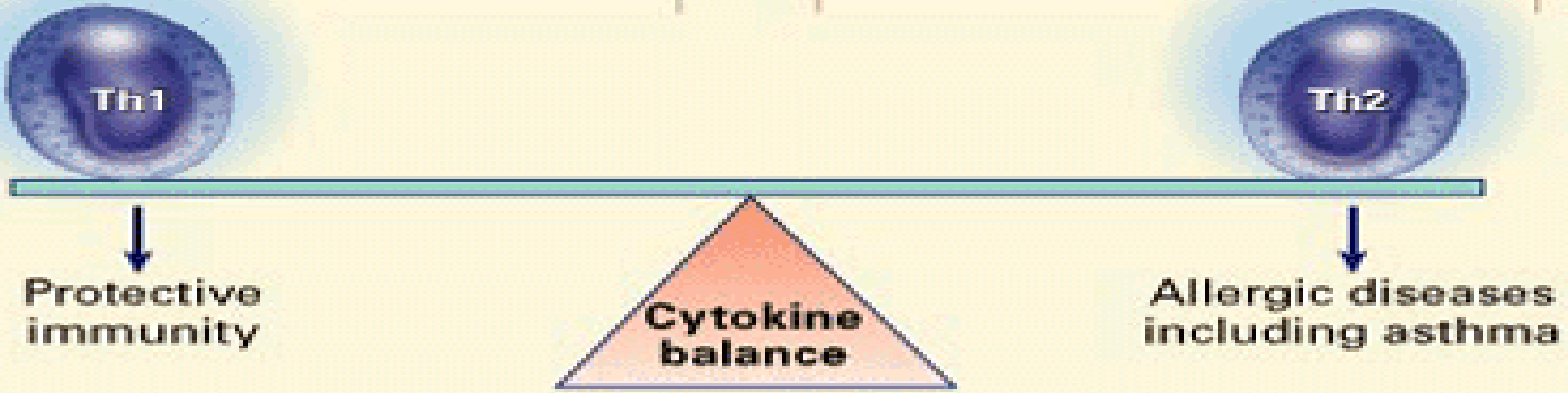
Widespread use of antibiotics

Western lifestyle

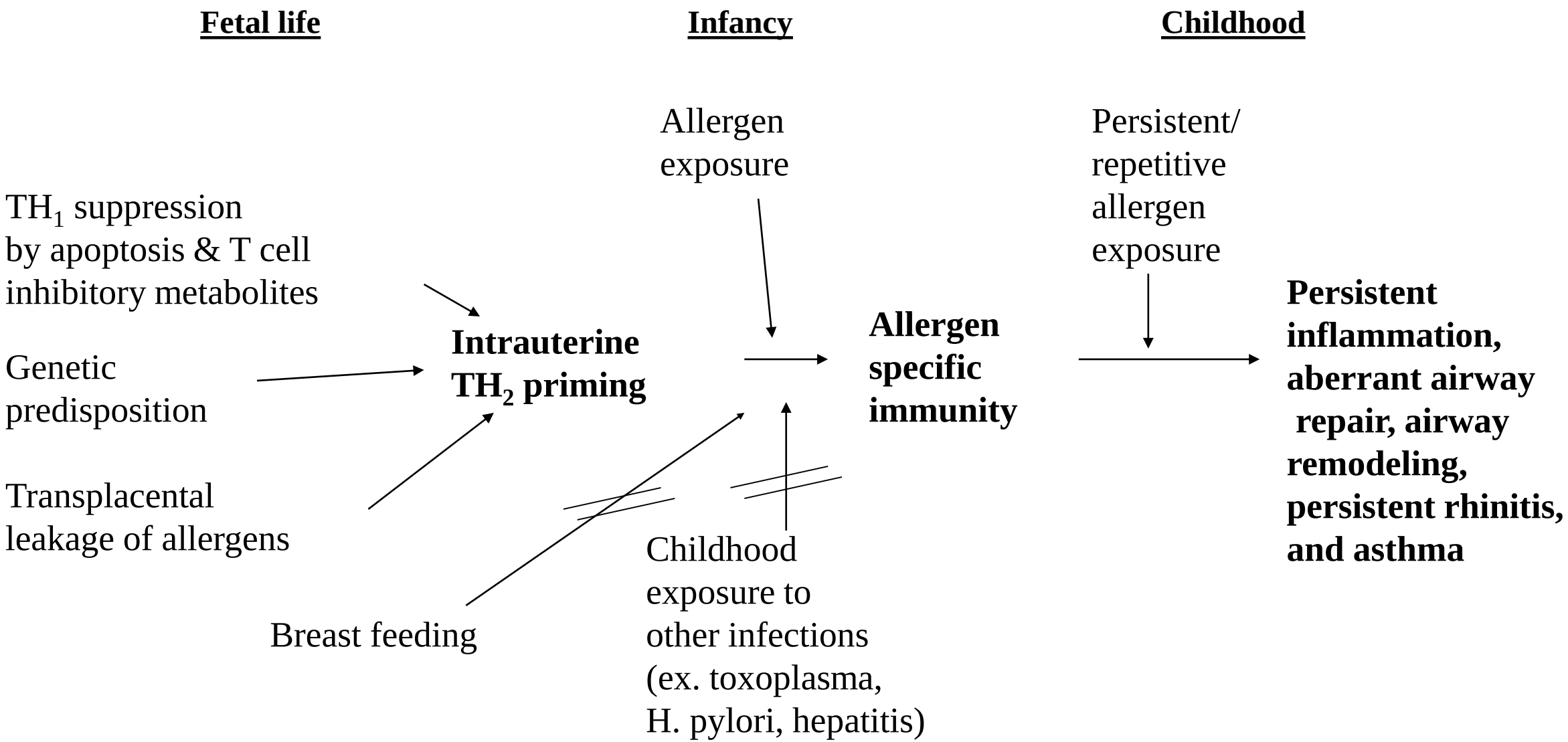
Urban environment

Diet

Sensitization to house-dust mites and cockroaches



Etiology of allergy



Allergic rhinitis

- Inhale allergen → antigen presentation→ TH_2 interleukins → IgE production & mast cell binding → histamine, prostaglandins, leukotrienes → PMNs & eosinophils → inflammation → mucosal edema & mucus production → nasal obstruction, interaction with bronchi

Diagnosis, history

- Detailed history
- Nature, duration & time course of symptoms.
- Possible triggers
- Response to medication
- Comorbid conditions e.g. asthma
- Positive familial history of allergic diseases
- Environmental, occupational exposures
- Effects on quality of life

Comorbid states

- Asthma: 20% of AR patients have asthma
- Atopic dermatitis (eczema)
- Atopic rhino – conjunctivitis
- Non –Allergic Rhinitis (NAR): hypothyroidism, sarcoidosis

Symptoms

- Sneezing
- Itching (ears, eyes, nose, palate)
- Rhinorrhea
- Post – nasal drip
- Congestion
- Anosmia
- Headache
- Earache
- Tearing
- Red eyes
- Eye swelling
- Fatigue
- Drowsiness

Facial stigmata of allergy

- Allergic shiners/ periorbital venous congestion
- Nasal creases
- Allergic salute

Nasal features of allergy

- Pale boggy mucosa
- Erythema
- Mucus
- Polyps
- Septal deviation
- Enlarged inferior turbinates

Systemic features

- Ear (otoscopy): tympanic membrane retraction, prominence of hand of malleus, air fluid levels, bubbles, tympanic membrane prominence & loss of land marks on the tympanic membrane
- Eye injection, lacrimation, Dennie Morgan Folds
- Cobble – stoning of posterior pharynx
- Tonsillar hypertrophy
- Malocclusion: overbite
- High arched palate
- Chest auscultation: rhonchi, loud P2 in the event of cor pulmonale

Complications

- ***Rhinosinusitis***: sinonasal ostia are closed
- ***OME*** because of blockage of ET → negative pressure in the middle ear
- ***OSA*** → mucosal hypertrophy
- Dental (***overbite***) & ***palatal abnormalities*** → chronic mouth breathing
- ***Nasal polyposis***

Diagnosis

- In vitro tests
  - ✓Radioallergosorbent test (RAST)
  - ✓Elisa
  - ✓Serum IgE
- In vivo tests
  - ✓Skin prick tests
  - ✓Nasal provocation tests

Therapy overview

- Prevention of sensitization
- Prevention of IgE allergen interaction
- Prevent mast cell mediator release
- Blockade of mediator – receptor interaction
- Suppression of resultant inflammation

Management of allergic rhinitis

Allergen avoidance

Pharmacotherapy

Immunotherapy

Medications

- Antihistamines
- Decongestants
- Antihistamines + decongestants
- Corticosteroids
- Mast cell stabilizers
- Anticholinergics
- Anti – leukotrienes

Immunotherapy

- An ***etiological treatmen***t for allergic disease
- More effective for ***grass pollen and cat dander less so for HDM***
- Safety concerns
- Acceptability
- Duration of Administration

Cont.

- Novel Forms Of Immunotherapy
- Recombinant allergens
- DNA immunization → allergens administered as naked DNA
- Alternative routes of administration
  - ✓Sublingual
  - ✓Intranasal

ARIA Guidelines for management of intermittent symptoms

1. Mild: ***PO/ intranasal antihistamine (e.g. cetirizine)*** OR ***LT antagonist (e.g. montelukast)***
2. Moderate to severe: ***PO or intranasal antihistamine*** OR ***intranasal steroid***

❖Review 2 – 4 weeks if improved; Continue for 4 weeks; If failure step up therapy.

ARIA Guidelines for management of persistent symptoms

1. Mild: ***oral antihistamine*** OR ***ICS***
2. Moderate to severe: ***Intranasal steroid***

❖Review 2 – 4 weeks; if improved step down; if no improvement:

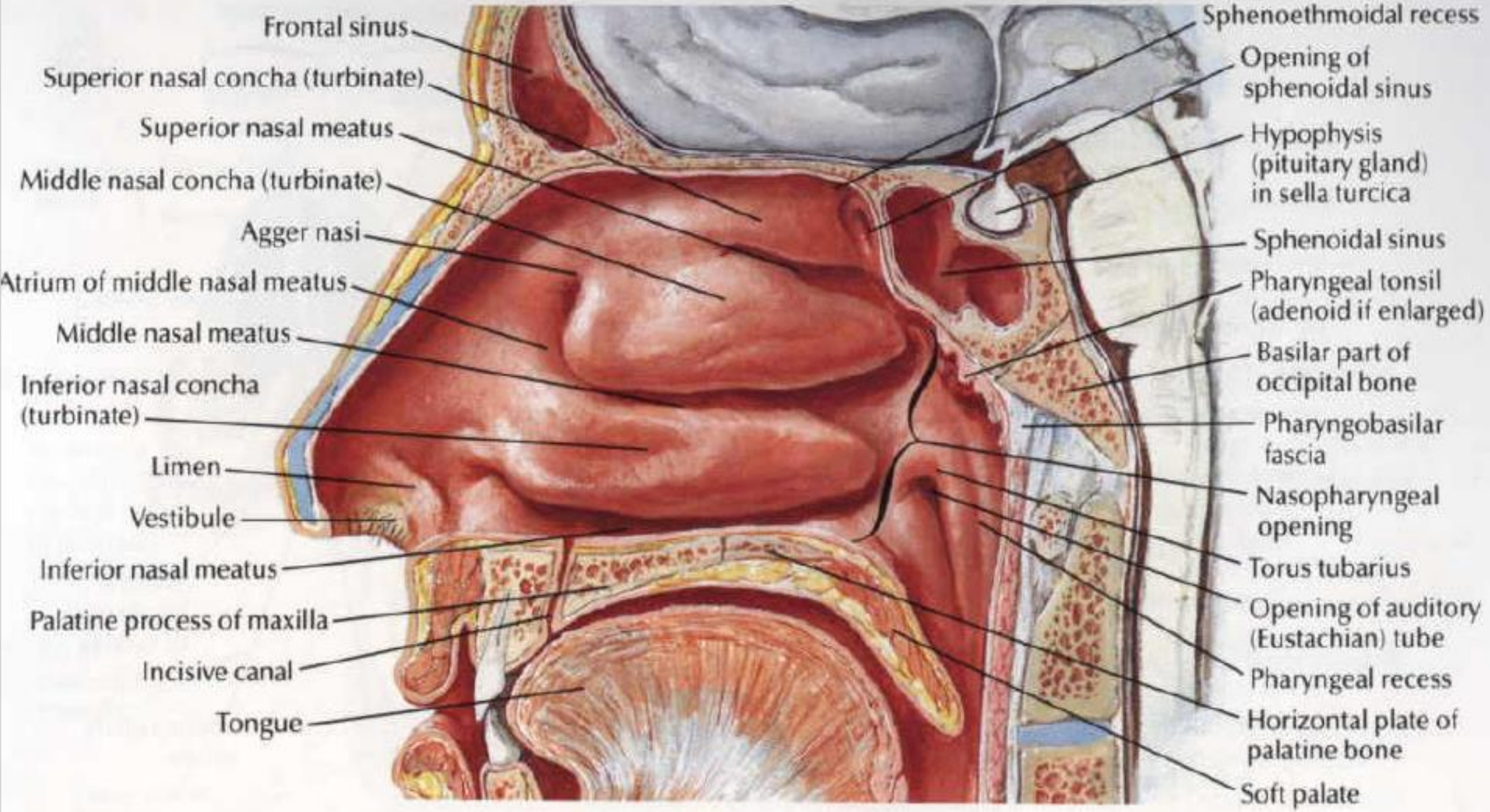
- ✓Review diagnosis
- ✓Check compliance
- ✓Query infections or other causes

❖Consider: increasing the steroid dose & administering a H_1 blocker for itch or sneeze.

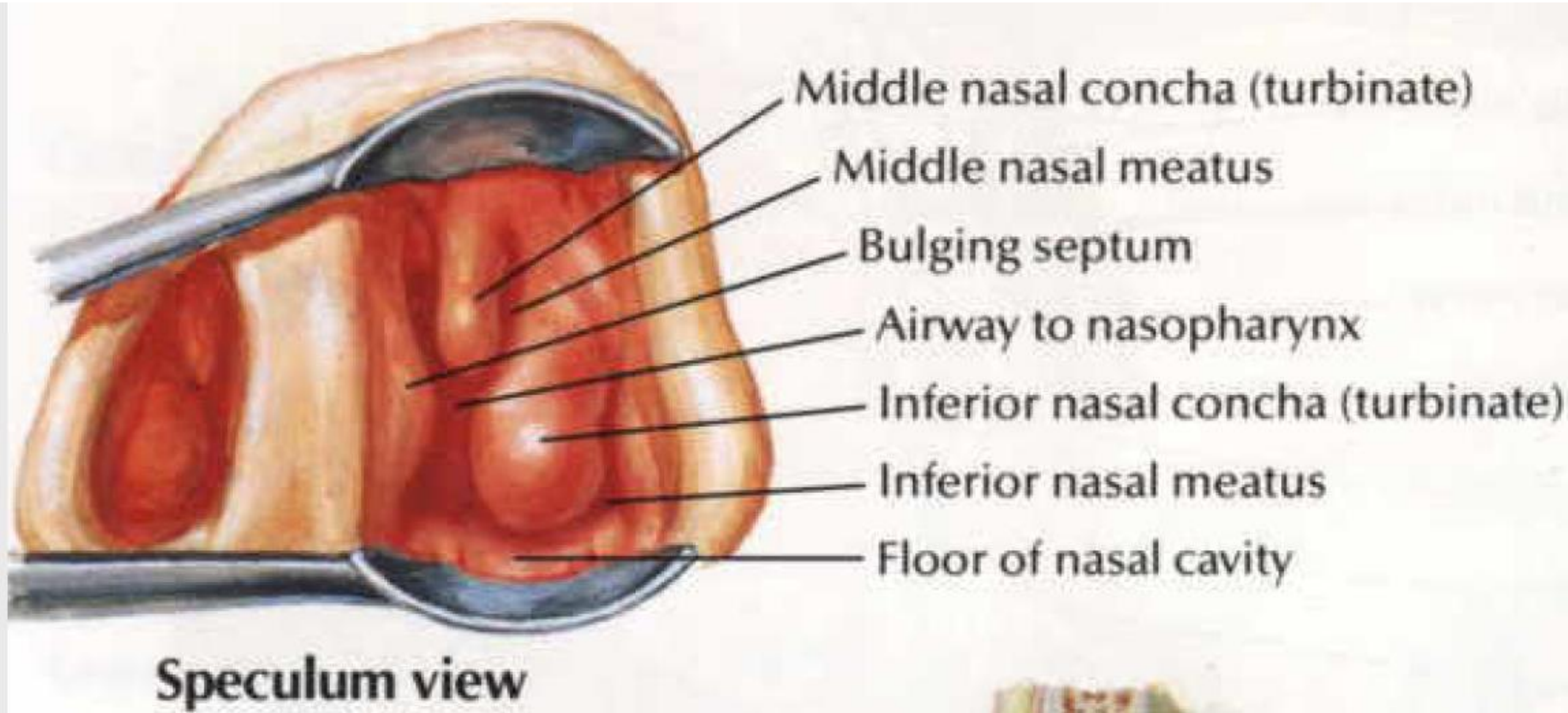
❖Failure of medical treatment: review diagnosis, consider surgical review.

NASAL POLYPOSIS

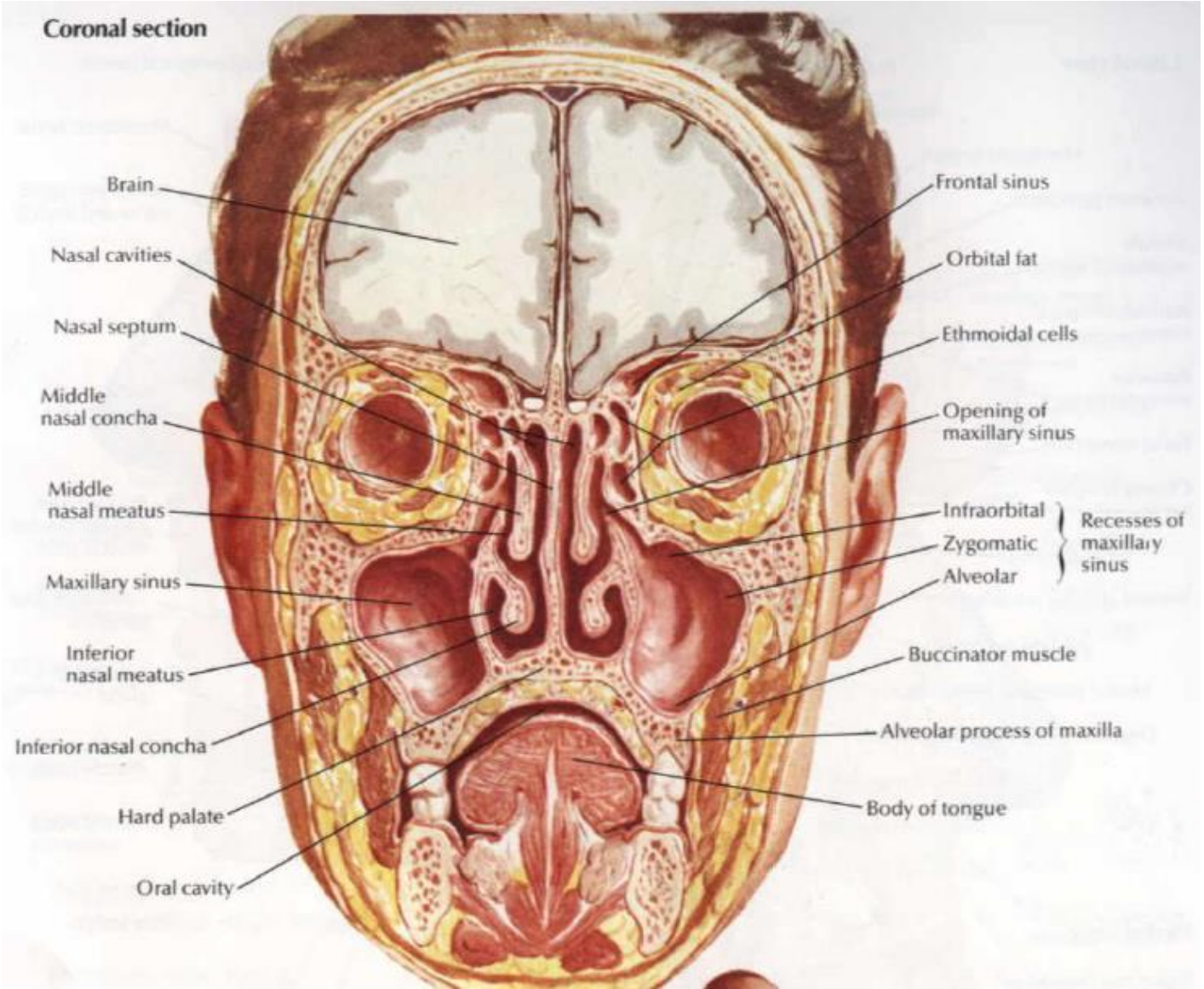
- Definition: Bags of ***edematous lamina propria*** surrounded by ***hyperplastic secreting nasal mucosa*** prolapsing into the nose or paranasal sinuses.
- Very rare in allergic children (<20yrs); less rare in children with cystic fibrosis
  - ✓If present, consider encephalocele with CSF rhinorrhea
- Non – allergic > allergic adults with rhinitis or asthma
  - ✓Incidence is ***5% in non – allergic people & 1.5% in people with allergic rhinitis***.
- Nasal polyps usually arise from the ***ethmoid sinus*** & the region of the ***middle meatus***


Frontal sinus
Superior nasal concha (turbinate)
Superior nasal meatus
Middle nasal concha (turbinate)
Agger nasi
Atrium of middle nasal meatus
Middle nasal meatus
Inferior nasal concha (turbinate)
Limen
Vestibule
Inferior nasal meatus
Palatine process of maxilla
Incisive canal
Tongue
Sphenoethmoidal recess
Opening of sphenoidal sinus
Hypophysis (pituitary gland) in sella turcica
Sphenoidal sinus
Pharyngeal tonsil (adenoid if enlarged)
Basilar part of occipital bone
Pharyngobasilar fascia
Nasopharyngeal opening
Torus tubarius
Opening of auditory (Eustachian) tube
Pharyngeal recess
Horizontal plate of palatine bone
Soft palate

Thudichum nasal speculum view



Paranasal sinuses



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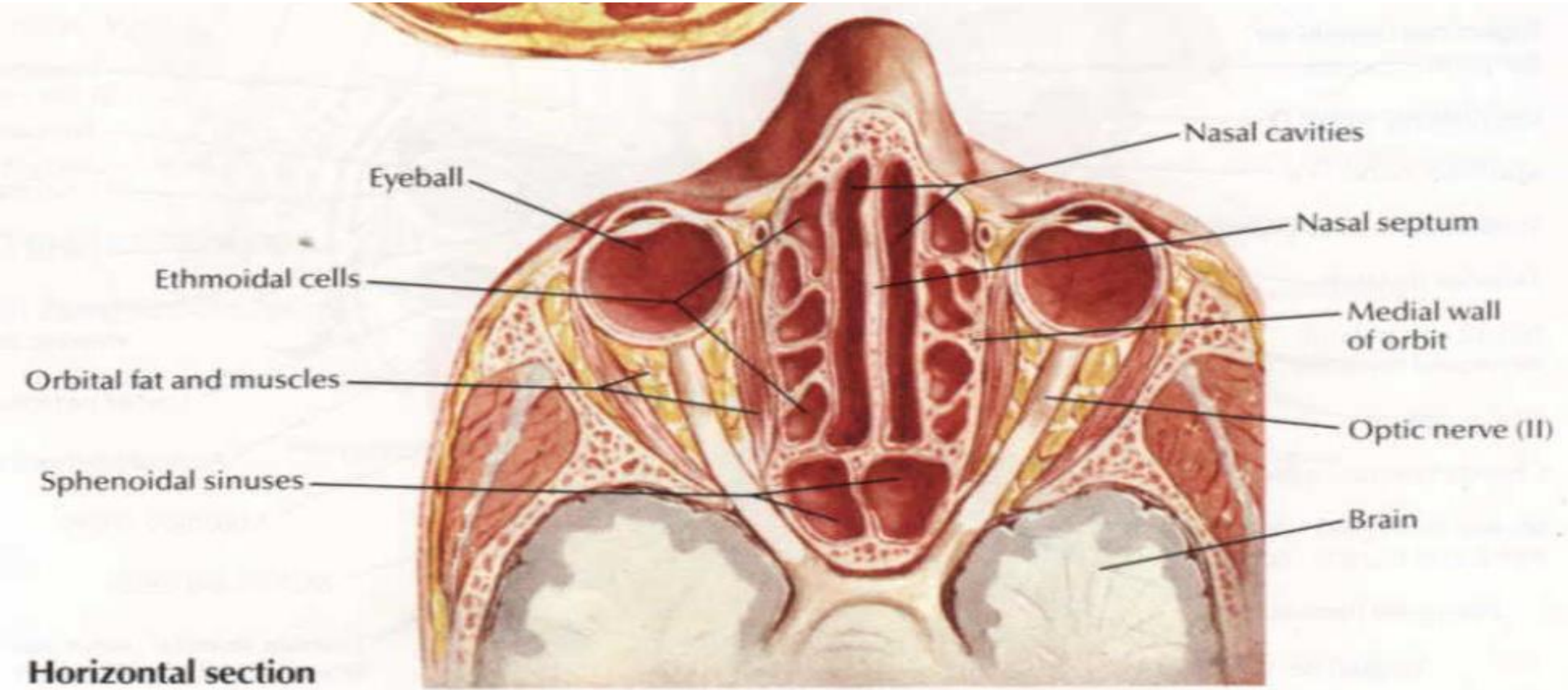

Nasal cavities
Eyeball
Nasal septum
Ethmoidal cells
Medial wall of orbit
Orbital fat and muscles
Optic nerve (II)
Sphenoidal sinuses
Brain
Horizontal section

Epidemiology

- Age: Nasal polyposis occurs in people aged > 20 yrs. ↑ incidence with age peaking at > 40 yrs.
  - ✓Incidence in adults 1 – 4%; Incidence in children < 0.5%.
  - ✓R/O cystic fibrosis in children <10yrs.
- Gender: M:F → 3:1 in adults
  - ✓Females with asthma have a higher prevalence

Cont.

- Allergy: Incidence is ***5% in non – allergic people & 1.5% in people with allergic rhinitis***.
- Associated conditions: the prevalence is increased in patients with:
  - ✓Samter's/ Aspirin hypersensitivity triad: chronic condition defined by ***asthma, sinus inflammation with recurring nasal polyps & aspirin sensitivity***.
  - ✓Cystic fibrosis
- No racial or sexual predilection is reported.

Factors predisposing to nasal polyps

Non – Modifiable

- Increasing age > 40 yrs.
- Male gender

Modifiable

- Chronic infection
  - ✓GABHS; *S. aureus*; *S. pneumoniae*, *H influenzae*
  - ✓Mainly neutrophilic infiltration rather than eosinophilic.
- Allergy: mainly eosinophilic infiltration (***most evidence is against this)***
- Aspirin hypersensitivity
- Cystic fibrosis
- Nasal mastocytosis

Conditions that are associated with multiple benign polyps

- **Allergic fungal sinusitis** (85%)
- **Allergic conditions**: bronchial asthma (20 – 50%), AR, aspirin intolerance (8 – 26%), alcohol intolerance (50%).
- **Cystic fibrosis** (6 – 48%)
- **CRS**
- **Primary ciliary dyskinesia**
- **Churg – Strauss syndrome**: Asthma, Peak eosinophilia > 1.5 X 10^9 cells/ L, systemic vasculitis, ≥ 2 extra pulmonary sites (***Lanham's criteria***)
- **Young syndrome**: CRS, nasal polyposis, bronchiectasis, azoospermia
- **Non – allergic rhinitis with eosinophilia syndrome (NARES)**

Frequency of nasal polyps in various diseases

Disease	Group	%
Allergic rhinitis	In children	0.1
	In adults	1.5
Non – allergic rhinitis		5
Asthma in adults	Allergic	5
	Non – allergic	13
NSAID intolerance		36 – 72
NSAID intolerance & asthma		80
Allergic fungal rhinosinusitis		>80
Churg – Strauss syndrome		50
Cystic fibrosis	In children	10
	In adults	40
Primary ciliary dyskinesia		40

Types

1. Nasal polyps
2. Antrochoanal polyps
3. Neoplastic polyps
4. Miscellaneous polyps

Pathogenesis

- The precise mechanism is incompletely understood.
- Numerous pathogenic theories
  - ✓Final manifestation of ***chronic inflammatory disease***.
  - ✓Autonomic Nervous System dysfunction.
  - ✓Genetic predisposition.
  - ✓Allergic vs. non – allergic causes.

Cont.

- New theory speculates on ***non – allergic eosinophilic reaction to fungi***.
- *S. aureus* is the MC bacteria
- B – cell stimulation results in production of specific IgE to the S. aureus enterotoxin
- Autonomic dysfunction of nasal mucosal blood vessels

Stammberger classification

Antrochoanal/ Killian's polyp.

Large isolated polyp.

Polyps associated with CRS, non – eosinophil dominated, non – related to hyperactive airway syndromes

Polyps associated with CRS, eosinophil dominated

Polyps associated with specific disease (***CF, non – invasive/ non – allergic fungal sinusitis, fungal ball, malignancy***)

Antrochoanal polyp

- Uncommon (4 – 6% of all nasal polyps).
- Usually arises from the ***mucosa of the maxillary antrum***
- More common in children 33%
- Unilateral
- Most arise from the posterior part of the antrum
- 70% emerge through an accessory ostium
- Prolapses in nose usually posteriorly towards PNS

Cont.

- Symptoms worse on expiration (ball valve effect)
- Usually present late when mass is very large
- Surgical excision via the nose
- Most often in children and young adults
- Cystic part in antrum solid part in the nasal cavity

Cont.

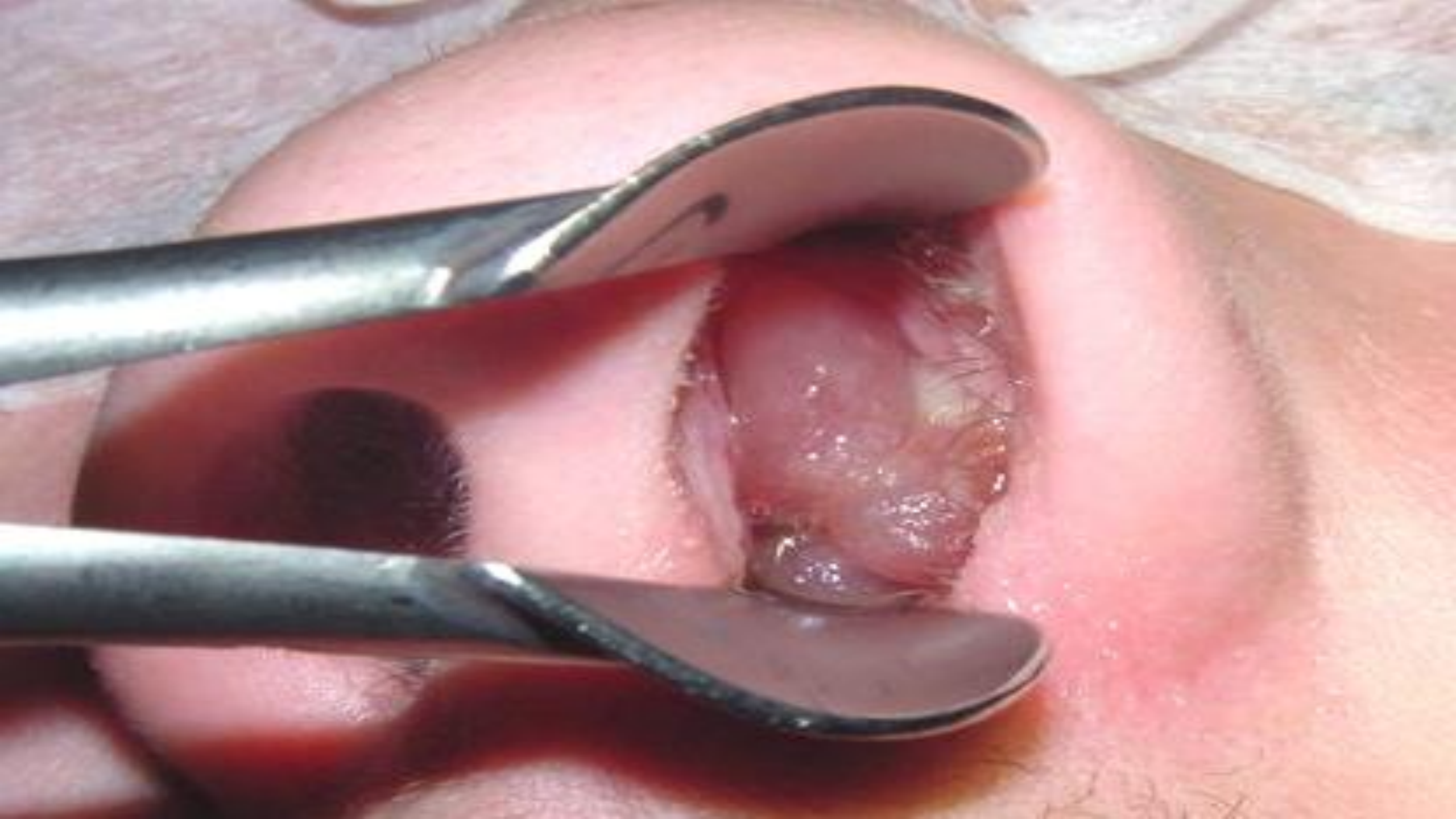
- Stammberger found that:
  - ✓80% of nasal polyps arise from ***middle meatus mucosa***, ***uncinate process*** & ***infundibulum***.
  - ✓65% originated from ***ethmoid bulla*** & ***hiatus semilunaris***
  - ✓48% originated from the ***frontal recess.***
- Polyps were found inside the ethmoid bulla in 30%

History

- Asymptomatic – small polyps
- Airway obstruction & OSA
- Post – nasal drip
- Dull headaches or facial pain
- Snoring
- Rhinorrhea
- Hyposmia/ anosmia
- Craniofacial abnormalities
- Optic nerve compression

O/E

- Anterior rhinoscopy
- Otoscopy for any otological symptoms
  - ✓In small children, use a hand held otoscope & otologic speculum.
- Examination of oral cavity and pharynx:
  - ✓Large polyps may prolapse into postnasal space & be visible in the oral cavity.



Cont.

- A single, unilateral polyp originating high in the nasal cavity or with a stalk that is not clearly visible may represent an ***encephalocele or meningocele***.
  - ✓Visible pulsations on endoscopy & enlargement of the mass with ipsilateral internal jugular vein compression (***Furstenberg's sign***) help to confirm the diagnosis.
- As a rule, ***if the intranasal mass does not have the characteristic appearance of a polyp, is unilateral, bleeds easily, or has a stalk that is not clearly identified***, imaging studies are indicated before proceeding with management.

Nasoendoscopy

- Flexible
- Rigid
- Rule out DDX: encephalocele, tumors

Differential diagnosis

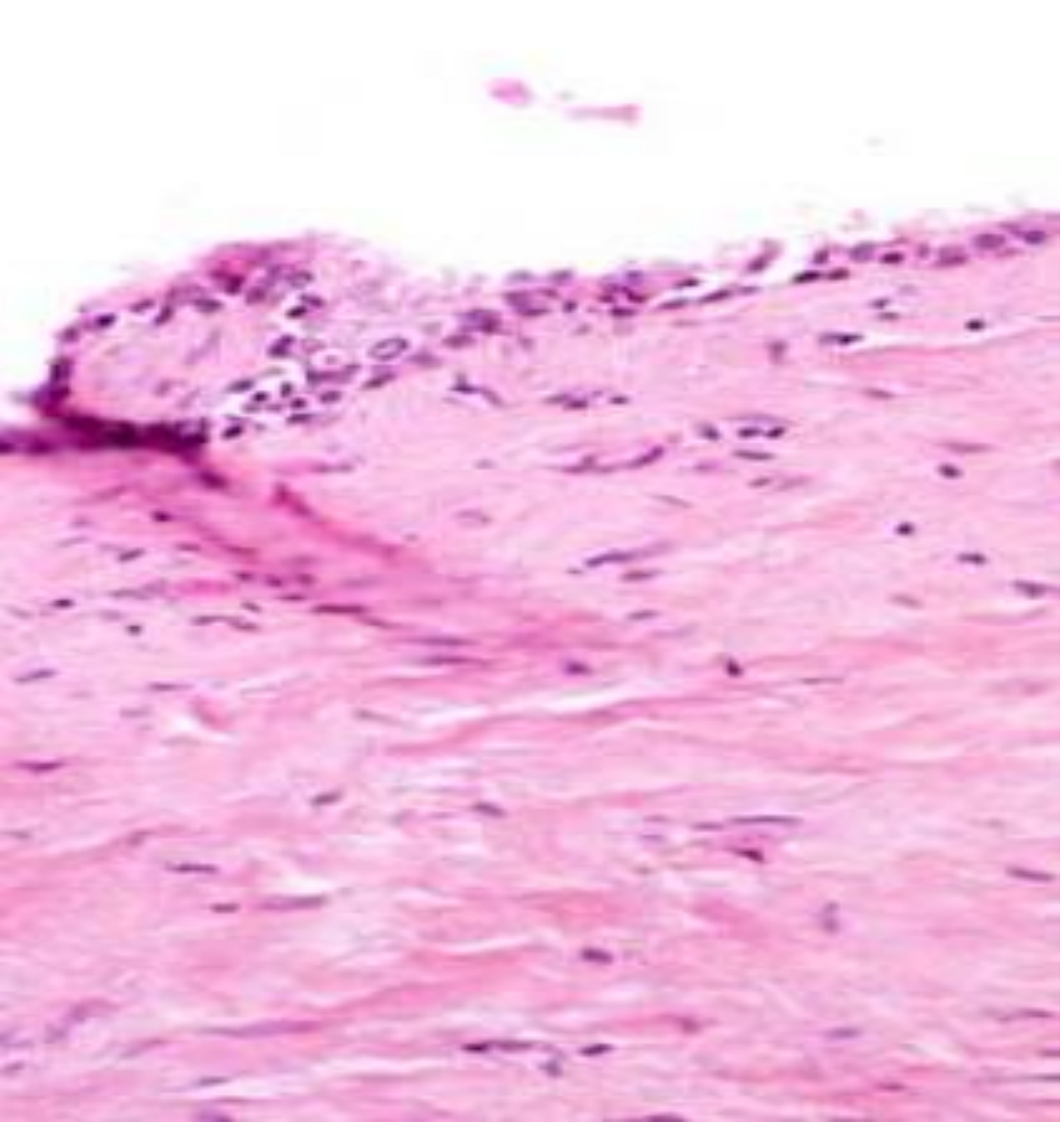
- Encephaloceles
- Gliomas
- Dermoid tumors
- Hemangiomas
- Papillomas/ transitional cell papilloma
- Rhabdomyosarcoma
- Lymphoma
- Neuroblastomas
- Sarcomas
- Chordomas
- Nasopharyngeal carcinomas
- Juvenile nasopharyngeal angiofibroma

Endoscopic staging of nasal polyps

- No polyps → 0
- Restricted to middle meatus → 1
- Below middle turbinate → 2
- Massive polyposis → 3

Laboratory tests

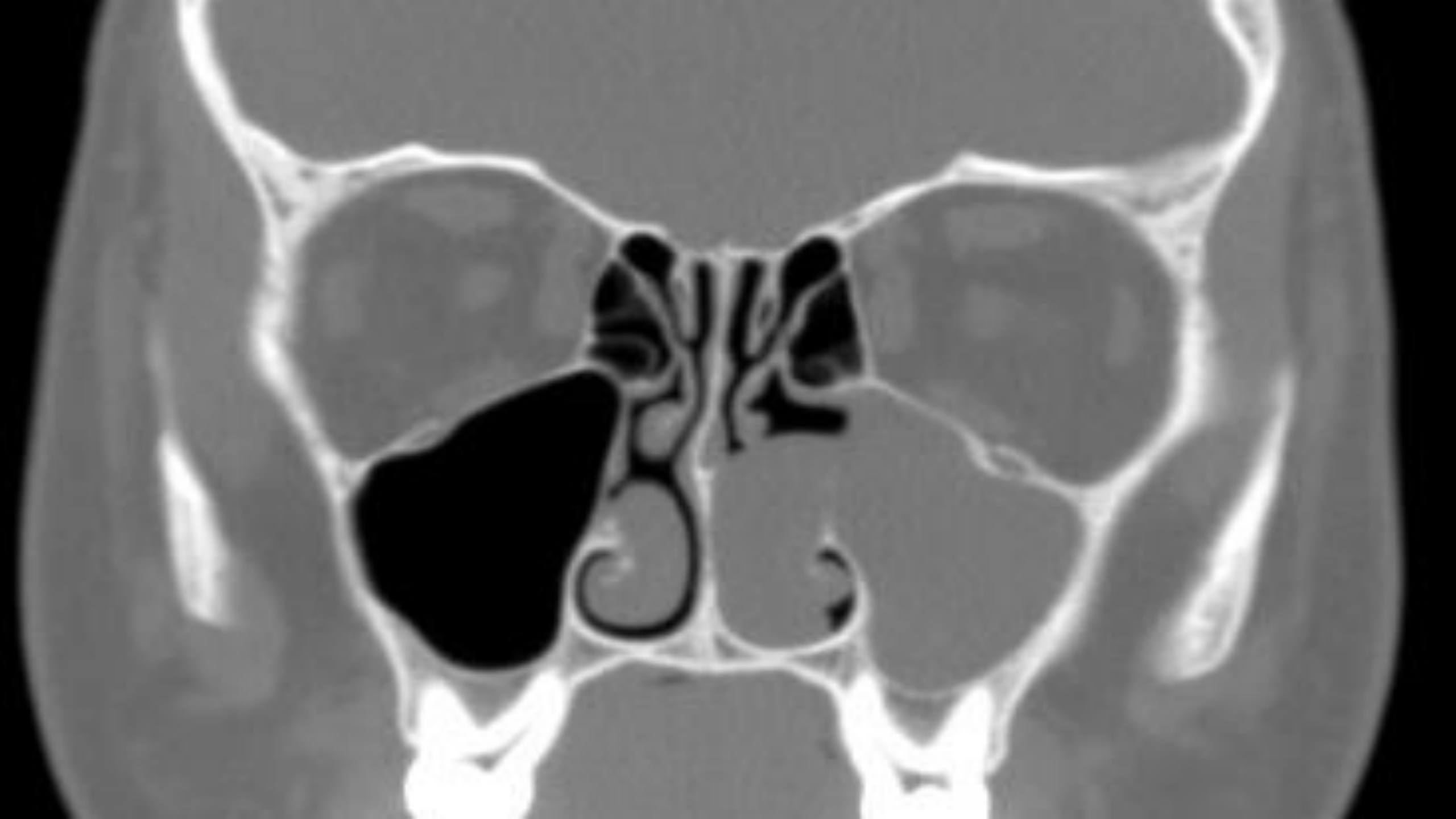
1. ***RAST/ skin testing***: A thorough allergy evaluation in patients with history of environmental allergies or a strong family history of allergies.
2. ***Nasal smear***
   - ✓Microbiology
   - ✓Eosinophils (allergic component)
   - ✓Neutrophils (chronic sinusitis)
3. Nasal polyposis in children: test for cystic fibrosis with either a ***sweat chloride test*** or with ***hematologic genetic testing***.

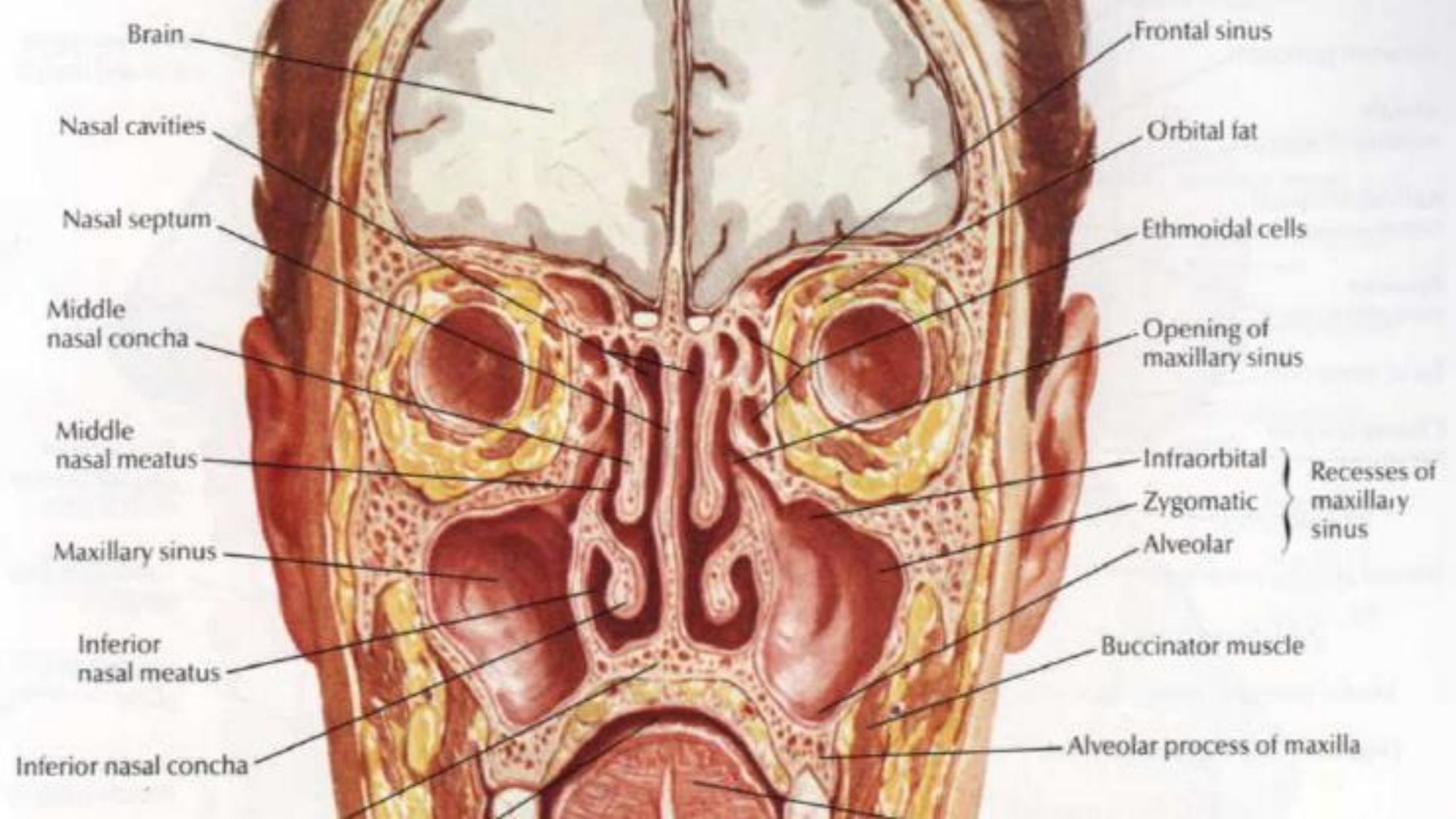


- Pseudostratified ciliated columnar epithelium
- Thickened epithelial basement membrane
- Edematous stroma.

Imaging studies

- ***Coronal sinus CT***: gold standard
  - ✓Shows the primary pathology, extent of disease and possible bony destruction
  - ✓Also aids in the assessment of the anatomy of the paranasal sinuses in the event of surgical intervention.
- ***MRI***: only appropriate if intracranial extension suspected; bony details of the paranasal sinus anatomy are poorly visualized on MRI.
- ***Radiography with Waters views*** may show opacification of the sinuses




Brain
Nasal cavities
Nasal septum
Middle nasal concha
Middle nasal meatus
Maxillary sinus
Inferior nasal meatus
Inferior nasal concha
Frontal sinus
Orbital fat
Ethmoidal cells
Opening of maxillary sinus
Infraorbital
Zygomatic
Alveolar
Recesses of maxillary sinus
Buccinator muscle
Alveolar process of maxilla

Medical treatment

- Aimed at non – specific treatment of inflammation.
- ***Management of allergies in atopic individuals does not reduce or eliminate polyps.***
- ***Oral corticosteroids*** – most effective short term treatment for nasal polyps.
- Intranasal steroid sprays – small intranasal polyps
- Intrapolyp steroid injections – reduce growth of polyp
- Macrolide antibiotics

Surgical management

- Endoscopic sinus surgery & polypectomy
- Recurrence
  - ✓***Multiple small polyps are more likely to recur*** than large & Antrochoanal polyps

Complications

- Recurrent acute sinusitis
- Chronic sinusitis
- Acquired Nasal deformities
- Proptosis, diplopia
- Meningitis
- Encephalitis

Clinical objectives

- Detailed history
- Stigmata of atopic disease
- Comorbid conditions
- Complications
- Allergen testing
- Interventions

People may hear your words, but they feel your attitude.

~ Anon

ACUTE OTITIS MEDIA AND OME.

MBCHB VI. 21.03.2019

DR S.GITONGA

ENT CONSULTANT KNH

- Acute inflammation of the middle ear, <3 weeks.
- Often associated with viral URTI.
- Most common in children <5 years.
- Recurrent AOM is :

✓ atleast 4 episodes/year

✓ Atleast 3 episodes in 6 months

Epidemiology

- 50-85% of children under 3 years will have atleast 1 episode.
- Peak incidence 6-15months of age
- Most common in the cold seasons of the year
- Occurs in only 20% of adults

pathology

- Eustachain tube and nasopharynx are lined with respiratory mucosa.
- Both respond to triggers with : edema- narrowing of lumen- negative middle ear pressure
- Influx of pathogens from the nasopharynx

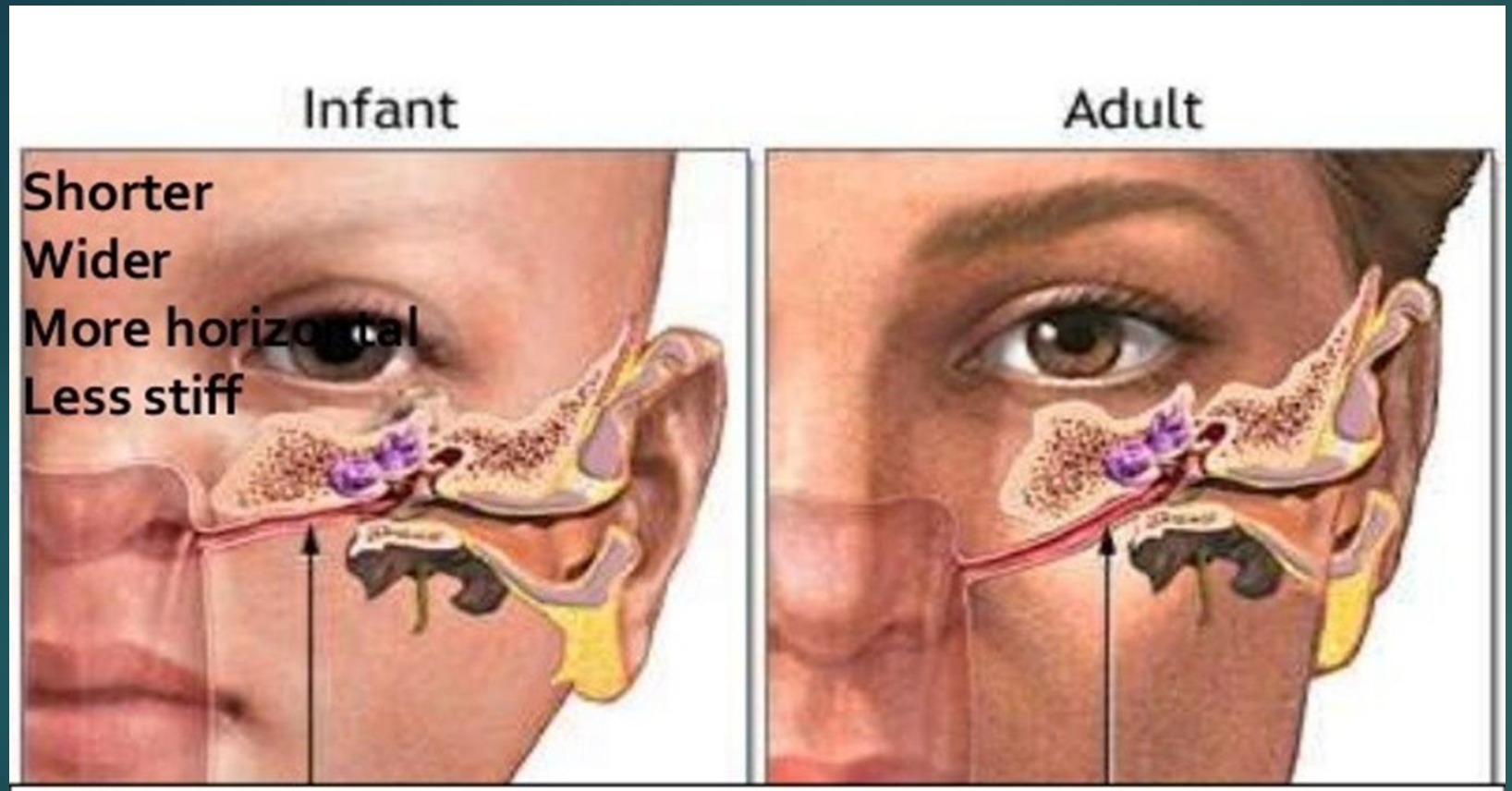
Triggers

1. URTIs
2. Allergy
3. GERD
4. Adenoid hypertrophy

Aetiological agents

1. Viruses (30-70%)
   - rhinovirus
   - RSV
   - Influenza/ parainfluenza
2. Bacterial (55%)
   - Strep. Pneumonia (44%)
   - H. influenza (41%)
   - M.catarrhalis (14%)
   - Gram negative enteric bacteria
   - Staph aeuris
3. Combined (15%)

Risk factors in children



- Children ET 10°
- Adults ET 45°
- ISTHMUS(narrowing of ET)
  - junction of the cartilaginous and bony part.
  - It is only present in adults.

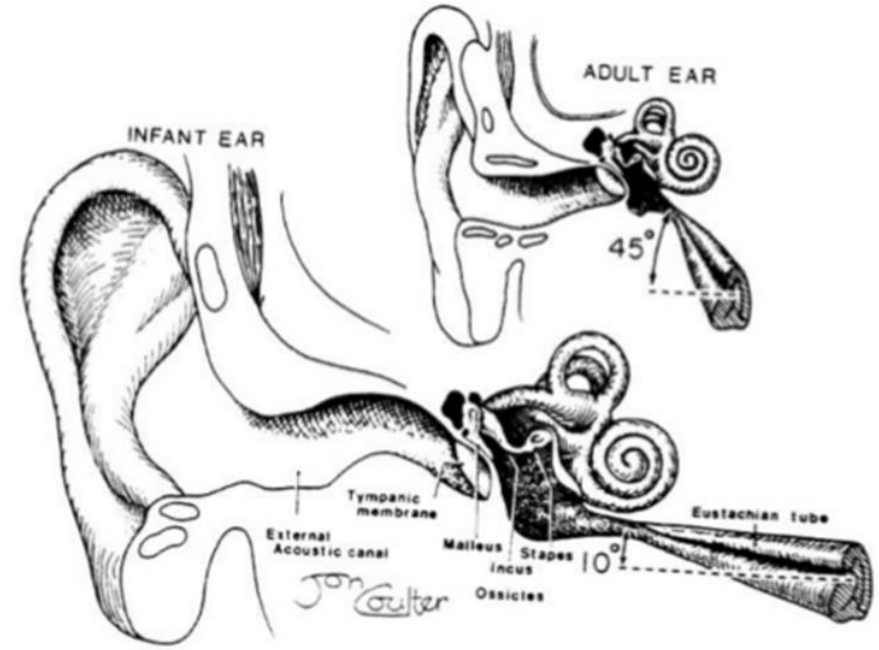


Fig. 3. Eustachian tube anatomy differences between a child and an adult.

Handbook of Pediatric Otolaryngology : A Practical Guide for Evaluation and Management of Pediatric Ear, Nose, and Throat Disorders

2. Genetic predisposition
   - Eustachian tube dysfunction
   - Allergy spectrum
3. Bottle feeding in the first 3 months.
   - Breast milk has oligosaccharides, lactoferrin and surface immunoglobulin A which prevent bacteria colonization.
4. Incorrect posture while breast feeding
5. Cleft palate
6. Parental smoking
7. overcrowding- large families or daycare
8. immunosuppression

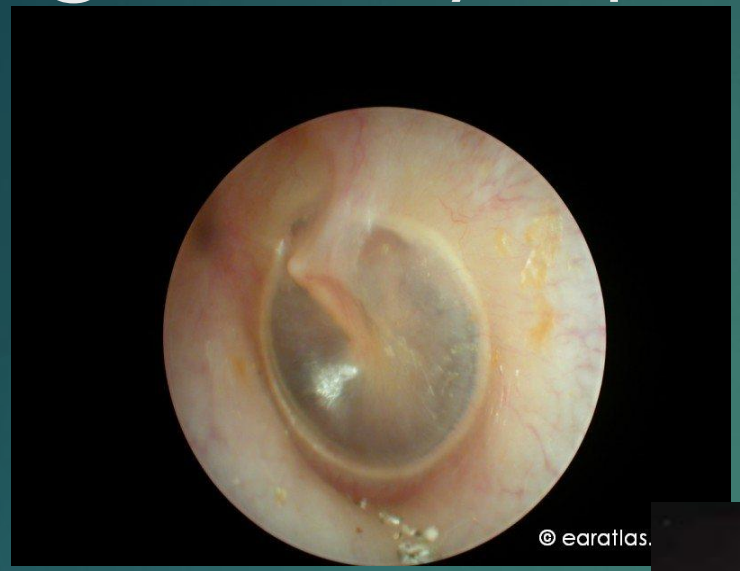
Signs and symptoms

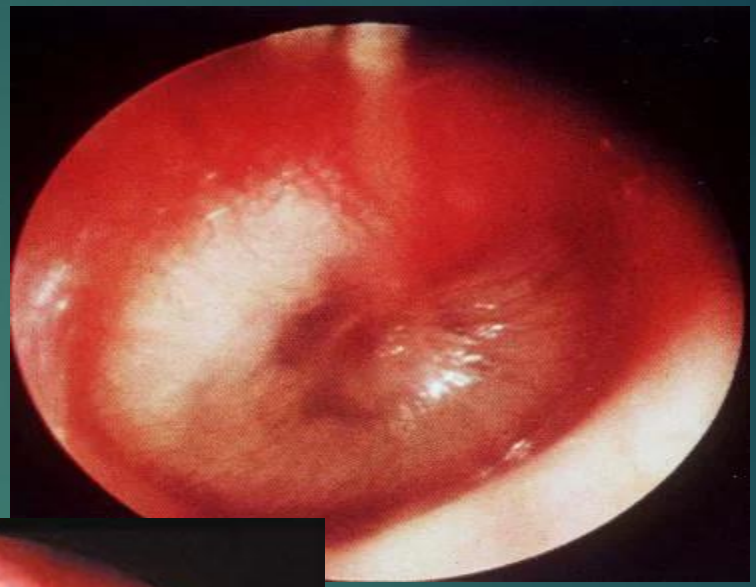
1. Otalgia- not always common
2. Fever
3. Hearing loss/ delayed speech
4. Tagging of ears
5. Headache
6. nausea
7. Cough
8. Rhinitis
9. conjuctivites

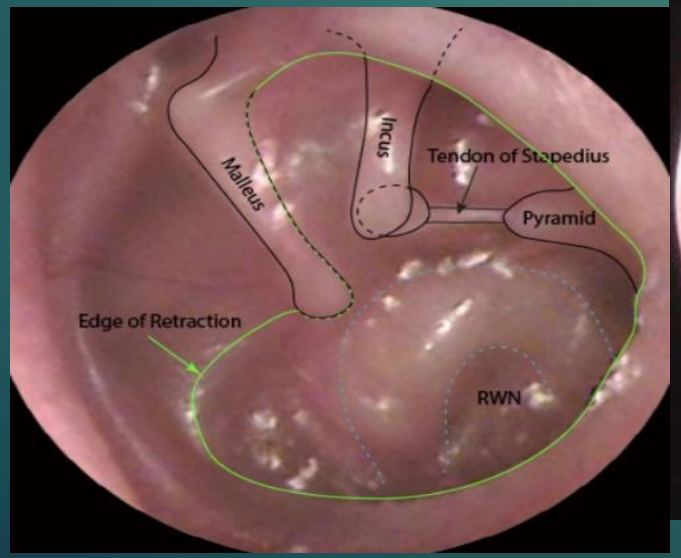
Physical examination

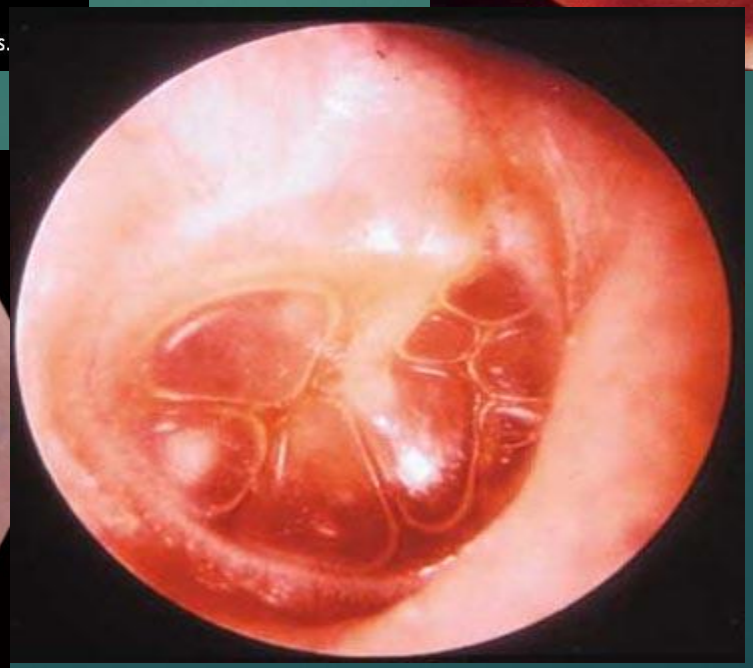
- Red/ opaque TM
- Retracted TM
- Immobile/ hypo- mobile TM
- Fluid effusion
- Retraction pockets
- Bullous myringitis

Images of tympanic membrane



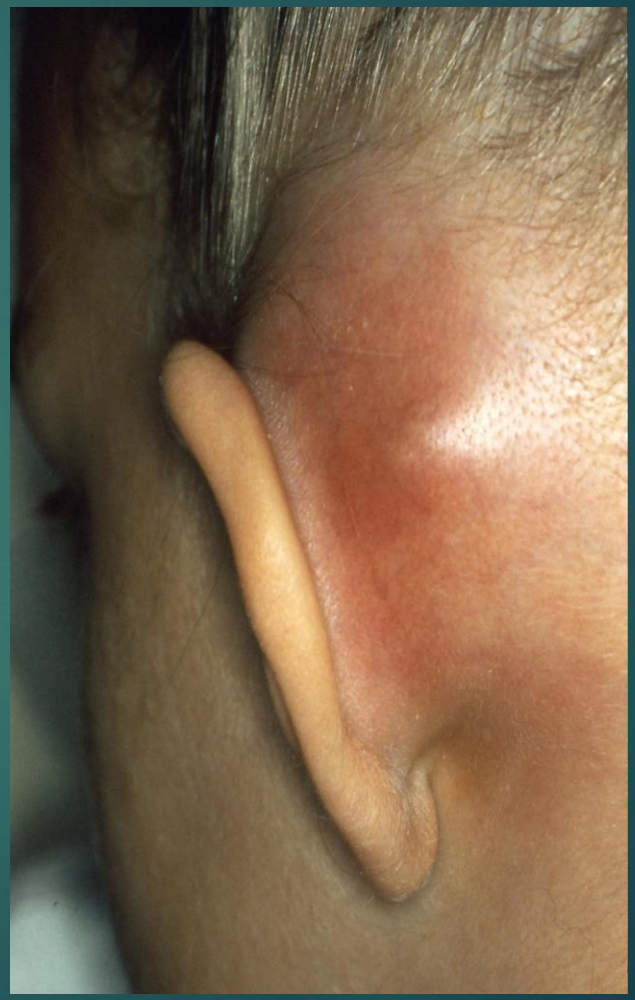






Physical examination

1. Anteriorly displaced pinna
2. Mastoid tenderness
3. otorrhoea



complications

1. Acute mastoiditis
2. Facial nerve palsy
3. Otitis media effusion
4. Hearing loss
5. Abscess formation
6. Recurrent otitis media
7. Perfusion otitis media
8. Tympanic membrane perforation

Rare complications

1. Intracranial complications
- Meningitis
- Extradural abscess
- Lateral sinus thrombosis
- Otitic hydrocephaleus
- Encephalitis

2. Intrtemporal
- Labyrinthitis
- petrositis

treatment

1. Antibiotics- indication
   - Children less than 6 months
   - Bilateral and/ or recurrent AOM
   - Children 6 months to 2 years with severe illness
   - Tympanic membrane perforation
   - Persistent fever >72 hours
2. analgesics- paracetamols, NSAIDs
3. Decongestants +/- antihistamines and nasal steroids

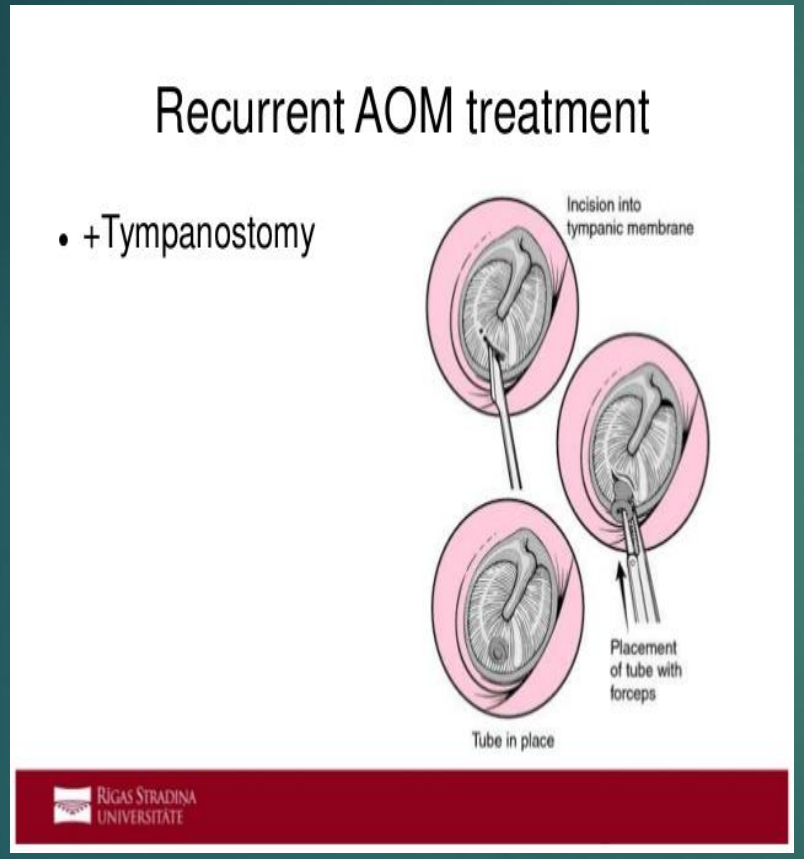
2-48 hours/ 48-72 hours

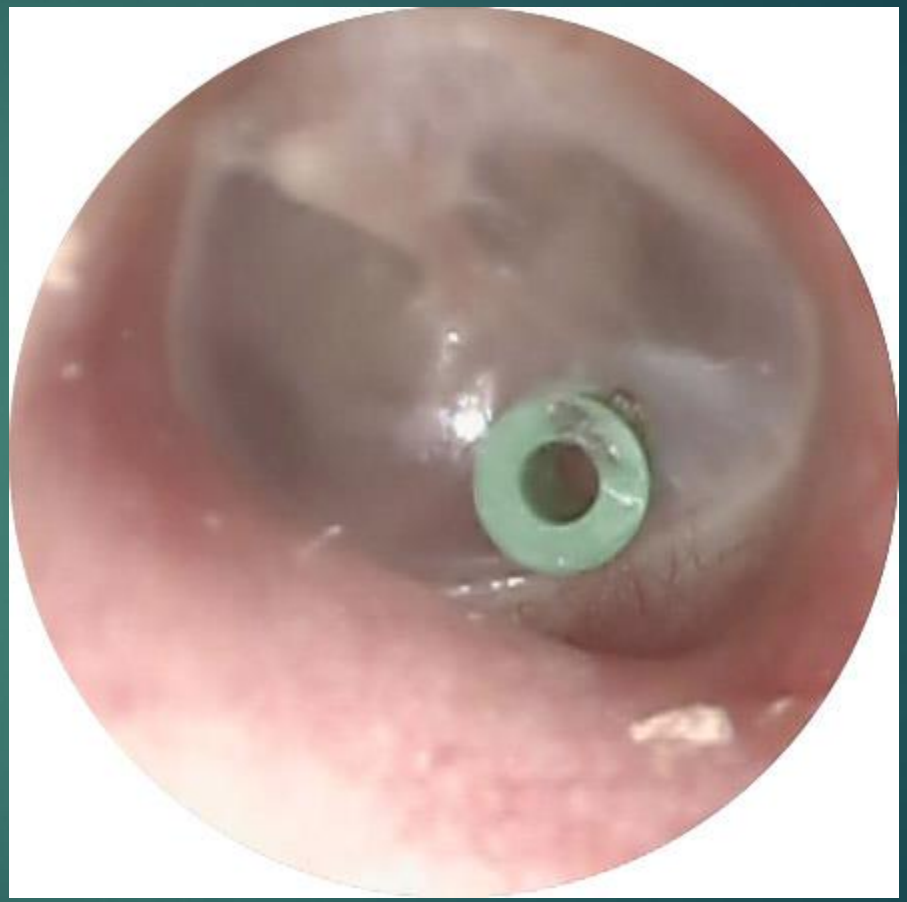
- If no Improvement

1. No antibiotics > antibiotics
2. On antibiotics> change antibiotics

- ✓ Amoxicillin 50-100mg/kg body weight/day
- ✓ Amoxicillin-clavulanic acid 875/125 mg/day ; 90/6.4mg/kg
- ✓ Ceftriaxone 50mg/kg or 1-2 g in adults/day
- ✓ Macrolides in patients with penicillin resistance
- ✓ Treatment duration 5-10 days

Recurrent AOM





OTITIS Media with EFFUSION

- Also known as
- ✓ Middle ear effusion
- ✓ Serous otitis media
- ✓ Secretory otitis media
- ✓ Glue ear

definition

- Chronic accumulation of mucus within the middle ear cleft and sometimes mastoid air cells.
- Chronic = 12 or more weeks
- ✓ Fluid is sterile
- ✓ Insidious onset
- ✓ Commonly occurs in children
- ✓ Tympanic membrane is intact but retracted
- ✓ Hearing loss fluctuating or constant

epidemiology

- Bimodal prevalence at 2 and 5 years.
- Occurs commonly cold season
- Male> females
- 50% children before the 1st birthday and 80% before the 5th birthday

Aetiology

- Eustachian tube obstruction due to;

1. Adenoid hypertrophy
2. Tumors- Nasopharyngeal carcinoma
3. Palatal defects- cleft palate
4. Barotrauma
5. Hyperbaric oxygen therapy
6. Edema secondary to radiation of HN

- Spread of Infections

1. Chronic adenoiditis
2. Chronic rhinosinusitis
3. Chronic tonsillitis
4. HIV

Contd.

- Increased secretions

1. Allergy
2. Smoking
3. GERD

- Infections

1. Unresolved AOM
2. Viral infections

Host risk factors

- Same as AOM
- Race- Caucasians
- Sibling with history OME
- Down's syndrome, cleft palate, ISS
- Cystic fibrosis
- Maxillectomy
- Prolonged intubation
- Poor mastoid pneumatisation

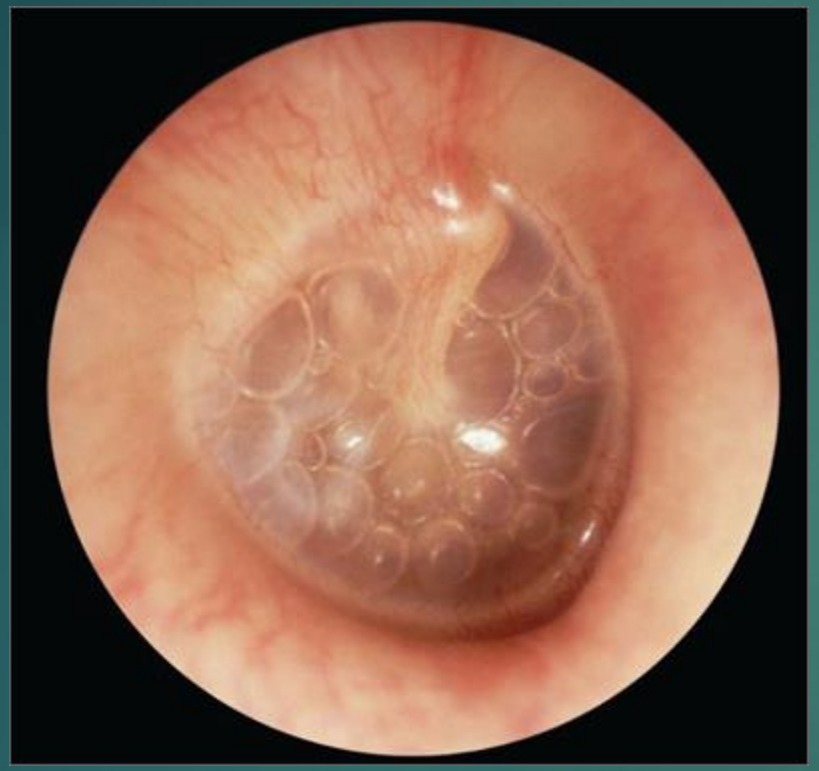
Environmental risk factors

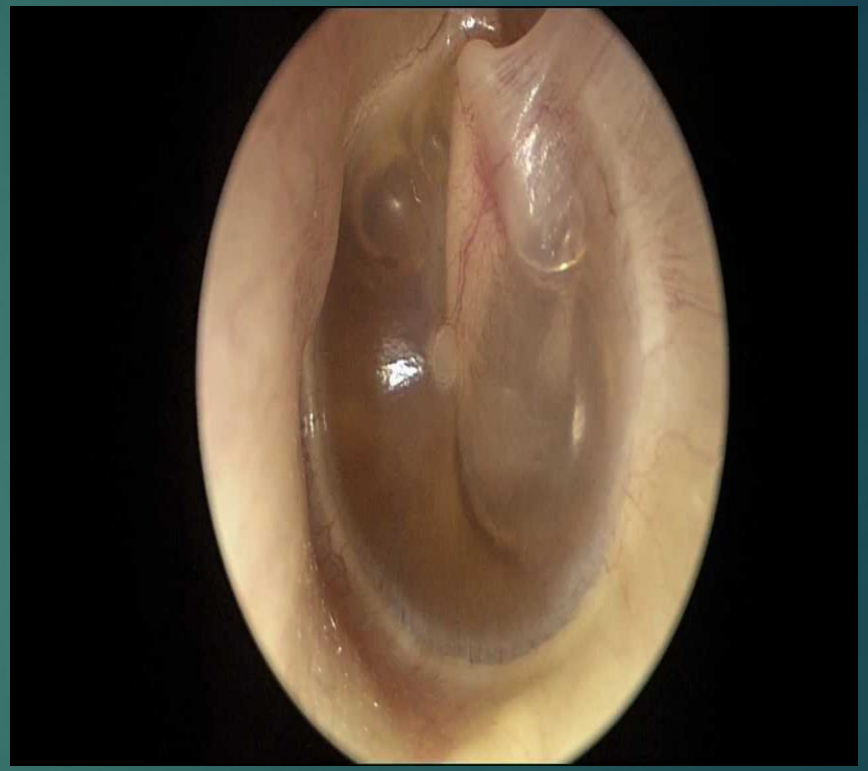
- Same as AOM
- Daycare centres with >4 children
- Use of pacifiers
- Low SES

Clinical presentation

1. Deafness
2. Tinnitus
3. Aural fullness
4. Hearing loss
5. Poor school performance
6. Delayed speech

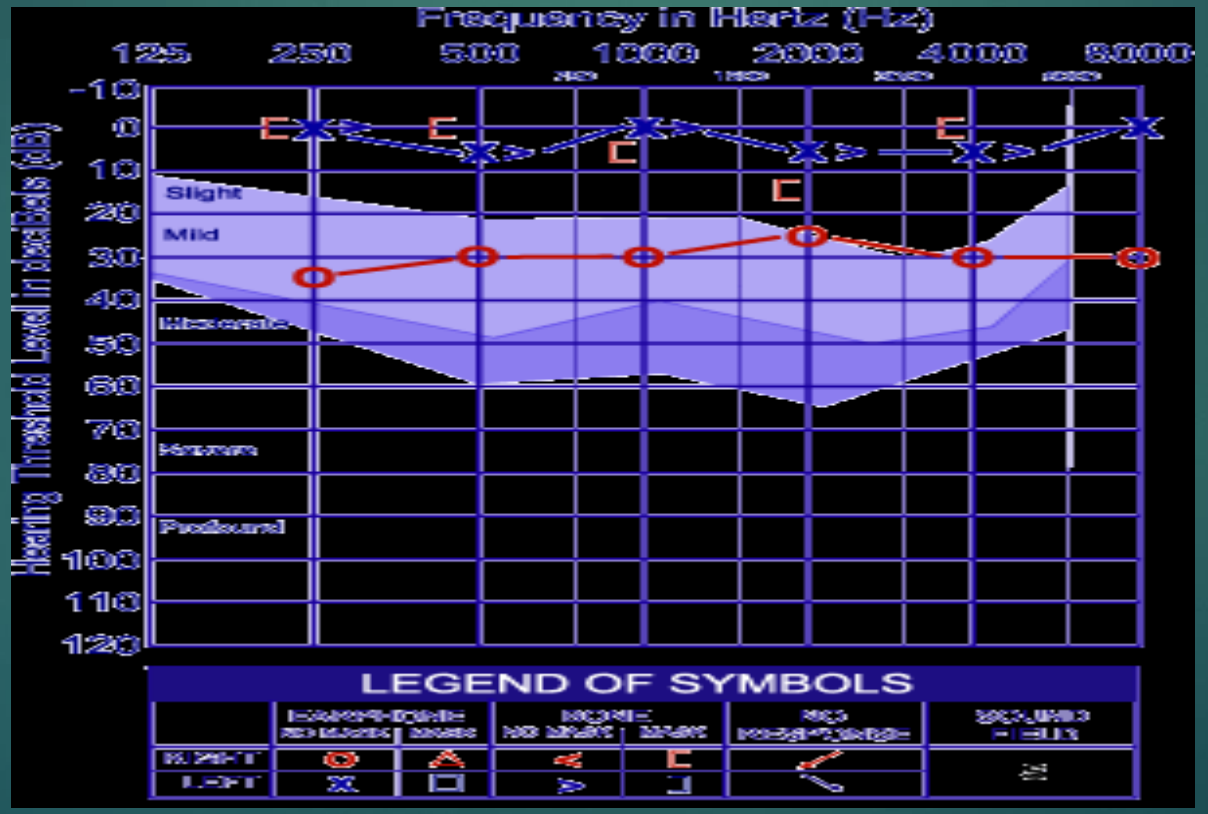
Otoscopy findings





Audiometry findings

- Conductive hearing loss with air- bone gap 20-40dB.



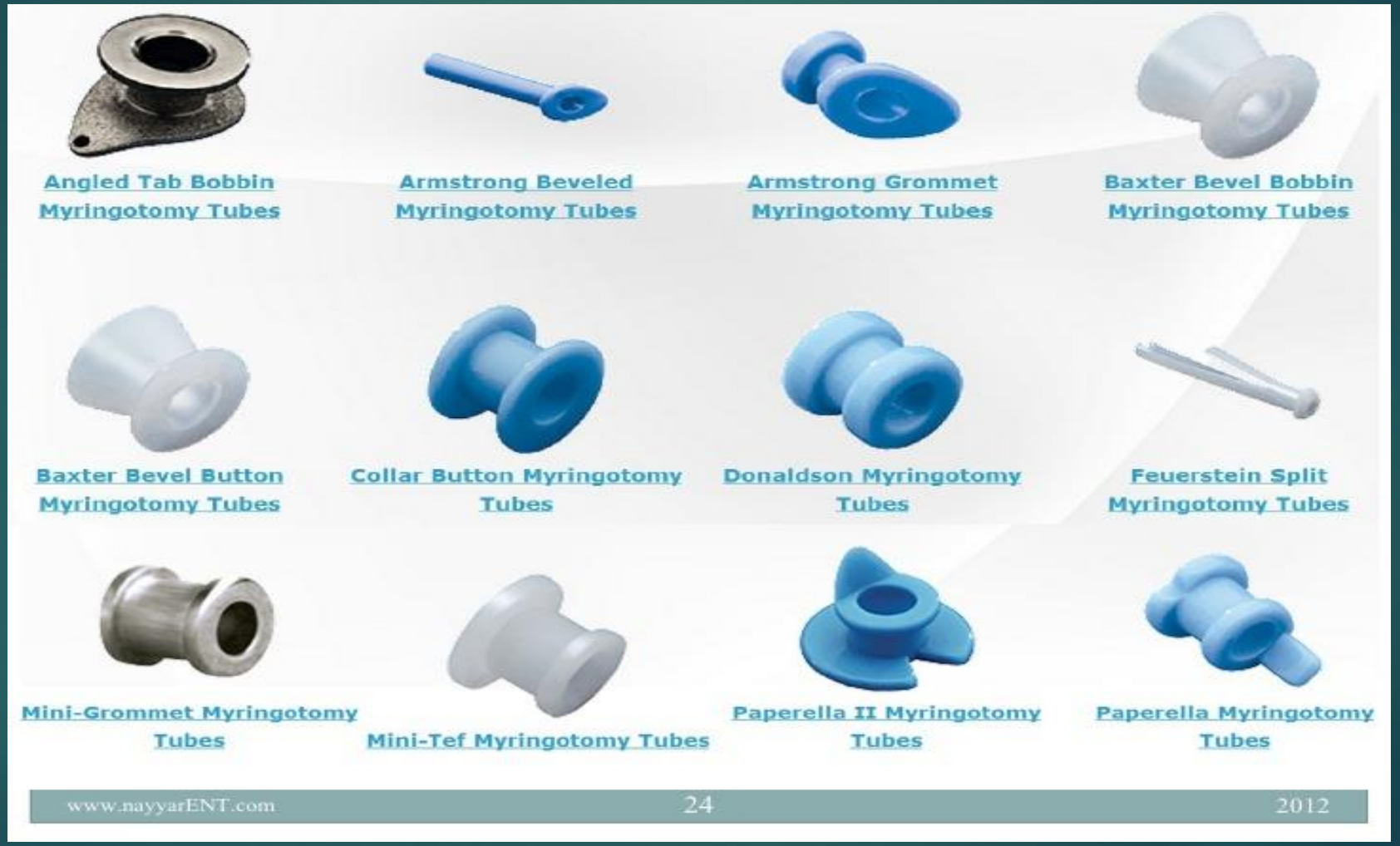
Medical treatment

1. Systemic and local decongestants
   - Pseudoephedrine, chlorpheniramine, oxymetazoline drops and xylometazoline drops
     - **NB: *Rhinitis medicamentosa***: this is a sequel of overuse of topical nasal decongestants and is characterized by rebound congestion, nasal hyperactivity & histological changes.
2. Antihistamines
3. Nasal steroid sprays and drops
4. Valsava maneuver: aeration of ET

Surgical management

- Insertion of grommets
- Adenoidectomy
- Tonsillectomy
- Cortical mastoidectomy
- Long stay Ventilation Tube in adults

Types of grommets



Complications of VT

- Displacement of tube into the middle ear
- Damage to ossicles
- Blockage of tube with blood
- Granulation tissue around the tube
- Ear infections
- Otorrhea
- Permanent tympanic membrane perforation
- Tympanosclerosis
- Tympanic membrane atrophy and retraction

Sequelae of OME

- TM atelectasis
- TM atrophy- retraction pockets- cholesteatoma
- Ossicular necrosis
- tympanosclerosis

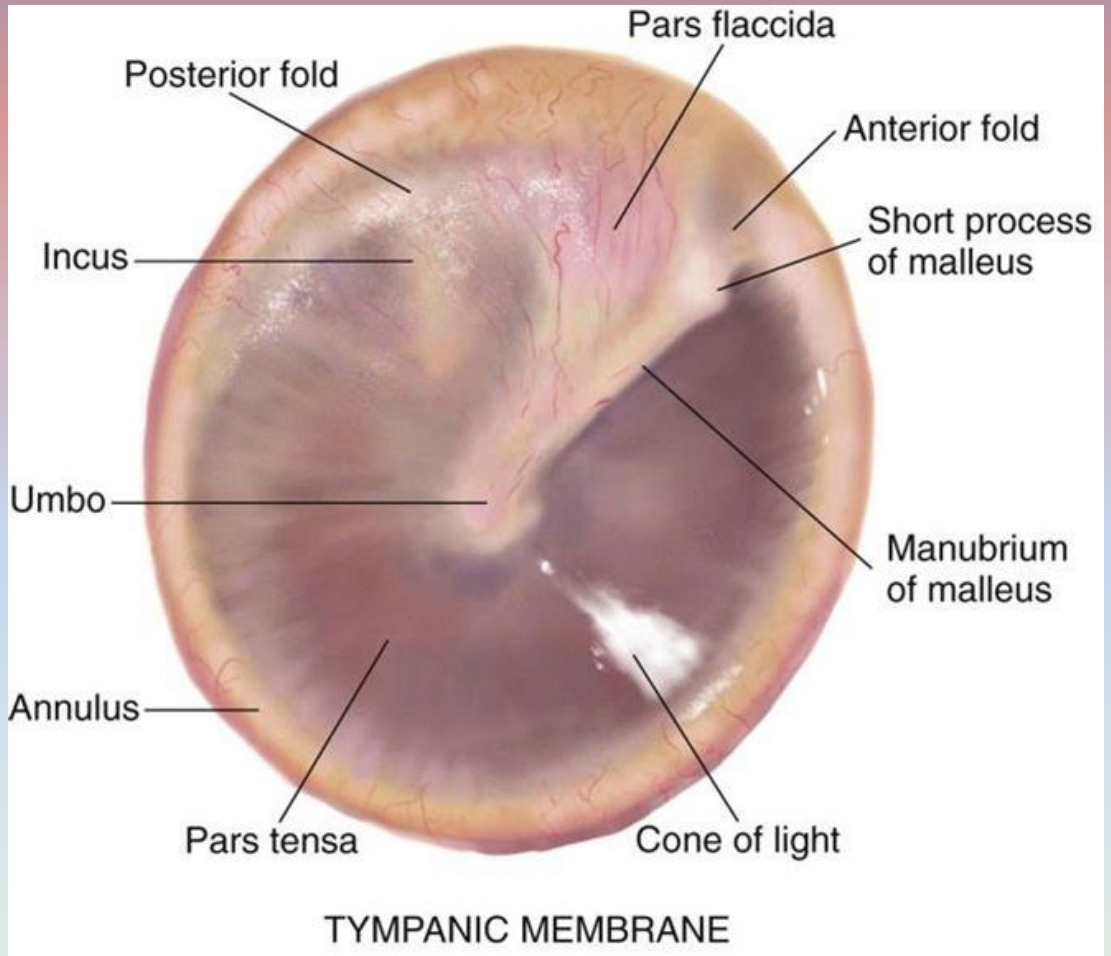
Acute Otitis Externa/ Swimmer's ear

- Definition: infection of the External Auditory Canal (EAC)
- Predisposing conditions: alkalization by retention of swimming water, trauma (cerumen removal or FB or scratching to relieve itching)
- MCC: ***Pseudomonas aeruginosa*** but *Staphylococcus & Streptococcus spp.* Also play an important role.
- P/C: severe otalgia, tenderness on pinna to movement, erythema & edema in EAC, purulent Otorrhea (sometimes)
- Rx:
  - Careful cleaning/ suction of canal
  - Topical antibiotic administration
  - Acetic acid to restore acidic environment
  - Severe obstruction: gauze wick to allow antibiotic drops to reach more medial aspects of canal.

CHRONIC SUPPURATIVE OTITIS MEDIA

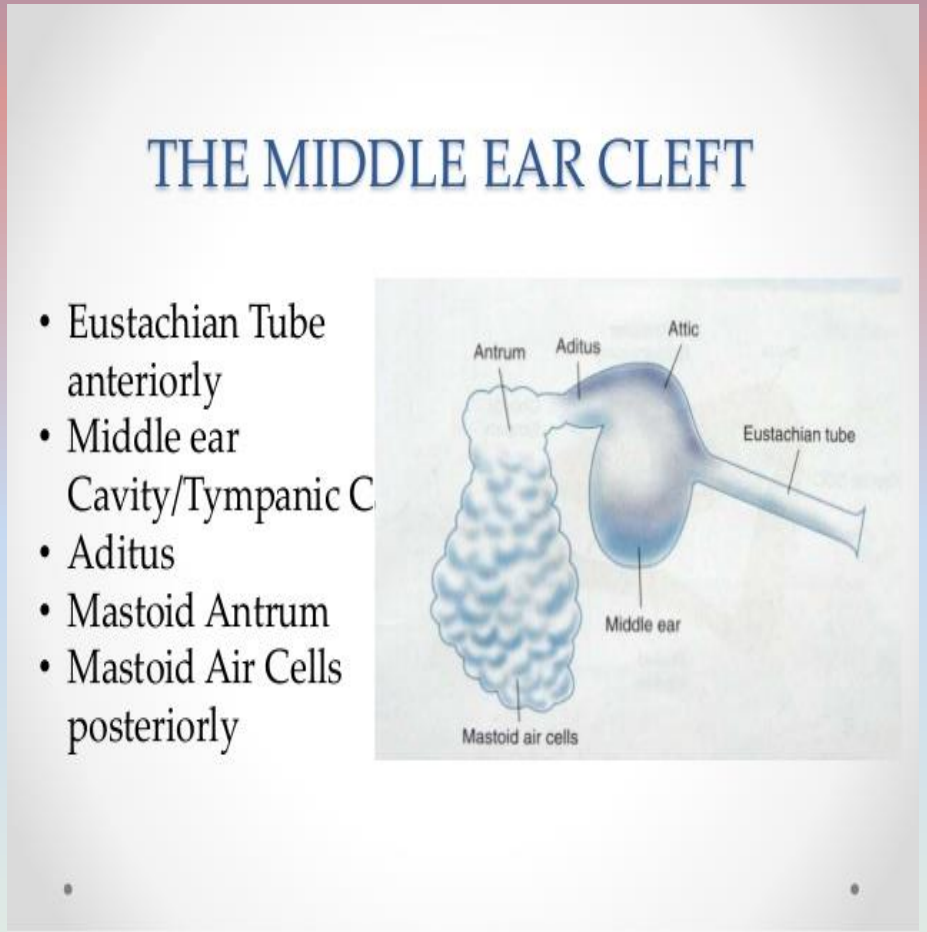
DR ELAINE YUKO

NORMAL TM


Pars flaccida
Posterior fold
Anterior fold
Short process of malleus
Incus
Umbo
Manubrium of malleus
Annulus
Pars tensa
Cone of light
TYMPANIC MEMBRANE

DEFINITION

- Chronic infection of the mucoperiosteal lining of the middle ear cleft.
- Granulation and associated unresolved and resistant bacterial infections



Introduction

- Poor populations increased incidence
- Most common cause of preventable deafness
- Developed countries mainly due to VT – no population based studies
- Risk factors
  - recurrent AOM
  - Traumatic perforations
  - Craniofacial anomalies
  - Lack of breastfeeding, overcrowding, poor hygiene and nutrition
  - High rate of nasopharyngeal colonization with potentially pathogenic bacteria with inadequate and unavailable healthcare
- Kenya: public health measures and appropriate medical care reduce incidence

Organisms

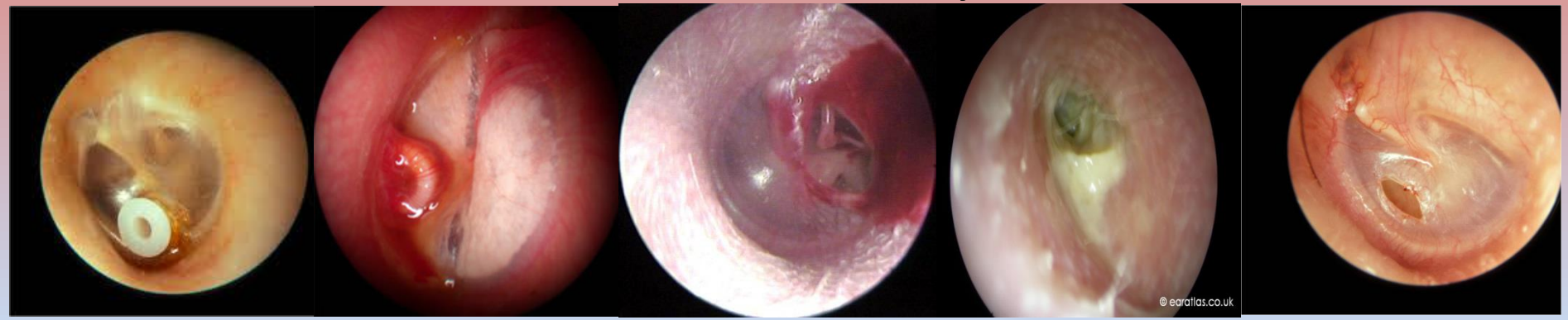
- Usually from skin of external auditory canal
  - Pseudomonas aeroginosa
  - Proteus
  - Staph spp.
  - Other gram negative and anaerobic bacteria

Clinical features

- Chronically discharging ear with TM perforation for over 2 weeks in absence of fever or significant pain
- Perforations become permanent when their edges are covered by squamous epithelium
- Otalgia or fever
  - R/o otitis externa esp. when patient attempts to clean purulent secretions with cotton bud
  - Mastoiditis or intracranial involvement
- Vertigo; calorific or from erosion of SCCs

Mucosal COM

- Chronic inflammation due to perforation
  - Active mucosal disease – wet
  - Inactive mucosal disease – dry



DRY GROMET

DISCHARGING GROMET +tympanosclerosis

TRAUMATIC PERFORATION

ACTIVE MUCOSAL

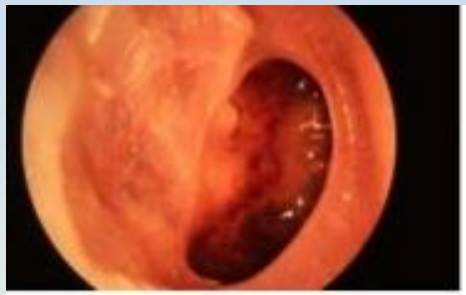
INACTIVE MUCOSAL

Squamous COM

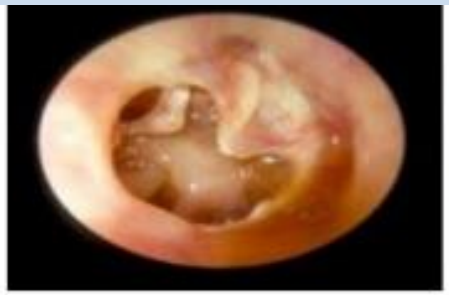
- Discharge due to cholesteatoma

Safe perforations/ tubotympanic

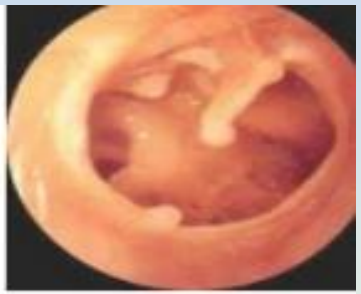
- (anteroinferior part of TM, central perforations), low risk of choleasteatoma
  - Rim of remnant TM or atleast the annulus (Central perforation).
  - Reniform (kidney shaped) because of poor blood supply to the affected portion of the tympanic membrane.
  - Mucopurulent discharge not foul smelling increases with increasing frequency of URTIs



Central perforation



Subtotal perforation



Total perforation

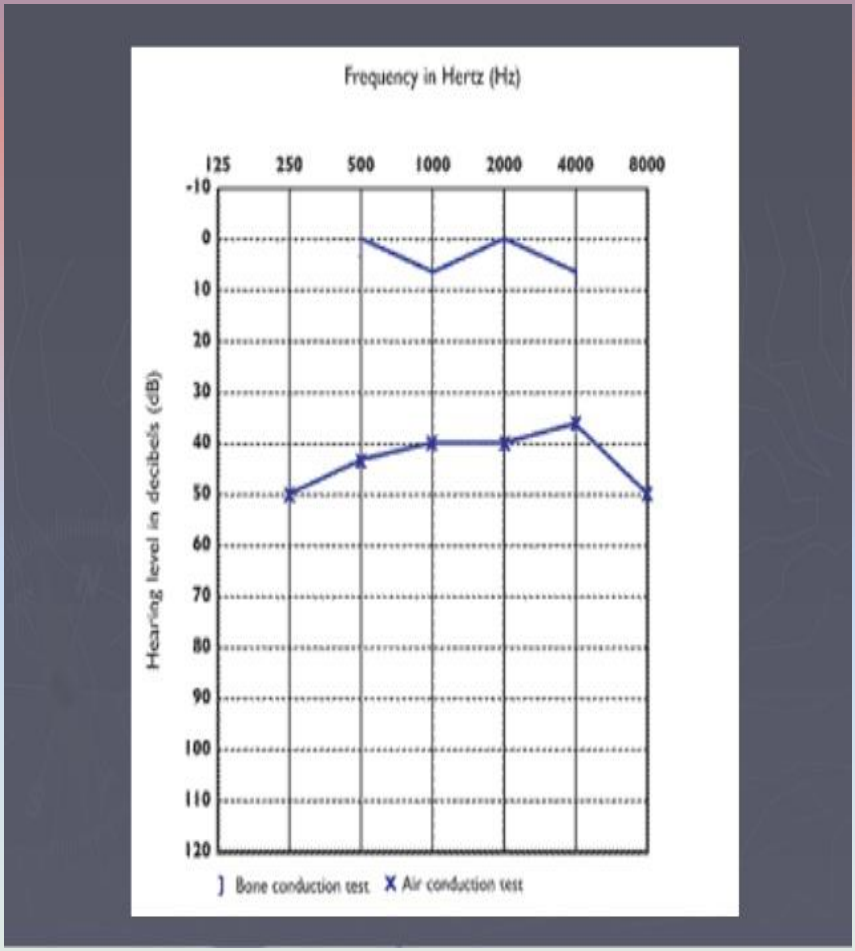
Safe perforations/ tubotympanic

- The ossicular chain rarely affected
- CHL: perforation and TM thickening, thickening of round window membrane due to secretions.
- Poorly pneumatised mastoids; repeated ME infections during childhood
- In pneumatised mastoid air cell system repeated ME infections causes sclerosis with new bone formation.

Stages of CSOM

- **Acute**: actively discharging. ME mucosa hypertrophied and congested.
- **Inactive**: dry perfortion, esp. antero inferior near ET orifice. ME mucosa is normal.
- **Quiescent**: Dry Perforation, mucosa normal or hypertrophied.
- **Healed**: neotympanun with thin scar +/- tympanosclerotic patches / chalky deposits. Intact ossicular chain.

Hearing assessment



- TFTs: Rinne - Negative on the affected side, Weber - Lateralised to the good ear, CHL,
- Absolute bone conduction test - Not reduced
- PTA: CHL; usually under 40 dB.

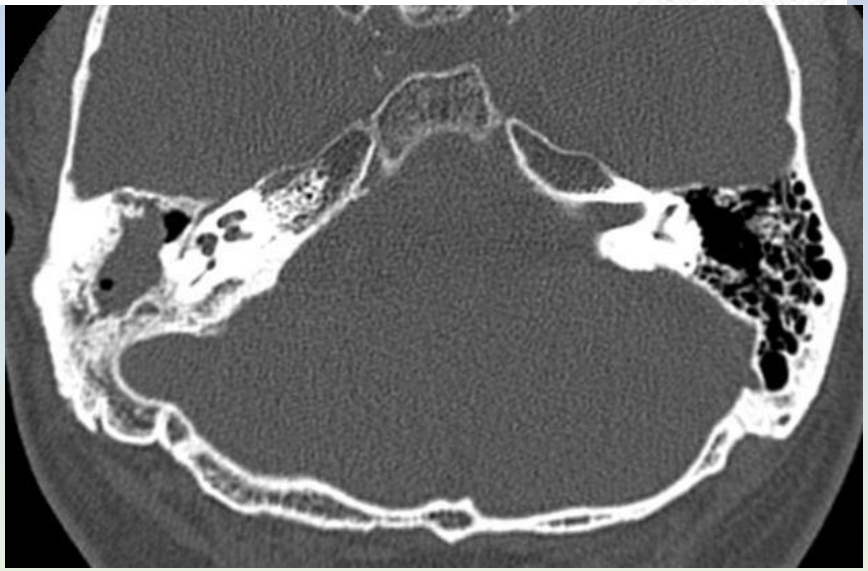
Unsafe perforations

- Atticoantral (posterosuperior part of TM;attic or marginal preforations)
- Attic is crowded, with head of the malleus + incus, High risk of granulation and cholesteatoma causing bone erosion via osteitic reaction. >complications
- Sclerosed mastoid cavities with granulation tissue

Cholesteatoma

Primary acquired cholesteatoma

www.drnkotb.com



- Skin in the wrong place, Ectopic epithelium usually in the attic
- Cystic bag lined by stratified squamous epithelium on a fibrous matrix with a sac of desquamated squamous epithelium
- Epithelial cells naturally shed within this pocket but cant escape ME
- Causes – retraction pockets in pars flaccida
- No metastatic potential, destructive if untreated

Cholesteatoma

- Scanty and foul musty smelling discharge; saprophytic infection and osteitis.
- Hearing loss
  - usually CHL
  - May have normal hearing ?bridging effects of cholesteatoma
  - TInnitus may precede SNHL: absorption of toxins through the round window membrane, long term use of ototoxic antibiotics
- OTALGIA: R/O otitis externa, or extradural abscess.
- Vertigo: erosion of lateral SCC by the cholesteatomatous matrix.
- Fistula test: a snugly fitting siegles pneumatic speculum and slowly applying pressure by compressing the pneumatic bulb. If present; dizzyness and nystagmus.
- Facial palsy : facial nerve canal with involvement of facial nerve.

Cholesteatoma

- Destruction of outer attic wall, with attic perforation. Cholesteatomatous flakes
- Sagging of the posterior superior meatal wall.
- At least a modified radical mastoidectomy +/- tympanopalsty +/- ossicualr chain reconstruction
  - To exteriorize the disease and create adequate ventilation to the ME
  - Create a permanent skin lined cavity exposed to the exterior.

Facial nerve palsy: House brackman classification

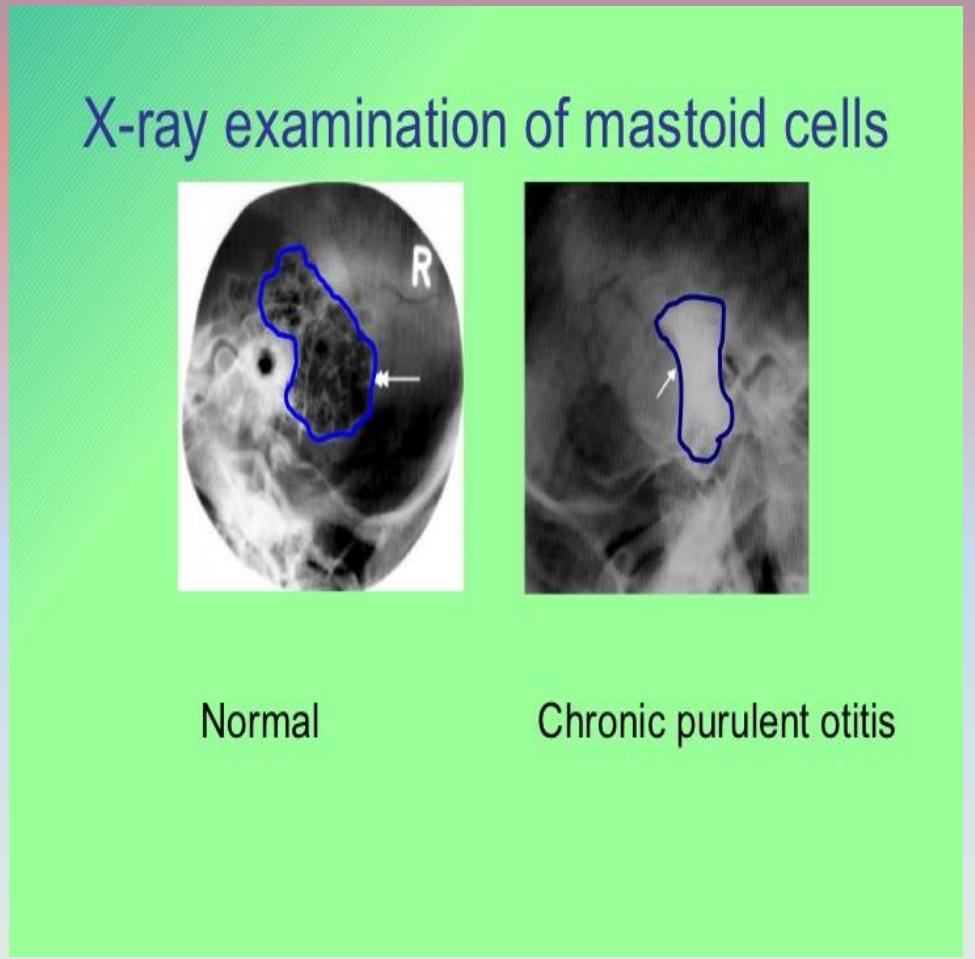
Grade	Description	Characteristics
I	Normal	Normal facial function in all areas
II	Mild dysfunction	Gross: slight weakness noticeable on close inspection; may have very slight synkinesis At rest: normal symmetry and tone Motion: Forehead—moderate-to-good function Eye—complete closure with minimum effort Mouth—slight asymmetry
III	Moderate dysfunction	Gross: obvious but not disfiguring difference between two sides; noticeable but not severe synkinesis, contracture, and/or hemifacial spasm At rest: normal symmetry and tone Motion: Forehead—slight-to-moderate movement Eye—complete closure with effort Mouth—slightly weak with maximum effort
IV	Moderately severe dysfunction	Gross: obvious weakness and/or disfiguring asymmetry At rest: normal symmetry and tone Motion: Forehead—none Eye—incomplete closure Mouth—asymmetric with maximum effort
V	Severe dysfunction	Gross: only barely perceptible motion At rest: asymmetry Motion: Forehead—none Eye—incomplete closure Mouth—slight movement
VI	Total paralysis	No movement

Examination

- General exam
- Look for previous surgical scars
- OTOSCOPY: via otoscope, microscope, rigid scope
- Cranial nerve exam; esp facial nerve
- TFTs
- Vertigo tests
- Neurological exam: r/o intracranial complications

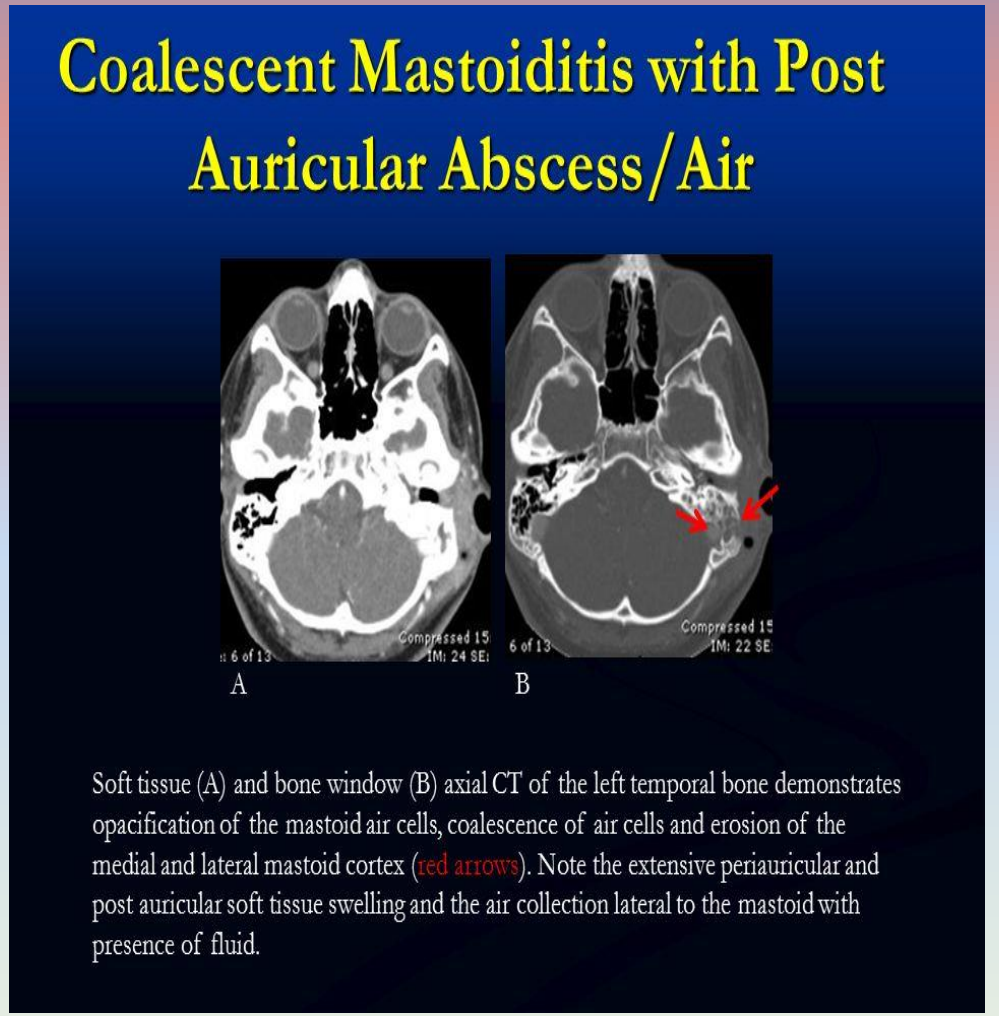
Investigations

- PTA, tympanometry, *BERA
- Xrays: Mastoid views, PNS
- HRCT TB – R/O cholesteatoma, mastoiditis, other complications
- M/C/S: Pus swab, deep aspirate
- Features acute on chronic infection: hemogram



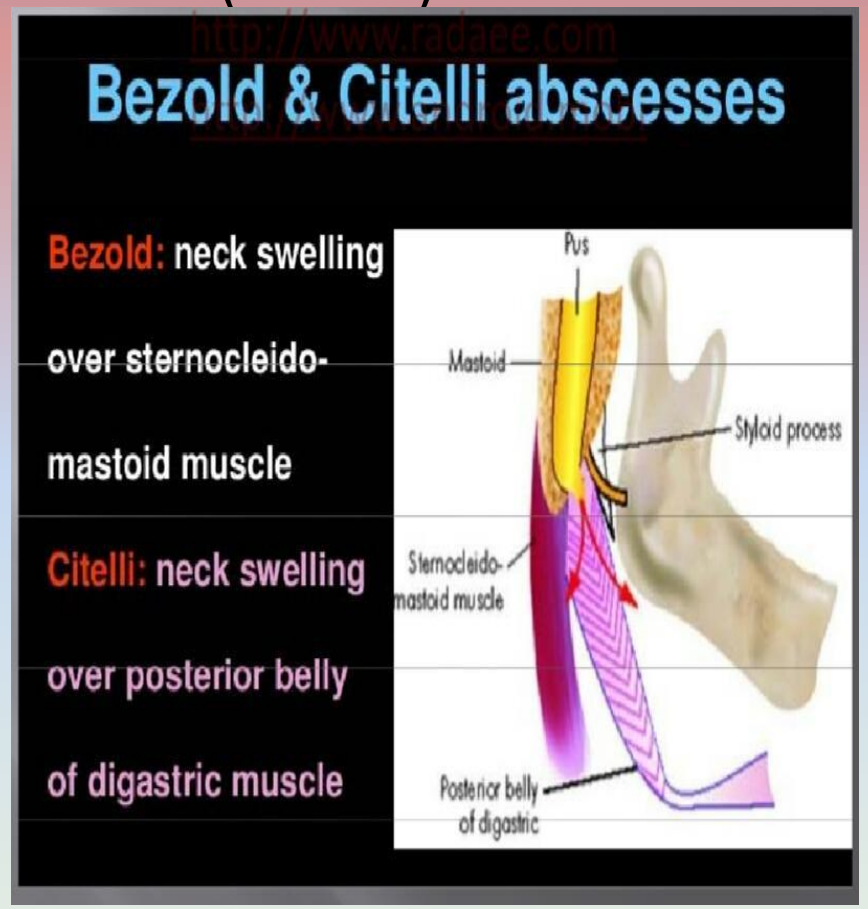
Complications

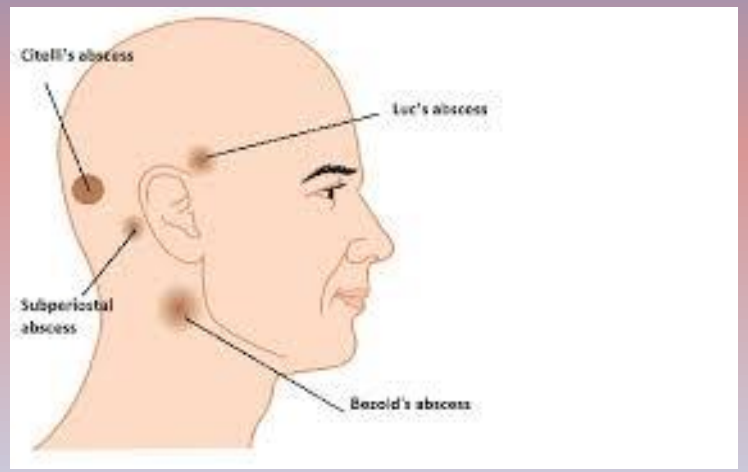
- Extracranial
  - Postauricual abscess
  - Mastoid abscess
  - Petrositis
  - Labyrinthitis
  - Facial nerve palsy

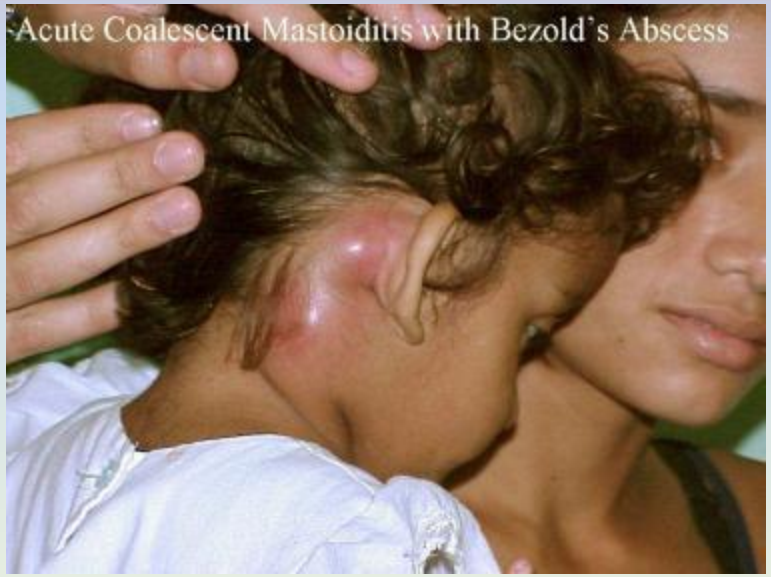


Extracranial complications

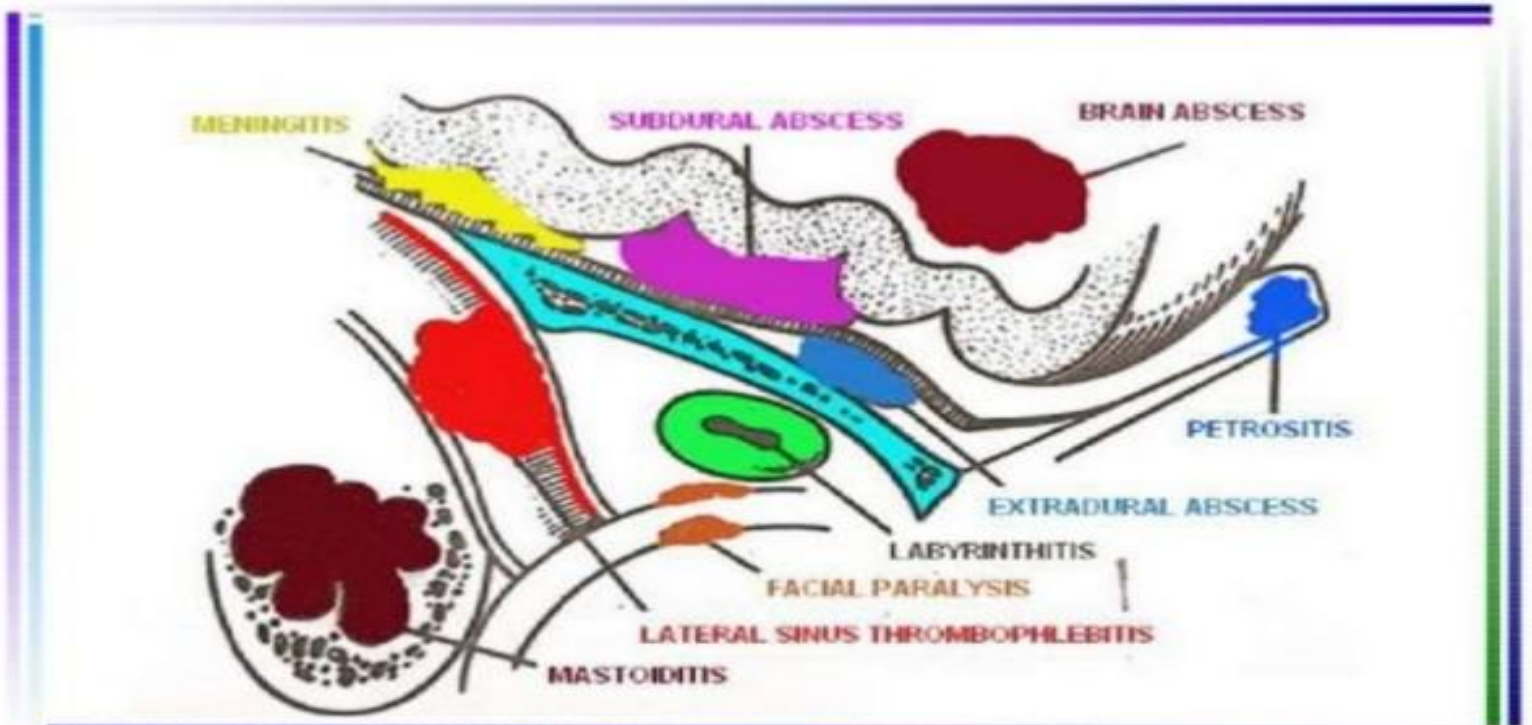
– Luc's abscess (meatal)







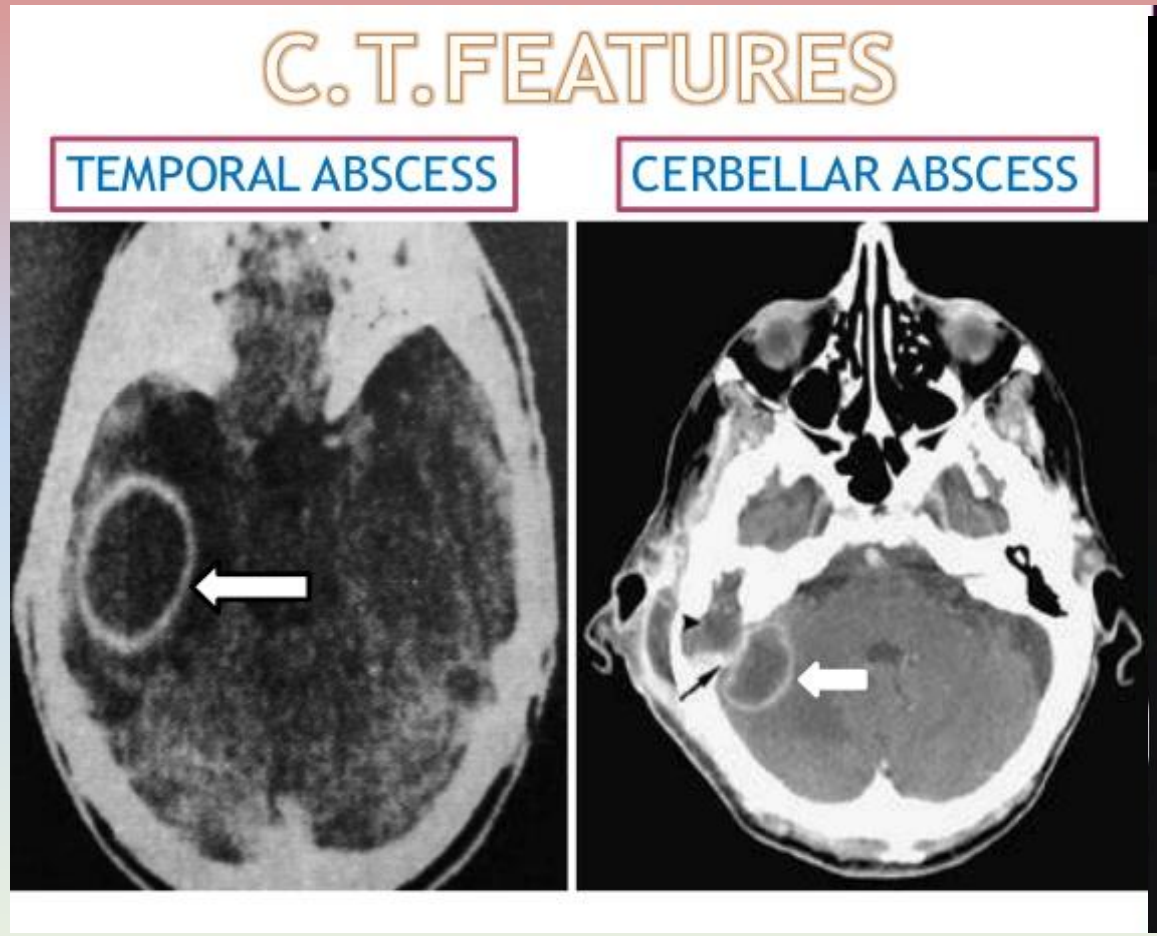
Intracranial complications

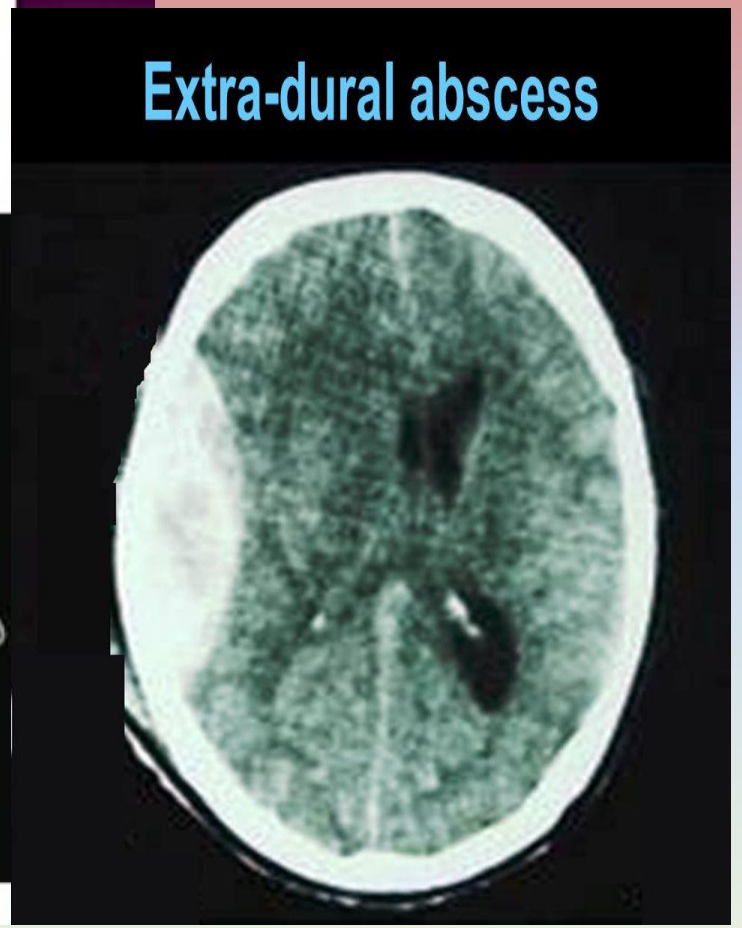


- Encephalitis
- Otic hydrocephalus

Intracranial abscesses

- Extradural, epidural, subdural, perisigmoid, brain abscess



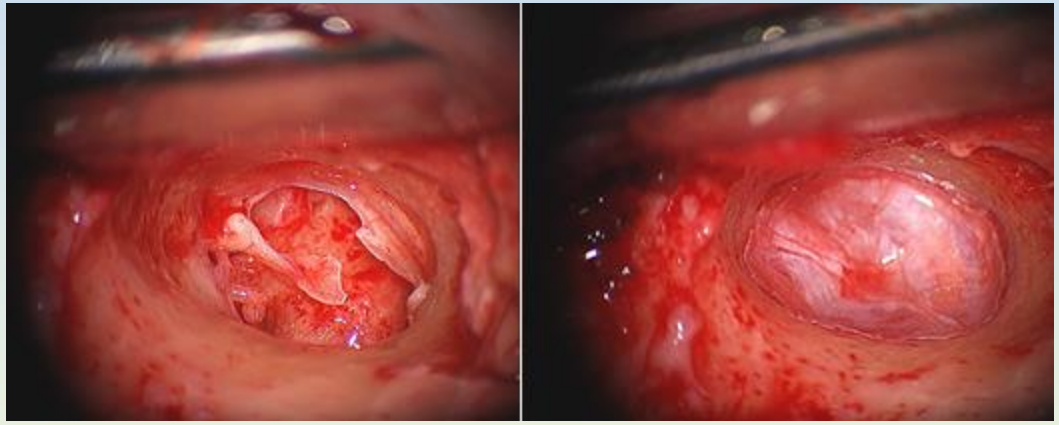


Treatment

- Aural toilet via:
  - Dry mopping and wicking
  - suctioning
  - Syringing: with warm saline mixed with acetic acid (1.5%) dissovles crusts and has bacteriostatic effect.
- Role of parenteral antibiotics: acute on chronic cases and complications
- Topical antibiotics: large central perforation enables adequate concentration of antibiotics to reach the ME mucosa.
- Topical quinolones; ciprofloxacin, ofloxacin
- Avoid ototoxic drugs: increased vascularity in the ME mucosa will cause easy absorption into the inner ear fluids causing SNHL
- KEEP EAR DRY/ protect ear
- Hearing aid assessment and augmentation
- Treat vertigo: vestibular suppressants

Treatment

- Surgery
  - Myringoplasty: closure of the perforation in pars tensa via autologous graft (fascia, cartilage, synthetic grafts) to allow scafold for neotympanicum to form
  - Tympanoplasty – Myringoplasty + ossicular chain reconstruction
  - Drainage of abscesses via I&D, mastoidectomy, cranoitomy for intracranial abscesses with neurosurgical team



HEARING LOSS

SERAH NDEGWA UON 21/5/2018

Outline

- Introduction
- Causes of hearing loss
- Hearing Loss Dimensions
- Hearing Assessment
- Management of Hearing loss

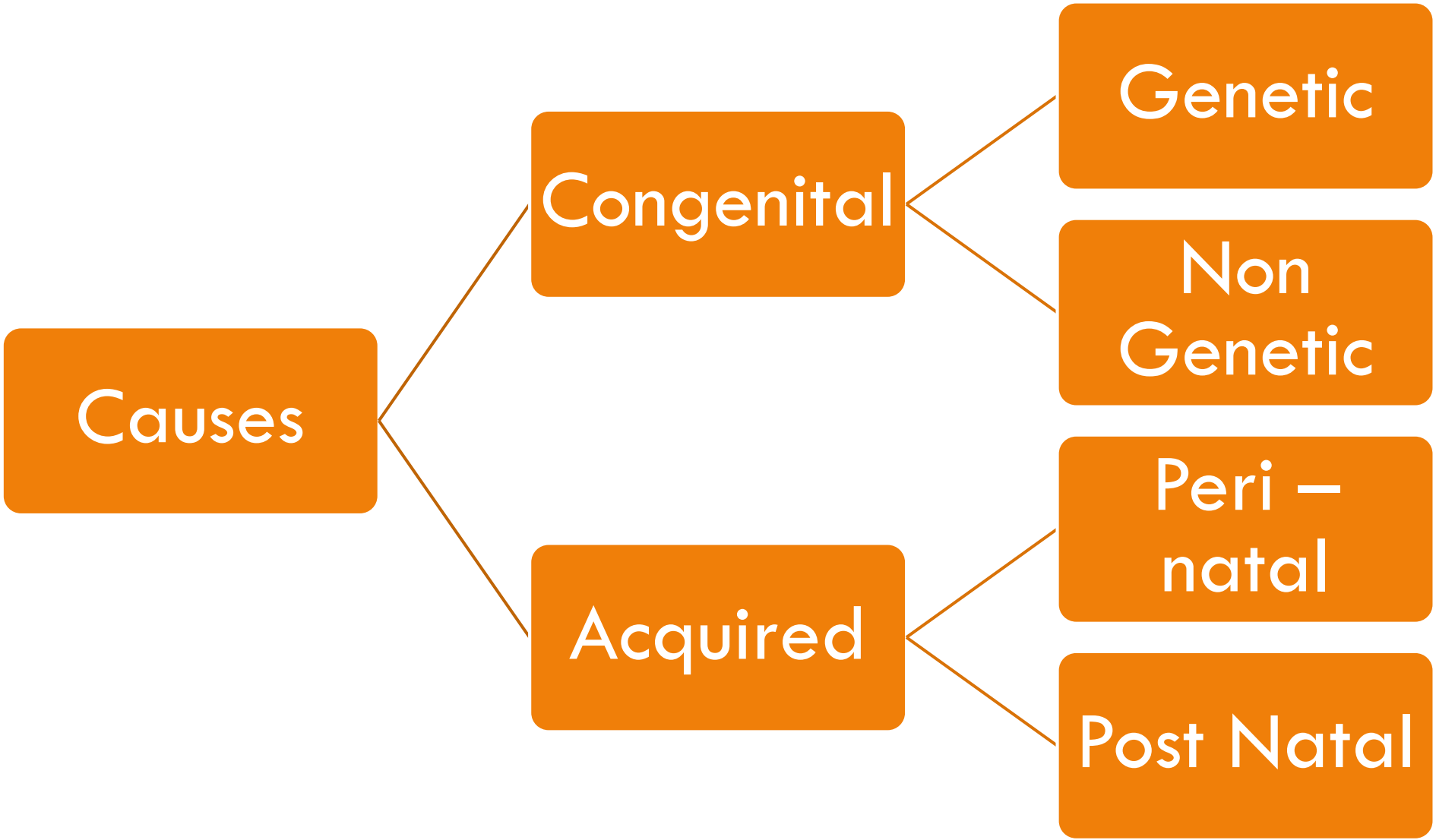
Introduction

- Hearing loss is often referred to as a ***hidden disability.***
- Depending on severity, it affects:
  - Interpersonal communication
  - Social interactions
  - Development of speech and language in children
  - Educational performance
  - Employment prospects
- Early diagnosis and intervention aims at minimizing these effects

Cont.

- 2/3 of the burden of hearing impairment is in developing countries & ***50% of the burden of Hearing loss is preventable***.

Causes of Hearing Loss



A. CONGENITAL CAUSES

GENETIC	NON GENETIC (TORCHES)
Branchio – Oto – Renal syndrome	Toxoplasmosis
Treacher Collins Syndrome	Rubella
Sticklers Syndrome	Cytomegalovirus
Crouzon syndrome	Herpes
Pendred syndrome	Syphilis
Waardenburg syndrome	
Alport Syndrome	
Jervell & Lange – Nielsen Syndrome	
Usher - Hallgren Syndrome: causes progressive hearing loss	

B. ACQUIRED CAUSES

Peri natal causes	Post Natal Causes
LBW < 1500g	Otitis Media: AOM, COM, OME, Cholesteatoma
Prematurity	Wax, foreign bodies
HIE	Meniere's disease, Labyrinthitis
Hyperbilirubinemia	Noise Induced Hearing Loss
Sepsis	Presbycusis
Infections: Measles, Mumps, Meningitis	Cerebrovascular disease: HTN
Ototoxic Drugs: Aminoglycosides, Quinine, Loop diuretics, cisplatin	Tumors: Acoustic Neuroma, Glomus tumor
	AIDS
	Trauma: Temporal bone fractures

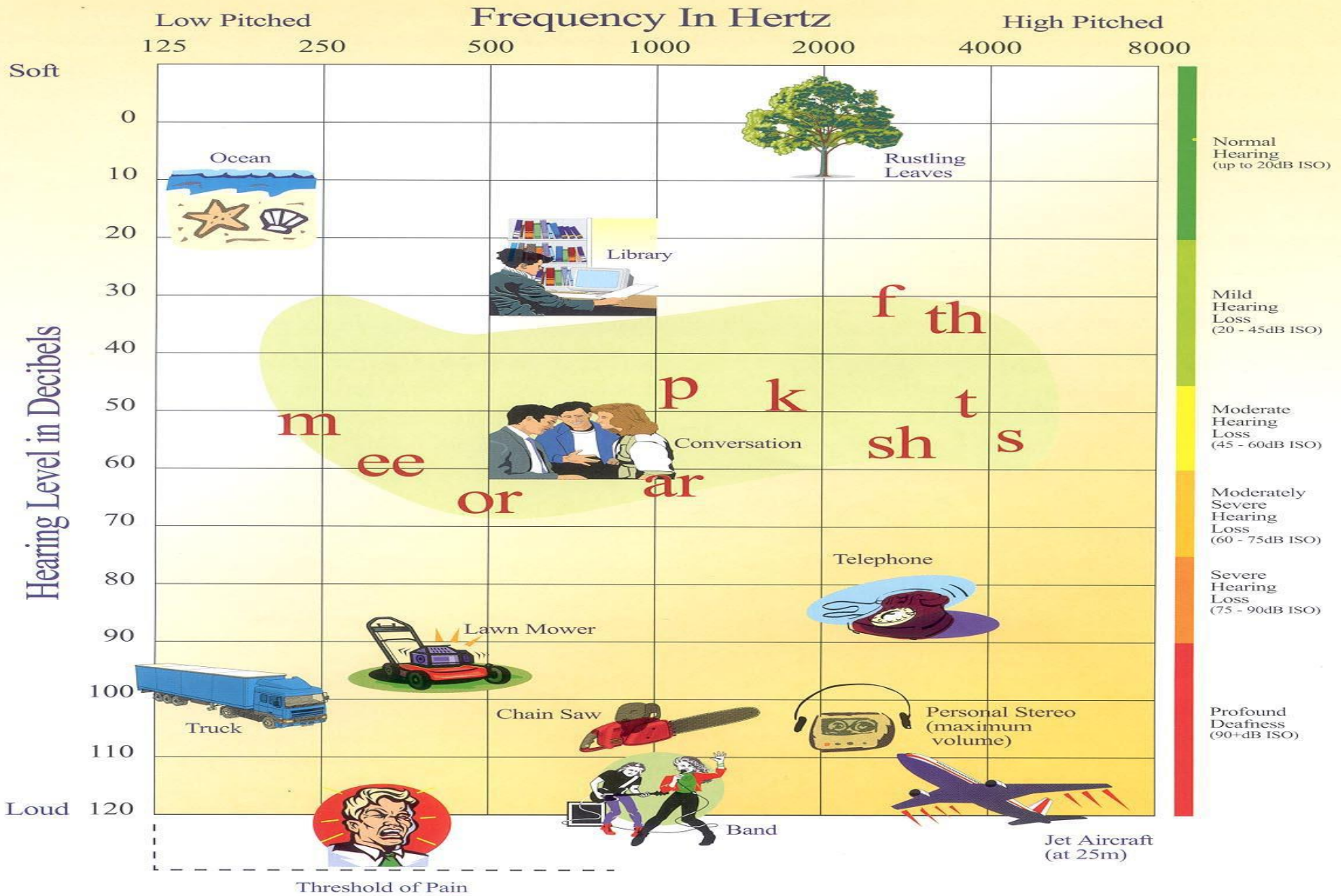
Summary of the main causes of hearing loss (WHO survey)

HIGH PROPROTION	MODERATE PROPORTION	LOW PROPORTION
Genetic Causes	Excessive Noise	Nutritional related
Otitis Media	Ototoxic drugs	Trauma related
Presbycusis	Antenatal & Perinatal problems	Meniere's disease
	Infectious causes	Tumors
	Wax & Foreign Bodies	Cerebrovascular disease

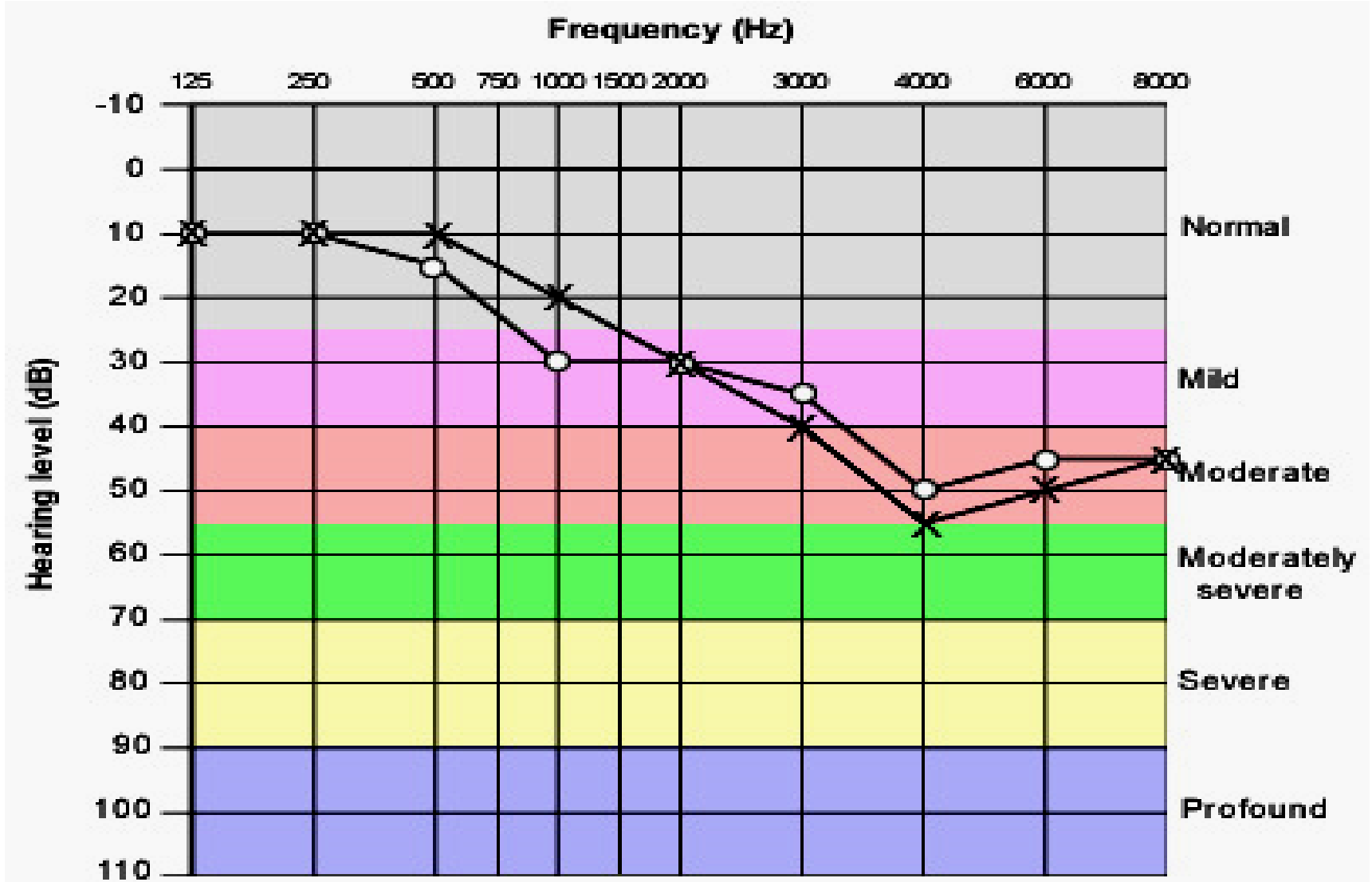
Hearing loss dimensions

Degree of loss	Type	Onset and Progression
• Mild • Moderate • Mod-severe • Severe • Profound	• Conductive • Sensorineural • Mixed	• Sudden or Progressive • Pre – lingual, Peri – lingual Post Lingual

Frequency and Intensity of Familiar Sounds



Degree of Hearing Loss



MILD HL (26-40 dbhl)

- **Difficulty hearing soft speech**
- Difficulty hearing in background noise.
- **Will acquire speech & language but may have some delay.**
- Can benefit from hearing aids.

MODERATE HL (41- 55 DBHL)
MODERATELY SEVERE (56- 70 dbhl

- Difficulty hearing conversation, especially in background noise.
- Usually hears vowels but great difficulty hearing consonants
- Hearing aids essential for speech & language development

SEVERE HL(71-90 DBHL)

- Loud environmental sounds audible.
- **Shouting may not be detected unless at close range.**
- Hearing aids and intervention essential for speech & language development

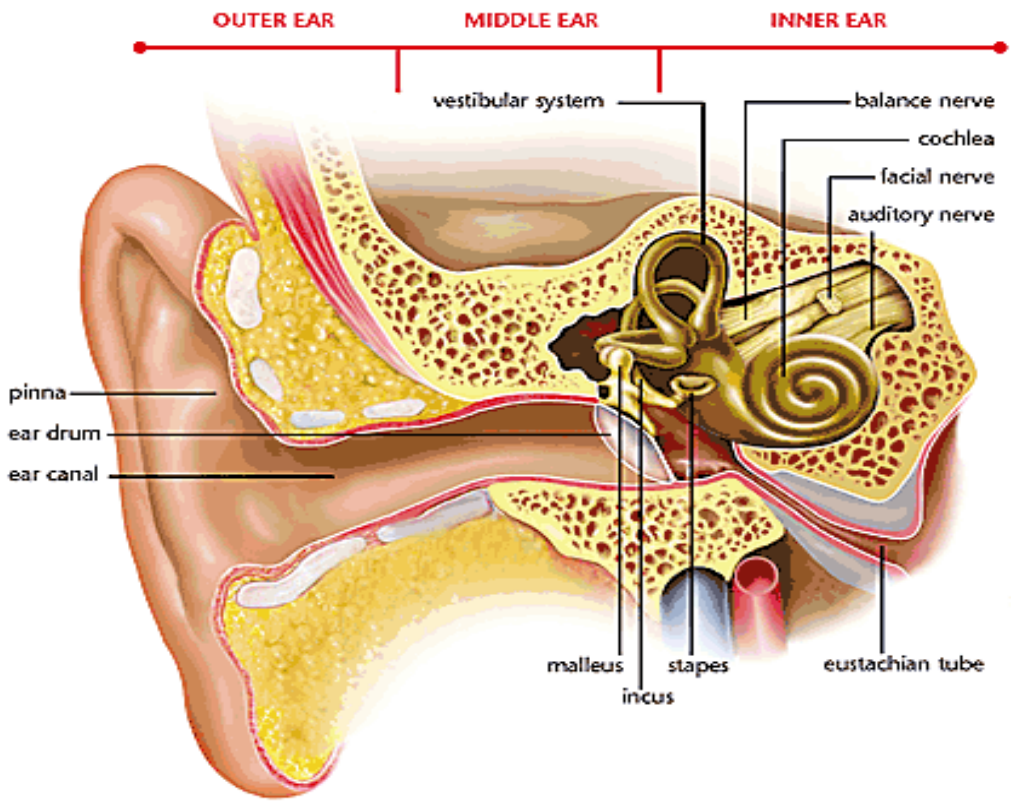
PROFOUND HL (91+ dbhl)

- All speech and most environmental sounds are inaudible.
- **Many will benefit more from a cochlear implant than from hearing aids.**
- Oral aural language will not occur without intensive speech language intervention

WHO Grades of Hearing Impairment

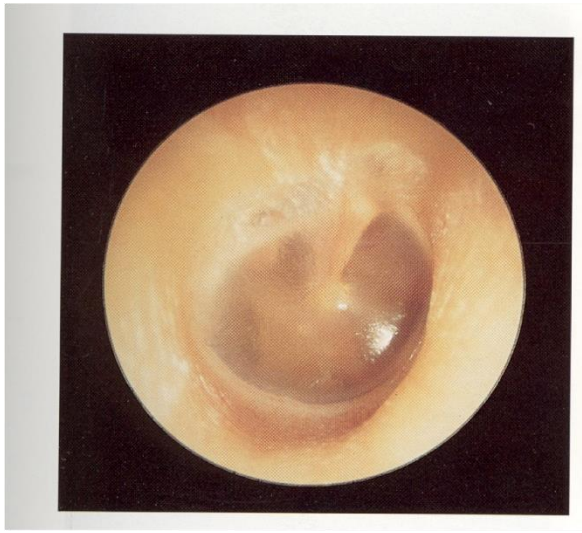
Normal Hearing	Hears whispers
Mild Hearing loss Grade 1	Hears and repeats words in normal voice at 1m
Moderate Hearing Loss Grade 2	Hears and repeats words in raised voice 1 m
Severe Hearing Loss Grade 3	Hears words shouted into better ear
Profound Hearing Loss Grade	Cannot hear/ understand shouted voice

Basic Anatomy



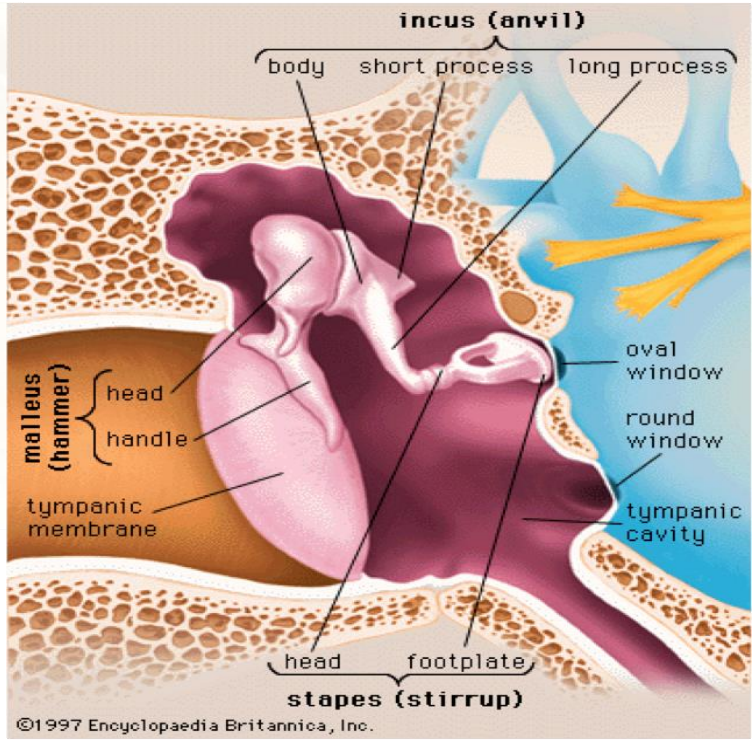
Middle Ear

Tympanic Membrane

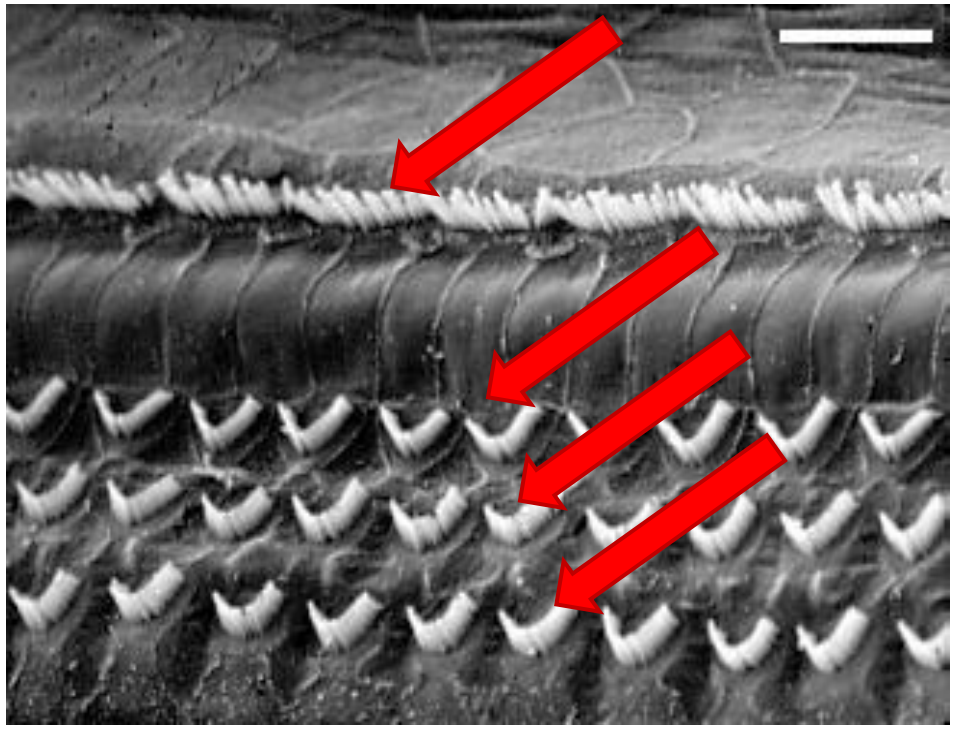


The Ossicles:

- Malleus
- Incus
- Stapes

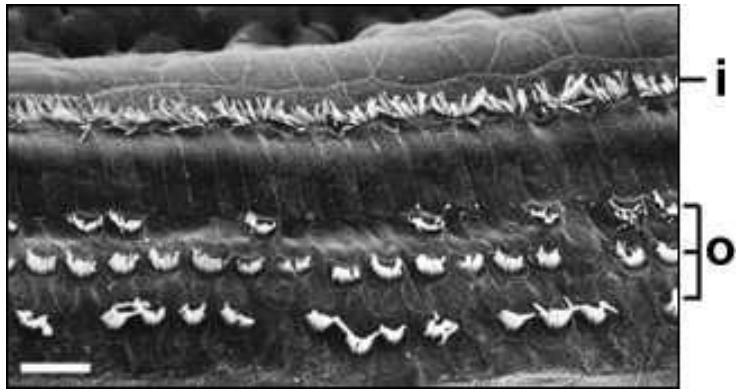


Normal Hearing Looks Like:



- Hair cells are the sensory cells of the auditory system
  - Outer Hair Cells
  - Inner Hair Cells
- Both inner and outer hairs cells have different functions in the auditory system
- Loss of either type of these hairs cells affects the way we hear speech
  - Outer Hair Cells: audibility
  - Inner Hair Cells: clarity

Sensorineural Hearing Loss



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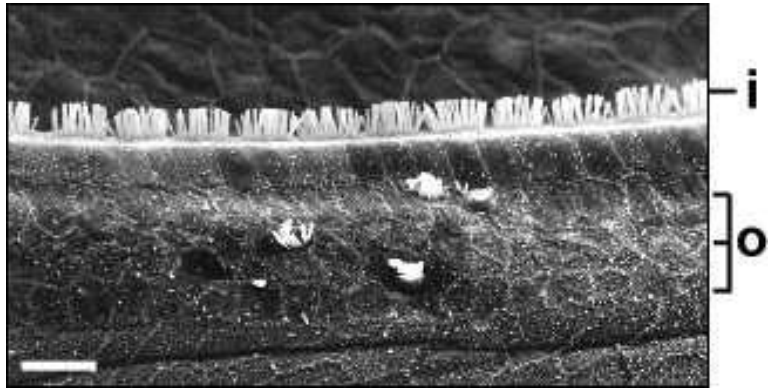

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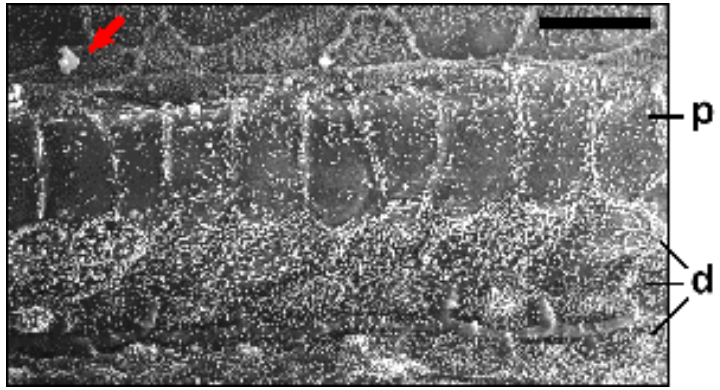
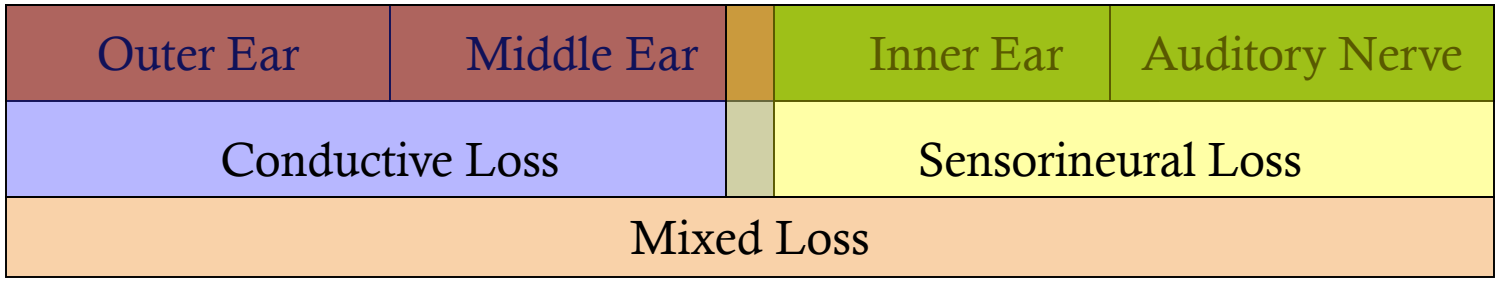
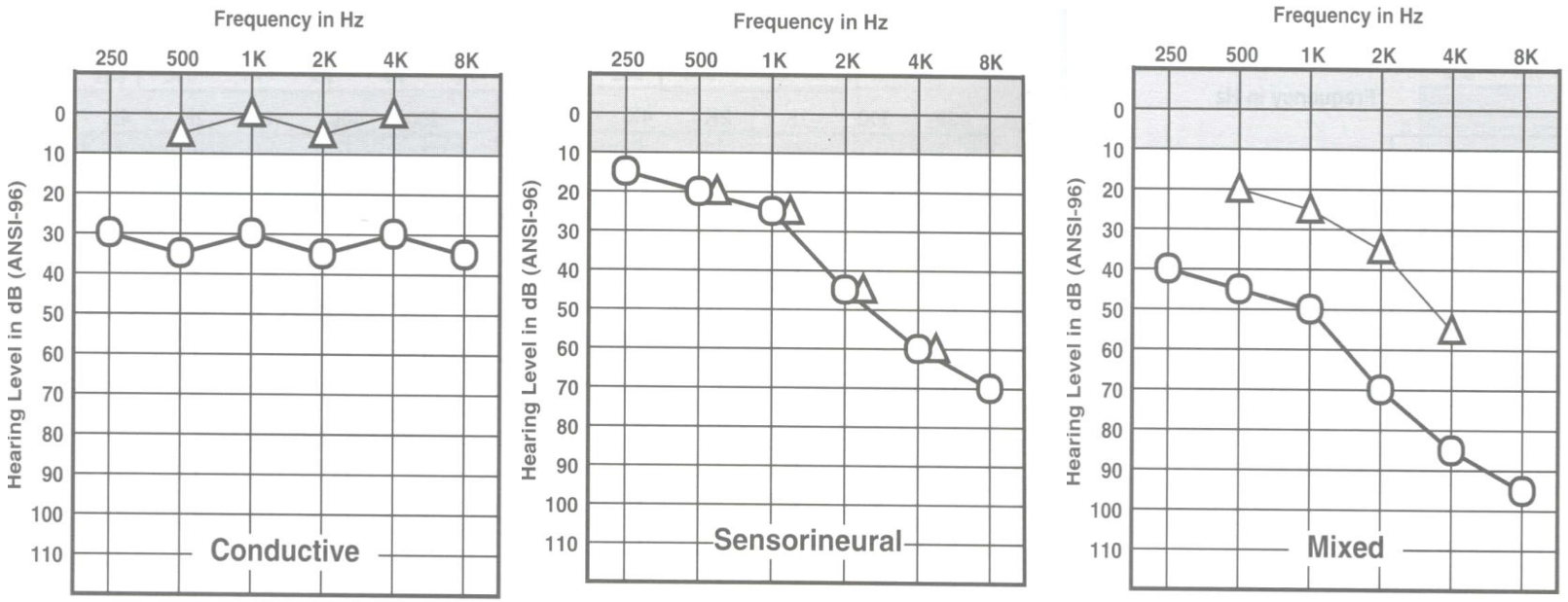


GONE

Type of Loss

- Compare air conduction and bone conduction thresholds





Audiological tests

SUBJECTIVE

ADULT

- Pure Tone Audiometry
- Speech Audiometry

PAEDIATRIC

- Behavioral Observation Audiometry (BOA), Distraction test,
- Visual reinforced Audiometry (VRA)
- Conditioned Play Audiometry

OBJECTIVE

- Auditory BrainstemResponse Audiometry
- Otoacoustic Emmissions
- Tympanometry

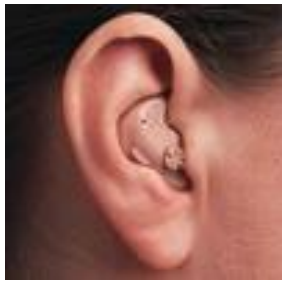
Hearing Loss Interventions

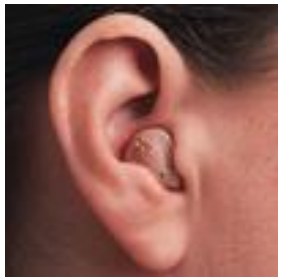
I. Medical
II. Surgical
III. Aural Rehabilitation
   - Hearing Aids
   - Cochlear Implants
   - Auditory brainstem implant (ABI)

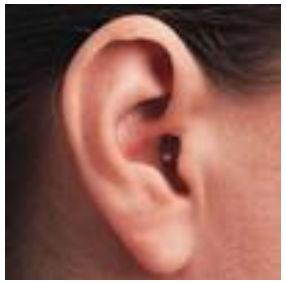
HEARING AIDS

- BTE Most common type used
- Digital or Analogue
- All degrees of hearing loss
- Binaural fitting advantages +++

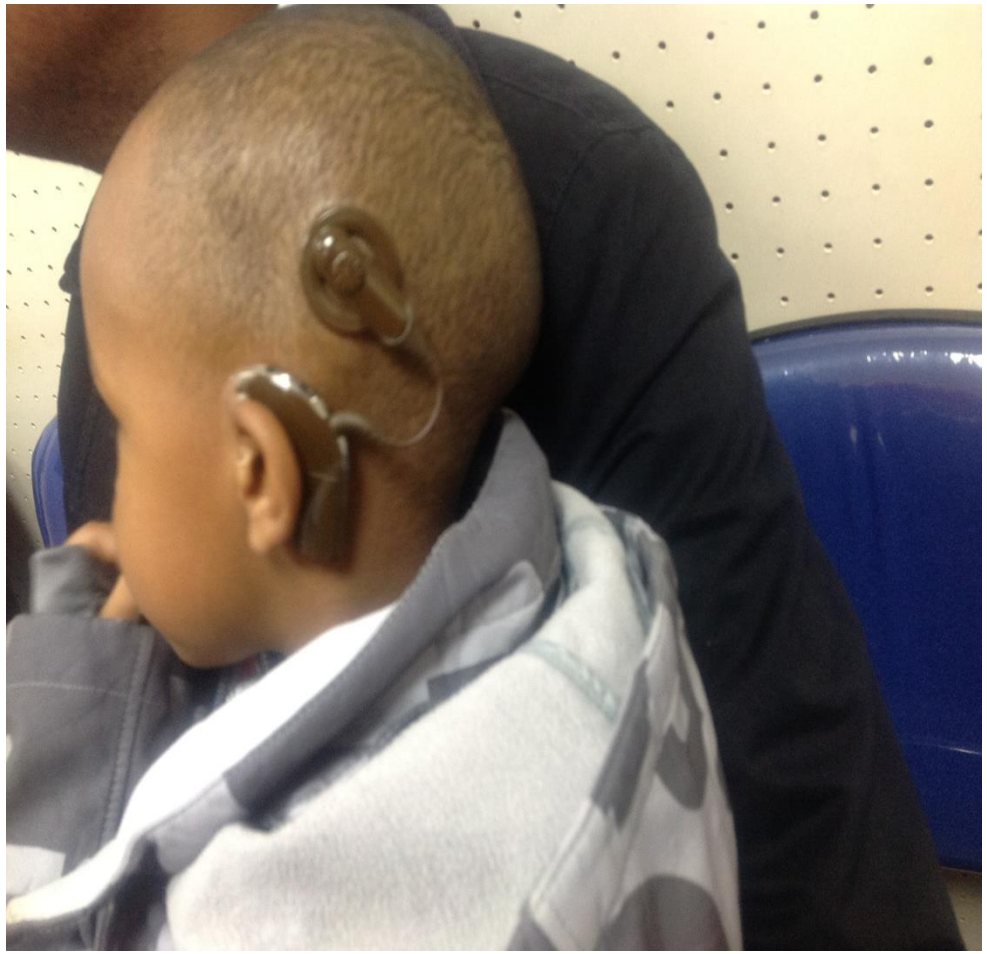
Hearing Aids

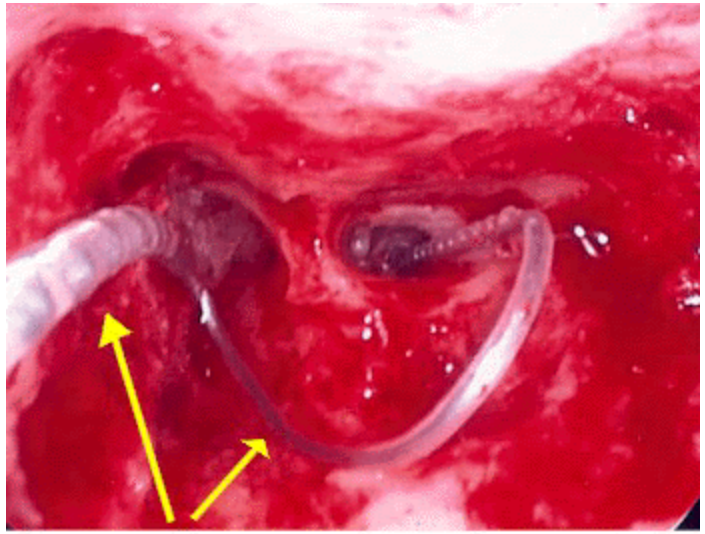






COCHLEAR iMPLANT





The electrode coils are inserted into the mastoid with plenty of slack for future growth

UPPER AIRWAY OBSTRUCTION 2ND/5/2019

BY: DR. NYAGAH

TYPED BY NAILA KAMADI

Anatomy of the upper air

- Nose
- Pharynx
  - Nasopharynx
  - Oropharynx
  - Hypopharynx/ laryngopharynx
- Larynx
- Extra – thoracic trachea

Classification of UAO

- Acute or chronic
- Partial or complete
- Acquired or congenital

Acute UAO

- Infectious
  - Viral
  - Bacterial
- Foreign bodies
- Trauma
  - Physical
  - Burns
- Anaphylaxis

Viral LTB/ croup

- Causes UAO in children below 2 yrs.
- Caused by parainfluenza virus in 75% of cases
- Presents with a barking cough, hoarse voice or cry +/- stridor
- Preceded by symptoms of a cold with a low grade fever
- Treatment:
  - ***Supportive***: analgesics, antipyretics
  - ***Systemic steroids & Nebulized adrenaline***: reduces nasal congestion & edema
  - ***ETT intubation***: depends on the patient's symptoms (<3% may require ETT intubation)

Infectious mononucleosis

- Caused by ***EBV***.
- Presents with ***sore throat, dyspnea, dysphagia & drooling.***
- Marked tonsillar enlargement (grade III & IV).
- Long term effects of EBV infection:
  - Nasopharyngeal carcinoma
  - Burkitt's lymphoma

Bacterial tracheitis

- Life threatening with 80% of children requiring intubation.
- Children affected are older than those with croup.
- Cause: ***S. aureus, S. pneumoniae, H. influenza***
- Starts as an URTI then suddenly deteriorates.
- P/C: Very toxic with high fevers, productive cough, hoarse voice, respiratory distress
- First line regiment: ***ceftriaxone***

Epiglottitis/ supra – glottitis

- Affects the false vocal cords & the epiglottis
- Affects both children and adults
- May present with severe UAO
- Caused by ***H. influenza type b*** that causes edema of the entire supraglottis.
- Presents with ***dysphagia, drooling, dysphonia & dyspnea***
- It is a very rapidly progressing disease that will require intubation.
  - At laryngoscopy: omega – shaped big epiglottis
- First line Rx: ceftriaxone

Abscesses

- Commonly:
  - ***Retropharyngeal*** in children
  - ***Peri – tonsillar (quinsy)*** in young adults.
- Caused by: ***staphylococcus aureus, Streptococcus pneumonia***
- Presents with: ***neck pains***, ***swelling***, ***trismus***, ***dysphagia***, ***fever, hot potato voice*** & ***may case UAO***
- Rx:
  - ABs
  - Sometimes surgical drainage

Tracheobronchial FB aspiration

- Develop a high index of suspicion in a previously well child who develops ***sudden onset, cough, choking*** or ***stridor***
- Presents less commonly with features of ***chronic cough*** & ***wheezes***. These cases have mistakenly been treated for refractory asthma, pneumonia or TB.
- Suspected FB, esp. in the acute setting will require ***immediate bronchoscopic removal***.
- X – ray may be of help if the FB is radio – opaque.
  - If it isn't, features suggestive of FB are ipsilateral atelectasis, abscess

Angioneurotic edema

- Ma be caused by allergy to foods, drugs, insect venom
- May be associated with anaphylactic shock
- Rx: immediate administration of ***IM adrenaline*** & ***O_2***
- Support of other systems if involved
- ***Hydrocortisone*** & ***chlorpheniramine*** may be helpful.

Trauma

- Blunt or penetrating
- Thermal/ inhalational burns
- Manage appropriately

Chronic UAO

- Inflammatory
- Anatomical
- Neoplastic

Inflammatory

- Infectious: Laryngeal TB (MCC)
- Non – infectious: granulomatous conditions e.g. Wegener's granulomatosis

Anatomical

- Children:
  - Enlarged adenoids and tonsils are commonest
- Adults:
  - Hypertrophied inferior turbinates/ concha, soft palatal collapse, large tonsils, enlarged base of tongue
  - Presents with snoring with or without sleep apnea

Neoplastic

- Benign:
  - Vocal cord polyps
  - Thyroid goiter
- Malignant:
  - NPC
    Laryngeal/ hypopharyngeal Ca.
  - Thyroid carcinoma etc.

Congenital UAO

- Presents with a congenital stridor
  - Laryngomalacia: stridor disappears in sleep; disappears with time
  - Glottic webs
  - Subglottic hematomas

General features of UAO

- Stridor: high pitched sound during breathing due to downward moving of the larynx
  1. Inspiratory
  2. Expiratory
  3. Biphasic
- Snoring: low pitched in the nasopharynx & oropharynx; associated with chronic conditions.
- Tachypnea, nasal flaring, use of accessory respiratory muscles, cough etc.
- Cyanosis, loss of consciousness, respiratory failure.

Management

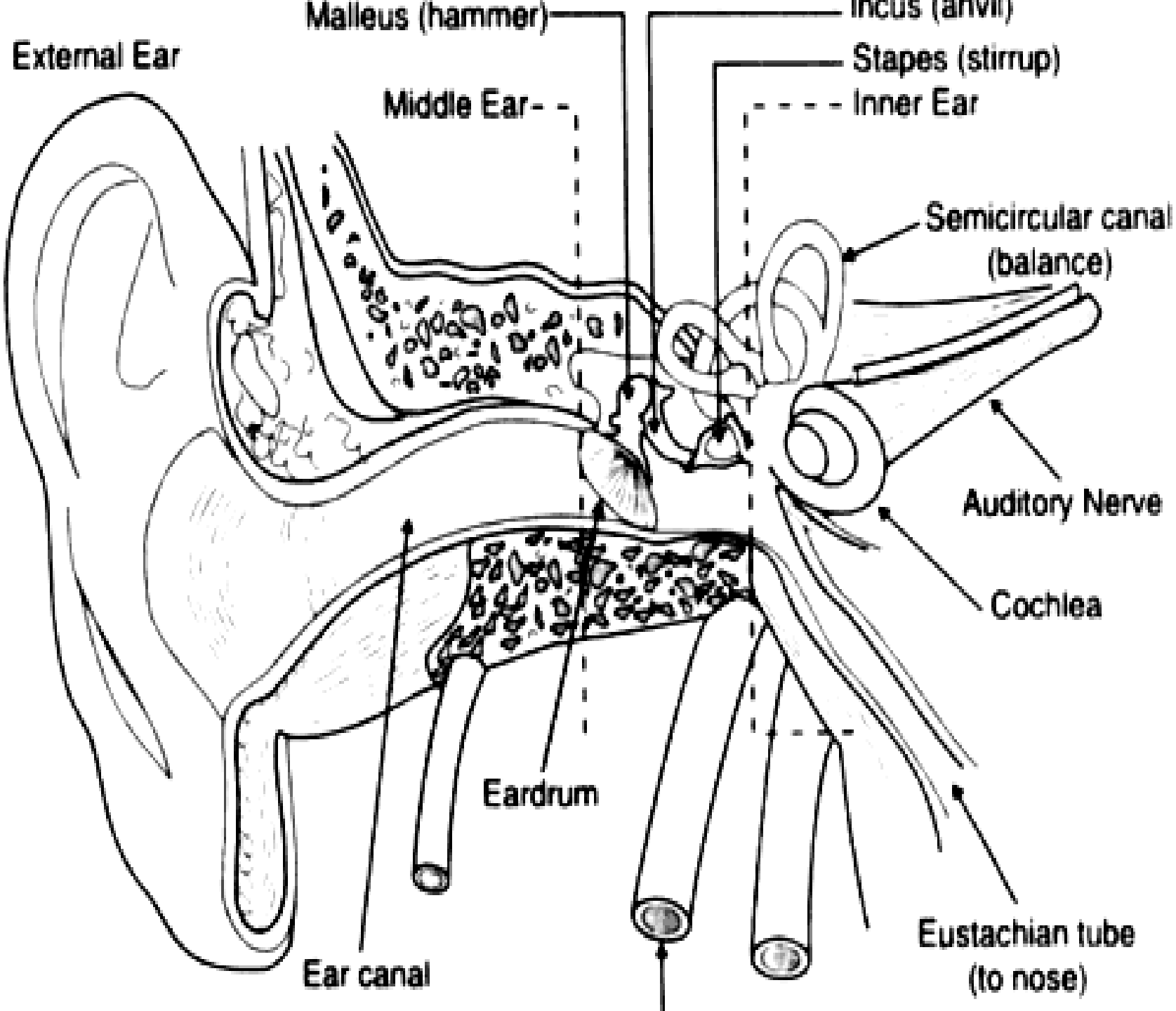
- UAO is an emergency
- Airway support in cases of severe partial or total collapse
  - Endotracheal intubation
  - Tracheostomy
- Manage cause or refer promptly.

FOREIGN BODIES IN THE ENT SYSTEM
7TH/2/2019

BY: DR. AYUGI

1. EAR

- Common in pediatric age group 2 – 4 yr.
- Rare in adults (cockroaches)
- M : F =1 : 1
- Types
  - Animate: insects, lice
  - Inanimate: vegetable (seeds) → they swell up when syringed)
  - Non vegetable (metal, plastic, stones, pebbles)
- Can present in hours, days or months.


Malleus (hammer)
Incus (anvil)
External Ear
Stapes (stirrup)
Middle Ear
Inner Ear
Semicircular canal
(balance)
Auditory Nerve
Cochlea
Eardrum
Ear canal
Eustachian tube
(to nose)

Site

- Canal, middle ear
- Right ear > left ear
- Bilateral is rare
- Method of removal depends on the nature, size, consistency site
- Needs good lighting

Cont.

- Syringing
  - Should be small
  - Should not be a vegetable: they swell when syringed
  - Should not impacted i.e. loose in the meatus
- A Hook for
  - Hygroscopic objects
  - FB not occluding whole meatus
- Bypass FB, turn hook, pull out.

Cont.

- Forceps: grasps
  - good for vegetable material, pebbles
  - Do not use for round substances
- Surgery: FB lodged in middle ear and beyond
- If possible no anesthesia unless
  - Uncooperative patient,
  - FB is impacted,
  - FB is in middle ear,
  - FB difficult to visualize
- Take child into theater, under electron microscope with GA.

Complications

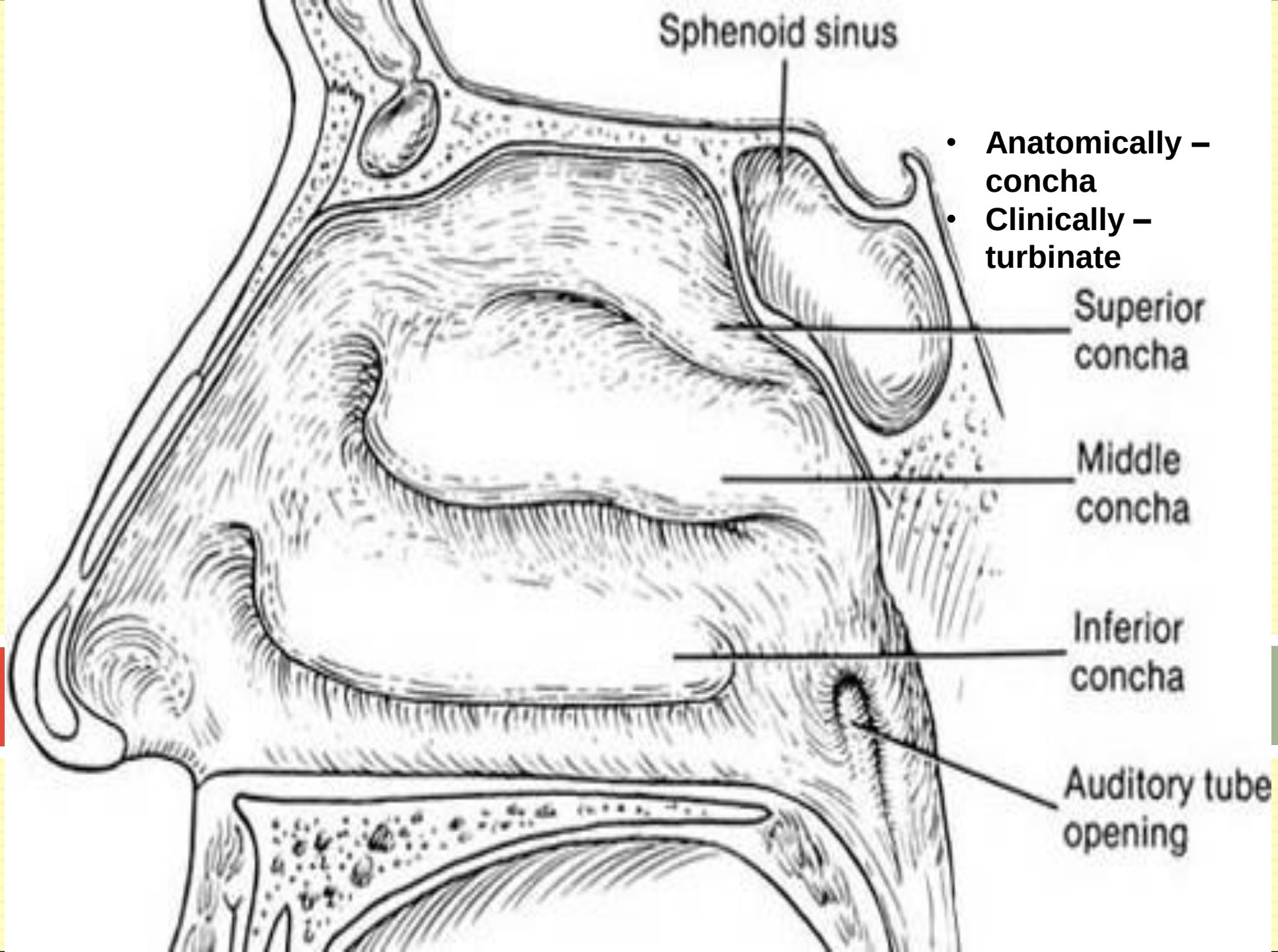
- Tympanic membrane perforation because of unskilled attempt at removal
- Conductive hearing loss (FB in canal, ossicular chain disruption)
- Trauma to canal
- Otitis externa (FB reaction)
- Vertigo: avulsion of stapes (leakage of perilymph)

2. NOSE

- Commoner in children, mentally retarded (older children , adults less)
- Penetrating injuries
- Post op FB (swabs)

Modes of entry

- Anterior nares
- Post nares: vomiting
- Penetrating wounds
- Palatal perforation e.g. cleft palate



- **Anatomically – concha**
- **Clinically – turbinate**

NB

- Review the boundaries of the:
  - Nasopharynx
  - Oropharynx
  - Laryngopharynx (clinically hypopharynx)
- Review the anatomy of the cricopharyngeus (muscle fibers & nerve supply etc.)

Polyp vs. turbinate [*OSCE*]

- Polyp
  - White
  - Not sensitive to touch
- Turbinate
  - Red
  - Sensitive to touch

Sites

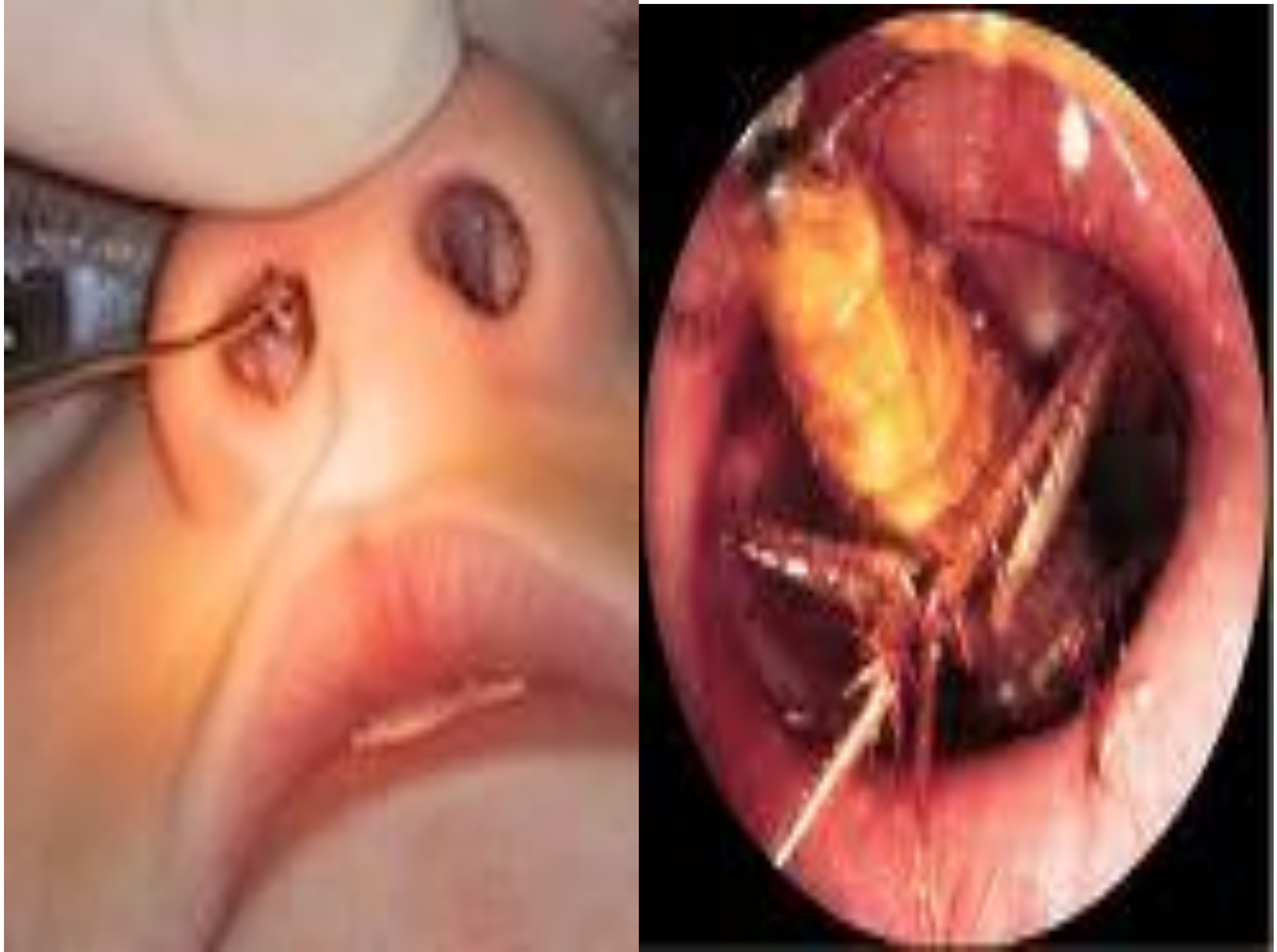
- Right > Left side
- Any part of nasal cavity ant, mid, deep
- **Types**
  1. Animate: roundworms, maggots
  2. Inanimate: vegetable material or non vegetable material (metals, toys, pebbles)

Signs and Symptoms

- Usually unilateral (animate usually bilateral)
- Vegetable FB: beans → unilateral fetid mucopurulent discharge, sometimes blood stained.
- Unilateral nasal discharge & nasal obstruction

Cont.

- Pain, epistaxis & sneezing
- Animate (headache, foul smelling discharge, feels things moving in the nose, nasal obstruction)



On examination

- Hold the **tip of the nose** (***not the forehead of the patient***) up & use a torch to visualize inside the nose.
- Congested red mucosa
- Mucus, pus
- Ulceration
- Granulation
- May see FB itself

How to remove

- Animate: 25% chloroform or paraffin drops
  - Tell patient to blow nose and remove with forceps
- Inanimate: hook (round objects) → pass hook on the floor of the nose, past FB it & pull

Cont.

- Usually under LA/GA (inhaled) if:
  - Uncooperative patient, apprehensive
  - FB firmly embedded in tissues
  - Posterior FB (difficult) can slip into hypopharynx & inhaled
  - If FB cannot be seen
- In theater use a **rigid endoscope** to look for the FB.

Rhinolith

- A FB that is missed in the nose forms into a stone in situ.
- This is rare.
- Removal requires GA to break up & remove piecemeal.

2. ESOPHAGUS/ HYPOPHARYNX

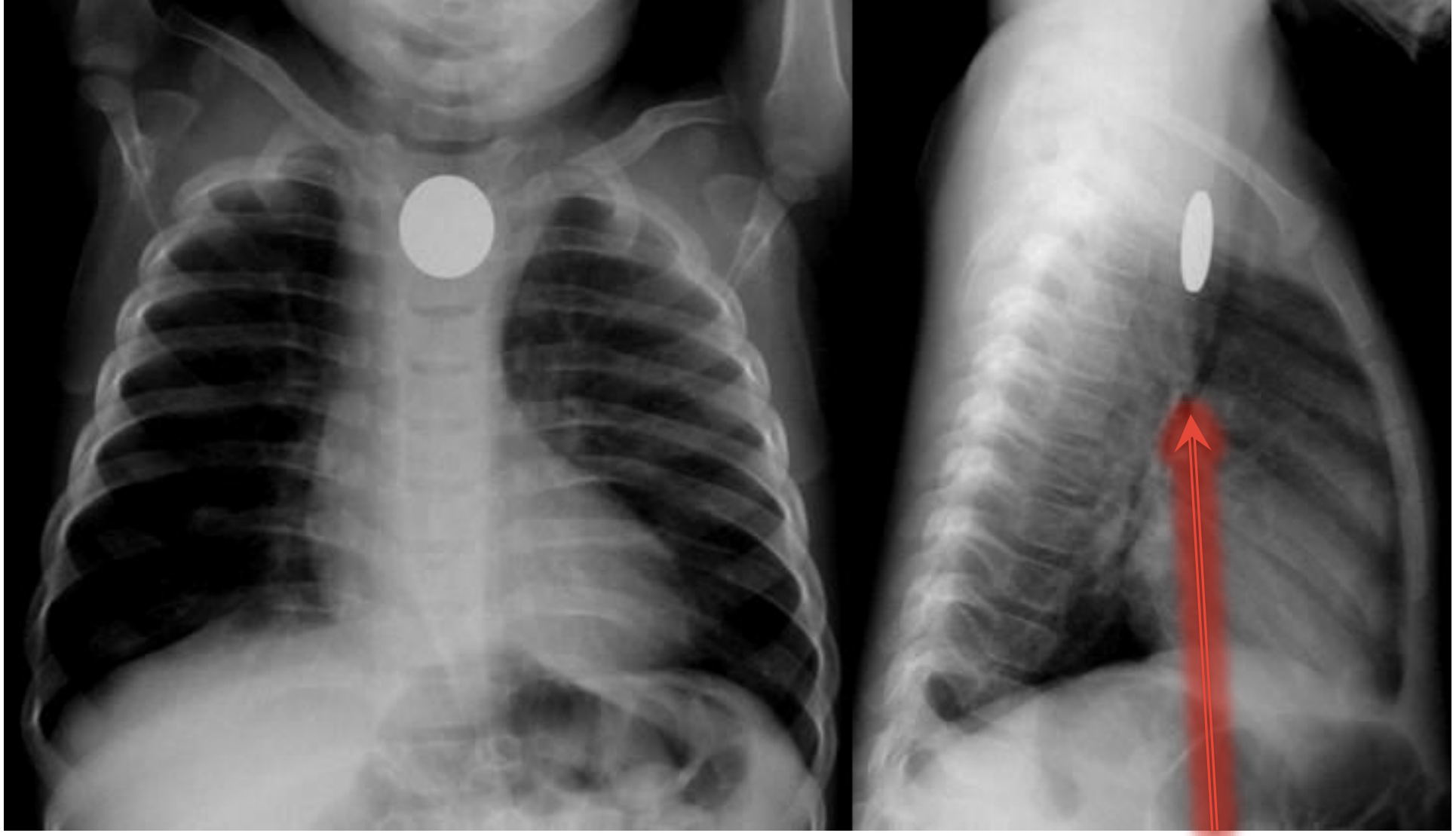
- Areas of constriction:
  1. Post – cricoid area (cricopharyngeus): 15cm from incisors → MC site where FB lodge.
  2. Aortic arch: 25cm from incisors
  3. Left main bronchus: 27cm from incisors
  4. Diaphragmatic hiatus: 40cm from incisors
- Most common site is the **post – cricoid region.**
  - Note that all the cartilage rings of the trachea are C – shaped but the cricoid cartilage is a complete ring.
- Pyriform sinus: fish bones

Pathological constrictions:

1. Peptic esophagitis
2. Corrosive stenosis: e.g. when the flat alkaline batteries are ingested. **These have to be removed in < 12 hrs** so always take a good history promptly to avoid complications due to delayed interventions.
3. Congenital stenosis

Clinical presentation

- History of ingestion of FB
- Choking & coughing in a child
- Dysphagia & Excessive salivation: differentiate between a FB in the esophagus and one in the bronchus.
- Hematemesis may occur in some cases



- ☐ Plain chest radiograph (AP & Lateral views)
- ☐ Biodata & date
- ☐ Radio – opaque FB
- ☐ Location: esophagus (likely at the cricopharyngeus)

Trachea is visualized anterior to the FB

Manangement

- X – ray
  - CXR
  - Barium swallow
- CT scan
- Endoscopy (diagnostic & therapeutic)
  - Rigid Esophagoscopy & removal
- If it is deeper observe & see whether it will pass.

Complications

Types of FB's: Fish bones, coins, meat, dentures.

- Ulcerations
- Stricture formation
- Tracheoesophageal fistula (TEF)
- Erosion through wall of esophagus to major blood vessel
- Mediastinal abscess
- Perforation of esophagus (air in mediastinum)

3. LARYNX, TRACHEA, BRONCHUS

- Commoner in 2 – 3yrs because they like putting things in the mouth.
- Rare in adults
- Forced inspiration during chewing, eating (e.g. crying with an FB)

Types

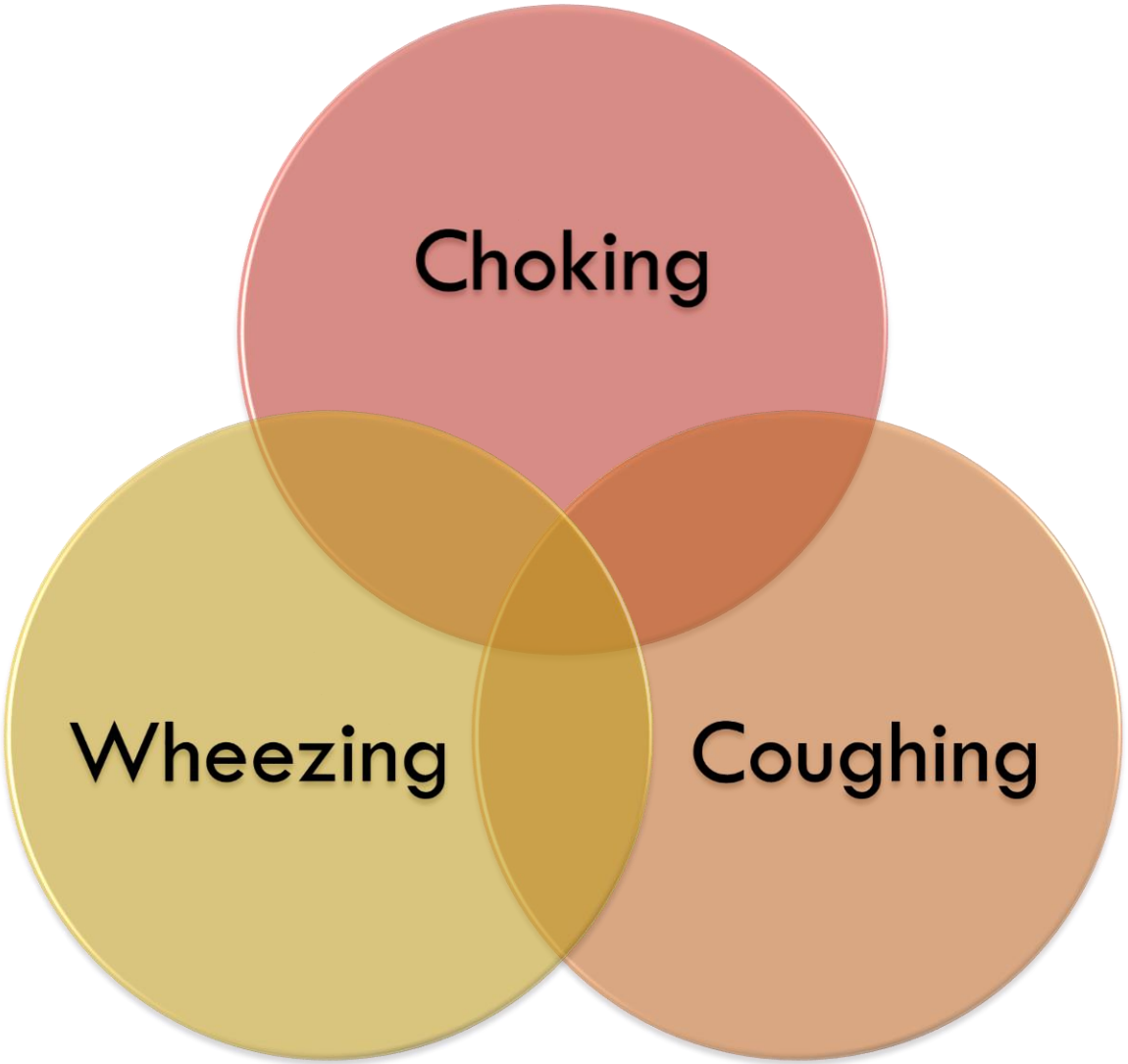
- Vegetable material (mostly), groundnut, maize, beans, bone, popcorn, fruit seeds.
- Non – vegetable: metals, pins, beads, stones, charcoal.

History

- Sudden **choking** followed by paroxysmal (on and off) **cough** & **wheezing.**
- In hospital: unexplained persistent fever.
- ARDS
- Triad: choke, cough, wheeze, (history of FB) → likely FB in the airway
- Can present in days, hours or weeks.
- 84% in laryngotracheal tree present early (hrs. – days)

FB in the airway:

A history of FB with a triad of →



On examination

- Respiratory distress
  - Cyanosis
  - Change of voice or cry
  - Stridor

Cont.

- Auscultation
  - Audible click as FB moves up and down
  - Unilateral expiratory wheeze.
  - Decreased air entry bilateral (trachea) or unilateral (bronchi)
  - Pneumonic changes: crackles & crepitations

Sites

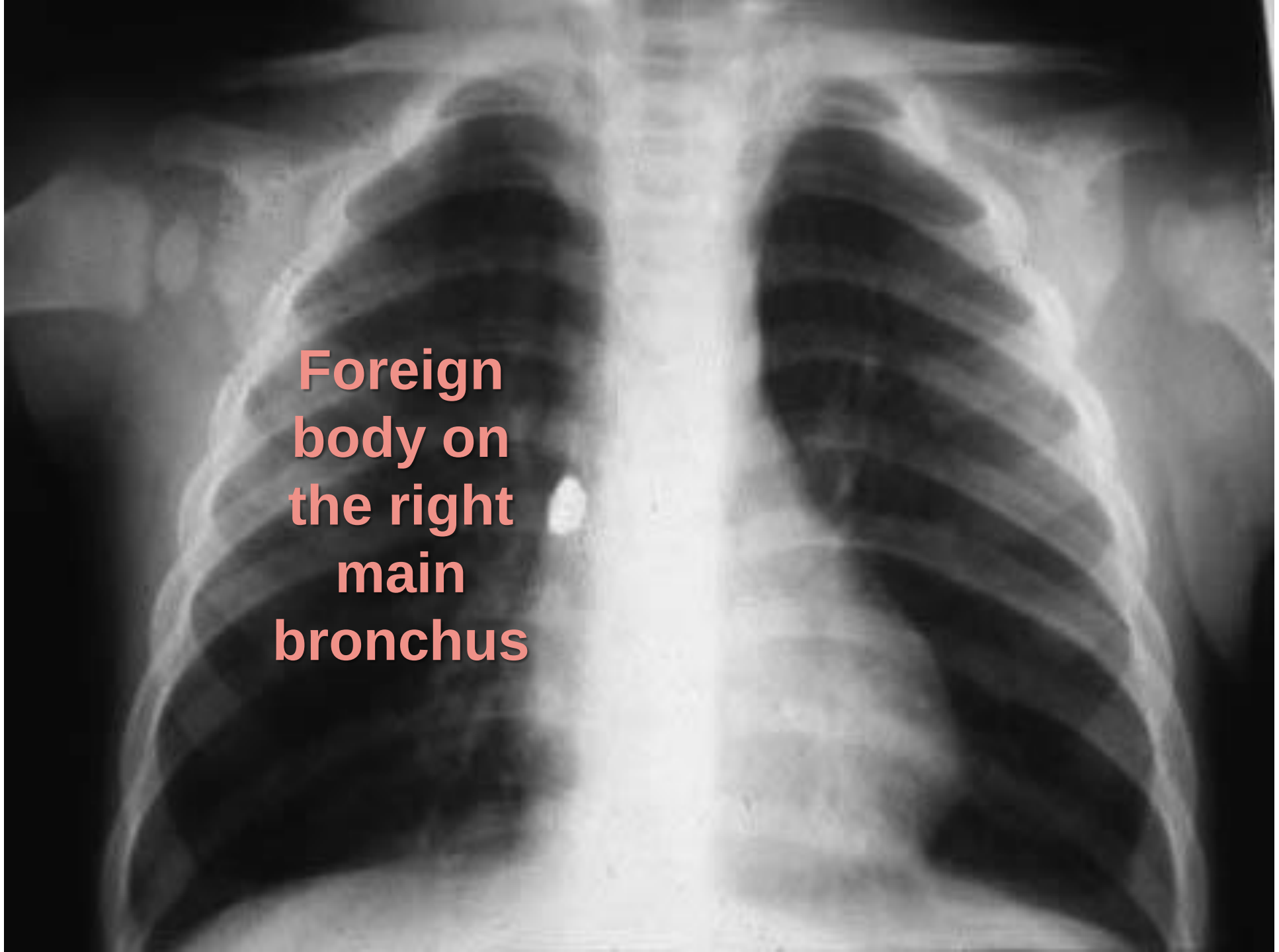
SITE	PERCENTAGE
Right main bronchus	51.6% (MC site)
Trachea	35.1%
Lt main bronchus	12.4%
Larynx	0.9%
Bilateral	0%

Cont.

- Why is the **Right Main Bronchus** the MCC site where a FB can lodge [*OSCE*]
  1. It is **wider**
  2. It is **more in line with the trachea**
  3. The **inter – bronchial septa (carina) is more to the left**
- Remember, the FB may change position e.g. coughed up from right bronchus to the left.

Management

- CXR (AP/LAT)
  - X – ray: may be normal when early or FB not radio opaque
  - Lung collapse
  - Mediastinal shift
- FB may be seen
- Obstructive emphysema: ball – valve mechanism
- Pneumonic changes.
- CT Scan


Foreign body on the right main bronchus

Treatment

- First aid: **Supplemental O_2** & **propping up** if the FB is not completely obstructing the upper airway. If it is, take to theater immediately and remove it.
- Rigid bronchoscopy
  - *Flexible bronchoscopy is mostly for diagnosis e.g. lung Ca etc.*

Cont.

- If in the larynx (remove via laryngoscopy)
- Cough out FB: conservative management before treatment.
- ***FB is an ENT emergency***: assess, treat or refer in a timely manner.

Complications

- Laryngeal edema
- URTI
- Death
- Pneumonia, lung abscess
- Bronchiocutaneous fistula
- If FB is impacted: an ENT surgeon can refer to a cardiothoracic surgeon to do a thoracotomy.

THE END

EDITED BY NAILA KAMADI

JESUS_TRANSFORMS_HEARTS

TRACHEOSTOMY

BY: DR. CHARLES MUREITHI

HISTORY

- 1st known reference- rig veda dated 2000 BC.
- Ebers papyrus (dated 1550 BC)- Egyptian medical papyrus mentions tracheotomy
- Alexander the Great
- Antyllus (2 AD), Greek surgeon- performed tracheostomies in oral surgeries
- Tracheostomy well documented in Indian and Arabian literature in middle ages.

- Tracheostomy gained popularity in the 1800s.
- Two methods:
  - High – by dividing cricoid
  - Low – trachea entered directly
- Significant problems associated with high Method.
- Till the end of 19th century tracheostomy was considered hazardous.
- Chevalier Jackson in 1923 established principles of tracheostomy.

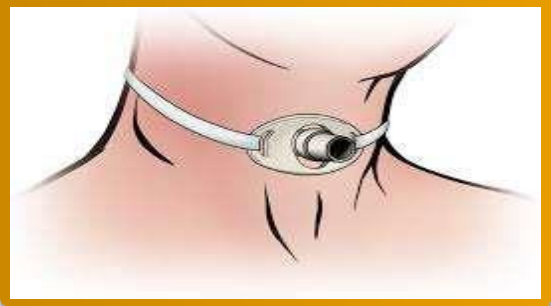
PHYSIOLOGICAL EFFECTS

Nasociliary clearance & humidification lost

Laryngeal bypass

Redundant area between stoma & larynx

Reduction in respiratory dead space



Disruption of normal swallowing mechanism

Indications of tracheostomy

Upper airway obstruction:

- **Congenital**: craniofacial anomalies, bilateral Choanal atresia, laryngeal webs/cysts, sub – glottic/ tracheal stenosis, tracheo – esophageal fistula.
- **Infective**: acute epiglottitis, Diphtheria, acute laryngotracheobronchitis, Ludwig's angina
- **Trauma**: external injury to larynx or trachea, maxillofacial injury, corrosive injury, inhalational injury
- **Neoplasm**: laryngeal, pharyngeal, tongue & upper tracheal tumors
- **Foreign body** lodged in larynx
- **Vocal cords**: bilateral abductor paralysis, bulbar palsy.

NOTE ABOUT UAO

- UAO is characterized by stridor:
  - Inspiratory: supra glottic obstruction
  - Biphasic: glottic obstruction
  - Expiratory: sub – glottic obstruction

Cont.

Removal of secretions & protection of tracheobronchial tree from aspiration in neurological disease:

- GBS, MS, bulbar palsy
- Coma: head injury, poisoning, tumor

- In such situations of laryngeal/ pharyngeal incompetence, a **cuffed tube is useful**.

Cont.

- **Respiratory failure**
  - Pulmonary diseases: exacerbation of chronic bronchitis, emphysema, pneumonia
  - Chest wall disease: Flail chest
  - Neurological diseases: MS, Motor neurone disease
- Tracheostomy reduces dead space & effort of breathing by:
  - Increasing alveolar ventilation
  - Ease of removal of secretions

Cont.

Prolonged ventilation

- T – tube is more secure than an ETT & is easier to wean off the vent.
- After > 3 weeks intubation, tracheostomy placement is associated with reduced length of ventilation & hospital stay.
  - Early tracheostomy placement is advised if it is expected that the patient will be ventilated for more than 11 – 14 days.

Cont.

As part of other procedures

- Temporary tracheostomy in head & neck surgery.
- Tracheal lavage
- Total laryngectomy

TYPES

- **TEMPORARY/ PERMANENT:**

1. ***Temporary tracheostomy:*** elective or emergency
2. ***Permanent tracheostomy*:** as part of laryngectomy e.g. in laryngeal carcinoma.

- **HIGH/ MID/ LOW:**

1. ***High*: above isthmus via 1st tracheal ring.**
2. ***Mid*: through 2nd – 3rd tracheal ring; preferred**
3. ***Low*: below level of isthmus**

CONT.

- **METALLIC/ SILASTIC**
  - ***Metallic*: fitted in theater**
  - ***Silastic*: can be fitted in the ward.**

PREOPERATIVE ASSESSMENT

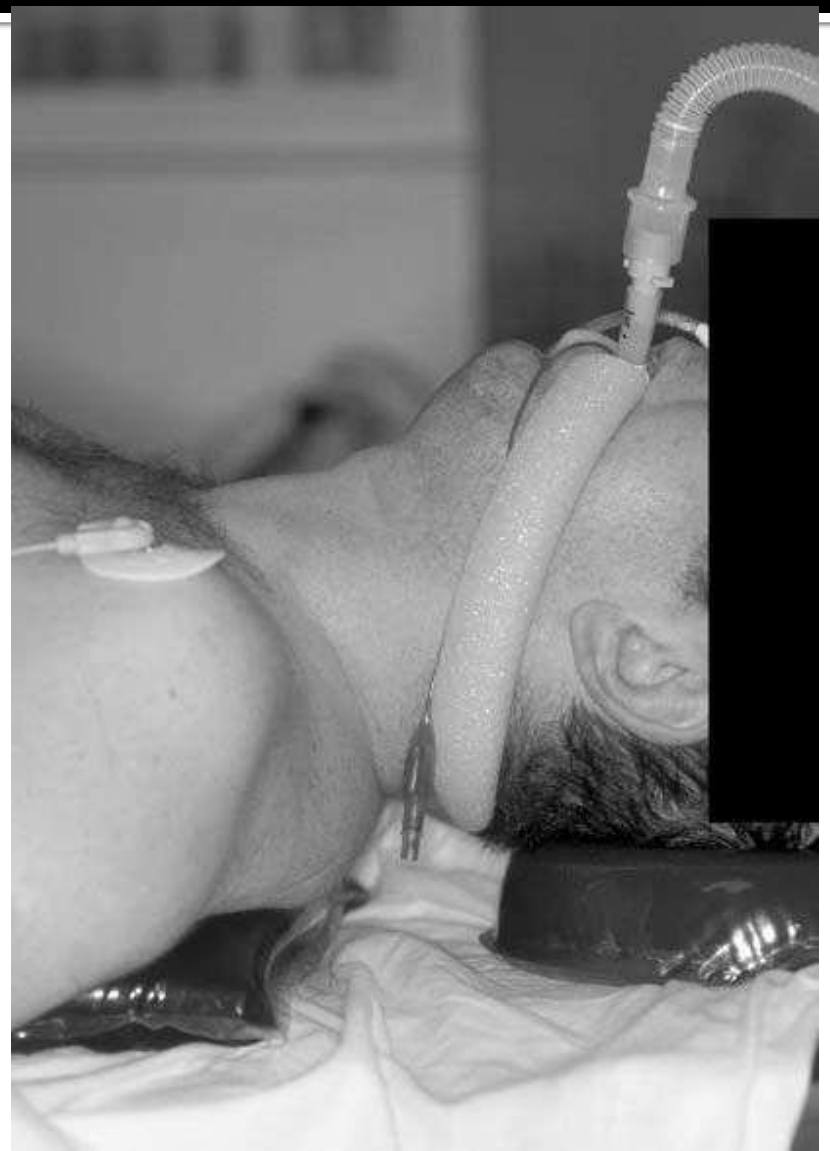
Informed consent

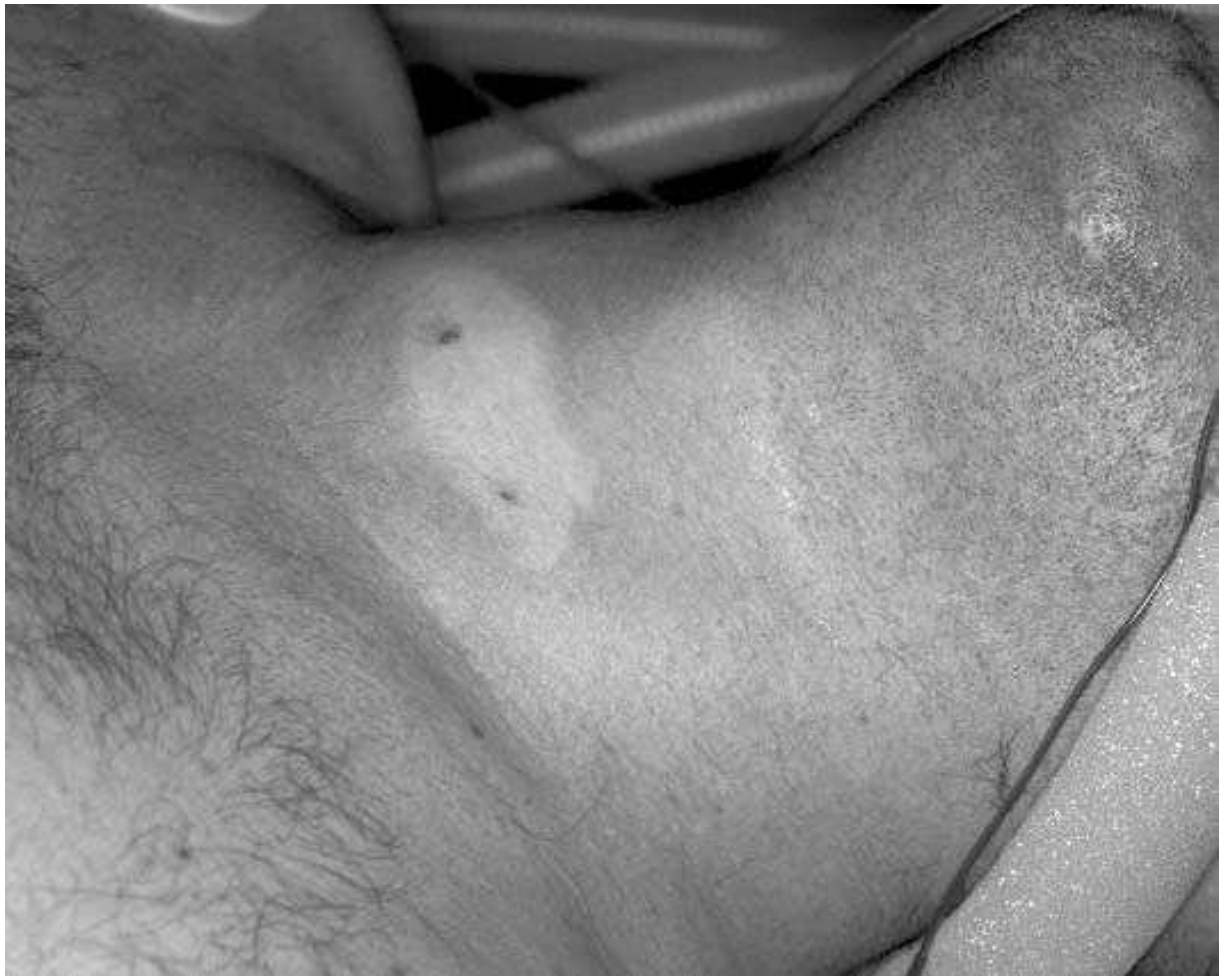
- **Coagulation profile** adequate, **platelet count** >50000/cumm.
- **Neck examination** to anticipate difficulties in procedure as in enlarged thyroid, limited neck extension.
- T – tube arranged, checked & prepared

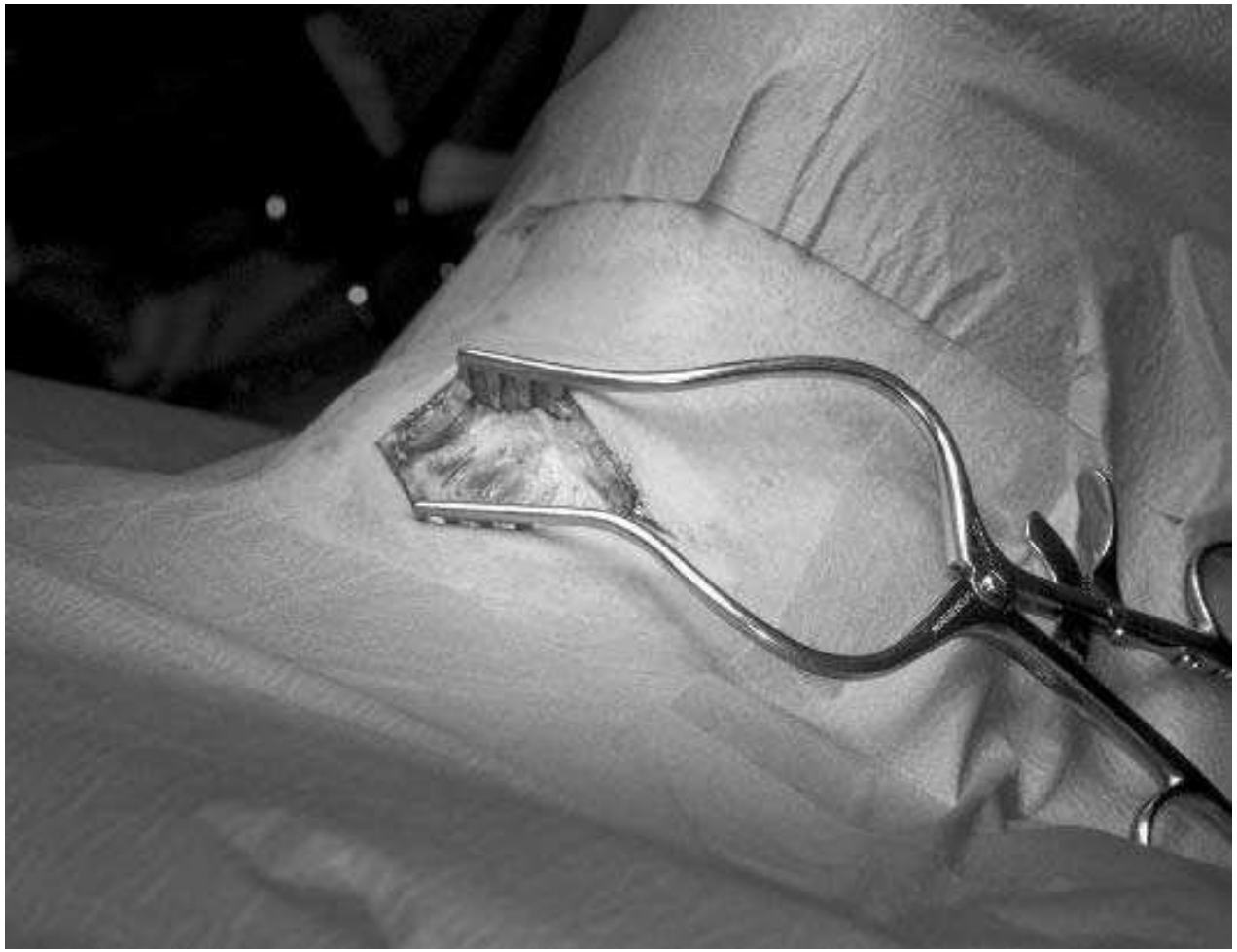
CONT.

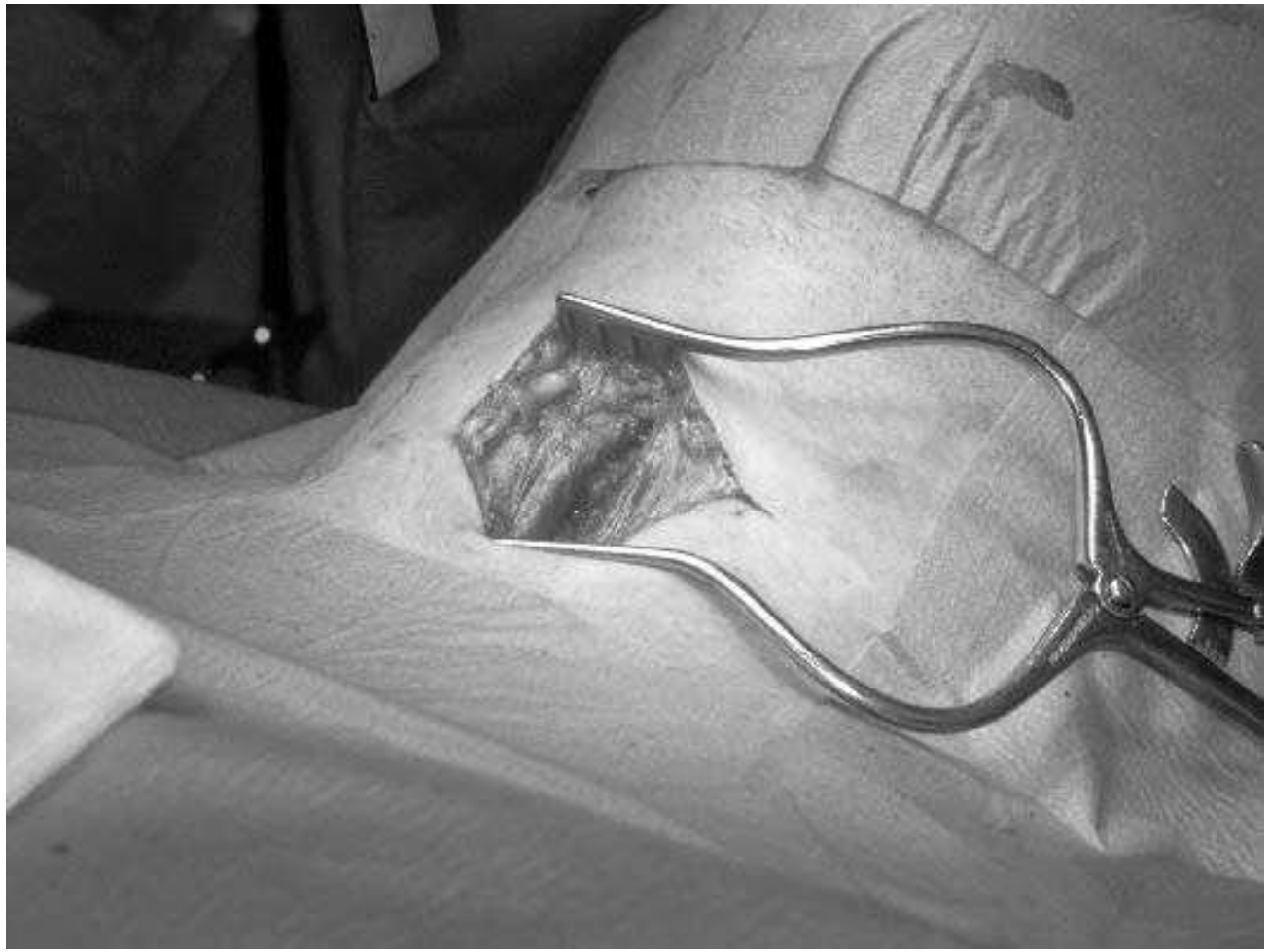
1. Surgical tracheostomy
2. Mini – tracheostomy
3. Pediatric tracheostomy
4. Percutaneous dilatational tracheostomy

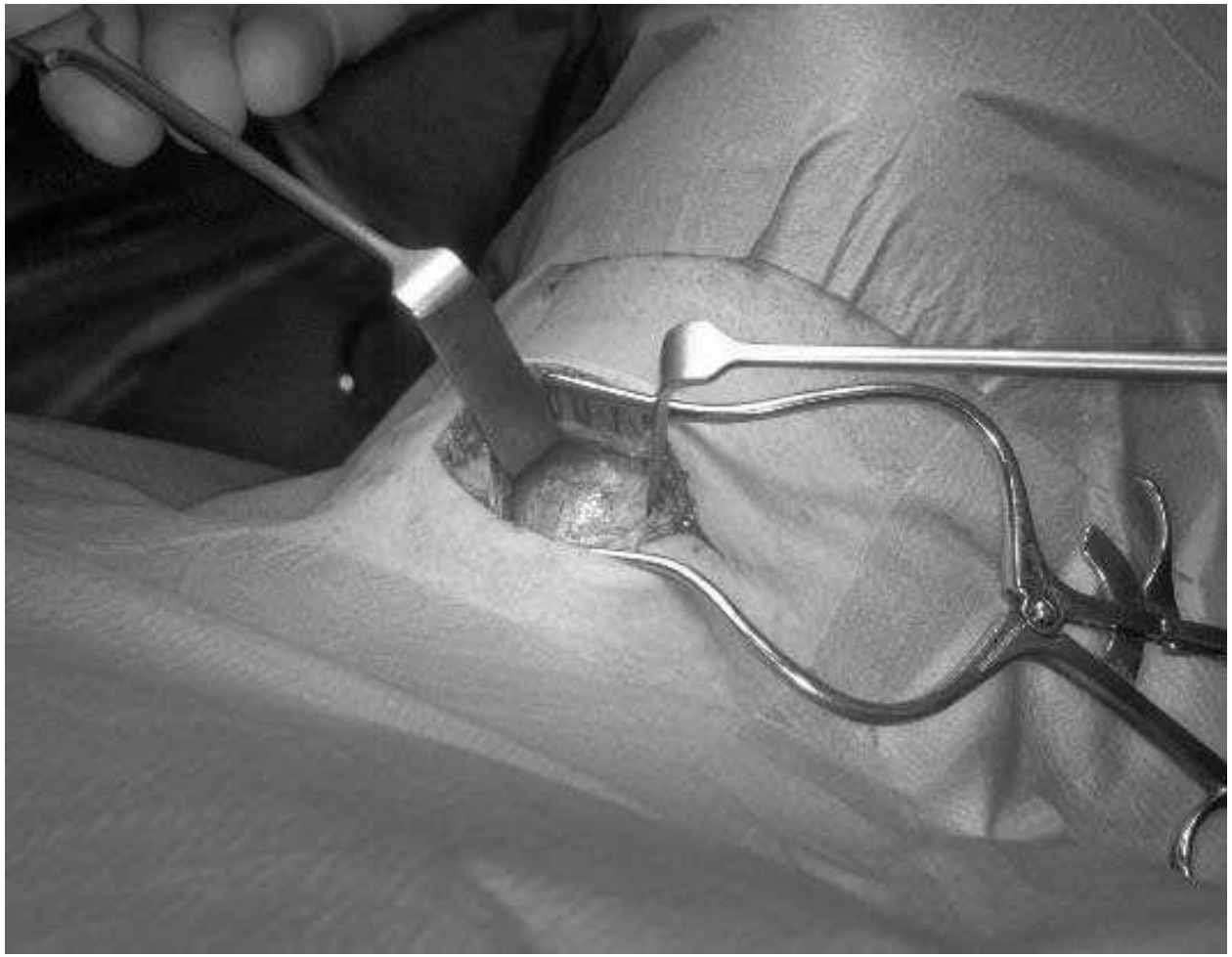
SURGICAL TRACHEOSTOMY

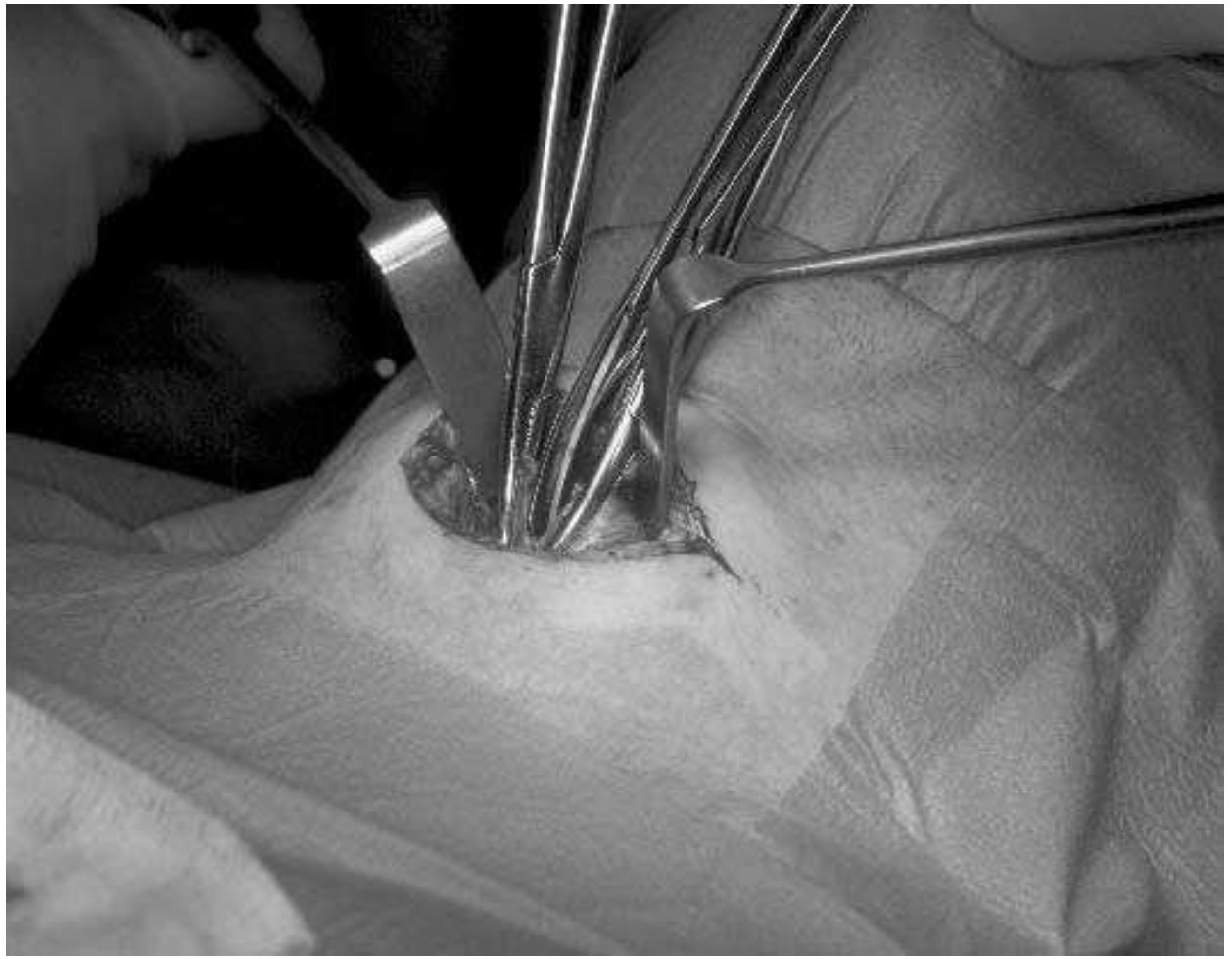


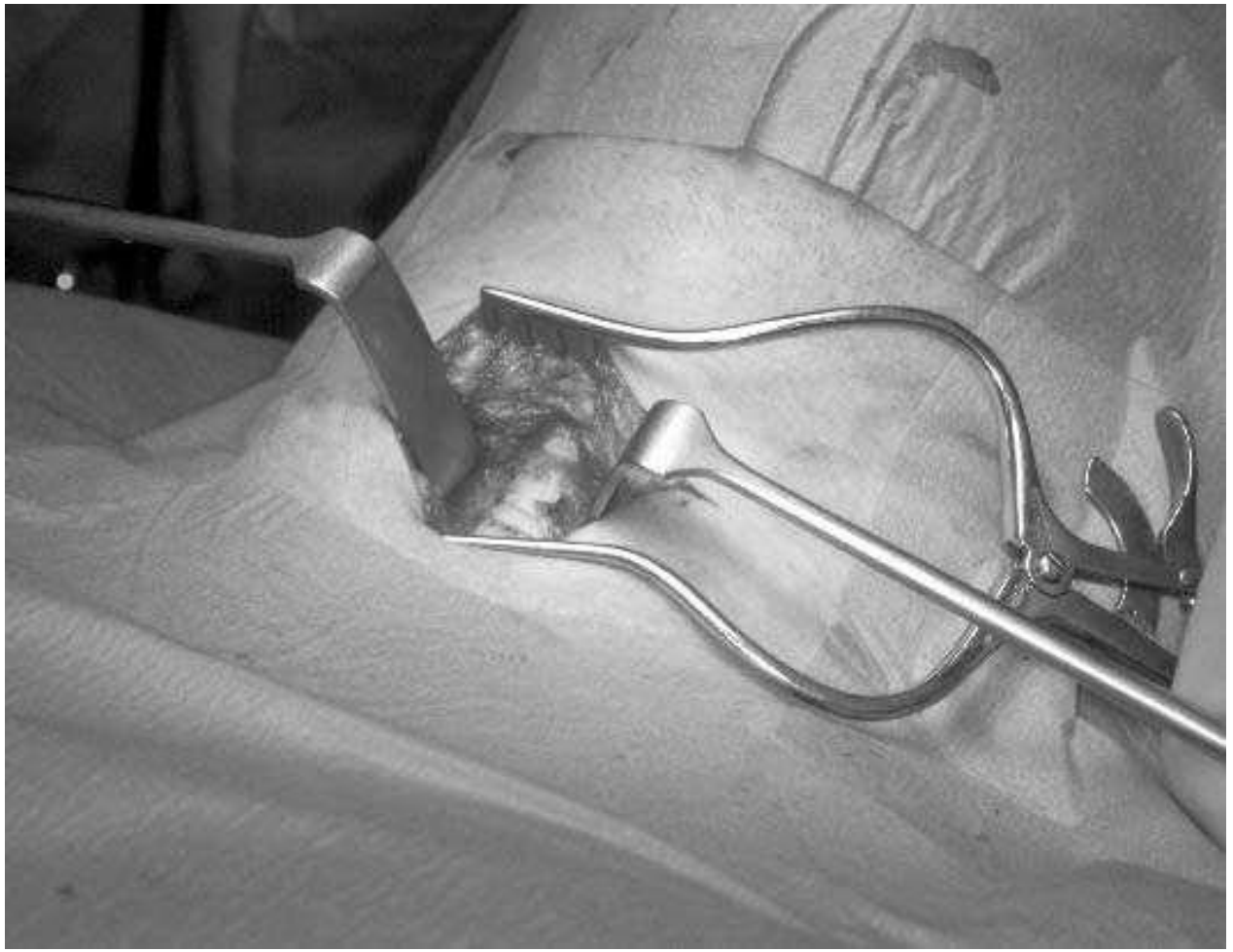


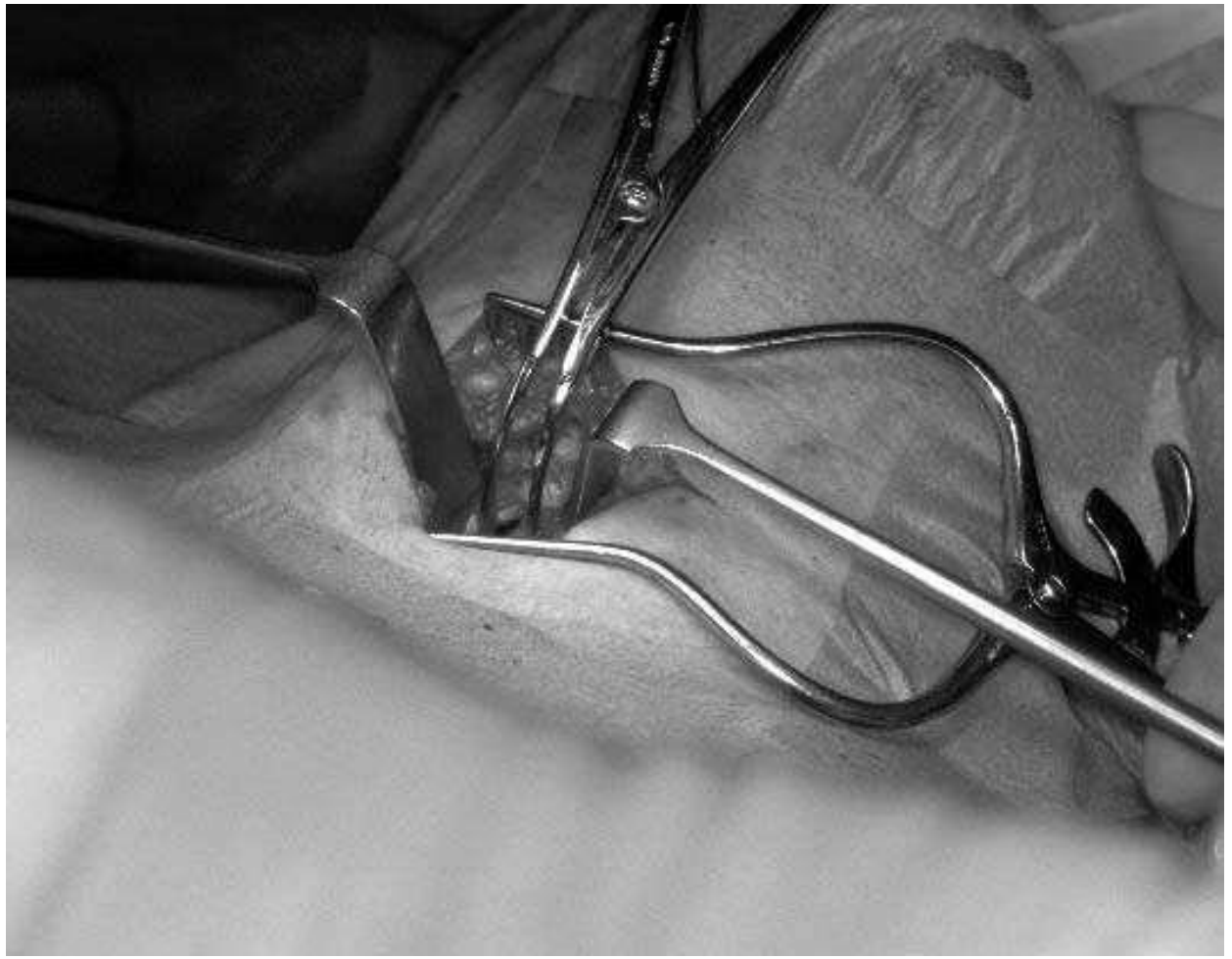


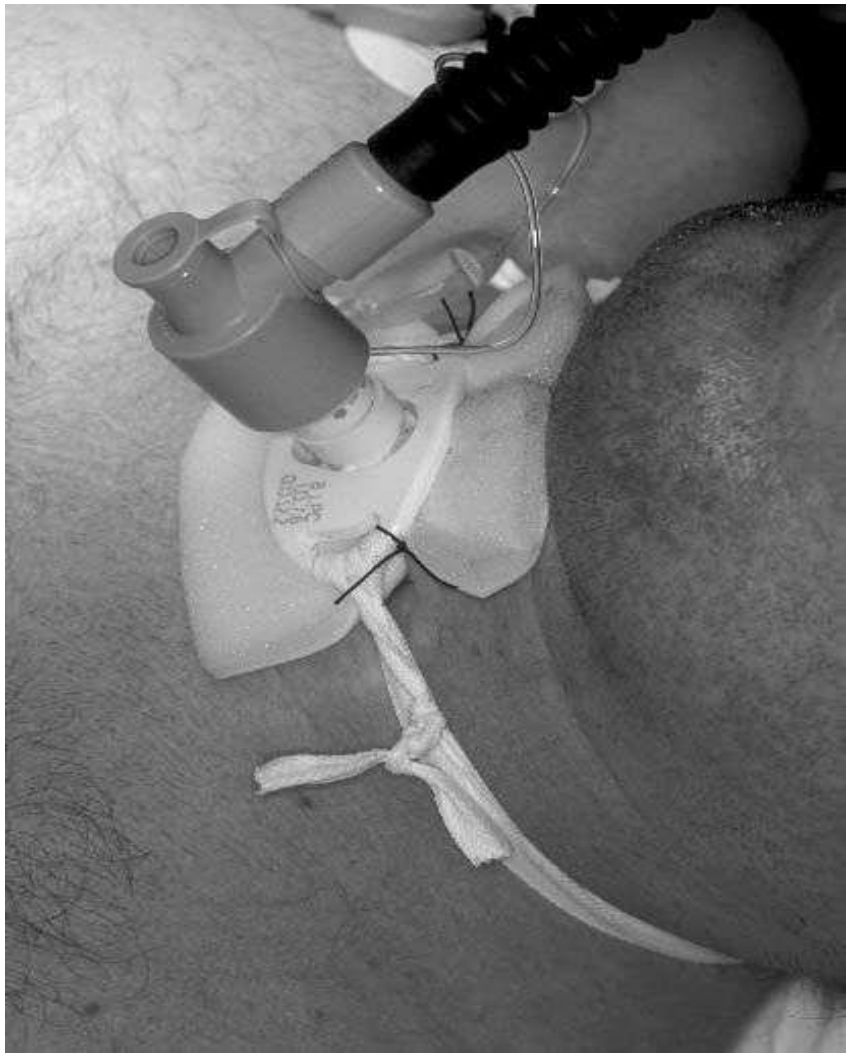












PROCEDURE

- After taking a history & examination, obtain an **informed consent**.
  - Let the patient know they won't be able to speak as the tracheostomy is below the vocal cords, they may experience a feeling of impending doom as the trachea is cut (cooperate), offer them an alternate form of communication e.g. paper & pen; let them know for how long the tube will be in place e.g. lifelong in laryngeal carcinoma; ensure they sign the consent.

CONT.

- Prepare patient for theater.
- Infiltrate jungle juice (lignocaine, epinephrine & water for injection)
- Position: hyper extend neck
- Incision:
  - 2 finger breadths above sternal notch (corresponds to tracheal rings 2 – 3)
  - Incision may be vertical (MC done in emergency) or horizontal (cosmetic)

CONT.

- LAYERS
  - Skin incision
  - Subcutaneous tissue: from this point do blunt dissection while remaining in the midline & palpating for the trachea.
  - Isthmus of thyroid: free fascia & retract upwards
  - Skeletonize trachea using a gauze
  - While palpating, aim for 2 - 4 cartilaginous rings.
    - < 2 cartilaginous rings → subglottic stenosis
    - > 4 cartilaginous rings → may enter the thoracic inlet; risk of injury to innominate vessels or pressure necrosis of the vessels → bleeding.
  - Make a button hole incision
    - ***Bjork flap: inferior flap is sutured to skin.***

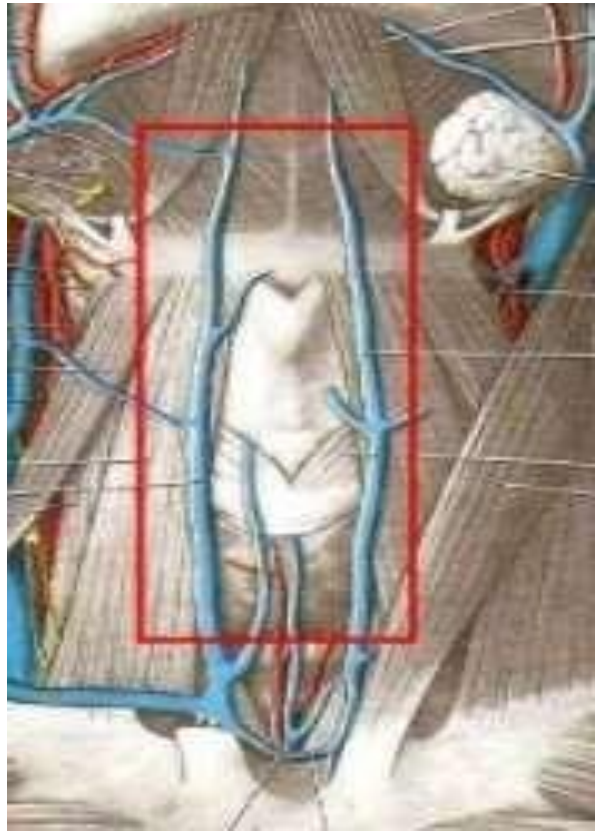
SIZING (INTERNAL DIAMETER)

- Adult males: 7.5 mm
- Adult females: 6.5 – 7.0 mm

COMPLICATIONS

Immediate

- Hemorrhage: local or hemothorax when innominate vessels are involved.
- Local injury to: cricoid cartilage, 1st tracheal ring, carotid artery, recurrent laryngeal nerve, thyroid cartilage
- Pneumomediastinum
- Air embolism
- Apnea
- Cardiac arrest
- Fires triggered with cauterization

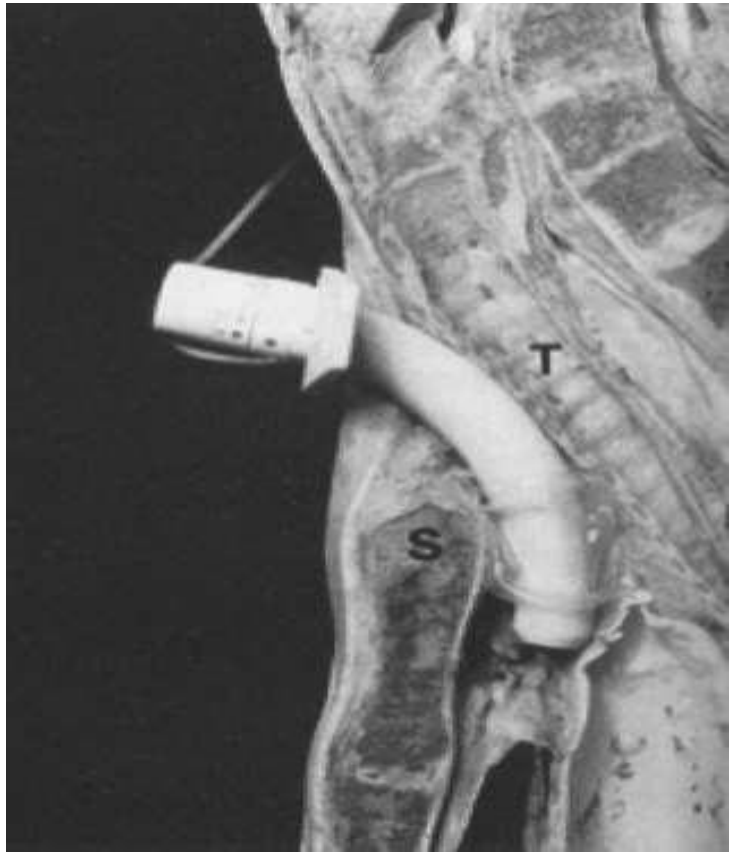


CONT.

- **Intermediate (1st few hrs. or days)**
  - **Secondary hemorrhage**
  - **Tube displacement**
  - **Tube blockage**
  - **Subcutaneous emphysema**
  - **Pneumothorax**
  - **Infection**
  - **Tracheal necrosis**

CONT.

- **Late**
- Hemorrhage
- Granuloma formation
- Tracheo – esophageal fistula
- Tracheo – cutaneous fistula
- Laryngotracheal stenosis
- Difficult decannulation
- Tracheostomy scar
- Dependence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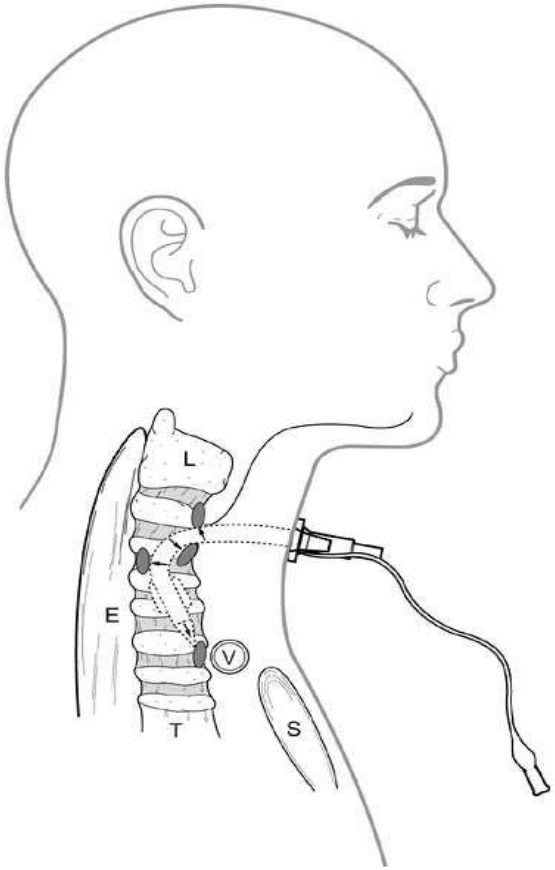
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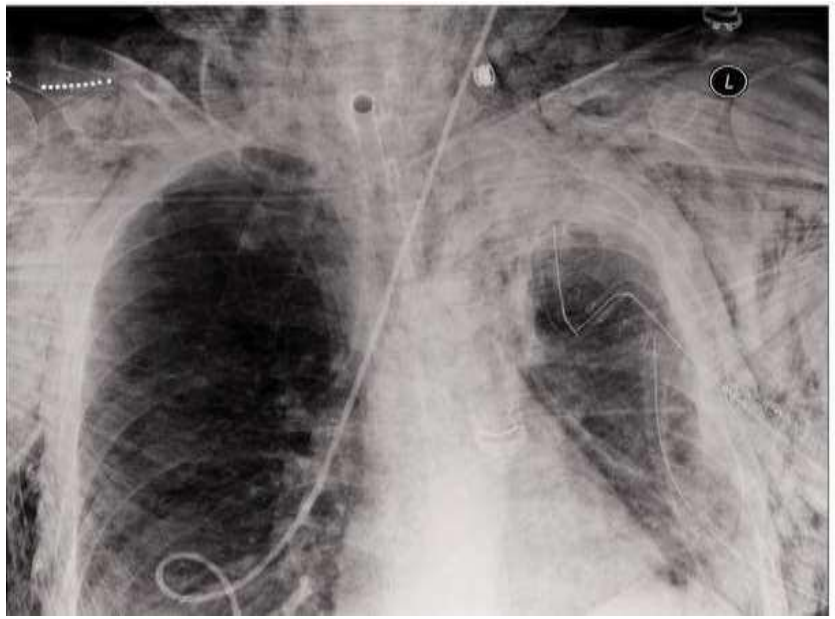

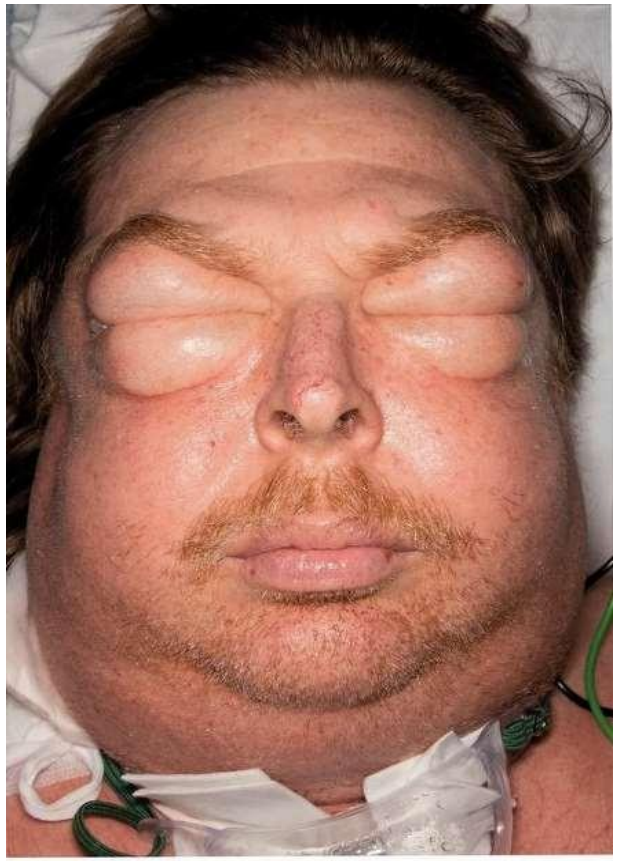

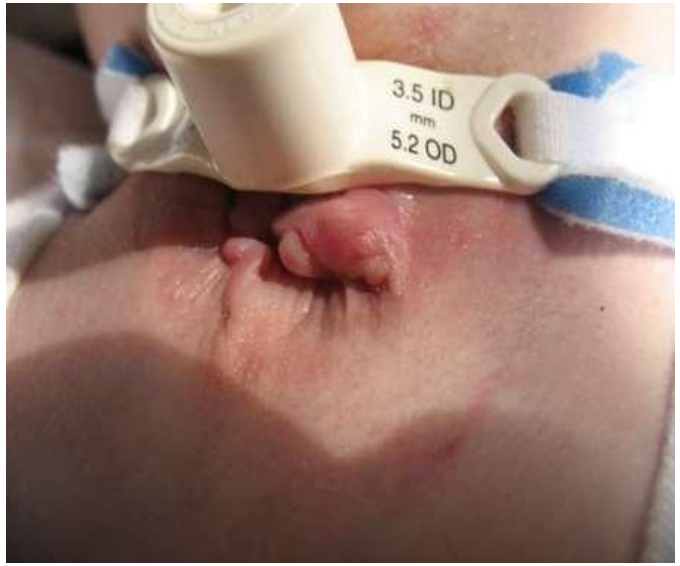
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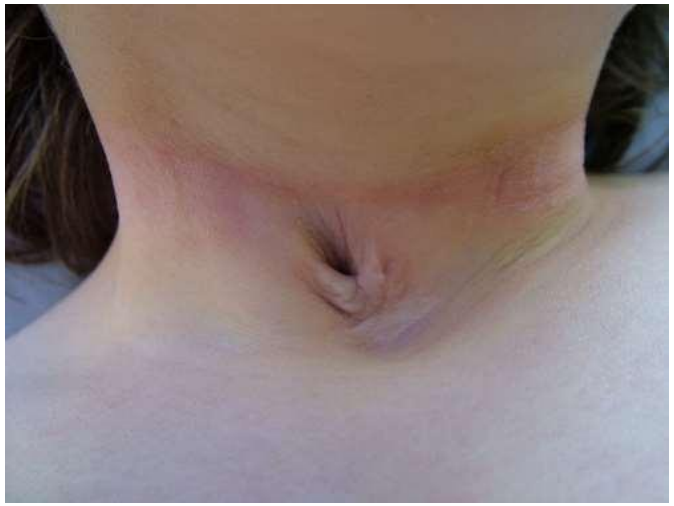
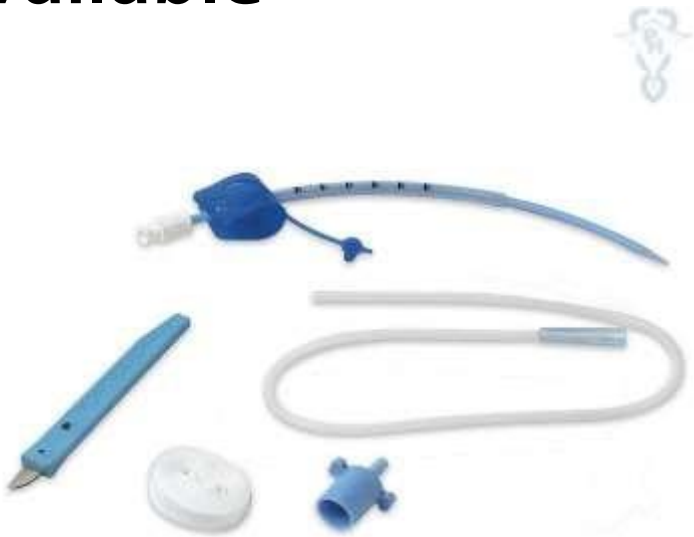


MINITRACHEOSTOMY OR CRICOTHYROTOMY

- Procedure for opening airway through cricothyroid membrane.
- Mini tracheostomy kits commercially available



PAEDIATRIC TRACHEOSTOMY

- Anatomy of pediatric upper airway is different from adults.
- Age of child is critical when deciding appropriate size of tube.
- Standard of pediatric intensive care facilities have improved in the last 2 decades.
- Reduced rate of tracheostomy in pediatric Population
- *Speech development may be impaired* in long term tracheostomies

INDICATIONS

➢ Upper airway obtruction

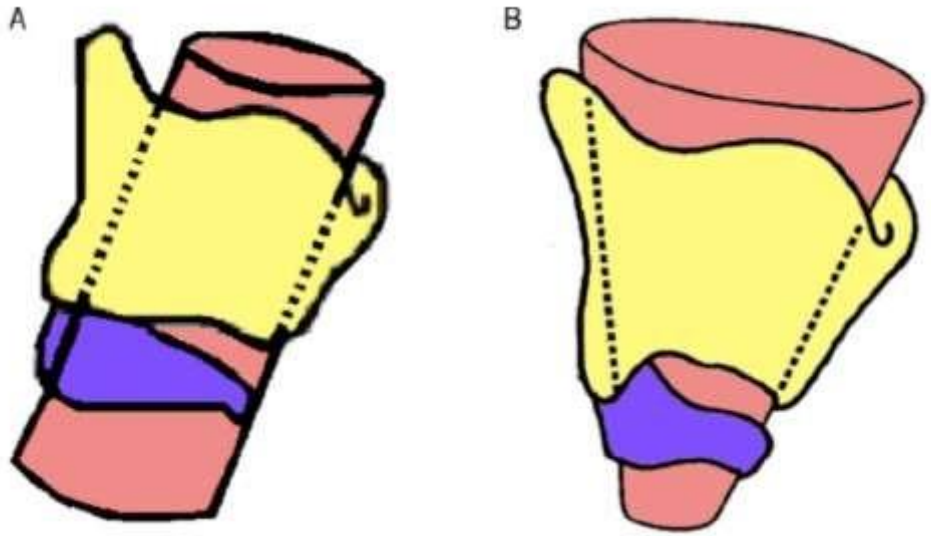
Oropharynx, Tongue base	Macroglossia, Treacher Collins syndrome, Goldenhar syndrome, Cystic hygroma, Diphtheria
Nose, Nasopharynx	B/L choanal atresia
Supraglottis	Supraglottic cyst, Acute Epiglottitis
Glottis	Vocal cord palsy, Laryngeal oedema, Physical trauma, Juvenile respiratoty papillomatosis
Subglottis	Subglottic stenosis, Hemangioma
Trachea	Acute laryngotracheobronchitis, Tracheomalacia, Tracheal stenosis

- **Prolonged (> 3 weeks) intubation**
  - Indicated for patients requiring long term PPV such as: preterm neonate, CNS disease, severe burns.
  - Long term intubation leads to complications and difficult decannulation

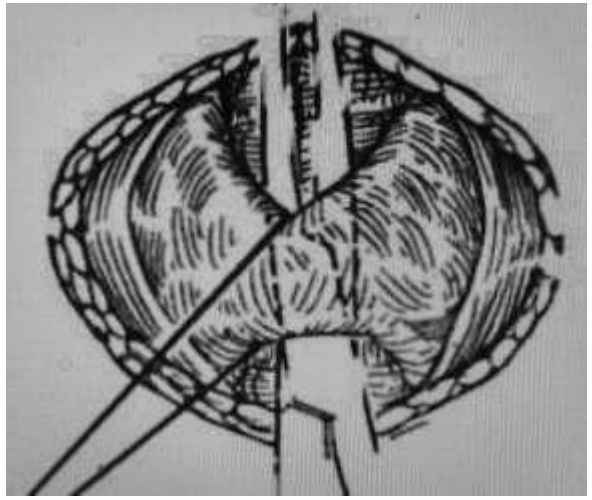
- **Pulmonary toilet**
  - For intractable aspiration: decreases dead space & eases work of pulmonary toilet

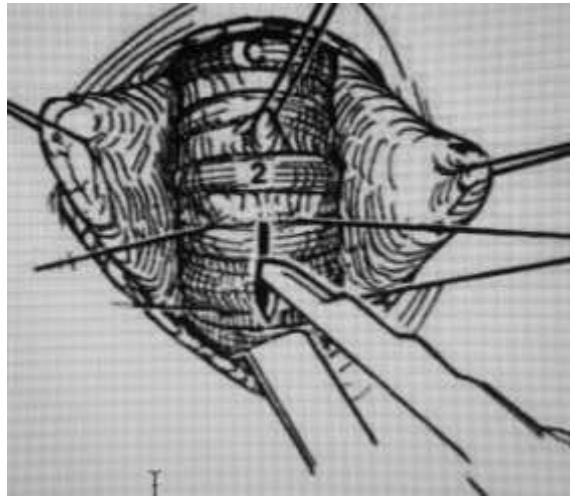
ANATOMICAL CONSIDERATIONS IN PAEDIATRIC TRACHEOSTOMY

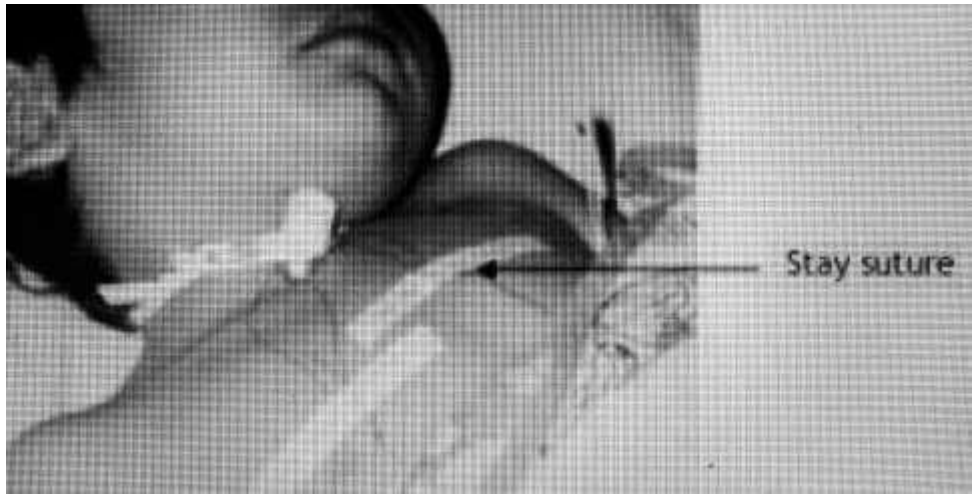
- Structures lie higher up
- Soft & compressible airway
- Structures from superior mediastinum pulled up during extension of neck.
- Small tracheal lumen
- Trachea is a developing structure.
- Funnel shaped larynx with *narrowest part being subglottis*



TECHNIQUE





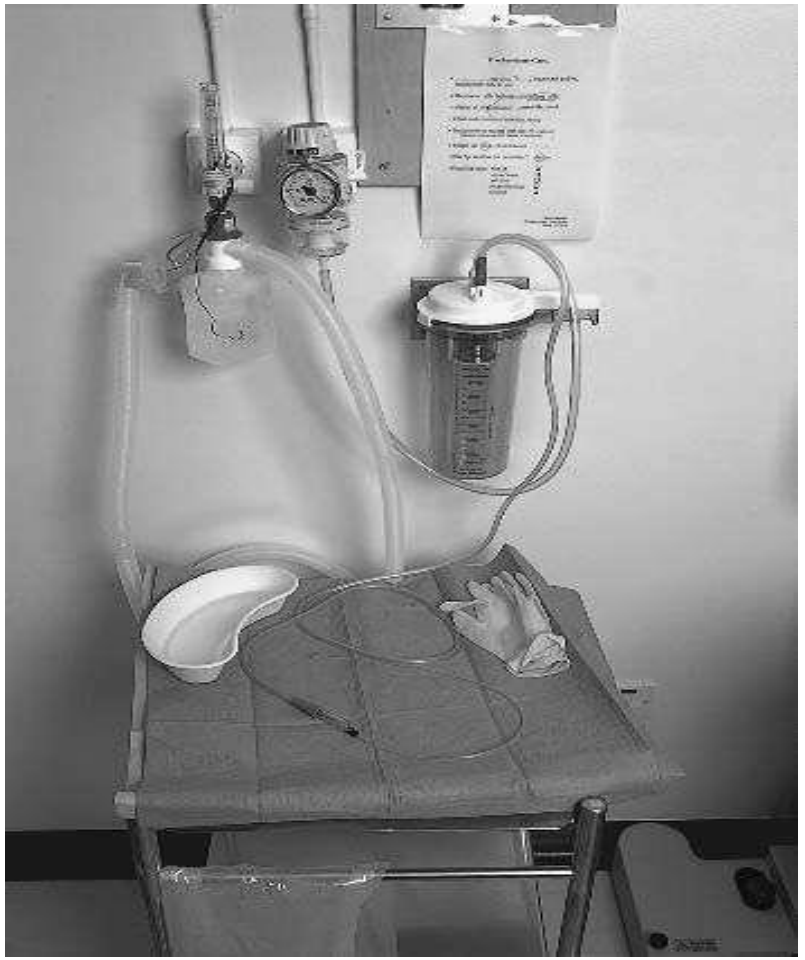


TRACHEOSOMY CARE

- Immediate post – operatively
  - **Deflate cuff** q2h for 15 minutes with monitoring
    - Cuff pressure should be around 25 cmH_2O (easily pliable pilot tube). At >30cmH2O → pressure necrosis → TEF, mediastinitis)
  - Change tube from cuffed to Uncuffed/ double barrel after 72 hrs. (tract has epithelialized). This change can be done earlier if a Bjork flap was in place.

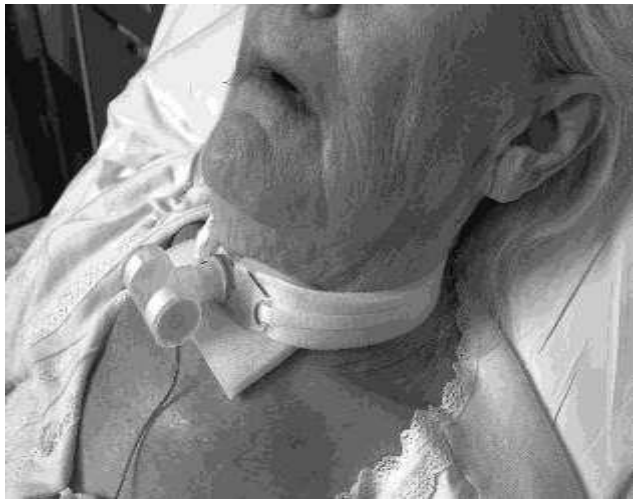
TRACHEOSTOMY CARE

- **Suction PRN**
- Regular suctioning
- Frequency depends on individual basis
- Indications
- Appropriate size of suction catheter & method.



➢ Humidification

- Upper respiratory tract bypassed, conditioning of inspired gas lost.
- Different preferences in different set ups
- Types: cold water humidifiers; hot water humidifiers; heat & moisture exchangers; stoma protector.
- A wet woolen cloth can be placed on neck over trachea.
- Nebulization

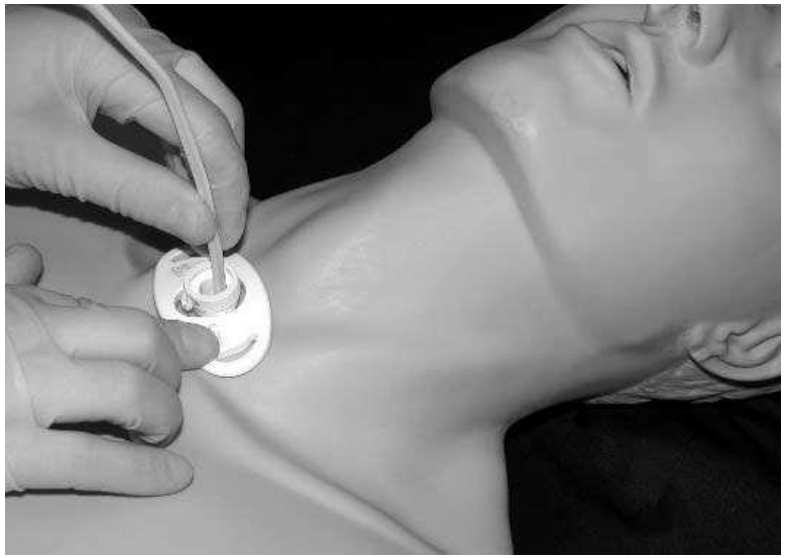


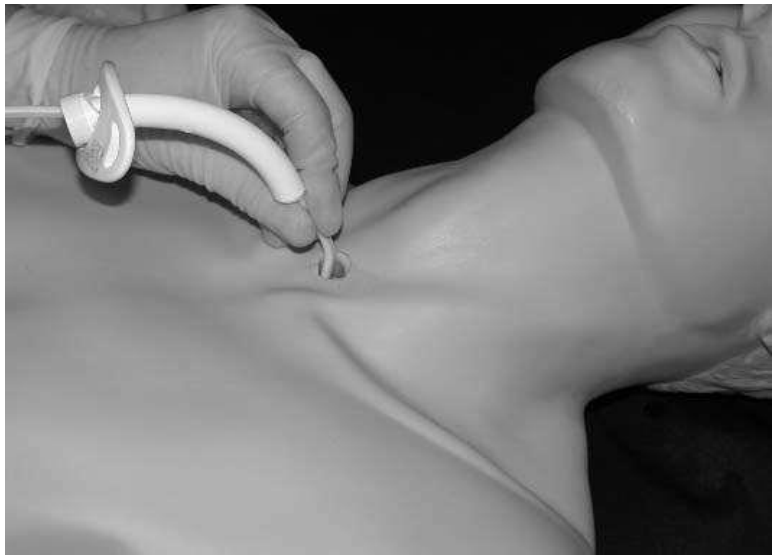
CONT

- **Mucolytics**: to thin secretions & make it easy to cough.
- Encourage coughing to remove secretions.

➢ Tracheostomy tube change

- 1st tube change: 5 – 7 days
- Frequency of tube change- no standard interval
- '*if you can hear (they can speak) a tube, you should change it*'
- Bougies or guidewires

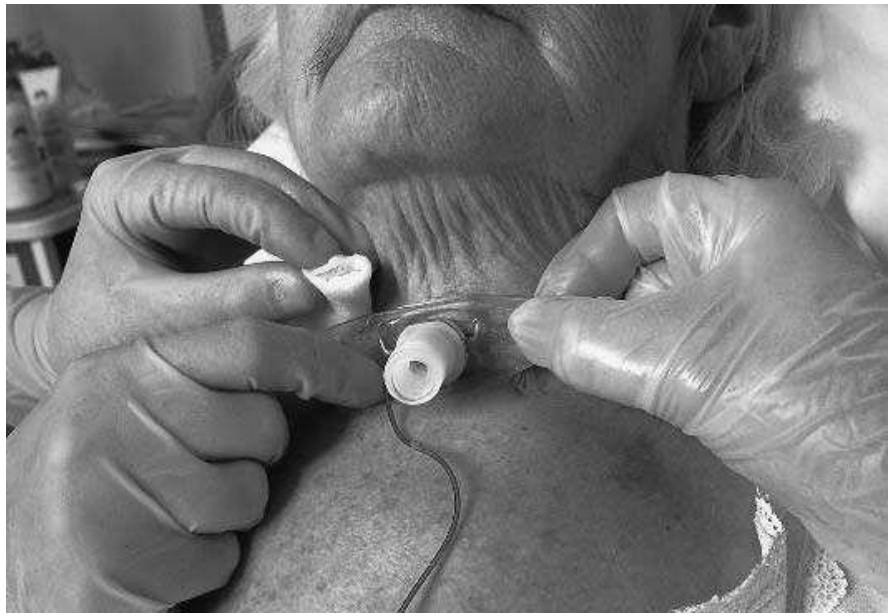


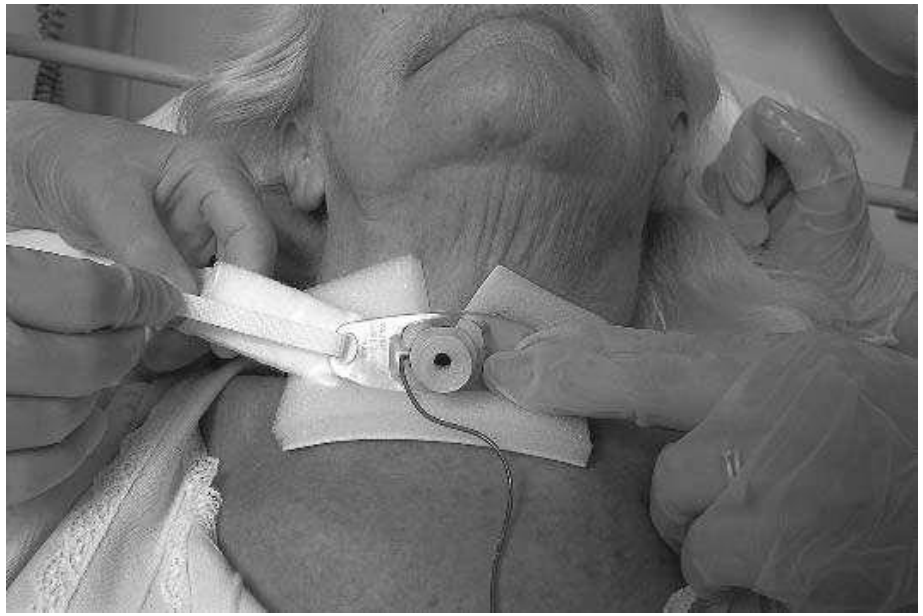


Decannulation

- Day 1: use fenestrated tracheostomy tube. Block for 12h (during the day) & see if the patient can breathe via their own trachea.
- Day 2: block the tracheostomy tube completely for 24 hrs. while monitoring.
- Day 3: remove the tube or release the Bjork flap (leave skin to contract)
- Alternative methods: downsizing

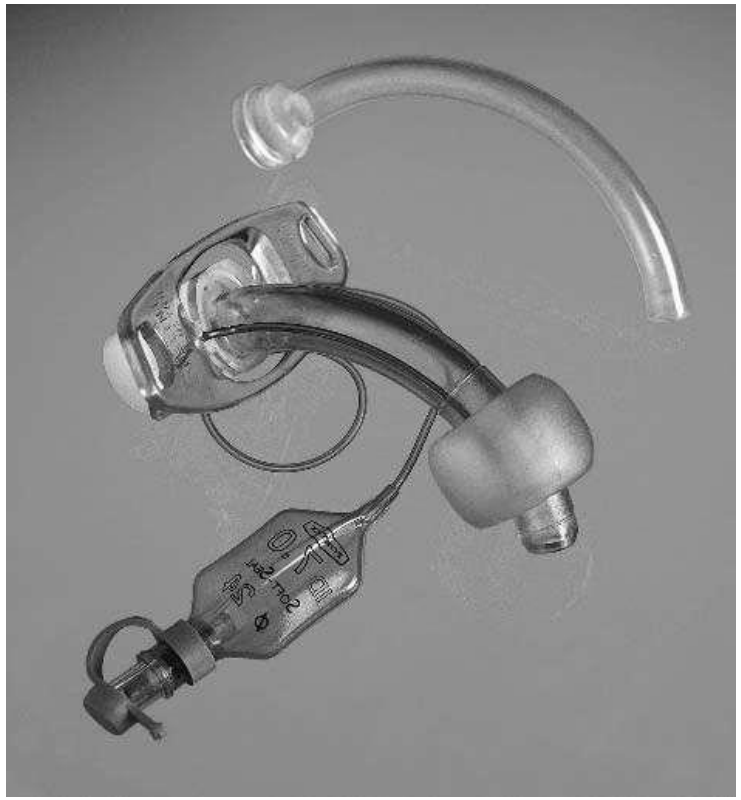
➢ Wound care





TYPES OF TRACHEOSTOMY TUBES

- **Cuffed or uncuffed**



- **Single or double lumen tubes**

- **Adjustable flange long tube; Suction aid tracheostomy tube**

- **Tracheostomy with speaking valve**

- **Types of tubes based on material:**
  - **PVC**
  - **Silicone**
  - **Siliconed PVC**
  - **Silastic**
  - **Silver**
  - **Armored**
  - **Fullers tube**

PERCUTANEOUS DILATATIONAL TRACHEOSTOMY

- 1st described by Shelden & Pudenz (1957)
- Tracheostomy: Indications & complications
- **Contraindications:**

- Absolute:
  - Cervical injury
  - Coagulopathy
  - Emergency airway
- Relative :
  - Sort fat neck/obesity
  - Enlarged thyroid
  - Inability to extend neck

(cervical injury/prior tracheostomy)

DECANNULATION

- Considered when original condition requiring tracheostomy has improved .
- Approached in a *step – wise manner*.
- In pediatric group endoscopic assessment prior to decannulation essential
- Fenestrated tube > occlusion cap > occlusion cap for 12 hrs > 24 hrs. > decannulation

EPISTAXIS
31/1/2019

BY: DR. J. AYUGI

Definition

- Epistaxis is defined as: acute hemorrhage from the **nostril, nasal cavity or nasopharynx**.

Epidemiology

- It is one of the MC **ENT emergencies**.
- Male to female ratio 1.6:1 (***slight male preponderance***).
- There is a higher incidence in ***older patients***.
- Clinically: the patient presents with bleeding from either the **lateral nasal wall** or from the **septum**.

Cont.

- 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 others can be life threatening e.g. in the elderly.

Cont.

- Minor epistaxis usually originates from the **anterior nasal septum**. It is often the result of **minor trauma** to the septal mucosa.
- In **children**, epistaxis is usually a result of **nose picking** whereas in **adults** it is usually as a result of **desiccation of the mucosa**.

Vascular Anatomy

- The nasal mucosa has a rich arborizing network of submucosal vessels.
- Arterial blood supply is from the **internal** & **external carotid arteries**.
- A confluence of the 2 systems occurs particularly at the caudal end of the septum. A number of arteries anastomose with each other in the **Little's area**.
  - Little's area is an anteroinferior part of the nasal septum where 4 arteries meet to form the Kiesselbach's plexus. This is a rich vascular anastomosis between the internal & external carotid artery systems.

The anterior septal/ Kiesselbach's plexus (Little's area) comprises a confluence of:

- From the ECA system: septal branches of →
  - Sphenopalatine artery.
  - Superior labial artery.
  - Greater palatine artery.
- From the ICA system: septal branches of →
  - Anterior ethmoidal artery.
- This is the **MC site of arterial epistaxis**.

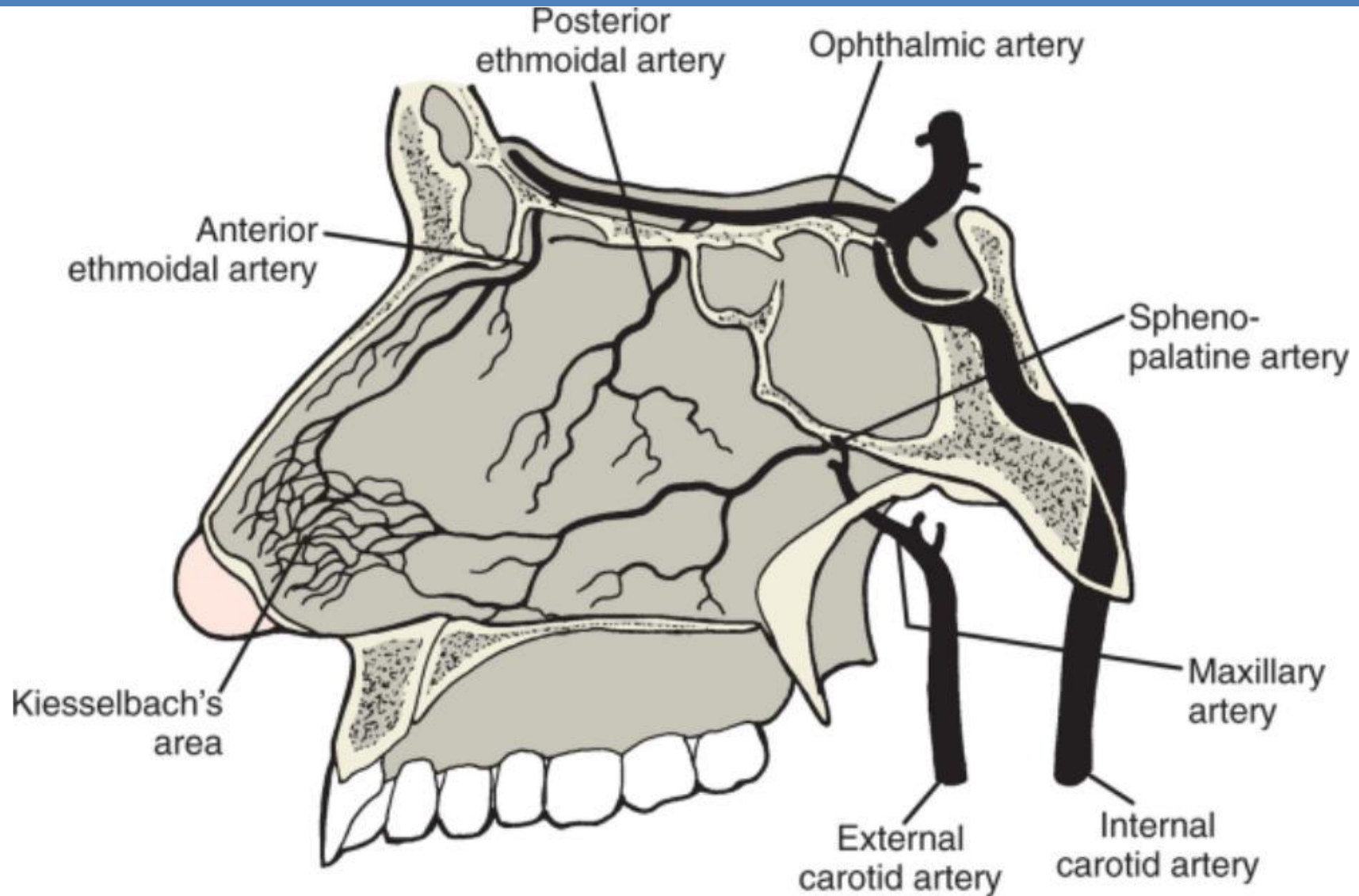
Cont.

- **Retrocolumellar vein** runs 2mm parallel & behind the columella (the anterior septum).
  - It is superficial & is a common cause for ***venous bleeding in children***.
- ***Venous epistaxis*** from Retrocolumellar vein tends to occur in ***subjects <35yrs.***
- Note that ***most nasal bleeding is venous*** & is usually short lived.

Cont.

- **Woodruff's 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

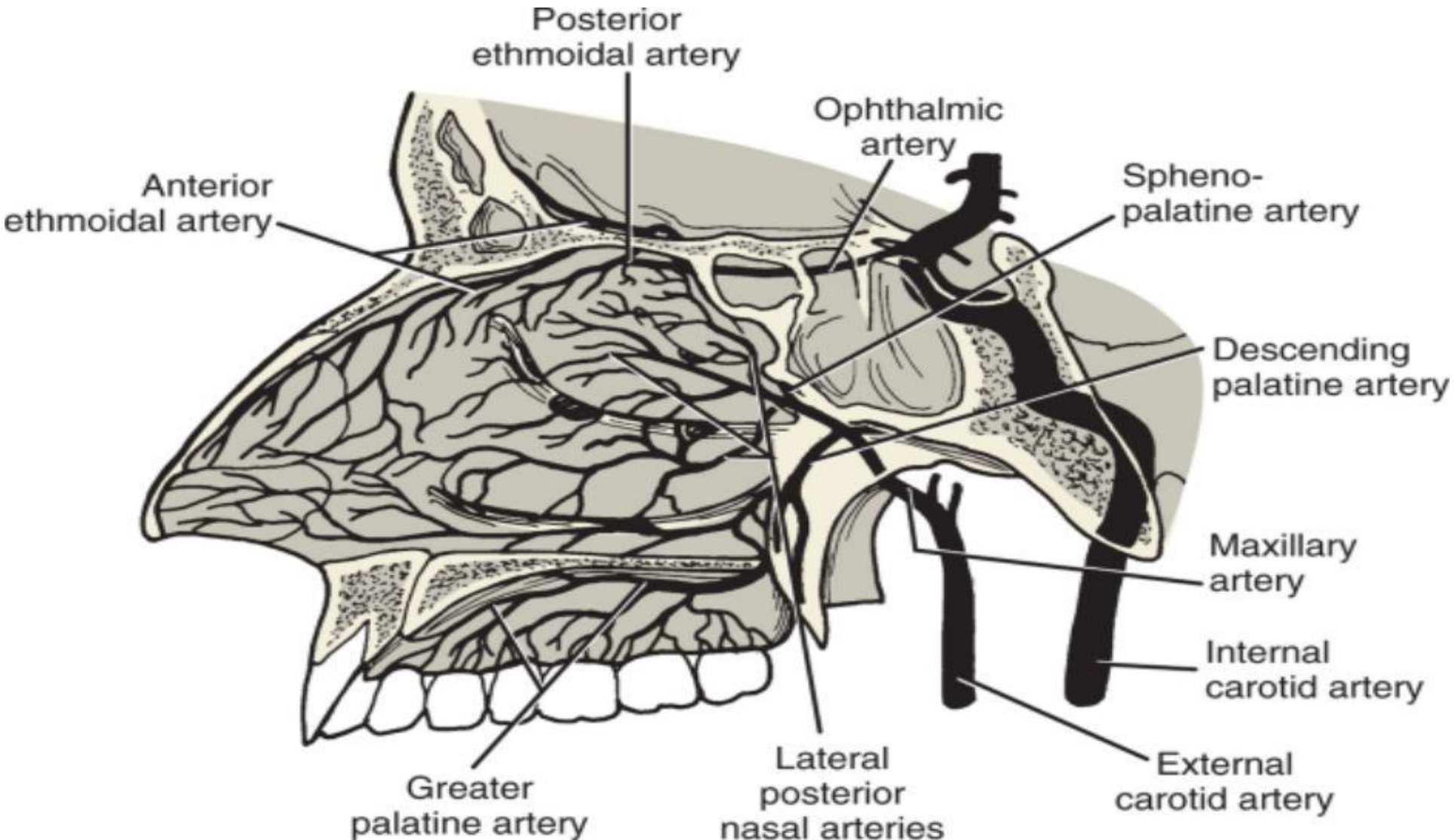
Fig 1: Vascular supply of the nasal septum



Cont.

- The MOST important arterial supply to the nose from the ECA is the **sphenopalatine artery.**
- It comes into the nose via the **sphenopalatine foramen** that is found just at the posterior end of the inferior turbinate.

Vascular supply of the lateral nasal wall



Etiology

Primary/ Idiopathic epistaxis (70 – 80%)

Secondary epistaxis

Local

General

Cont.

- Etiology of epistaxis may be: **primary or secondary**.
  - 70 – 80% of all cases are idiopathic, i.e., **primary epistaxis.**
- Standardized description:
  - Anterior: Bleeding from a source anterior to the plane of the **piriform aperture** (anterior septum, vestibular skin, mucocutanoeus junction.)
  - Posterior: Bleeding posterior to the piriform aperture
- May be multifactorial, with each factor playing a minor role.

Local causes

- **Trauma**: Nose picking, facial trauma, RTA, fracture base of skull.
- **Idiopathic** (from Little's area)
- **Inflammatory**: Rhinitis (infective, allergic), sinusitis, specific nasal infections (bacterial, fungal, TB)

Cont.

- **Anatomical/ structural deformities** of the nose:
  - Congenital or acquired
  - Deviated nasal septum: people tend to bleed from the most deviated part of the septum.
  - Nasal spur
  - Hypertrophied or rotated turbinates (paradoxical) drying, crusting, bleeding

General causes

- Neoplastic (Benign or malignant)
  - In the nose or paranasal sinuses & postnasal space tumors: **Nasopharyngeal carcinoma** is the MC tumor that presents with one – sided bleeding
  - **Juvenile angiofibroma** (exclusively in the adolescent males, recurrent & severe episodes of epistaxis. ***Never biopsy a mass in the nose with a history of bleeding since the patient will bleed excessively***)
  - Aneurysms of internal carotid artery

Cont.

- Environmental: high altitude, air conditioning, toxic or chemical irritant, Cold winter weather
- Foreign bodies:
  - Unilateral, purulent nasal discharge & bleeding
  - Usually in children or the mentally retarded
- Iatrogenic
  - Excessive prescription of **intranasal topical steroids** for allergic rhinitis → Can lead to changes in mucosa & bleeding
  - After nasal surgery (septoplasty, FESS)

Cont.

- HTN – associated with local factors:
  - Elderly: arteriosclerotic vessels do not contract well and the nasal mucosa becomes atrophic hence dries up & cracks easily and vessels may rupture esp. during a hypertensive episodes.
- Blood dyscrasias: vary in ability to cause epistaxis; usually diagnosed in early life by excessive bleeding after minor trauma.
  - Examples: deficiency of factor VIII (hemophilia A), factor IX (hemophilia B)
  - vWF; impaired PLT adhesiveness
  - Leukemia, lymphomas, ITP, Osler Rendu Weber Syndrome
- Alcohol abuse: poor diet esp. vitamin C, K deficiency
- Parenchymal liver damage: deceased fibrinogen & prothrombin

Cont.

- Pregnancy due to folic acid deficiency (decreased platelets)
- Drugs (anticoagulants, aspirin, NSAIDs, CAF, Carbenicillin).
- Systemic toxic agents: phosphorous, mercury.
- Infectious diseases (Typhoid, rheumatic fever, whooping cough)
- Cardiovascular disorders (MS, CHD, CCF, COA)
- ISS → HIV
- Allergic diseases
- Malnutrition

Clinical presentation

- Sudden onset
- Occasionally preceding headache
- May be unilateral
- Smell of blood in the throat, trickling in the throat
- If the blood trickles posteriorly, the patients swallows & eventually vomits fresh blood (*may be misdiagnosed as an UGIB*)
- Anxiety (increased PR, BP) increased bleeding
- Elderly decompensate very fast (hypovolemic shock)

Investigations

1. Medical history
2. Physical examination
3. Laboratory investigations: FBC, PBF, GXM
4. Radiological investigations
5. EUA/ Endoscopy

General management

- ABC:
  - Depends on the degree of hemorrhage
  - Site: anterior (anterior to pyriform sinus) & posterior
  - Age of patient
  - History of precipitating factors
- An accurate patient history (location, duration & frequency), trauma, nasal blockage, rhinorrhea.
- Family history, drug history, tobacco & alcohol usage
- History of prior bleeding is important, general state of the patient (e.g. 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)

Cont.

- **Rules of fluid replacement:**
  - Crystalloid fluid → 3:1
  - Colloids fluids → 1:1
- The patient is evaluated in the **seated position** with **adequate light suction anesthetic solution**.
- Packing materials & cautery are used.
- A topical vasoconstrictor & anesthetic agent: oxymetazoline & xylocaine.
- Most bleeding sites are anterior & accessible to local treatment.
- Bleeding sites that are not visible on anterior rhinoscopy most likely from posterior (sphenopalatine artery).

Cont.

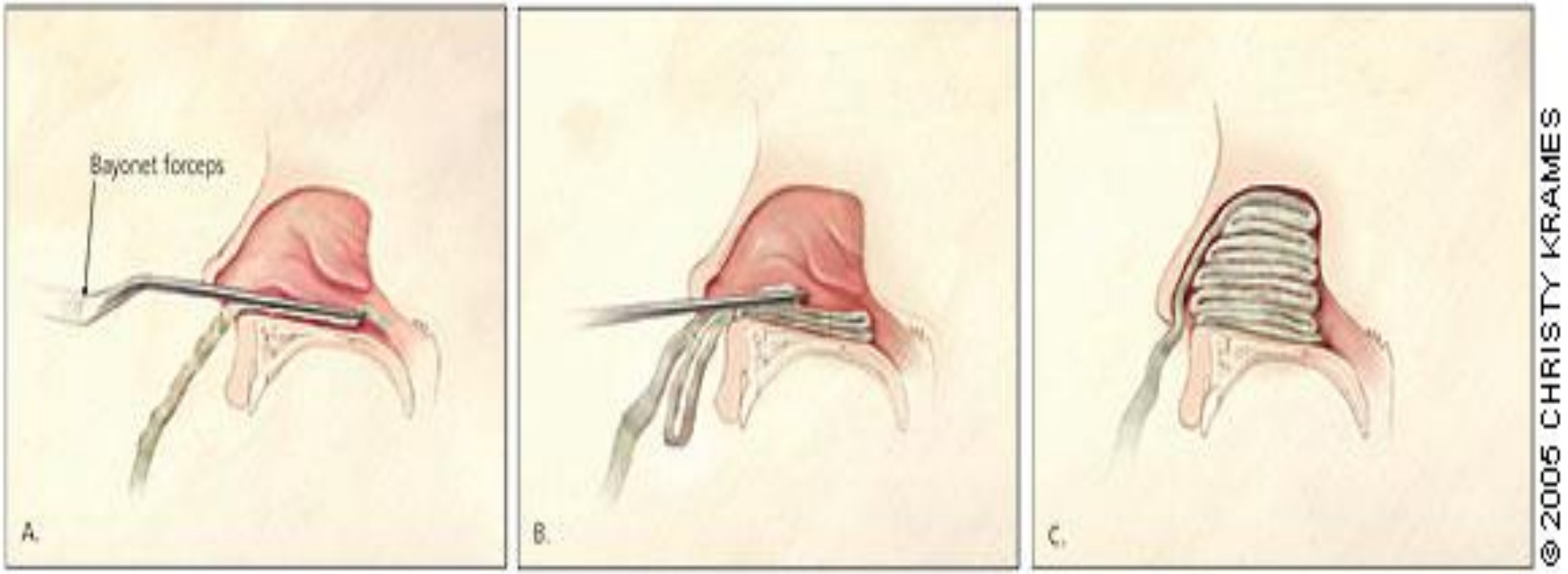
- Trivial hemorrhage: first aid measures.
- Mild – moderate (patient may develop shock)
- Main aim is to stop hemorrhage.
- Firm pressure to the nostrils 5 – 10 mins seated upright, head facing downwards.
- Advice the patient to breath through the mouth.
- Arrange for good light, nasal cannula & suction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

Non – surgical management

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

Nasal packing

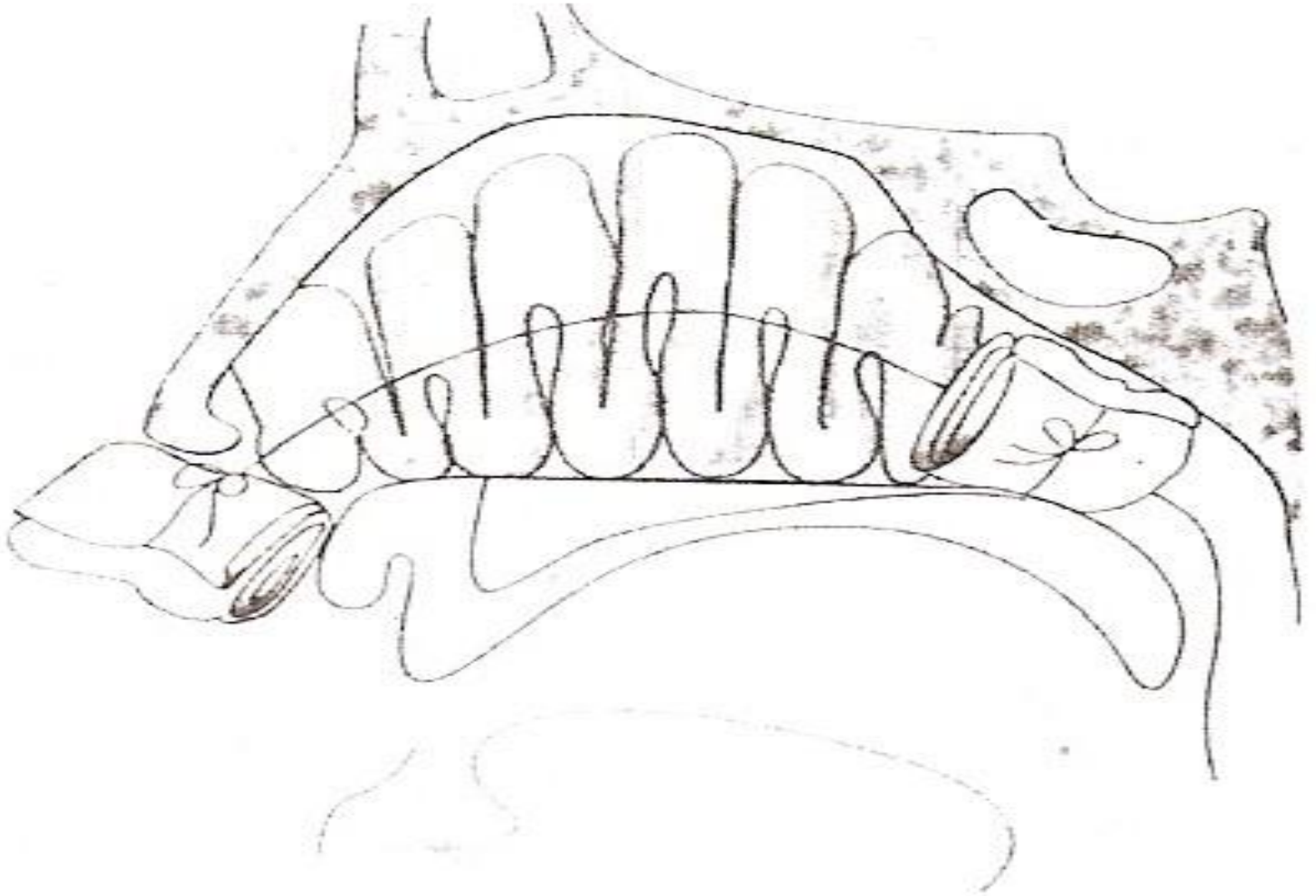
- **Anterior**
  - Ribbon gauze impregnated with petroleum jelly or bismuth iodoform paraffin paste (BIPP).
  - Left in situ for 24 to 72 hrs.
  - Complications include: ***sinusitis, septal perforation, alar necrosis & hypoxia.***
  - There are special nasal tampons and balloon catheters.
- **Posterior**:
  - Under GA preferably. Can also use Foley's catheter
  - Hot water irrigation at 50^0 (activates clotting system)
  - Systemic medications: Tranexamic acid → inhibits fibrinolysis

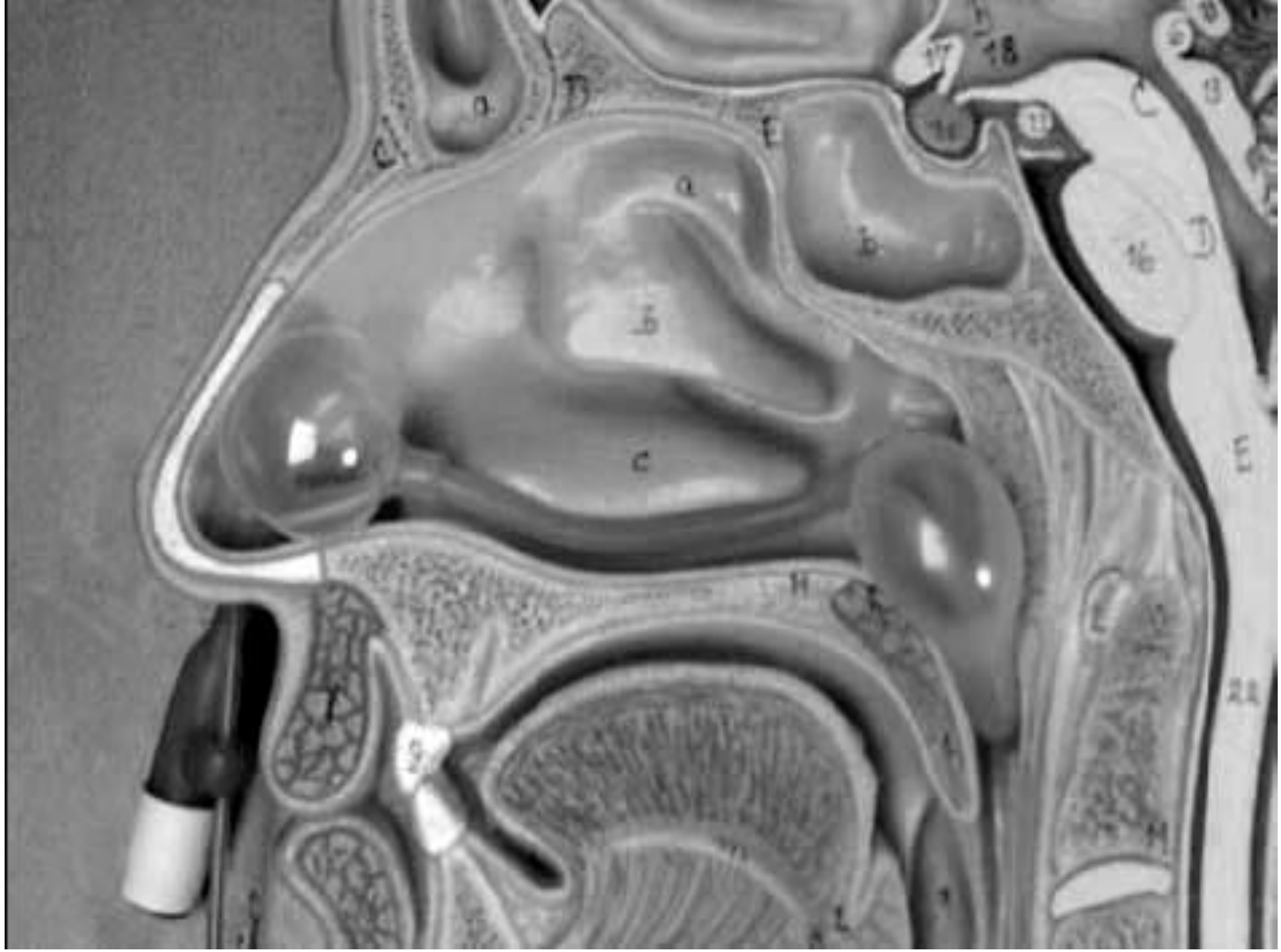


Anterior nasal packing

1.Take a gauze
2.Impregnate with Vaseline, sofratulle jell
3.Layer it from down to up
4.Live the pack in place for 24 – 48 hrs.

Posterior nasal packing: put in theater under GA

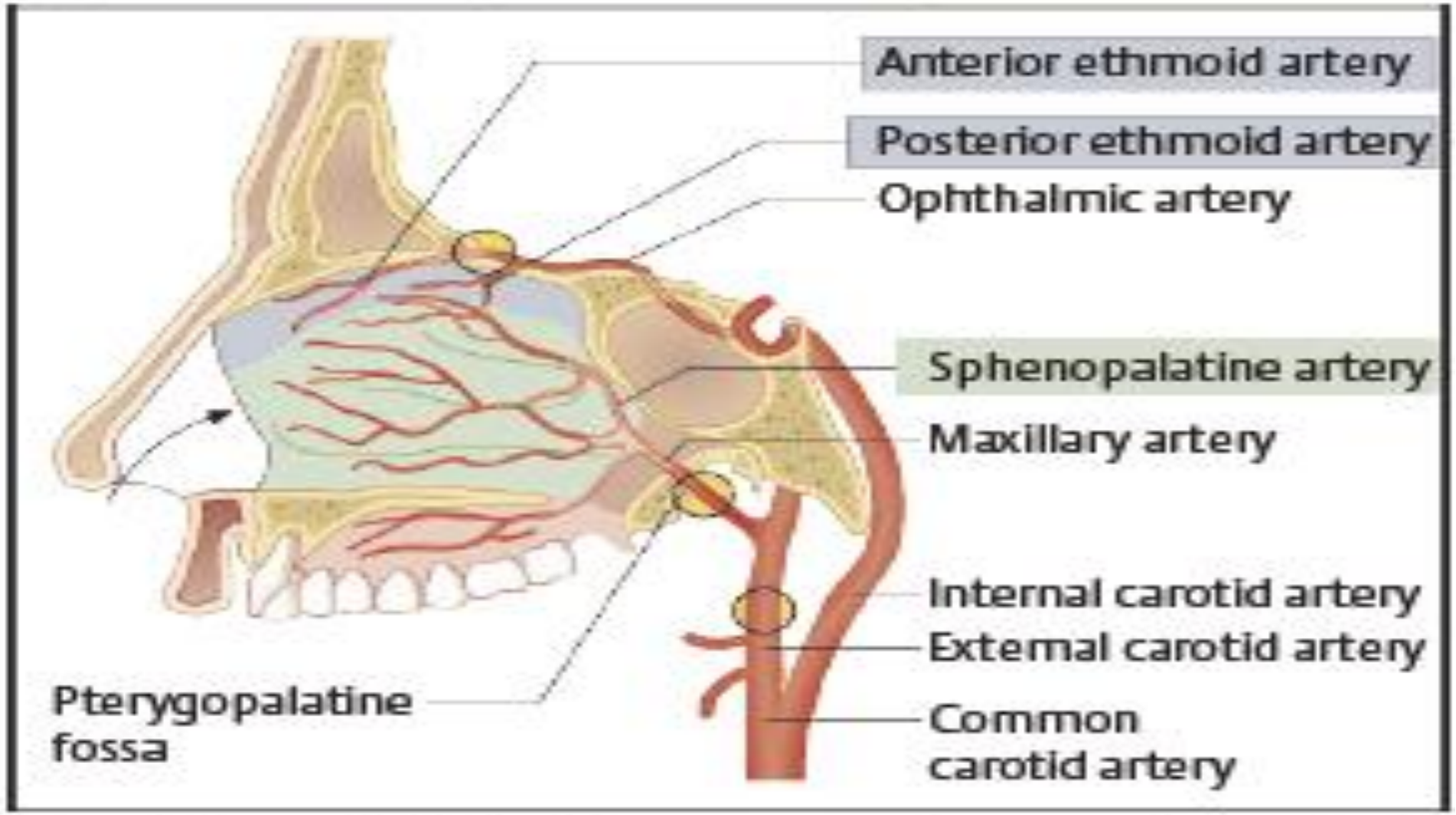




Surgical management

- In cases of intractable bleeding ligation of arteries is performed:
  - Sphenopalatine artery
  - Maxillary Artery
  - External carotid artery (percutaneous access) → rare
  - Ant. and Post. 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.

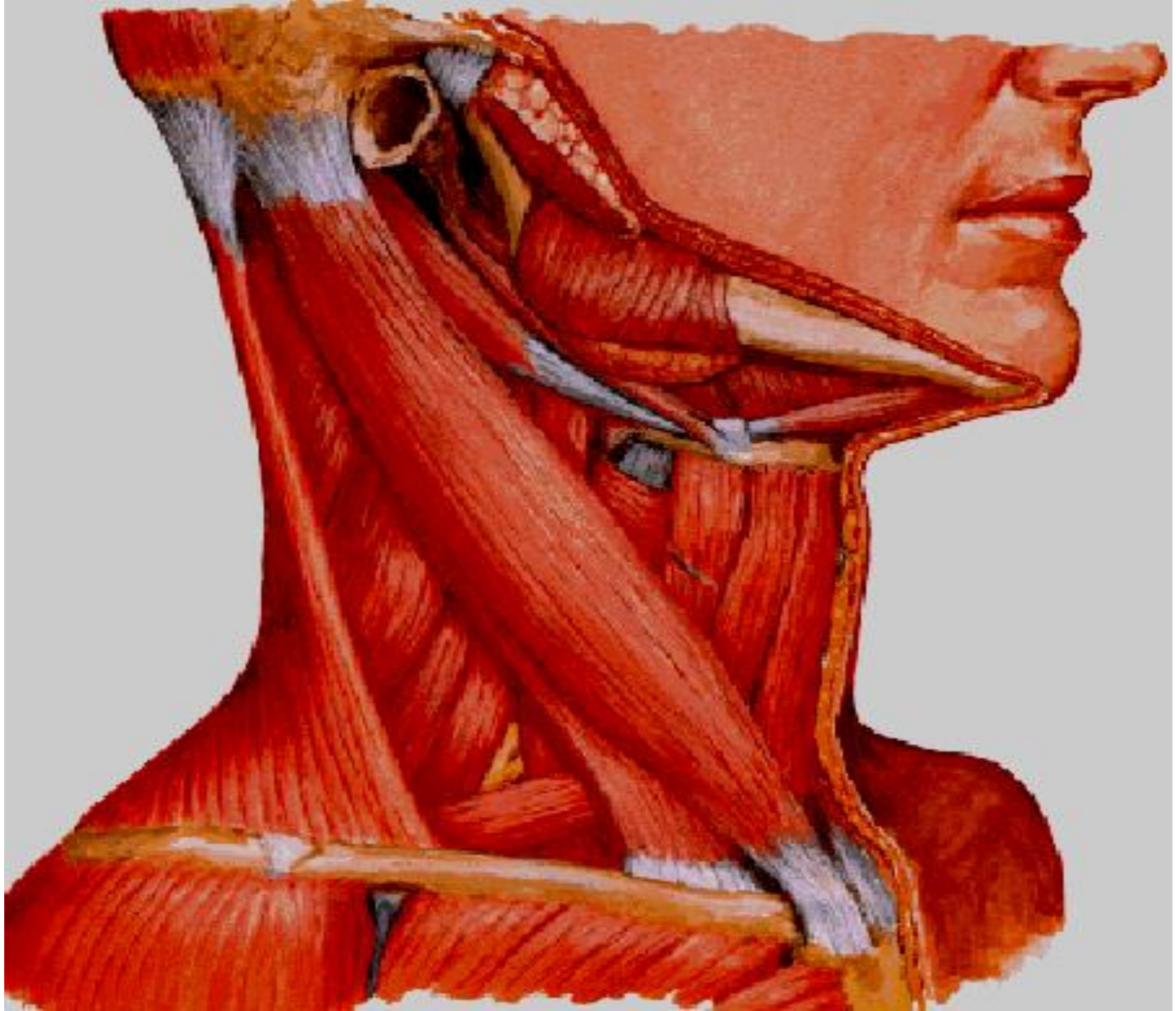
NECK TRAUMA

DR CATHERINE IRUNGU

HISTORY

- First recorded hanging - Judas Iscariot in 30 A.D.
- Innocuous injury may mask serious damage (UAO & hemorrhage)
- 1552- Ambroise Pare successfully treated a vascular neck injury
- 1803, Fleming ligated a lacerated common carotid artery

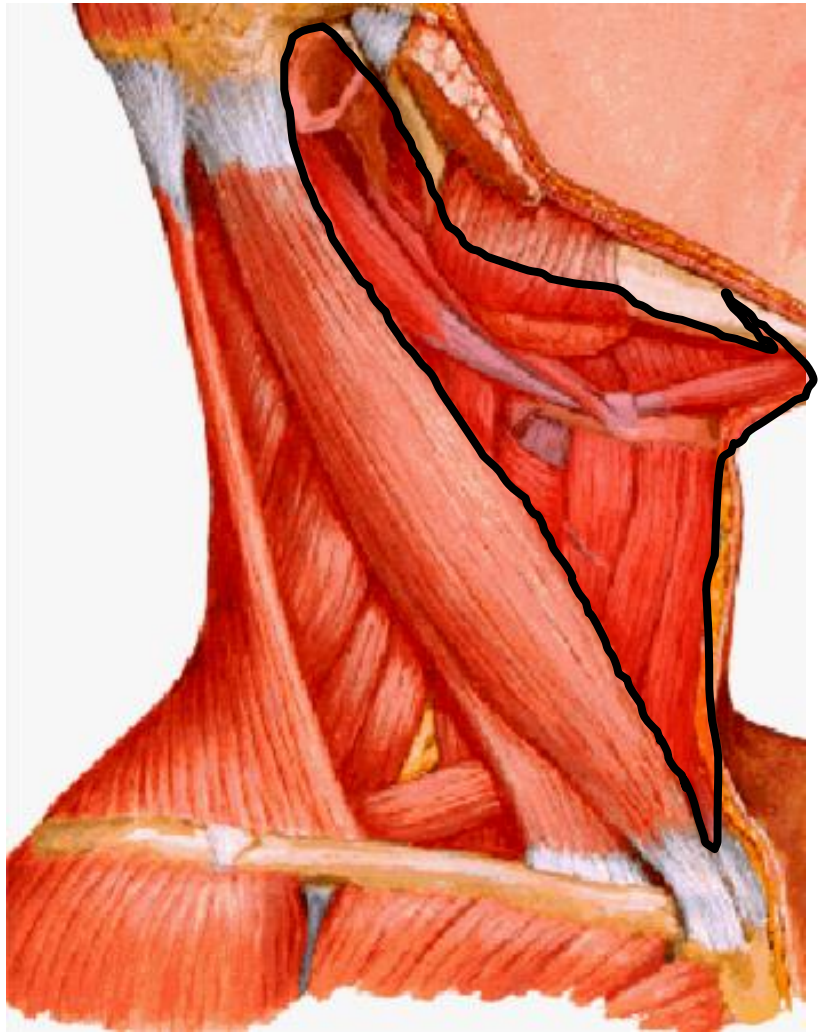
- At WWI all major vessels were ligated.
- Mortality rates of neck injuries were recorded as high as 60%.
- 1/3 - Significant neurologic impairment
- WWII- management changed to mandatory exploration of all penetrating wounds through the platysma.
- The present mortality for civilian wounds is 4-6%, with most deaths occurring from vascular injuries prior to arriving in the emergency room.



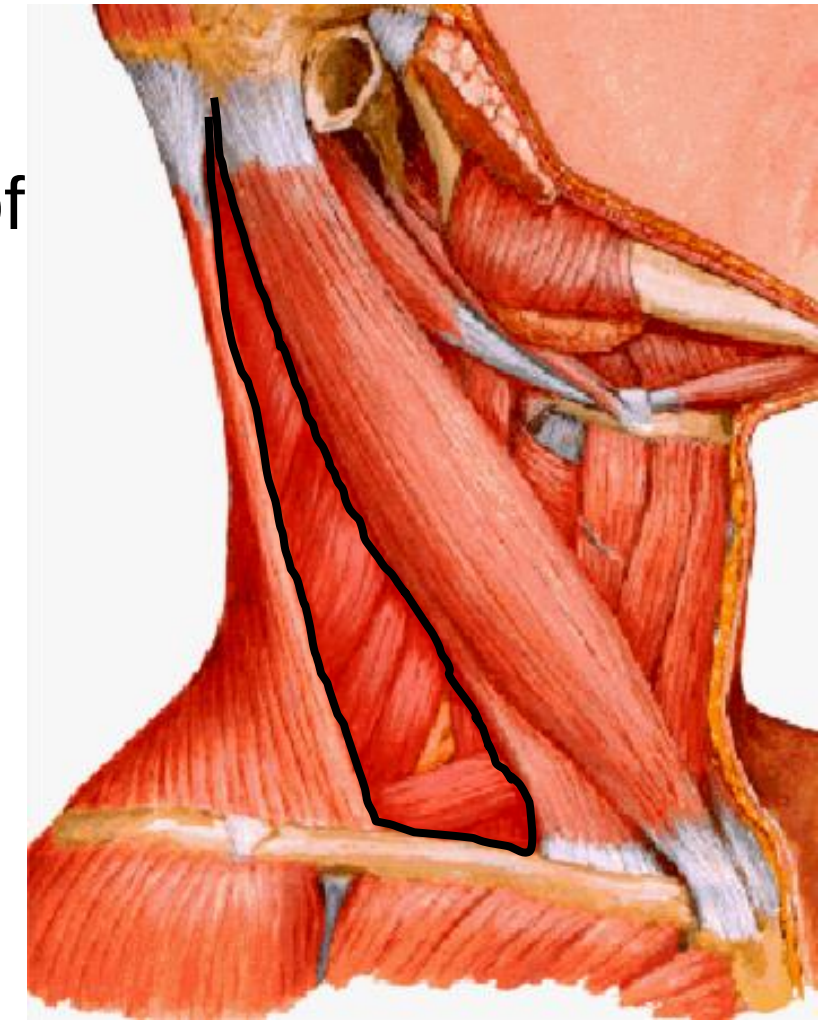
Anatomy

SCM divides cervical region into two

- The anterior triangle
  - Anterior margin of SCM
  - Inferior margin of mandible
  - Vertical midline from mental symphysis to the suprasternal notch.

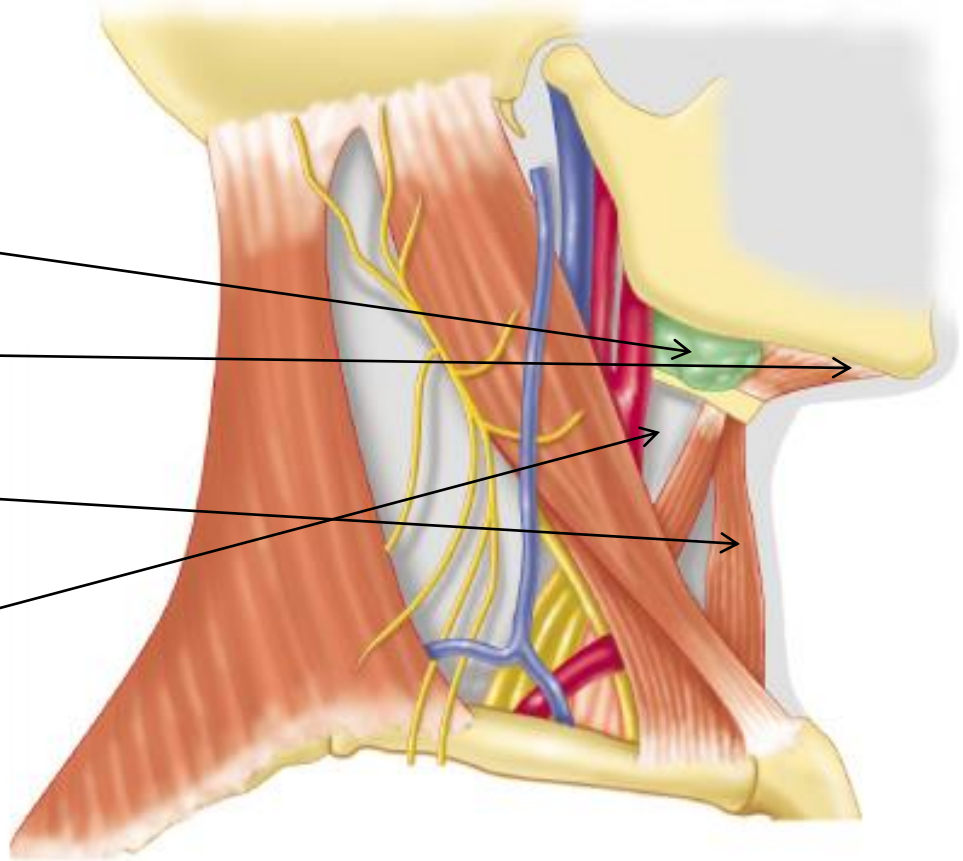


- The posterior triangle
  - anterior margin of the trapezius muscle
  - posterior border of SCM
  - middle third of the clavicle



- Suprahyoid and infrahyoid areas

- Submandibular,
- Submental,
- Muscular
- Carotid triangles



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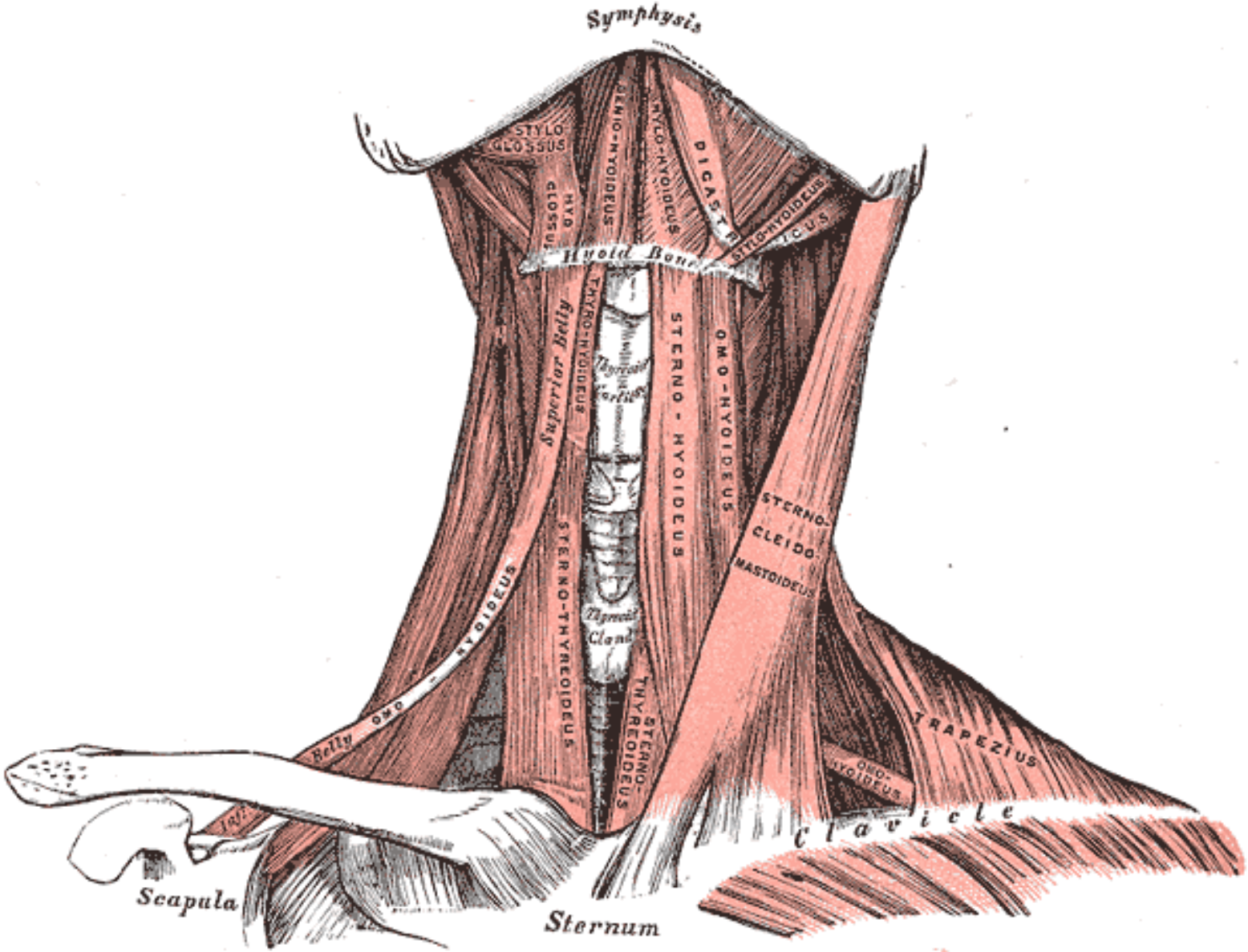

Symphysis
STYLO GLOSSUS
HYO GLOSSUS
GENIO-HYOIDEUS
MYLO-HYOIDEUS
DIGASTRIC
STYLO-HYOIDEUS
Hyoid Bone
Superior Belly
THYRO-HYOIDEUS
Thyroid Cartilage
STERNO - HYOIDEUS
OMO-HYOIDEUS
STERNO-CLEIDO-MASTOIDEUS
Thyroid Gland
STERNO-THYREOIDEUS
Belly OMO - HYOIDEUS
STERNO-THYREOIDEUS
TRAPEZIUS
OMO-HYOIDEUS
Clavicle
Scapula
Sternum

PosteriorTriangles of the Neck

Bounders:

Base

intermediate 1/3 of the clavicle

Apex

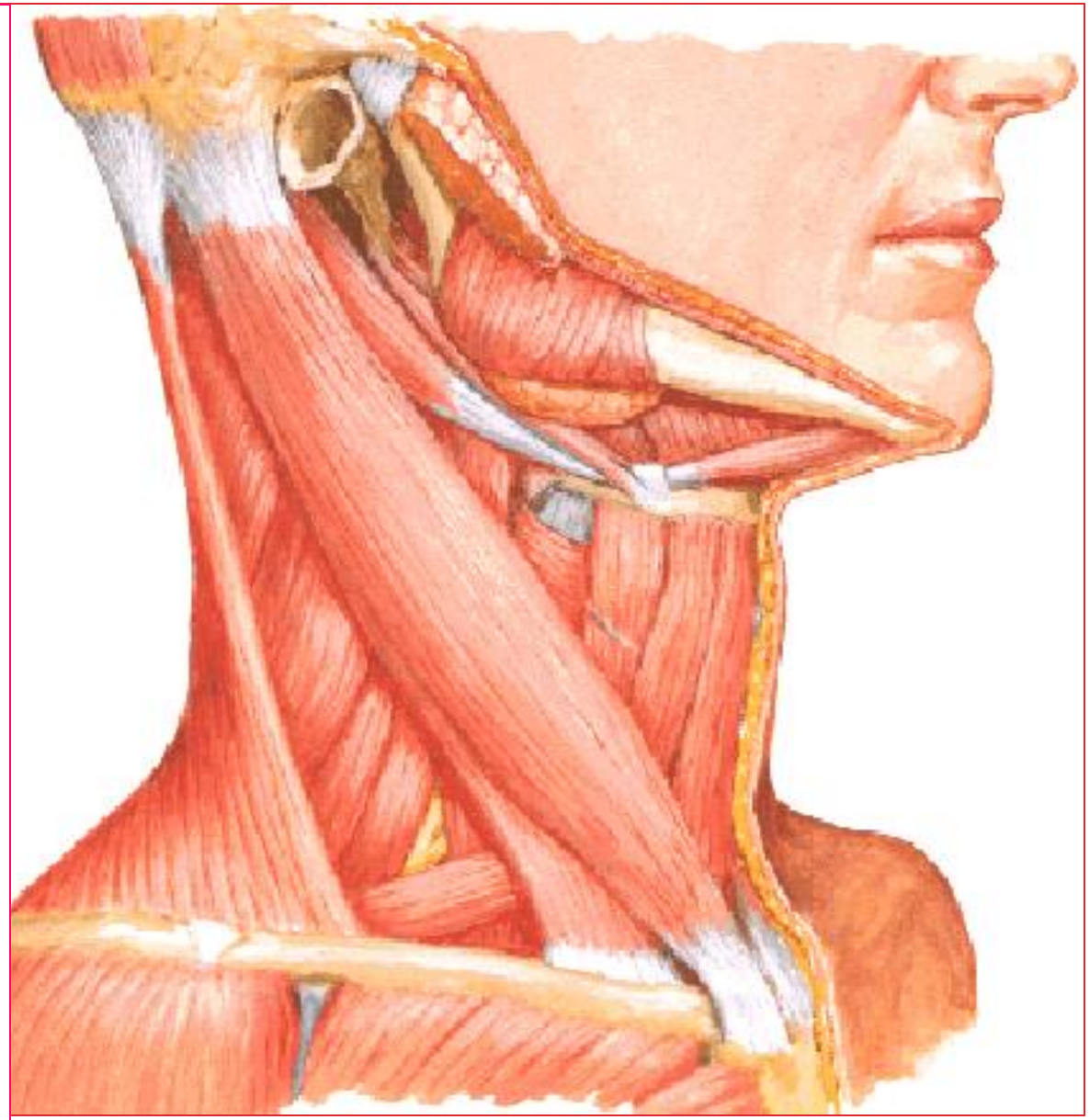
meeting of the anterior and posterior border

Anterior border

posterior border of the SCM

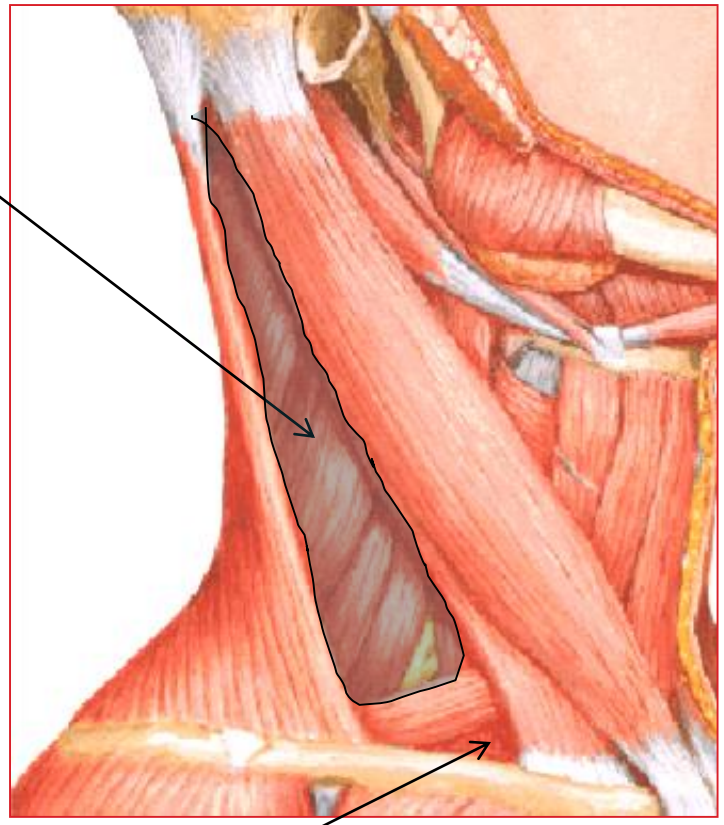
Posterior border

anterior border of the trapezius muscle



OCCIPITAL TRIANGLE

- Upper and larger part of posterior triangle
- **Spinal accessory nerve**,
- Cutaneous and muscular branches of cervical plexus,
- Upper part of brachial plexus
- Lymphnodes along posterior border of SCM



SUPRACLAVICULAR TRIANGLE

Its floor contains the first rib, scalenus medius and the first slip of serratus anterior
Supraclavicular nerves

Divided by the inferior belly of the omohyoid muscle into •Occipital •Subclavian triangles (supraclavicular)	
Muscle	Inferior belly of Omohyoid muscle
Arteries	The 3rd part of subclavian artery, Suprascapular artery, Transverse cervical artery and The 3rd part of the occipital artery
Veins	Subclavian vein, External jugular vein
Nerves	Spinal accessory nerve, Brachial plexus, Cervical plexus
Lymph nodes	Occipital lymph nodes and the Supraclavicular nodes

arteries

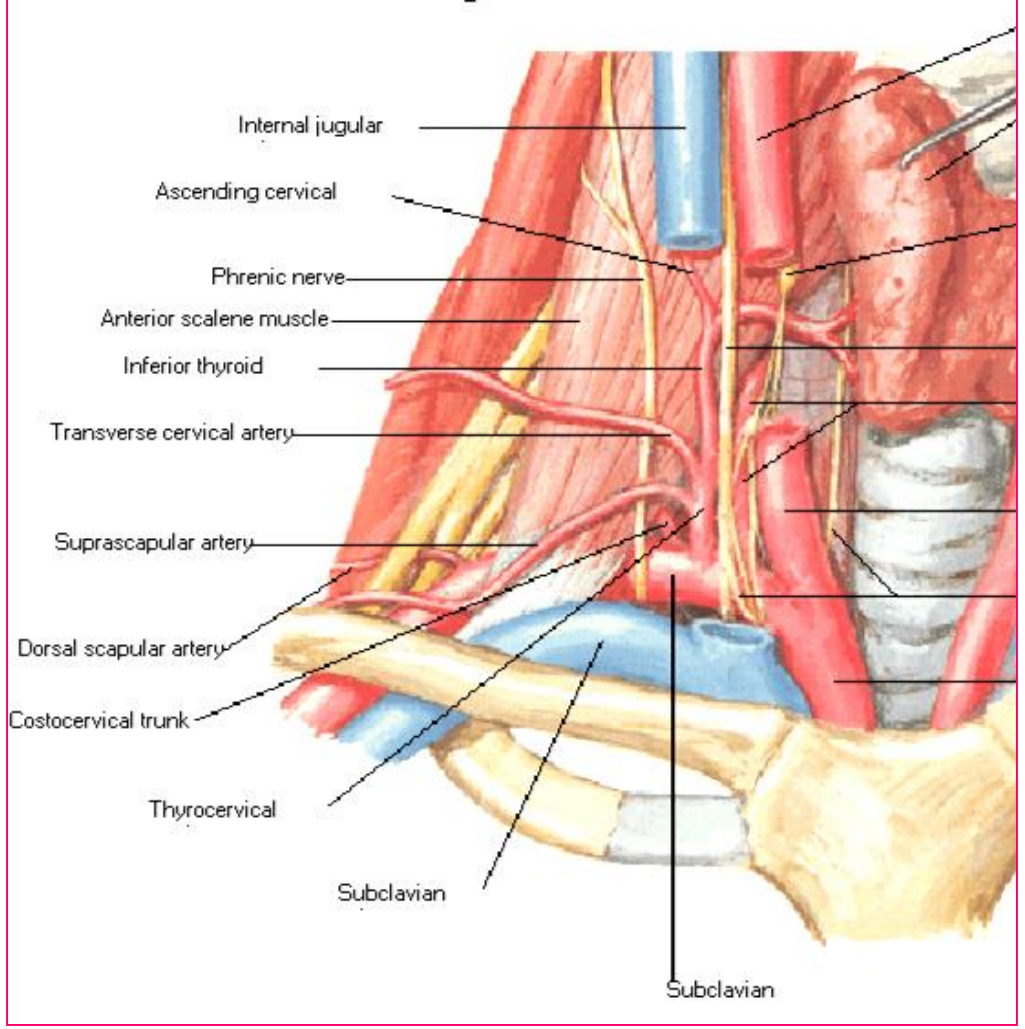
-Transverse cervical vessels

-Suprascapular vessels

-Subclavian artery crossing the first rib

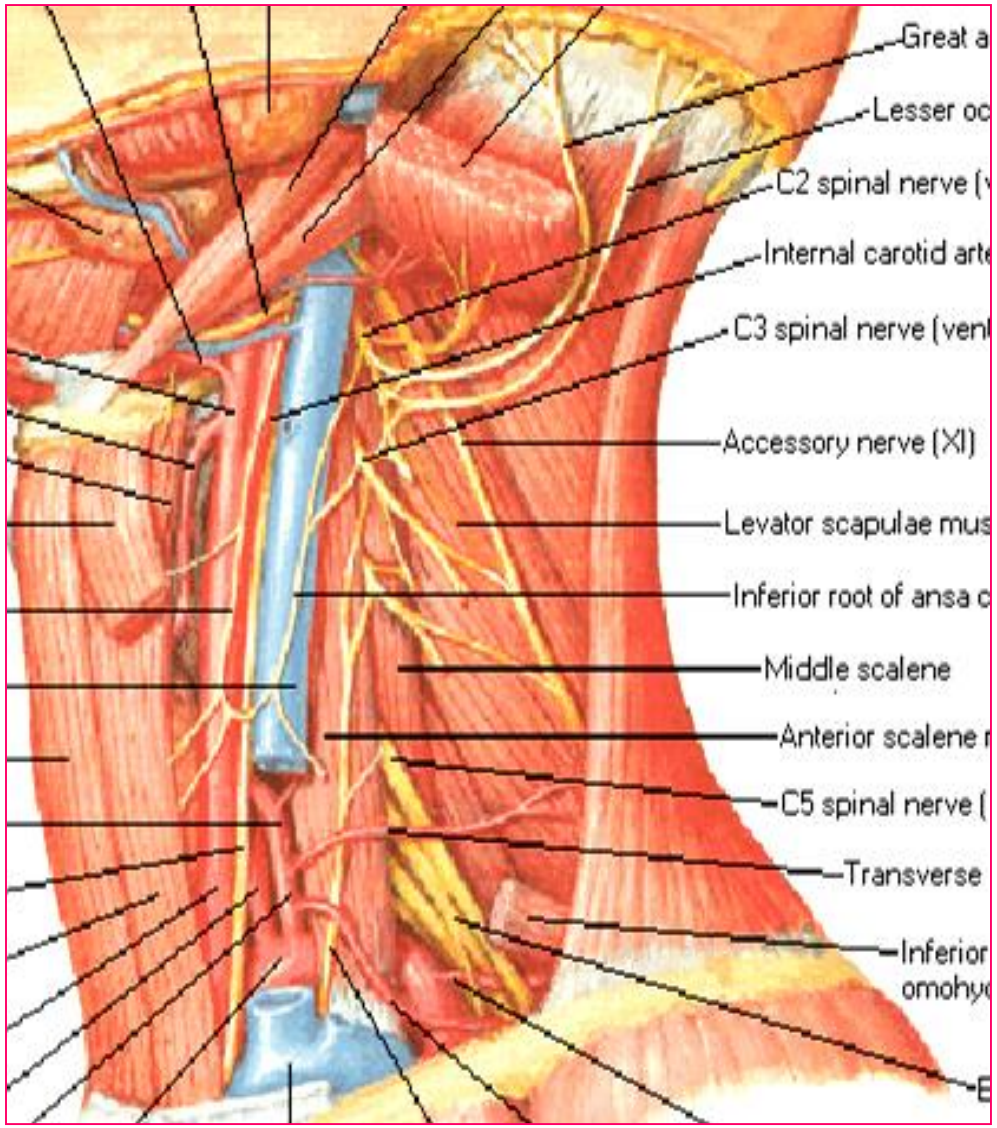
veins

External jugular vein



Nerves

1. *Accessory nerve
2. *Nerves to the levator scapulae
3. *Cutaneous branches of the cervical plexus
4. *Roots and trunks of the *brachial plexus
5. *Supraclavicular nerve
6. *Suprascapular nerve
7. *Dorsal scapular nerve *Long thoracic nerve
8. *nerve to subclavius

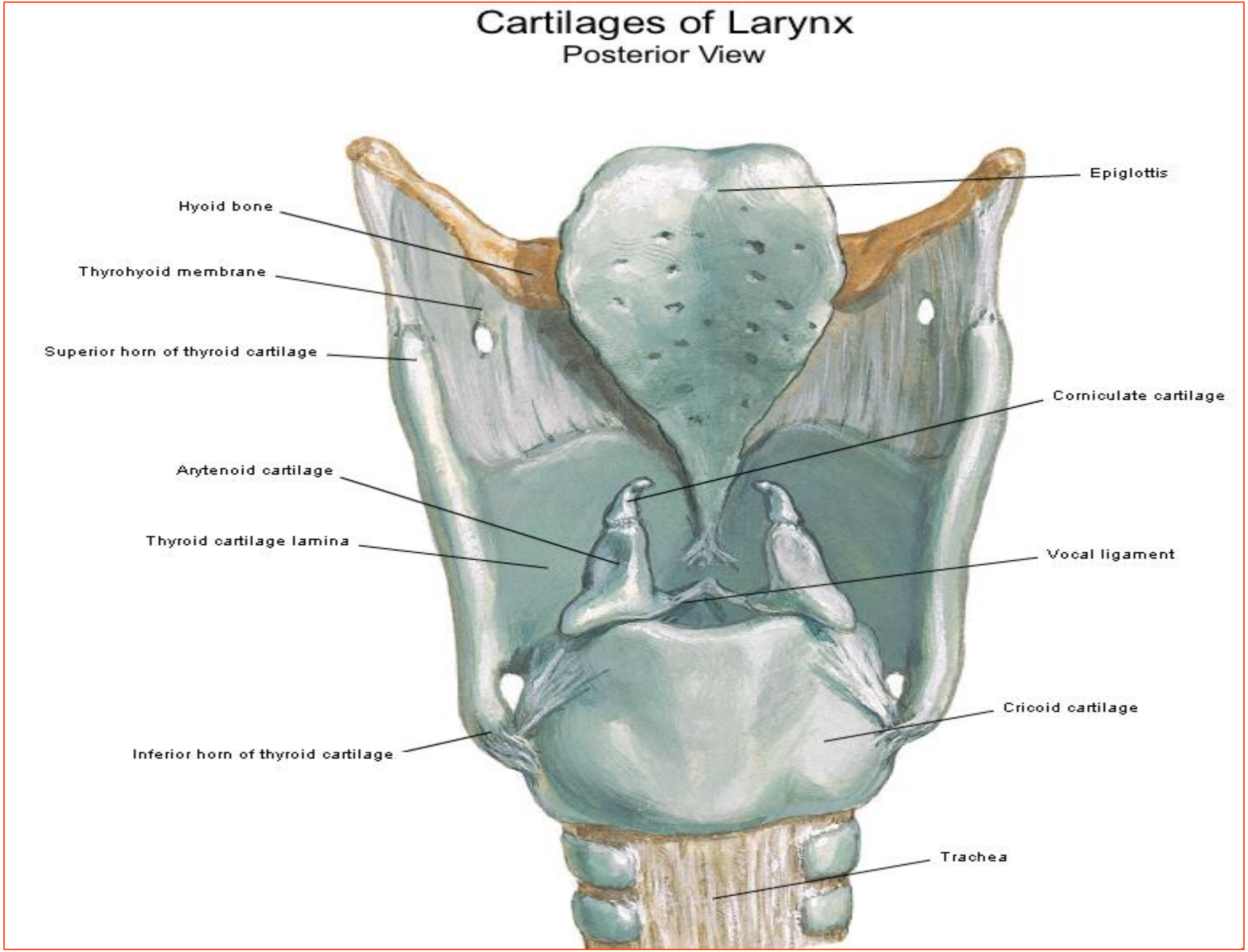


Posterior triangle

- Suprasternal Space
- Space b/w the 2 layers of deep fascia just above the manubrium which encloses
    - Sternal head of SCMs
    - Inf. end of anterior jugular veins
    - Jugular venous arch
    - Fat and few LNs

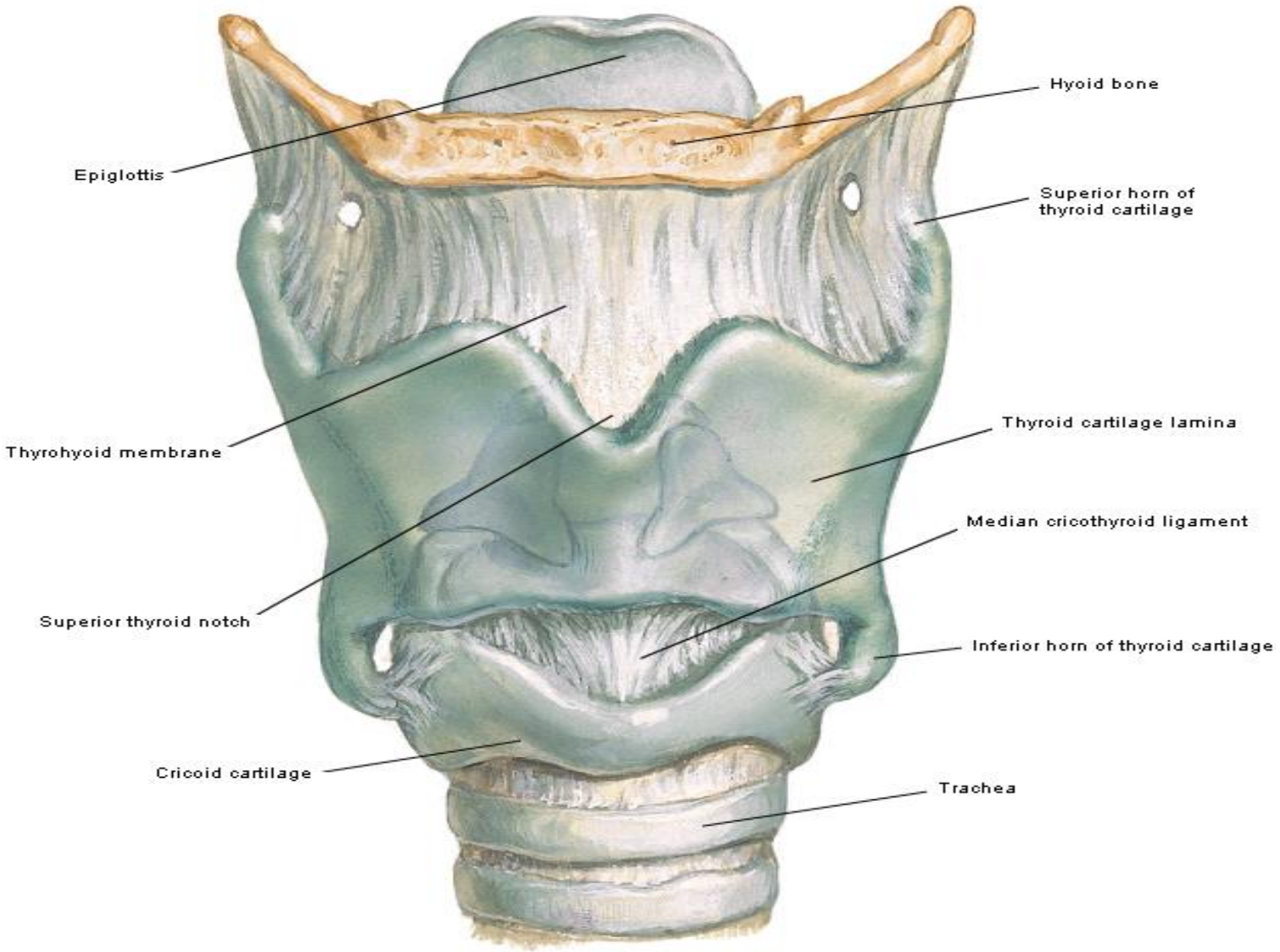
- Three layers surround the neck.
  - Superficial fascia.
  - Platysma.
  - Deep fascia’
    - Investing.
    - Visceral.
    - Prevertebral.

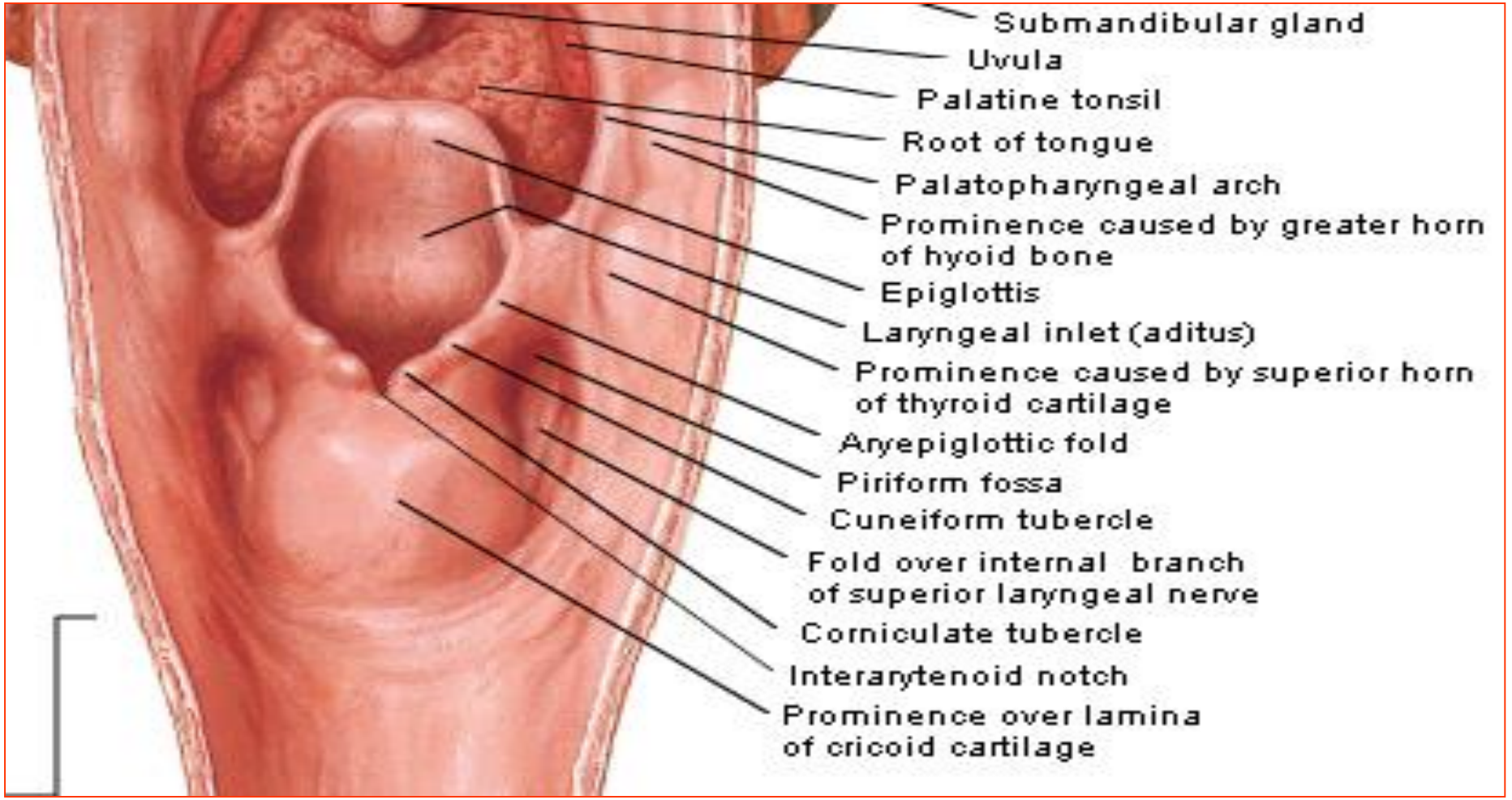
Cartilages of Larynx
Posterior View

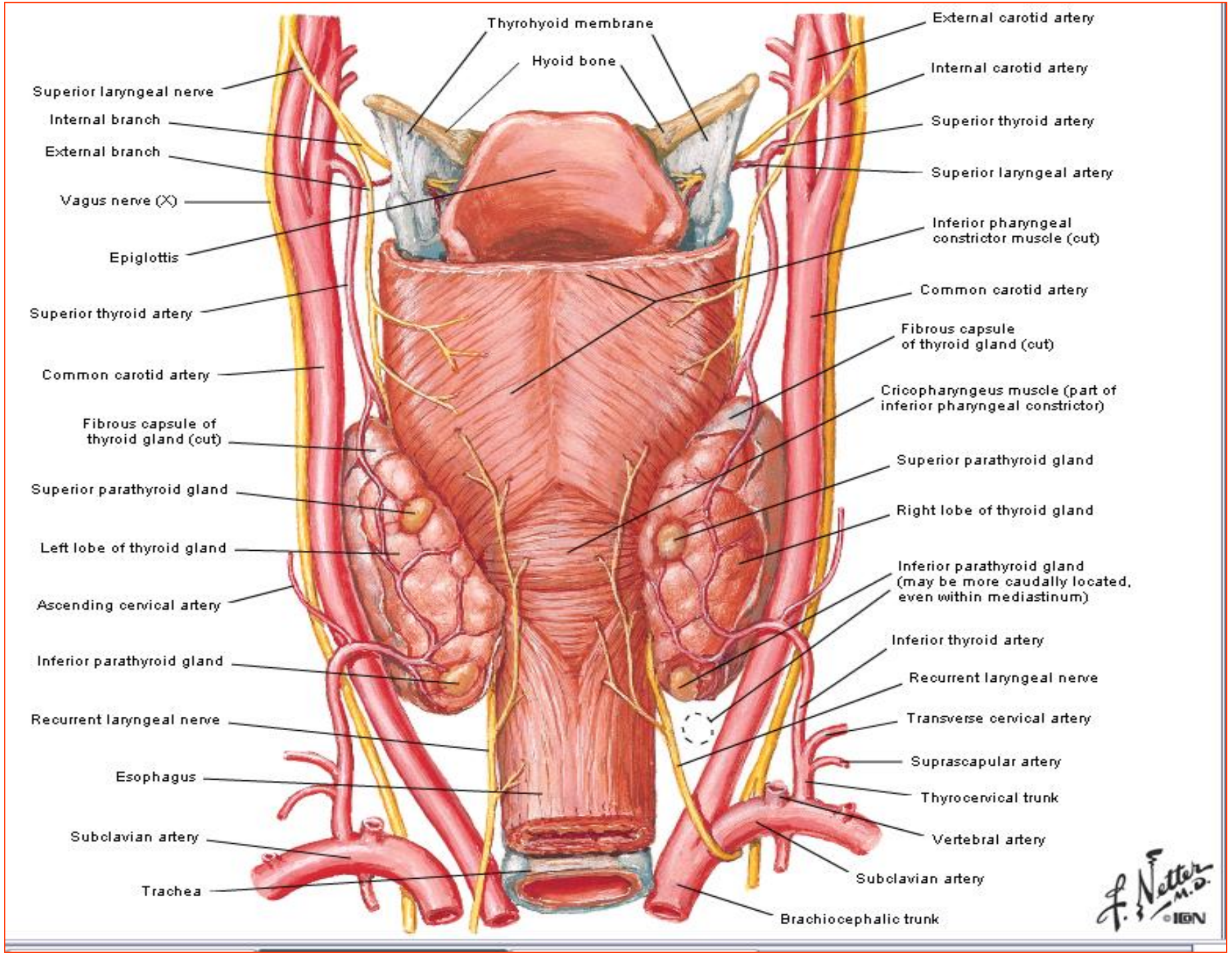


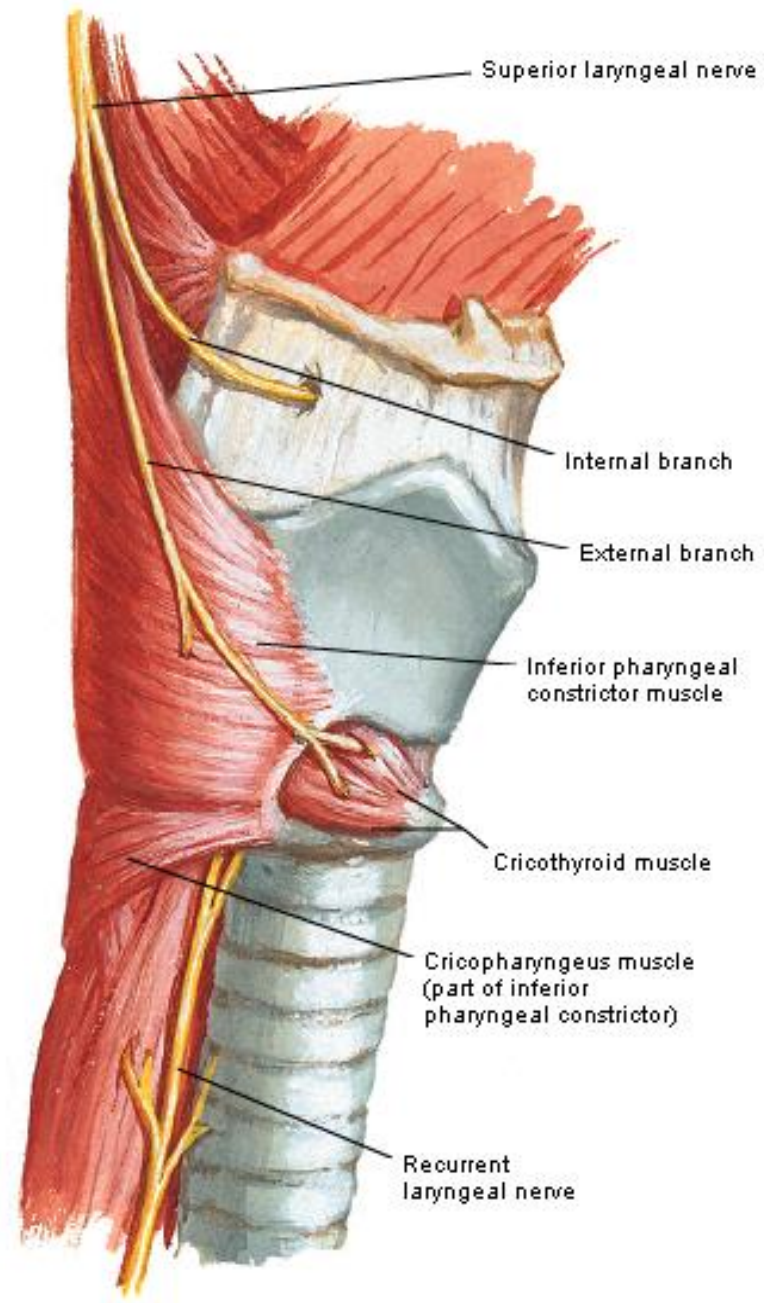
Cartilages of Larynx

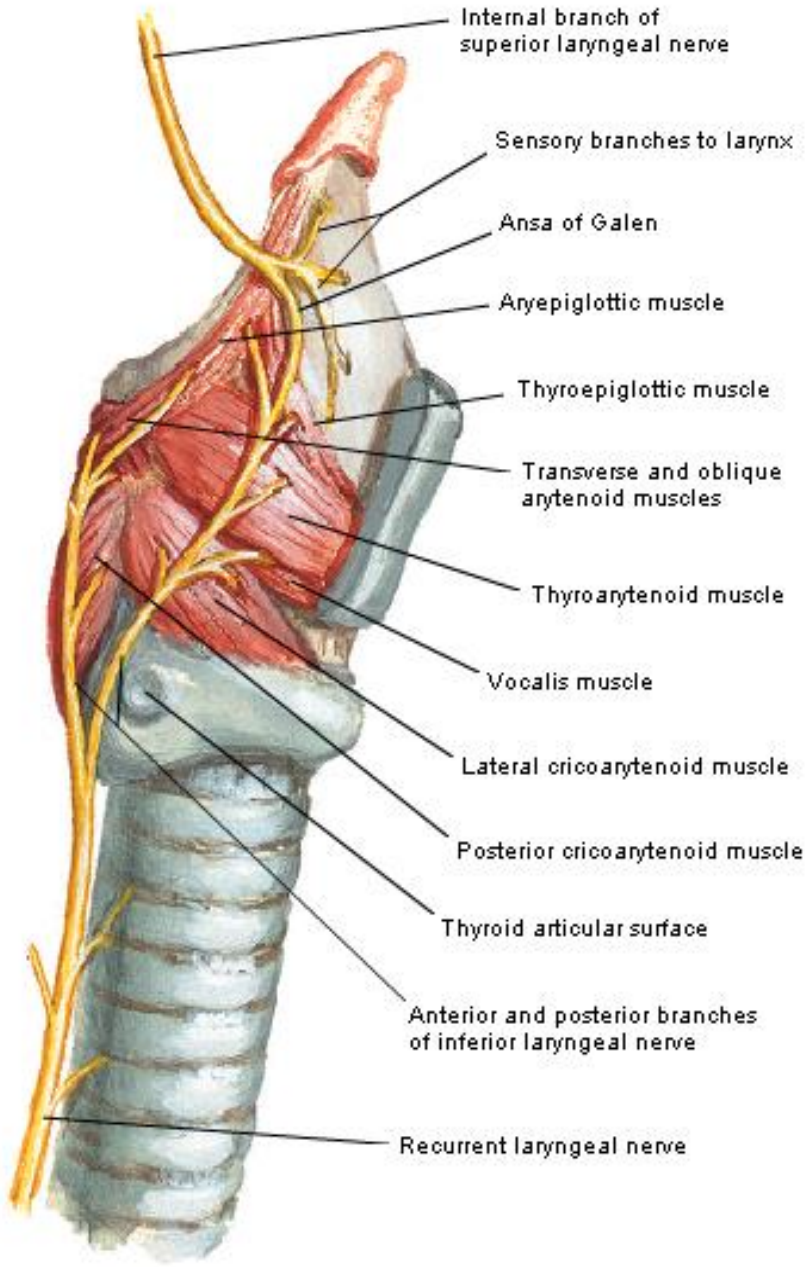
Anterior View




Submandibular gland
Uvula
Palatine tonsil
Root of tongue
Palatopharyngeal arch
Prominence caused by greater horn of hyoid bone
Epiglottis
Laryngeal inlet (aditus)
Prominence caused by superior horn of thyroid cartilage
Aryepiglottic fold
Piriform fossa
Cuneiform tubercle
Fold over internal branch of superior laryngeal nerve
Corniculate tubercle
Interarytenoid notch
Prominence over lamina of cricoid cartilage

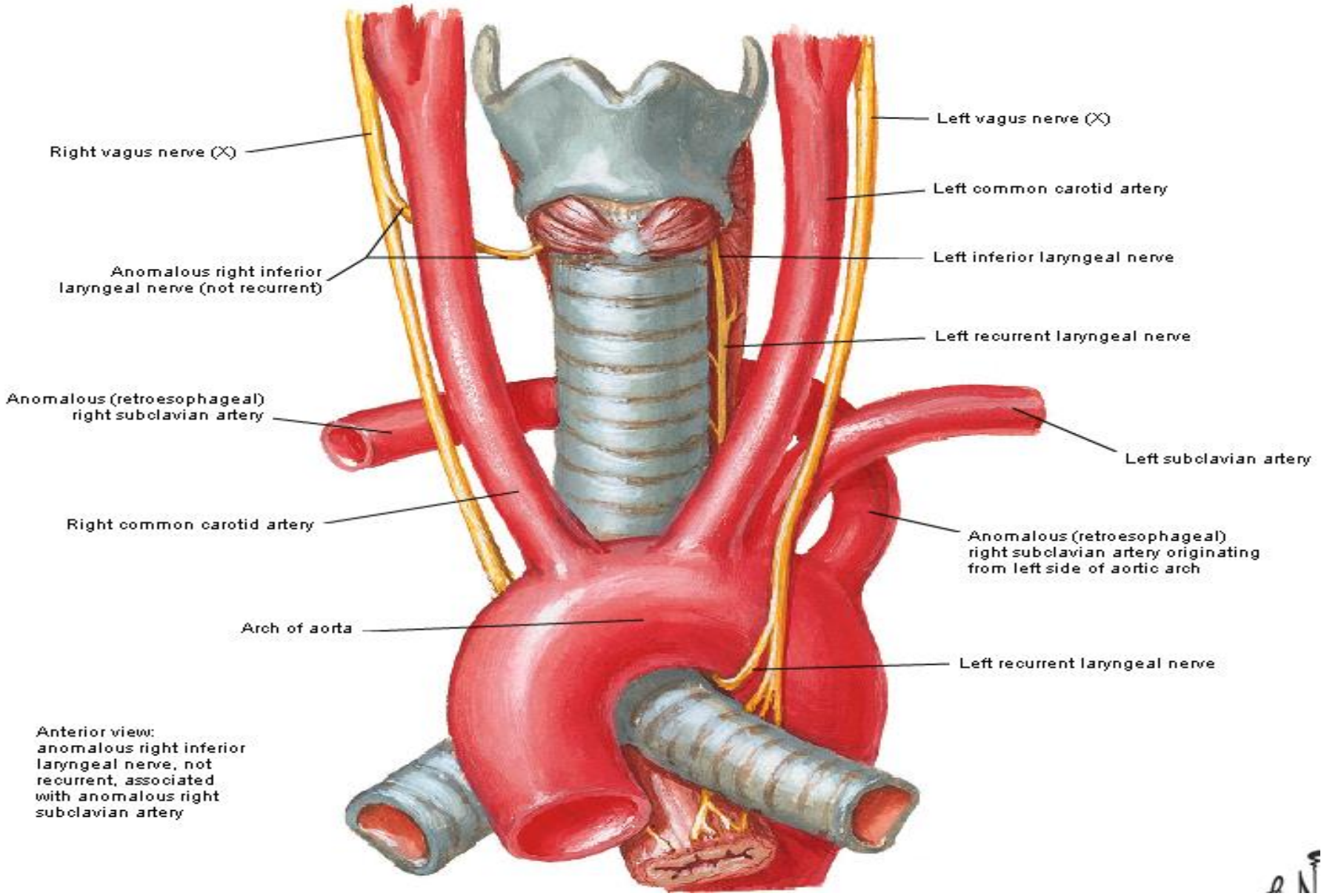

Thyrohyoid membrane
Hyoid bone
Superior laryngeal nerve
Internal branch
External branch
Vagus nerve (X)
Epiglottis
Superior thyroid artery
Common carotid artery
Fibrous capsule of thyroid gland (cut)
Superior parathyroid gland
Left lobe of thyroid gland
Ascending cervical artery
Inferior parathyroid gland
Recurrent laryngeal nerve
Esophagus
Subclavian artery
Trachea
External carotid artery
Internal carotid artery
Superior thyroid artery
Superior laryngeal artery
Inferior pharyngeal constrictor muscle (cut)
Common carotid artery
Fibrous capsule of thyroid gland (cut)
Cricopharyngeus muscle (part of inferior pharyngeal constrictor)
Superior parathyroid gland
Right lobe of thyroid gland
Inferior parathyroid gland (may be more caudally located, even within mediastinum)
Inferior thyroid artery
Recurrent laryngeal nerve
Transverse cervical artery
Suprascapular artery
Thyrocervical trunk
Vertebral artery
Subclavian artery
Brachiocephalic trunk
F. Netter M.D.
©IGN





Thyroid cartilage lamina removed

Nerves of Larynx
Anterior View



Anterior view: anomalous right inferior laryngeal nerve, not recurrent, associated with anomalous right subclavian artery

Background

- Protected by
  - spine posteriorly
  - head superiorly
  - chest inferiorly
- The anterior and lateral regions are most exposed to injury.
- Two fascial layers invest the neck:
  - The superficial fascia (enveloping the platysma muscle)
  - The deep cervical fascia (envelops the SCM and trapezius) & demarcates the
    - pretracheal region (trachea, larynx, thyroid gland, and pericardium),
    - the prevertebral area (prevertebral muscles, phrenic nerve, brachial plexus, and axillary sheath),
    - and the carotid sheath (carotid artery, internal jugular vein, and vagus nerve).

Mechanism of Injury

- Penetrating projectile- kinetic energy
- (KE): $KE=1/2MV^2$. high-velocity projectiles impart significantly larger amounts of energy into the tissue
- Firearms- muzzle velocity
  - Low velocity (less than 1,000 feet per second)
  - High-velocity (more than 1,000 feet per second).

Gunshot wounds cause tissue injury

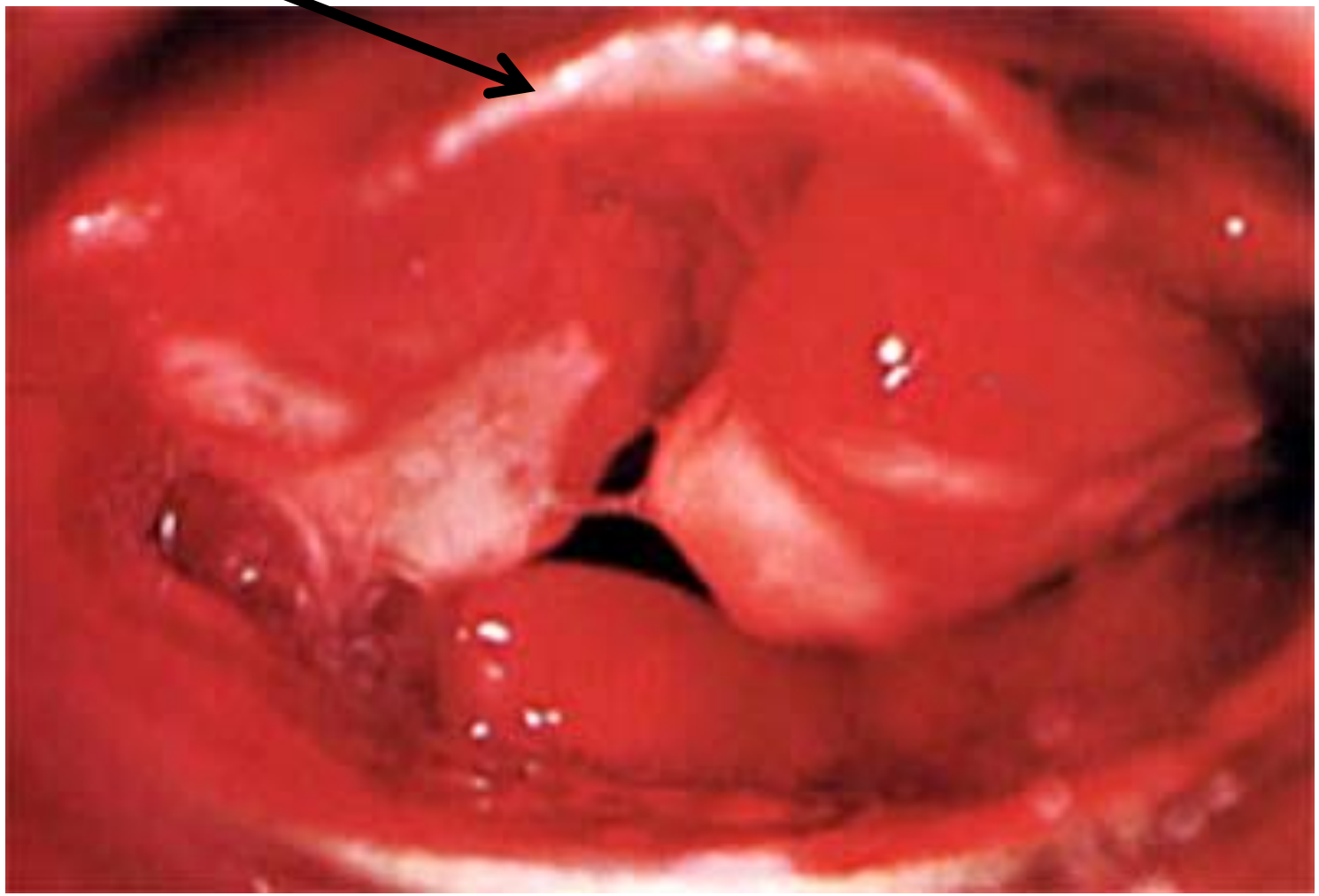
- Direct tissue injury
- Temporary cavitation, (results in tissue necrosis adjacent to the missile path)
- Transmission of shock waves (may result in adjacent or distant tissue damage)
- Projectiles have rotational characteristics, erratic course after impact
- Projectiles may shatter on tissue impact with secondary projectiles
- Impacted bone may shatter
- Secondary bone fragments may cause further damage.

Airway Injury

- More common with gunshot than stab wounds
- Respiratory distress and stridor
- Complete obstruction - cricothyroidotomy, or tracheostomy if the cricoid area is involved in the injury site.
- Tracheal injury- HOV, haemoptysis and subcutaneous emphysema.
- Laryngeal injuries - crepitus following fracture/dislocation

- A benign airway injury may lead to subsequent complete obstruction with progression of oedema and haemorrhage.
- Diagnosis is confirmed by direct or flexible largyngoscopy and tracheoscopy.
- These manoeuvers may lead to complete airway obstruction - able of performing an emergent intubation or a surgical airway as necessary.

EPIGLOTTIS



Vascular Injury

- Uncontrollable haemorrhage
- Expanding haematomas
- In shock

- Large-bore venous access should be gained and blood sent for rapid cross-match.
- A CXR - ?intra-thoracic penetration.

ZONE I

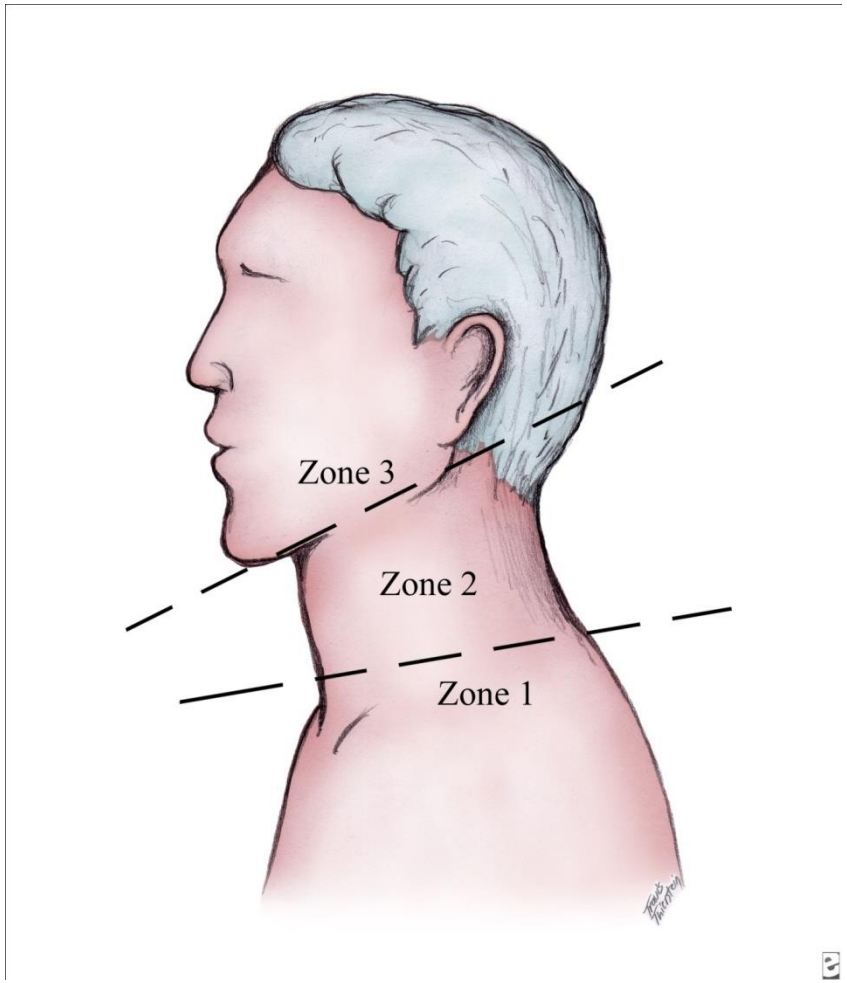
- clavicles to the cricoid cartilage.

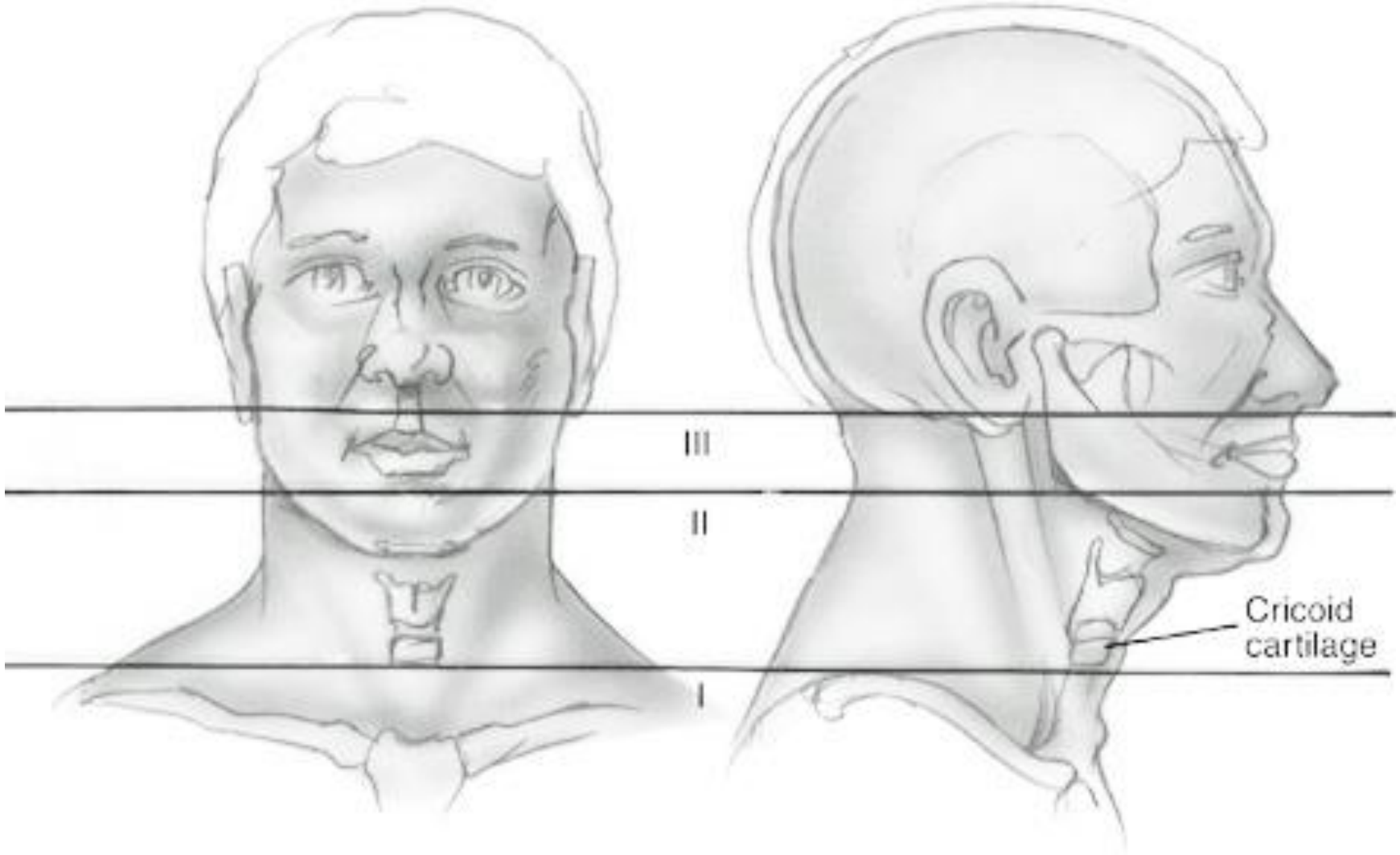
ZONE II

- cricoid cartilage to the angle of the mandible.

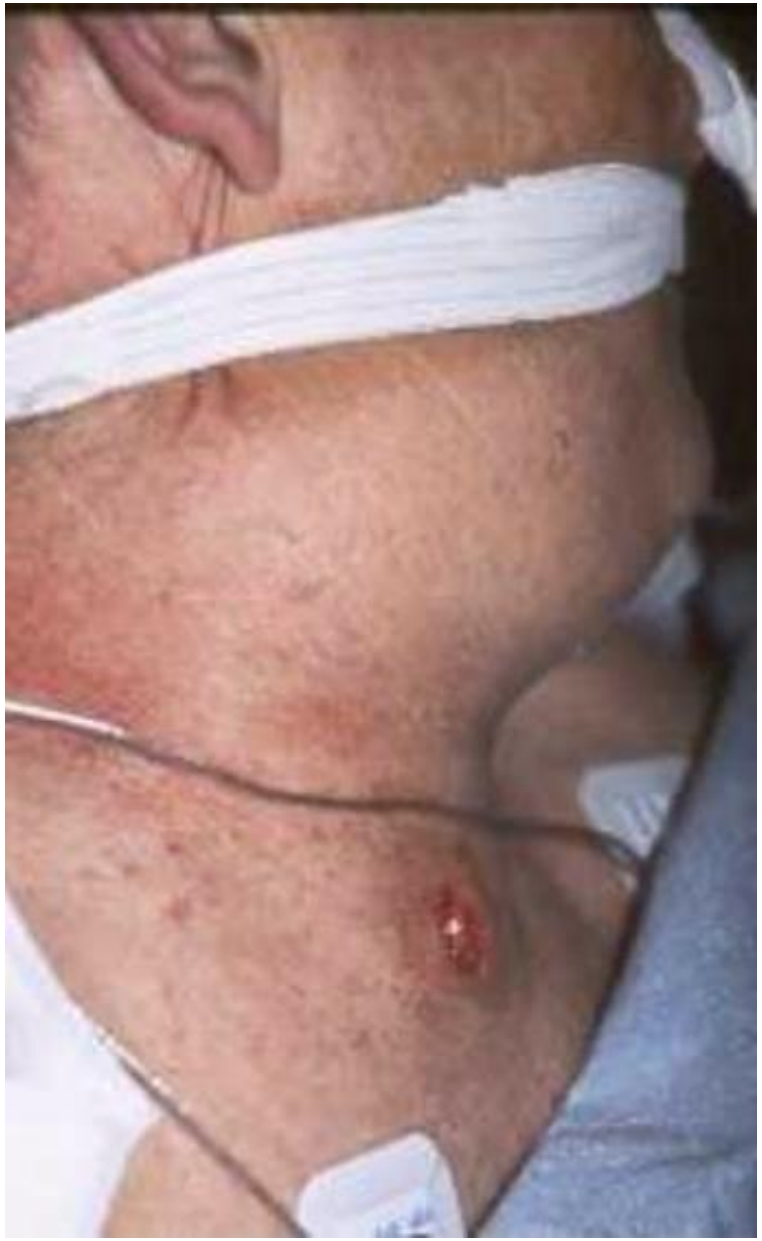
ZONE III

angle of the mandible to the mastoid process.

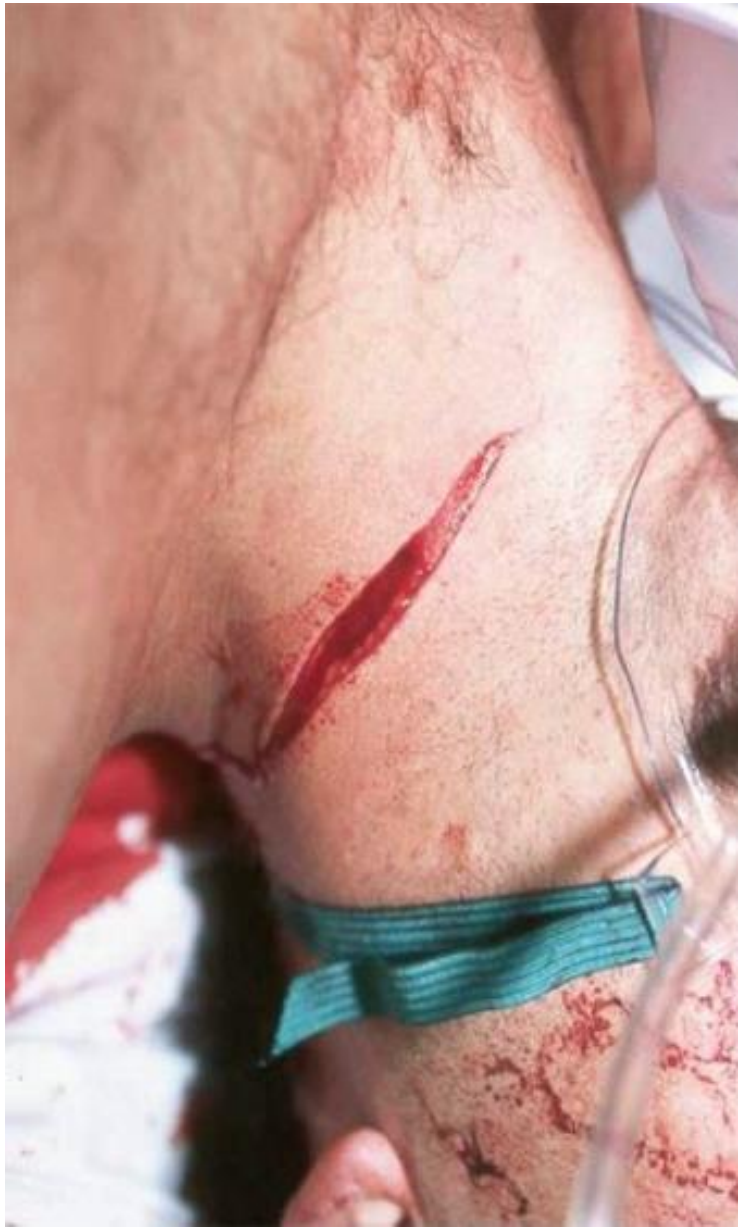



III
II
I
Cricoid
cartilage

ZONE I



ZONE II



ZONE III

ZONE I	ZONE II	ZONE III
•VESSELS •Carotid, Subclavian, Vertebral artery •Internal + External + •Anterior jugular + Subclavian vein	•Common, Internal & External carotid artery •Vertebral artery •Internal + External + Anterior jugular vein	•Internal carotid artery •Internal + External jugular vein
Trachea Dome of the lungs	Pharynx	Oropharynx
Cervical trunk Brachial plexus	Spinal cord Recurrent laryngeal nerve	Spinal cord
Oesophagus		
Thyroid gland Thoracic duct		Submandibular gland
Angiography first unless haemo- dynamically stable		Angiography first unless haemo- dynamically stable
Clavicles; first and second ribs; and hyoid bone are at risk		

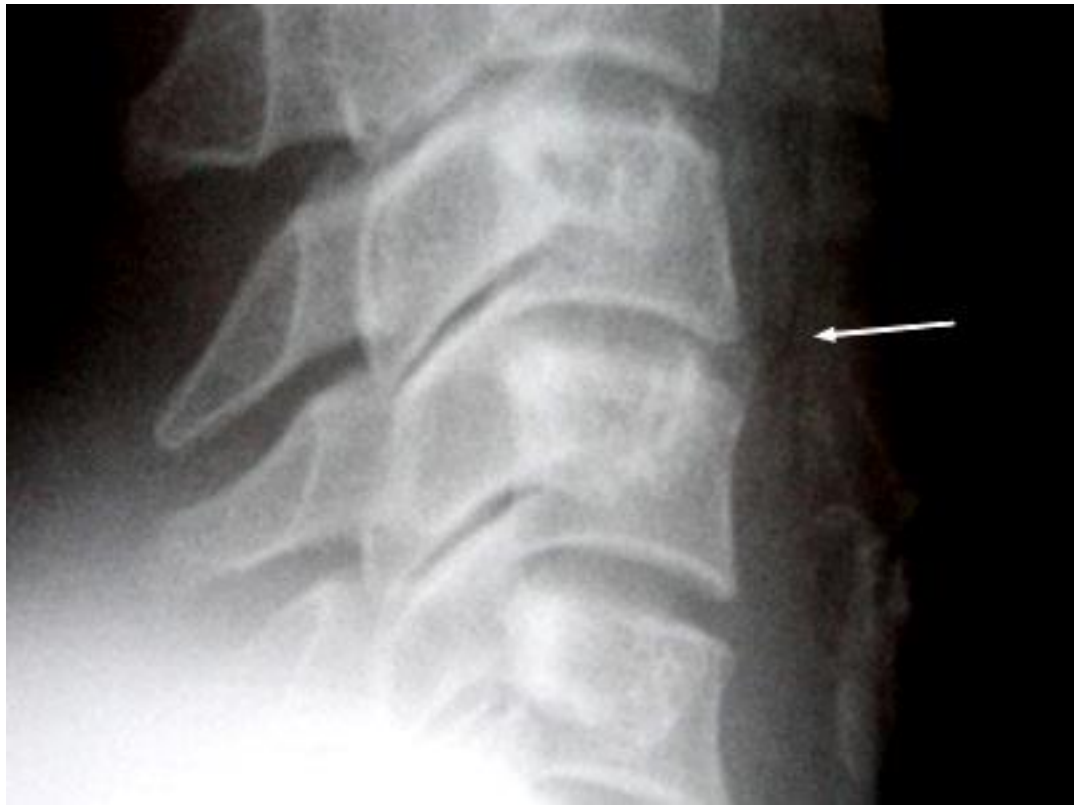
Vertebral Artery

- Vertebral artery injuries are more difficult to diagnose and treat.
- Stab wounds to the posterior neck that are bleeding extensively are likely to involve the vertebral artery.
- There may be evidence of a hemi-cord (Brown-Sequard) lesion on neurological examination.
- Exsanguination is usually best controlled by angiography and embolisation, although back bleeding from the basilar artery may continue.
- Operative control of the vertebral artery as it courses through the vertebral foramina can be exceedingly difficult.

Oesophageal Injury

- Oesophageal and pharyngeal injuries may be difficult to diagnose, but the morbidity and mortality of missed oesophageal injuries is high.
- Gunshot wound traversing the midline.
- Patients may complain of pain on swallowing (odynophagia) or haemoptysis/haematemesis.
- A lateral neck radiograph should be obtained, which may show prevertebral soft tissue swelling or subcutaneous emphysema.

- The presence of subcutaneous emphysema, in the absence of a pneumothorax, is an indication for surgical exploration.
- Otherwise, further investigation will be necessary when there is suspicion of a pharyneal or oesophageal injury.
- Oesophagoscopy and gastrograffin swallow are both employed. Each modality alone has a sensitivity of around 80-90%, while combined they have a sensitivity of approximatly 95%.
- Patients without physical or radiographic signs of oesophageal injury may be observed in a critical care area.



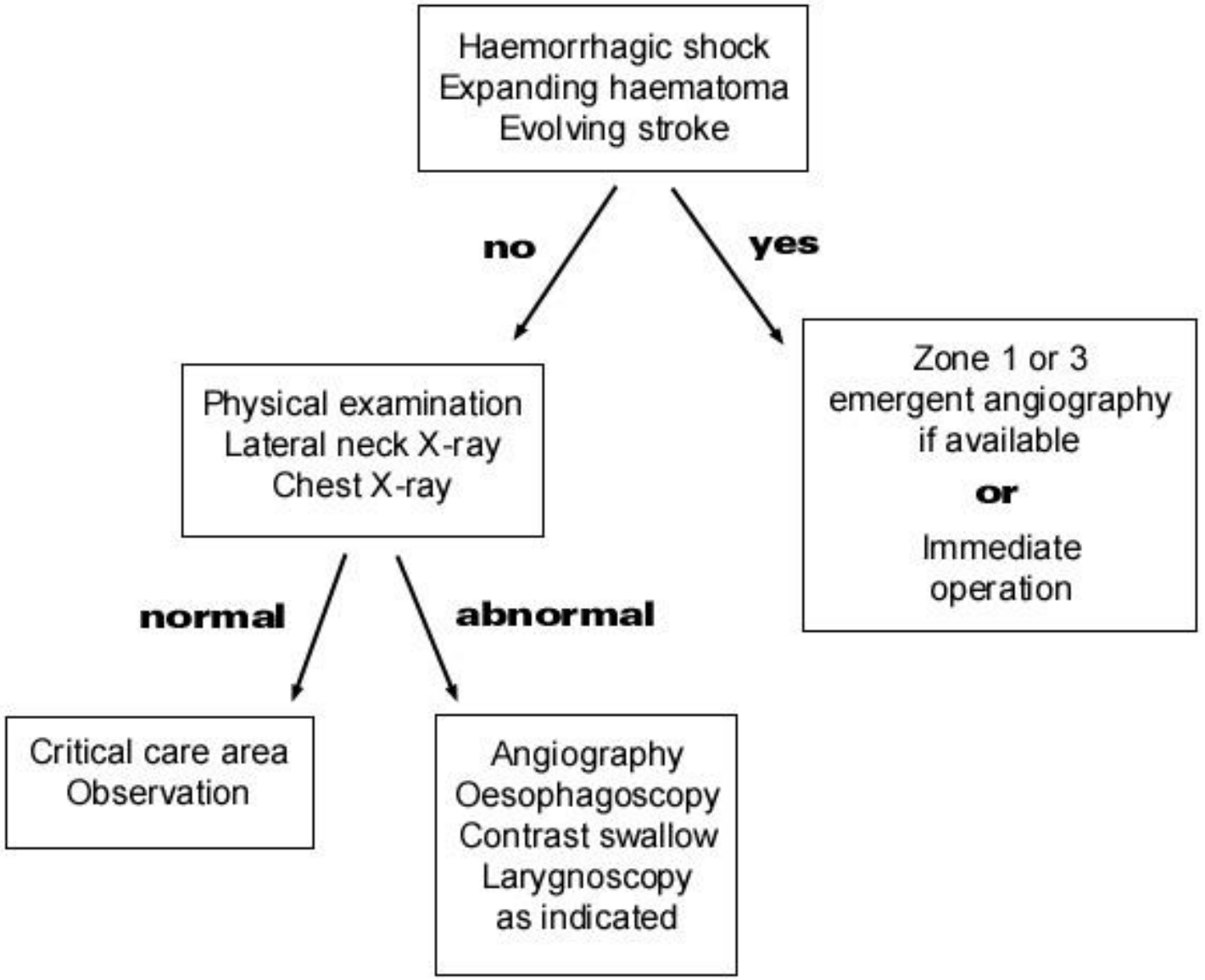
- **Brachial plexus**
- **Cranial nerves**
  - The lower branches of the facial nerve and
  - the hypoglossal nerve are at risk in upper cervical lesions.
  - Injury to the vagus is often only found at operation, but may manifest as hoarseness due to recurrent laryngeal nerve involvement.

Management of Penetrating Neck Injury

- Non-operative management
- Labour intensive and require close nursing and medical observations in a critical care environment.
- Angiography and endoscopy
- Where unavailable- surgical exploration to exclude significant injury.

- Physical examination
- Ct scan, angiography, endoscopy and contrast radiology.
- CT- Evaluating the unconscious patient, and in stable gunshot wounds of the neck to assess the path of the bullet track.

Penetrating Neck Injury



Penetrating trauma

- Mostly from guns and knives
- RTA, household injuries, industrial accidents, and sporting events.
- High-velocity bullet wounds (>2000-2500 ft/s) tend to follow a direct and predictable pathway, while low-velocity bullets travel a more erratic pathway, often demonstrating no direct relationship to the entrance or exit wounds
- A lateral transcervical GSW is more likely to cause a grave injury than a GSW involving injury to only one side of the neck.
- Close-range GSWs of the neck that produce massive destruction are usually fatal.
- After a GSW to the neck, surgery is indicated in 75% of cases, whereas only 50% of neck stab wounds require surgical exploration.

- Vascular injuries arising from penetrating trauma may occur directly, or inducing formation of an intimal flap, arteriovenous fistula, or pseudoaneurysm.
- Injury to the blood vessels can also result from external compression or mural contusion.
- Thrombosis is the most common complication of blood vessel injury, occurring in 25-40% of patients.
- The internal jugular vein (9%) and carotid artery (7%) are the most common sites of vascular injuries.

- Injury to the pharynx or the esophagus occurs in 5-15% of cases.
- The larynx or the trachea is injured in 4-12% of cases.
- Major nerve injury occurs in 3-8% of patients sustaining penetrating neck trauma.
- Spinal cord injury occurs infrequently and almost always results from direct injury rather than secondary osseous instability.

Blunt trauma

- RTA, sports-related injuries (eg, clothesline tackle), strangulation, blows from the fists or feet, and excessive manipulation (ie, any manual operation such as chiropractic treatment or physical realignment or repositioning of the spine).
- Basilar skull fractures may disrupt the intrapetrous portion of the carotid artery.
- Impact to the exposed anterior aspect of the neck may crush the larynx or the trachea, particularly at the cricoid ring,

- Strangulation may result from
  - hanging (partial or complete suspension of the body from the neck),
  - ligature suffocation,
  - manual choking, and
  - postural asphyxiation (eg, seen in children when the neck is placed over an object and the body weight produces compression).
- Significant cervical spine and spinal cord damage- a fall from a distance greater than the body height.
- Zone I injuries are associated with the highest morbidity and mortality rates.

- **Laboratory Studies**
- Baselines, blood type and crossmatching).
- CBC count and blood typing suffice in a previously healthy individual
- Coagulation profiles when indicated.
- **Imaging Studies**
- **Xray**
- (AP) and lateral films may help in localizing emphysema, fractures, displacement of the trachea, and presence of a foreign body (eg, missile fragments).
- **CXR**
- Any finding suggestive of a zone I wound or damage to the thoracic cavity- hemothorax, pneumothorax, widened mediastinum, mediastinal emphysema, apical pleural hematoma, and foreign bodies.

Supplementary tests

- CT
- MRI,
- color flow Doppler studies,
- contrast studies of the esophagus, (Gastrografin study)
- interventional angiography, and
- endoscopic images.
- CT - injuries of the larynx readily identifiable

Angiography

- **Stable** patients with penetrating wounds to zones I and III that pierce the platysma.
- 4-vessel study is a prerequisite.
- therapeutic embolization or occlusion of the harmed vasculature remains a better option in some
- dislodge a clot (eg, causing a stroke) or chance causing or exacerbating intimal damage, and even risk inauspicious perforation of the blood vessel.

Airway

- Intubating a patient with penetrating neck trauma may incite gagging or coughing, potentially dislodging a clot and setting off massive bleeding from a previously injured blood vessel.
- Existent bleeding and edema rapidly distort the surrounding anatomy, making oral intubation difficult, if not impossible.
- Acceptable suction apparatus and having multiple-sized endotracheal tubes
- supplies necessary to perform the surgical airway procedure close at hand.
- modified jaw thrust. Never do a head-tilt chin-lift maneuver in a patient with a suspected cervical spine injury.

- **Breathing**
- Signs or symptoms of respiratory embarrassment compel consideration for a hemothorax or a pneumothorax.
- Zone I injuries may breach the chest cavity.
- Ventilatory distress that persists beyond competent intubation indicates a possible tension pneumothorax- needle decompression and chest tube placement.
- ?Occlusion of the tracheobronchial tree, whether due to a foreign body or iatrogenic

- **Circulation**
- direct pressure
- **Do not** blindly clamp a transected vessel
- Never probe, cannulate, or locally explore these wounds in the ED - air embolus or dislodge a clot and provoke bleeding.
- Do not remove objects protruding from the neck in the ED.
- Establish intravenous access with at least 2 large-bore catheters (14 or 16 gauge).
- Uncontrolled bleeding - balloon tamponade.

- **Disability**
- Cervical spine injury
- phrenic, recurrent laryngeal, and lower lying cranial nerves, as well as the brachial plexus.
- carotid or vertebral artery -CNS ischemia.

MANAGEMENT OF VASCULAR PENETRATION

- Zone I vascular perforation requires thoracic surgery.
- Zone III injuries at the skull base can be temporarily stabilized by pressure, but once delineated, access to the injury via surgery
- All veins in the neck can be safely ligated to control hemorrhage.
- However, if both internal jugular veins are interrupted by the injury, an attempt to repair one is mandated.
- All external carotid artery injuries are easily managed by suture ligation because collateral circulation is good.
- Common carotid or internal carotid injury in zone II is explored

- Complications of vascular injuries include
  - aneurysm formation, dissecting aneurysm, and arteriovenous fistulas.
- Embolization procedures can help control arterial disruption.
- In areas of difficult vascular access at the skull base, detachable balloons or steel coils can be placed for carotid occlusion.
- Embolized bullets also can be retrieved by angiographic techniques.
- The possible complications of interventional angiography include blood vessel injury, inadvertent balloon detachment, ischemic events, pseudoaneurysm formation, and treatment failure.[13]

DIGESTIVE TRACT

- A negative Gastrografin study should be followed by a barium swallow if suspicion remains high.
- Fever, tachycardia, or widening of the mediastinum on serial chest radiographs requires that repeat endoscopy or neck exploration be considered.
- When an esophageal injury is found early, management involves a two-layer closure with wound irrigation, debridement, and adequate drainage.
- After repair of the mucosal perforation, a muscle flap may be interposed over the esophageal suture line for further protection.
- If an extensive esophageal injury is present, it may necessitate a lateral cervical esophagostomy and definitive repair at a later date.[13]

LARYNGOTRACHEAL INJURY

- Laryngeal mucosal lacerations from penetrating injury should be repaired early (within 24 hours).
- Significant glottic and supraglottic lacerations and displaced cartilage fractures need surgical approximation.
- Endoscopy and CT will differentiate between the patients that need only observation (small laceration, shallow laceration, nondisplaced fracture) and those that require a thyrotomy or open fracture reduction and mucosal approximation.
- A soft laryngeal stent may be needed for badly macerated mucosa.
- Simple tracheal lacerations that do not detach a tracheal ring or encroach on the airway can be repaired without a tracheostomy.
- More severe disruptions (gunshot wound directly to the trachea) imply more soft-tissue injury and a 6-week tracheostomy either below or through the tracheal injury is the safest procedure.

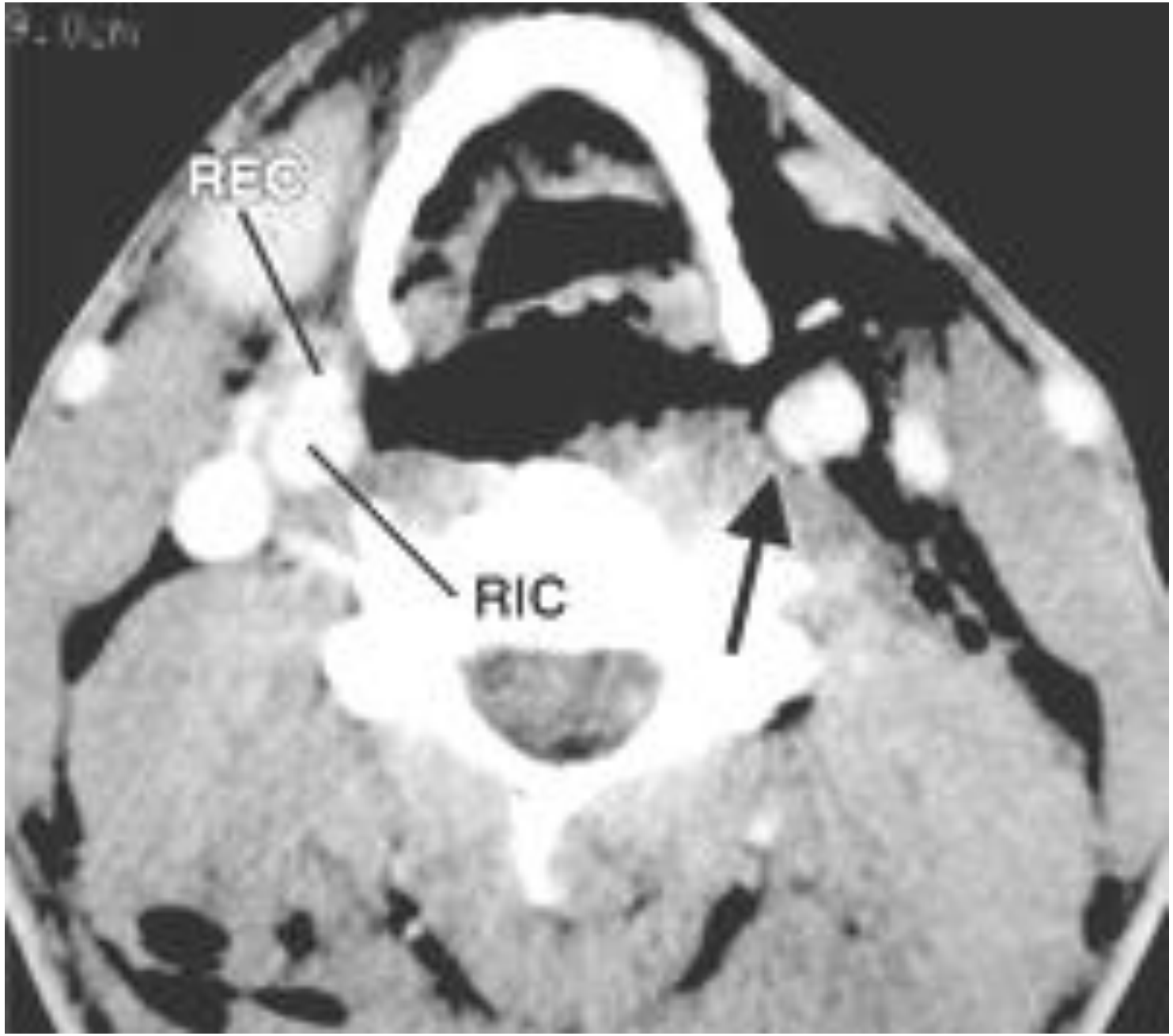
drugs

- Prophylactic antibiotics- adequate tissue levels immediately, preferably within 4 hours of injury.
- Corticosteroids- improving neurological function

Prognosis

- As a general rule, zone I injuries have the worst prognosis.
- Zone II injuries are the most prevalent penetrating neck wounds (accessibility= best prognosis.
- Zone III –secluded critical structures
- Complete disruption of the spinal cord above C4 is frequently fatal.
- Vascular injuries arising from blunt trauma are associated with a poor outcome.

- The prognosis is poor when severe neurological deficits (eg, hemiparesis, coma) occur subsequent to carotid artery damage.
- Identify digestive tract injuries early
- Strangulation patients who present in cardiac arrest have a dismal prognosis
- If the Glasgow score is greater than 8, the chances are good that the patient with a choking or strangulation injury will eventually be discharged neurologically intact.
- Overall, the present mortality rate for civilian wounds secondary to penetrating neck trauma is 2-6%. However, injury to a major blood vessel results in fatality almost 65%.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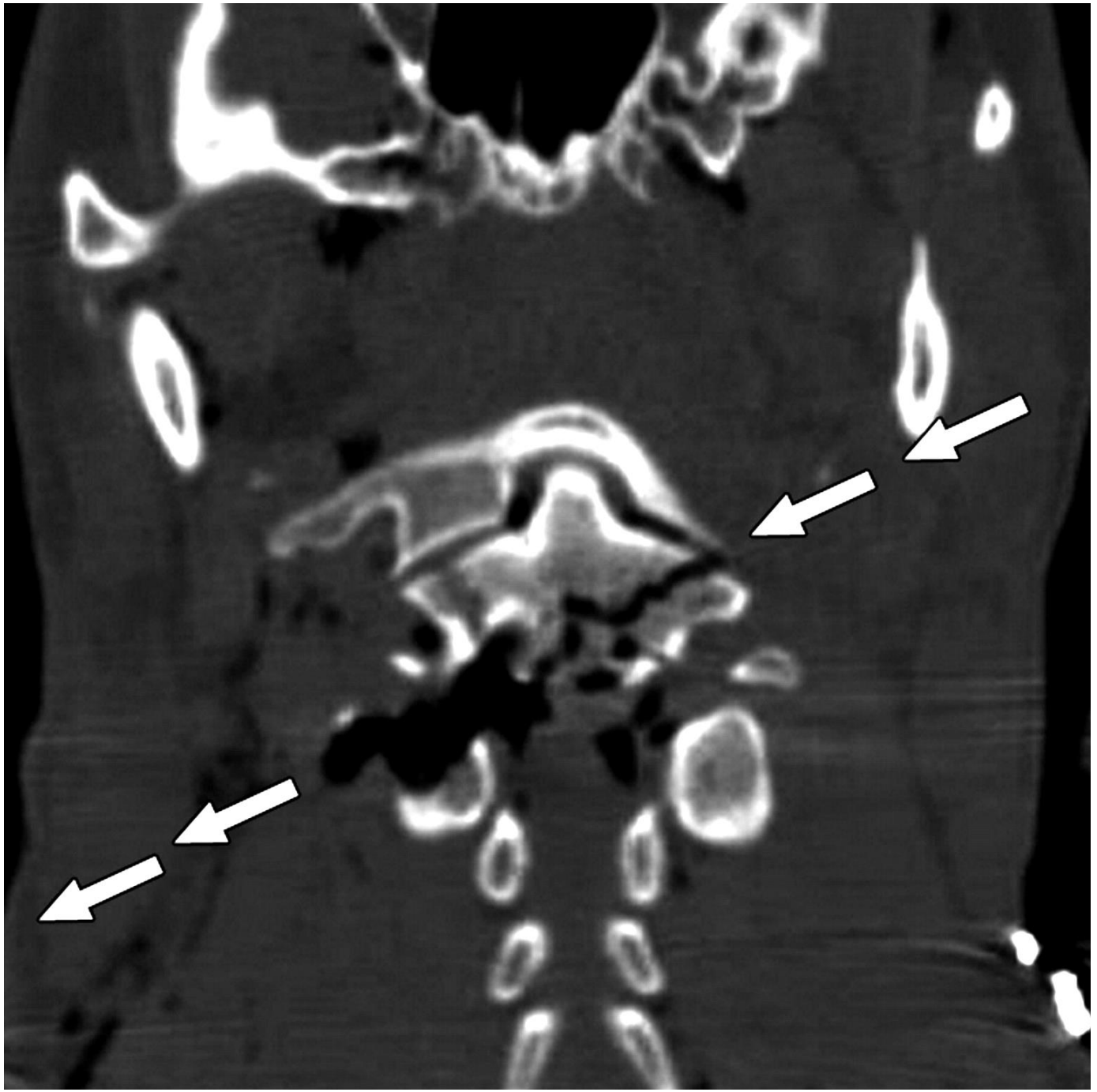
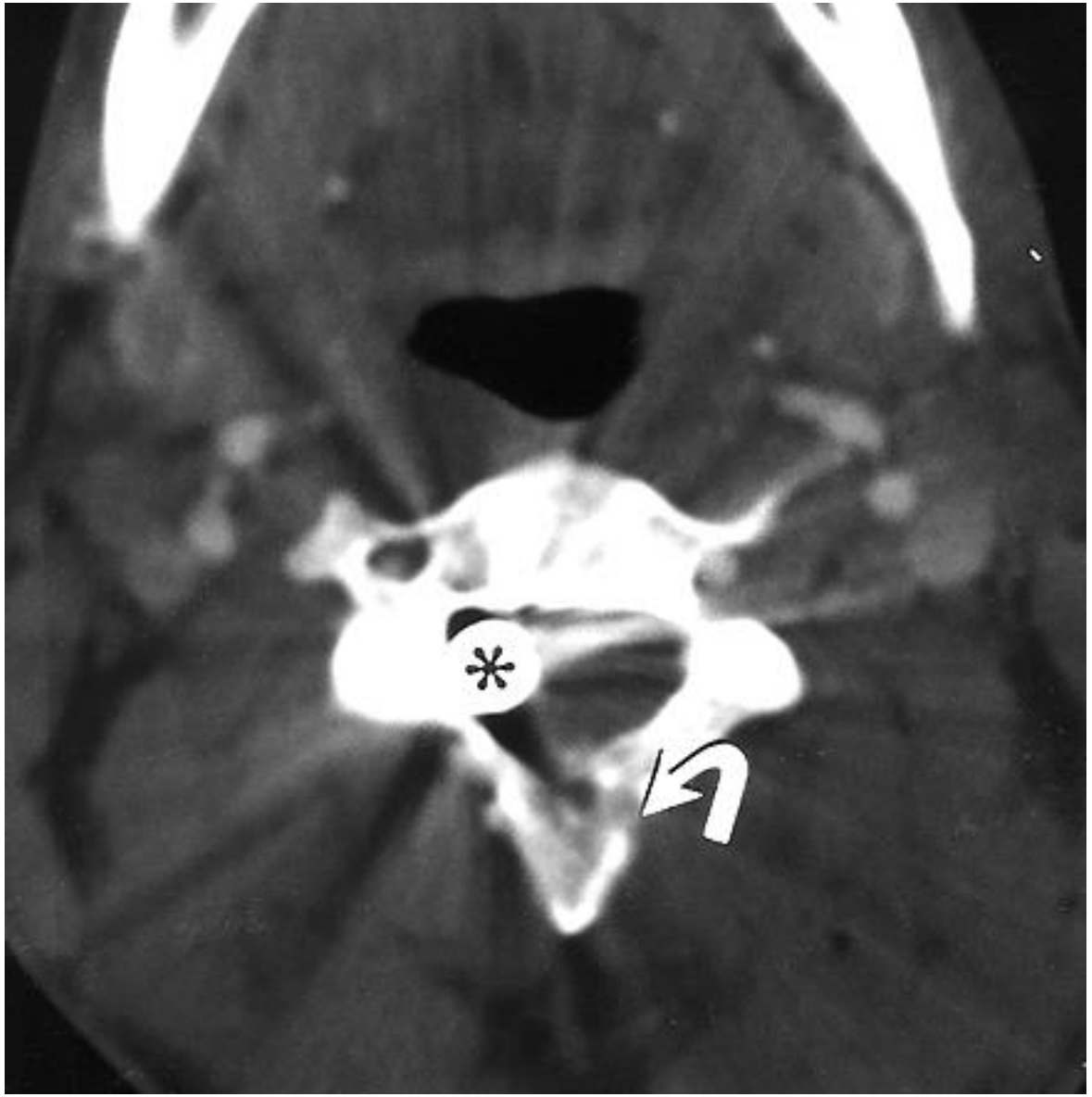
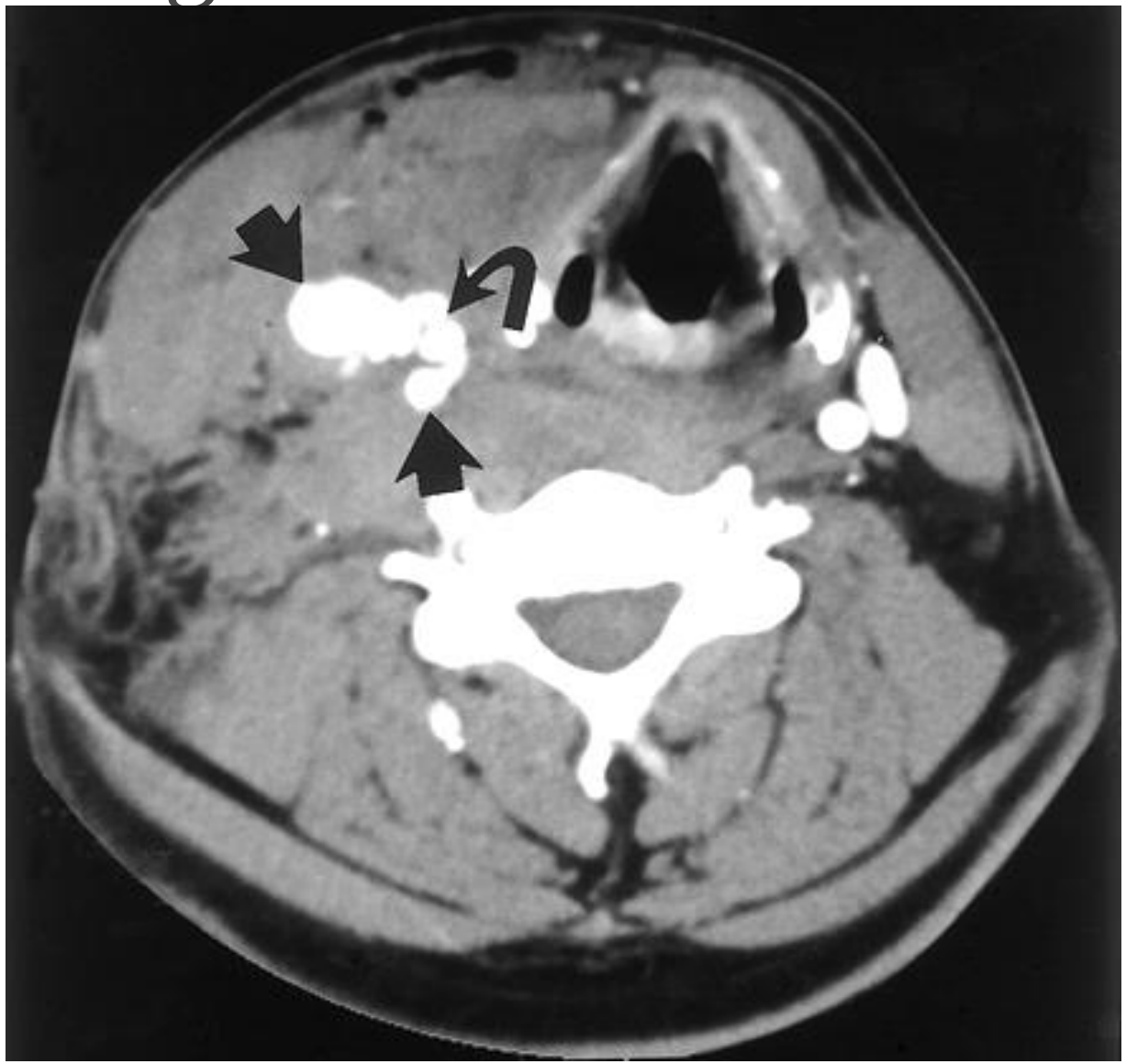


BULLET IN SPINAL COLUMN



Leak from right carotid



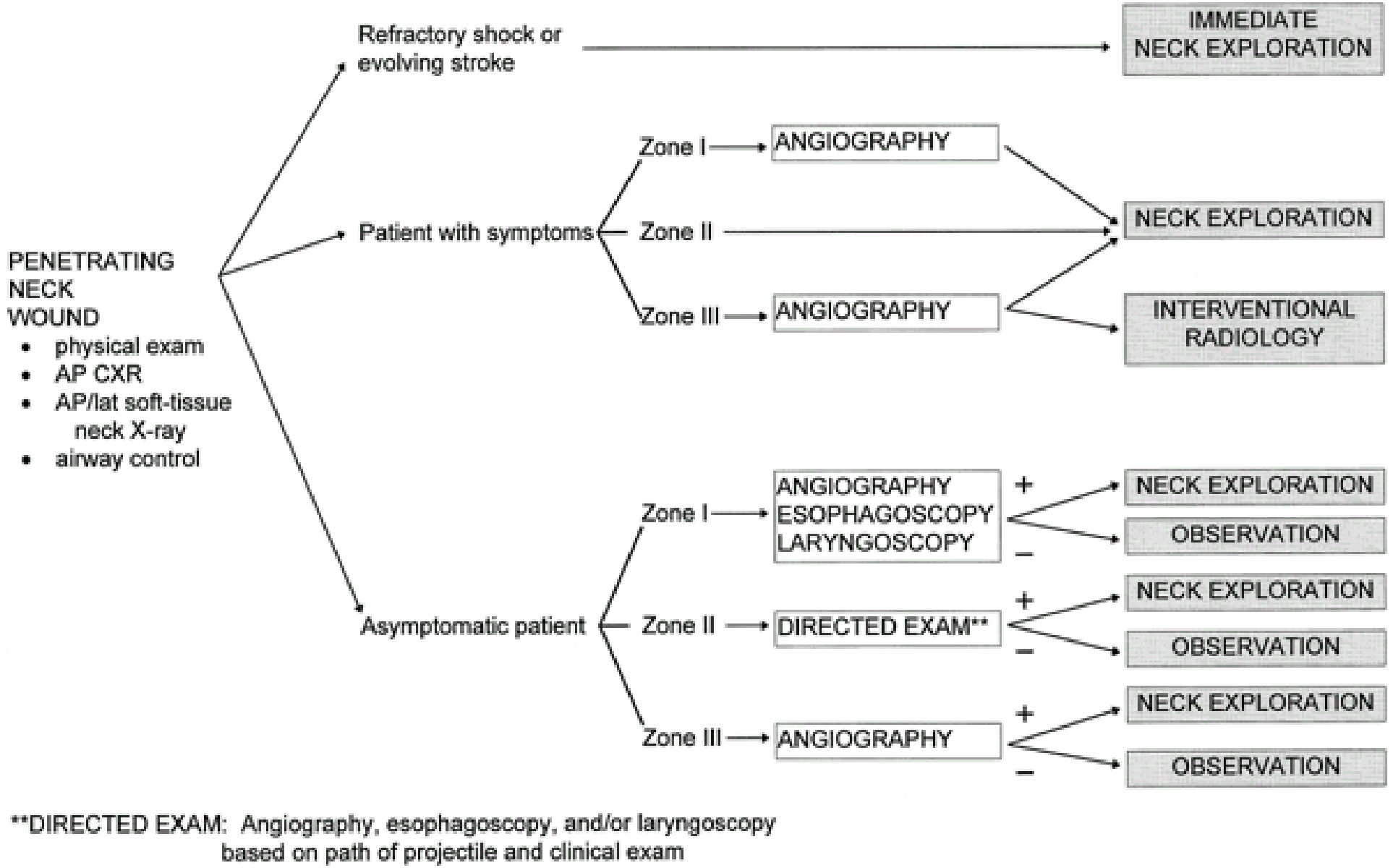


FIG. 73-9. Algorithm for the initial management of patients with penetrating injuries to the neck. (Modified from ref. 14, with permission.)

VERTIGO

SERAH NDEGWA

LECTURER DEPT SURGERY- ENT THEMATIC UNIT

11TH APRIL 2019

OUTLINE

- INTRODUCTION
- EPIDEMIOLOGY
- ANATOMY AND PHYSIOLOGY
- HISTORY
- EXAMINATION
- INVESTIGATIONS
- TREATMENT
- TAKE HOME MESSAGE
- REFERENCES

INTRODUCTION

- Vertigo is the sense of false motion which can be rotating, shifting or tilting (1,2).
- It is the commonest type of dizziness.
- Others include: Presyncope, lightheadeness and dysequilibrium.(3)

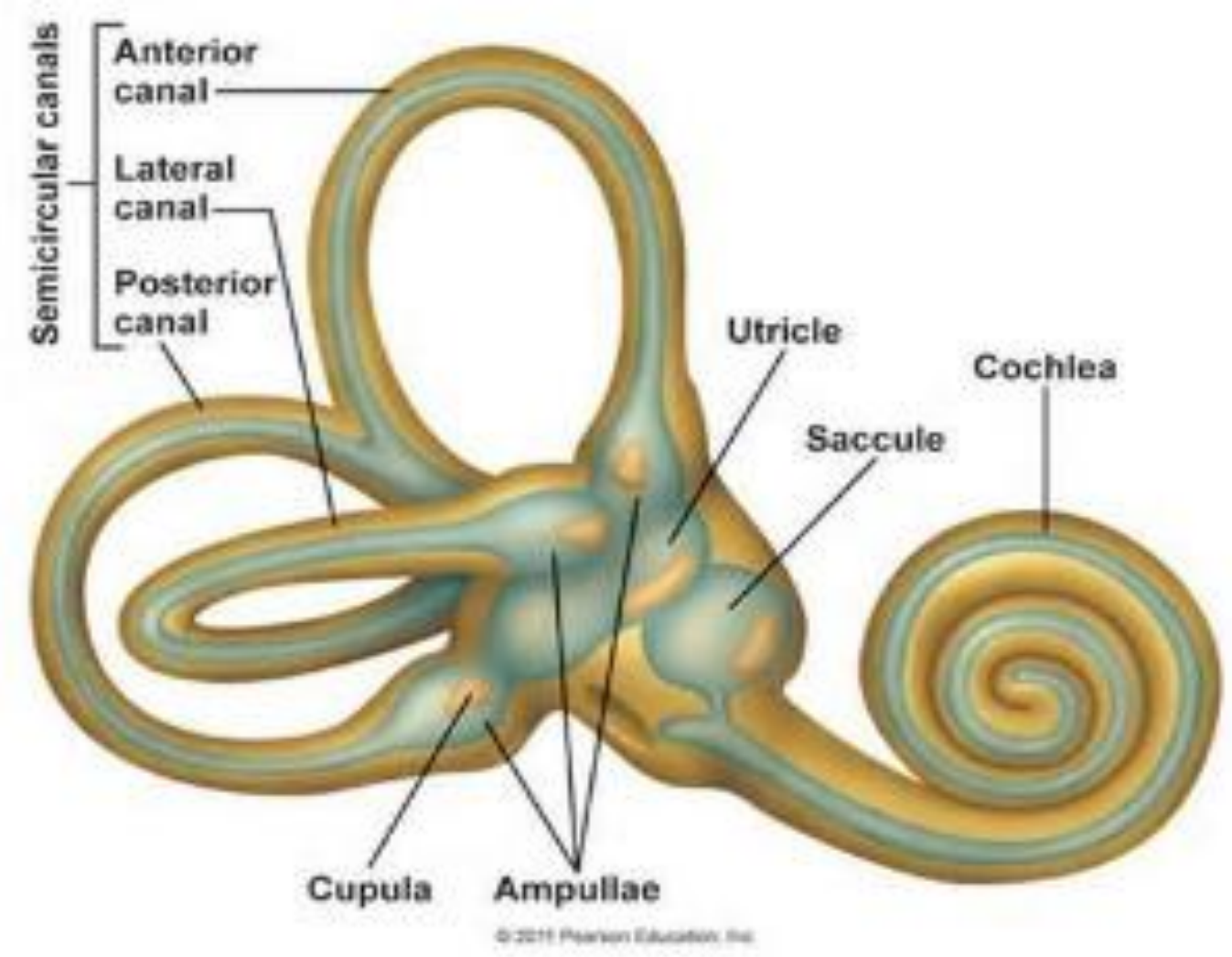
1.Labuguen RH. Am Fam Physician. 2006 Jan 15;73(2):244-51. Review
2. Glasscock .M, Gulia. A , Surgery of the ear, 5th Edition, 2003; page 79, Ontario
3. Erratum in: Am Fam Physician. 2006 May 15;73(10):1704.PMID: 16445269

EPIDEMIOLOGY

- Vertigo is a frequent symptom in the general population.(4)
- Prevalence rises with age.
- M:F, about 3x more in females
- accounts for 54 percent of reports of dizziness in primary care.(5)

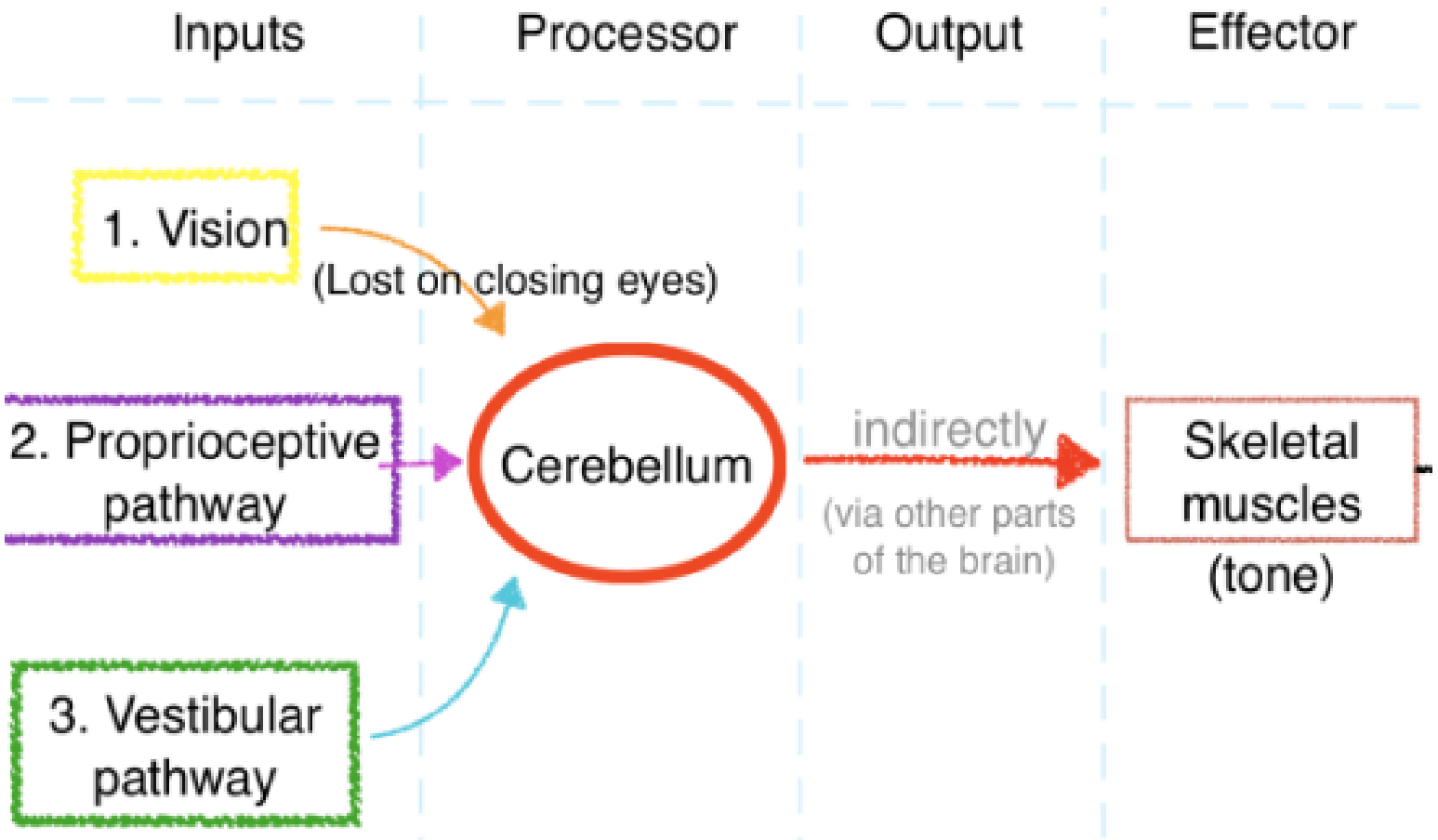
4. Neuhauser HK.Curr Opin Neurol. 2007 Feb;20(1):40-6. Review
5. Kroenke K, Lucas CA, Rosenberg ML, Scherokman B, Herbers JE Jr, Wehrle PA, et al. Causes of persistent dizziness. A prospective study of 100 patients in ambulatory care. *Ann Intern Med*. 1992;117:898–904.

Anatomy



From hearing and balance centre of Austin

Physiology


Inputs
Processor
Output
Effector
1. Vision
(Lost on closing eyes)
2. Proprioceptive pathway
3. Vestibular pathway
Cerebellum
indirectly
(via other parts of the brain)
Skeletal muscles
(tone)

Balance Control

- ✓ Balance control requires: visual, vestibular and proprioreceptive inputs.
- ✓ The primary function of the vestibular system is to provide information about our orientation in space
- ✓ **Vestibulocular Reflex**
- ✓ **Vestibulospinal Reflex**

HISTORY

1. Establish whether it is vertigo
2. Characteristics of the vertigo
3. Associated symptoms: hearing loss, tinnitus, aura, headache, aural fullness, visual disturbance
4. Precipitating, aggravating and relieving factors e.g.change in position of head/body, tullio phenonemon
5. Past Medical History: Hypertension, Diabetes, Migraines, Neurological diseases, Vascular disease
6. Drug History; cisplatin and aminoglycosides
7. Previous ear surgery
8. H/o Trauma
9. Family and social Hx

1. Dizziness evaluation

VERTIGO	PRESYNCOPE	DISEQUILIBRIUM	LIGHTHEAADEDNESS
False sensation of motion, possibly spinning sensation	Feeling of losing consciousness or blacking out	Off- balance/wobbly	Vague symptoms, possibly feeling disconnected with the environment
45 to 54%	Up to 16%	Up to 14%	10%

"When you have dizzy spells, do you feel light-headed or do you see the world spin around you?" An affirmative answer to the latter part of this question has been shown to accurately detect patients with true vertigo.

9. Kroenke K, Lucas CA, Rosenberg ML, et al. Causes of persistent dizziness. *Ann Intern Med*. 1992;117(11):898–904.

Description of the vertigo

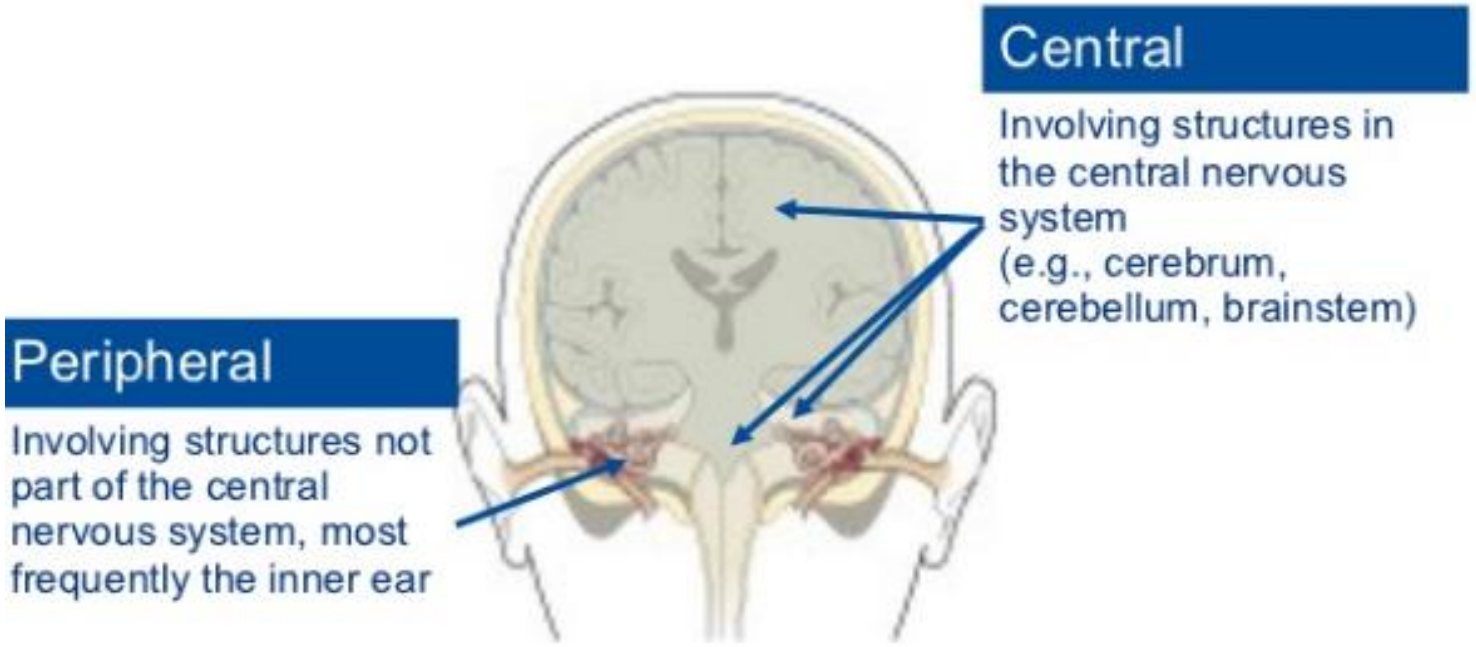
- Tilt illusion
- Drop attack
- Spatial disorientation
- Oscillopsia

Joseph M Furman, Jason JS Barton ; Assessmet of a patient with vertigo, Topic 5094 Version 9.0:;Uptodate Online

- **Other symptoms of vestibular dysfunction** — Patients with vestibular injury may not complain of vertigo. In fact, vertigo is unusual in chronic vestibular injury or acute, bilaterally symmetric vestibular injury. Other symptoms of vestibular injury may include:
- **Tilt illusion** — A tilt illusion in which patients feel that they and their environment are tilted with respect to gravity, even to the point of being upside down, usually reflects damage to otolithic organs (utricle and saccule) or their central connections. Otolith dysfunction may also cause lateropulsion or the tendency to fall to the side of the lesion.
- **Drop attacks** — Drop attacks are attributed to a sudden loss of tone mediated by vestibulospinal reflexes. Unlike presyncope and seizures, there is no faintness or loss of consciousness with drop attacks. Patients with drop attacks of vestibular origin often have a sensation of being pushed or pulled to the ground [17,18]. Drop attacks are an unusual feature of Meniere disease. Sometimes called Tumarkins' otolithic crises, this symptom is usually seen only in advanced cases. Superior canal dehiscence and aminoglycoside toxicity are also associated with this presentation [19,20].

- **Spatial disorientation** — A fleeting spatial disorientation with rapid head turns often remains after the patient has recovered from an acute attack of vertigo. The most astute patients will observe that this is more pronounced with head turns to the side of the lesion.
- **Oscillopsia** — Oscillopsia, a visual illusion of to-and-fro environmental motion, and blurred vision whenever the head is in motion is a manifestation of an impaired vestibuloocular reflex. Affected patients notice that everything jiggles when they are walking or driving on rough pavement; they often have to stop and stand still to read signs.
- **Impaired balance without vertigo** — This is a common manifestation of acute simultaneous bilateral vestibular loss such as that occurring with aminoglycoside antibiotic toxicity. Vertigo does not occur because there is no marked vestibular asymmetry. Symptoms are most marked in the dark when visual cues to position in space are not available. Aminoglycoside toxicity is the most common identified etiology of bilateral vestibulopathy, followed by Meniere disease and meningitis [21]. Most cases, however, are cryptogenic in origin. This presentation is also characteristic of acute midline cerebellar lesions or thiamine deficiency.

Peripheral vs Central Vertigo ?



BalohRW, Lancet996;352:1541-6. Mukherjee A et al. JAPI 2003;51:1095-101

2. Characteristics

i) Duration

Duration Of Episode	Differential Diagnosis
A few seconds	Peripheral cause: unilateral loss of vestibular function; late stages of acute vestibular neuritis; late stages of Meniere's disease
Several seconds to a few minutes	BPPV; perilymphatic fistula
Several minutes to one hour	Posterior transient ischemic attack; perilymphatic fistula
Hours	Meniere's disease, perilymphatic fistula, migraine
Days	Stroke, migraine, Multiple sclerosis, Labyrinthitis
Weeks	Psychogenic, Acoustic Neuroma,

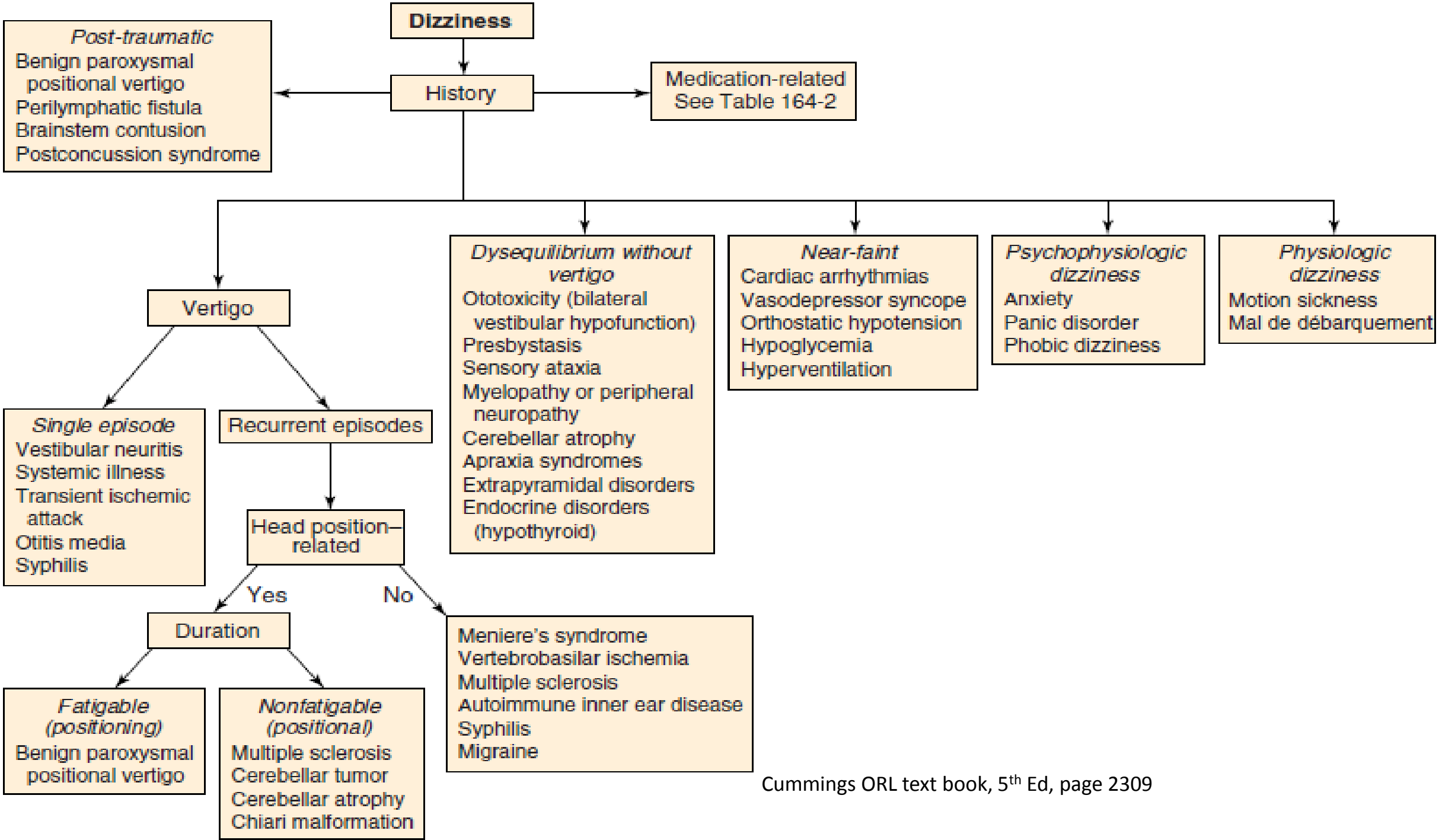
3. Associated symptoms

Symptom	Diiferential Diagnosis
Hearing loss	Meniere's, superior canal dehiscence
Tinnitus	Peripheral / central cause
Aura	Central ; migraine
Headache	Migraine
Aural fullness	Meniere's
Visual disturbance	Oscillopsia
Nausea and Vomiting	Peripheral cause

4. Aggravating/ Relieving factors

Provoking factor	Differential Diagnosis
Change in head position	BPPV; acute Labyrinthitis
Recent upper respiratory viral illness	Acute vestibular neuronitis
Mental Stress	Migraine, psychological
Immunosuppression	Herpes Zoster oticus
Changes in ear pressure, head trauma, excessive straining, loud noises **(Tullio phenomenon)**	BPPV,Perilymphatic fistula, Superior canal dehiscence

Kaski D, Seemungal BM; Bedside assessment of Vertigo.doi: 10.7861/clinmedicine.10-4-402Clin Med August 1, 2010 vol. 10 no. 4402-405



Cummings ORL text book, 5th Ed, page 2309

Physical Examination

Be orderly and systematic!

Standing

1. Gait
2. Romberg test
3. Fukuda step test
4. Tandem gait walk

Sitting

5. General Physical Examination

Vitals: BP**(including orthostatic**) in both arms,

6. Head and neck, Ear, nose Throat

Ear: scars, otoscopy, fistula test

7. Neurological Examination

Cranial Nerves and Cerebellar function

8. Eye examinations

i) Spontaneous nystagmus

ii) Gaze test

iv) Saccades

v) Smooth pursuit

9. Head thrust
10. Head shake
11. dynamic visual acuity
12. Orthostatic BP

On the couch

13. Positional test: Dix hallpike
14. Caloric test

Test Name	Procedure	Interpretation
1. Gait	Assess patient walking	severe gait impairment ;neurologic disorder. Off balance, can walk with assistance: peripheral lesion.
2. Romberg	patient stands with feet together, arms folded, first with eyes open and then with eyes closed	Positive when the patient demonstrates an increased sway or falls when eyes are closed. vestibular lesions: fall or sway to the side of lesion. Cerebella lesions: little enhancement with eye closure.
3. Fukuda step test	Arms extended, marches with eyes closed	vestibular malfunction tend to rotate towards affected side > 45egrees
4. Tandem gait walk	walk in a straight line heel to toe, first with eyes open, then with eyes closed.	Unilateral vestibular failure, sway towards affected side with eyes open. Bilateral vestibular

Cerebellar tests

- **Dysmetria**: inability to measure the distance for reaching intended target

  Test: Finger nose test

  Finger finger test

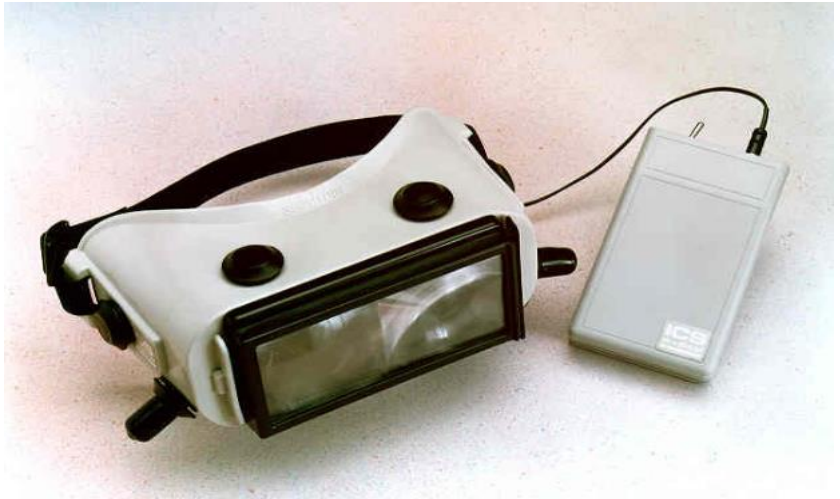
- **Dysdiadokinesi**a: inability to do alternate opposite movements; Supination and pronation
- **Heel shin test**

Eye examination;Good to know!!!

Nystagmus

	Peripheral	Central
Type	Typically horizontal and torsional	Usually vertical, Pendular
Direction	Fast beat away from side of lesion	Fast beat towards side of lesion
Visual fixation	Relieved by gaze fixation	Not relieved by gaze fixation
Associated Cerebellar signs	absent	Present

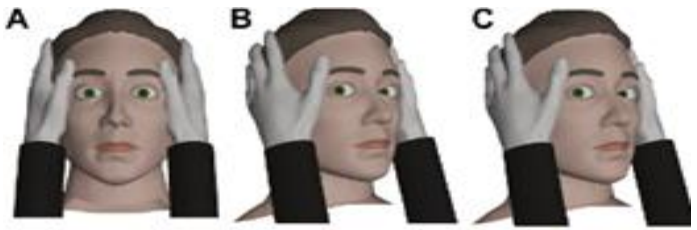
Alexander's Law
Ewald's Law



- Has 3 degress and applies to classical unilateral peripheral vestibular loss
- consist of the combination of magnifying glasses (+20 lenses placed in front of the patient), and a lighting system. When Frenzel's goggles are placed on the patient, and the room lights darkened, nystagmus can easily be seen because the patients eyes are well illuminated and magnified, and because fixation is removed as the patient can hardly focus through magnifying glasses on a dark room.

Examination	Procedure	interpretation
i) Spontaneous Nystagmus	Observe for nystagmus	Peripheral or Central. Spontaneous nystagmus for peripheral lesions beats in the direction of the fast phase
ii) Gaze test	Fix gaze on object	Right lesion has left beating nystagmus (towards the unaffected ear). Gaze to the left makes the nystagmus even worse.
iii) Saccades	Move object rapidly while patient is following it with eyes	Corrective saccades : central lesion
iv) Smooth pursuit	follow target without moving head. Take 4-5s to move from left to right	test results should resemble a smooth sinusoid. Abnormal pursuit + vertigo= central lesion Normal pursuit +vertigo = peripheral

Test	Procedure	Interpretation
8. Head thrust	Thrust the head and observe for nystagmus	Unilateral vestibular deficit causes slow phase nystagmus to the side of lesion
9. Dynamic visual Acuity	Use Snellen Chart; determine baseline VA. Head shake 2Hz and assess VA	Drop of > 2 VA indicates abnormal VOR
10. Orthostatic BP	BP on sitting and standing	Difference of >20mmHg SBP and 10mmHg DBP; Vascular

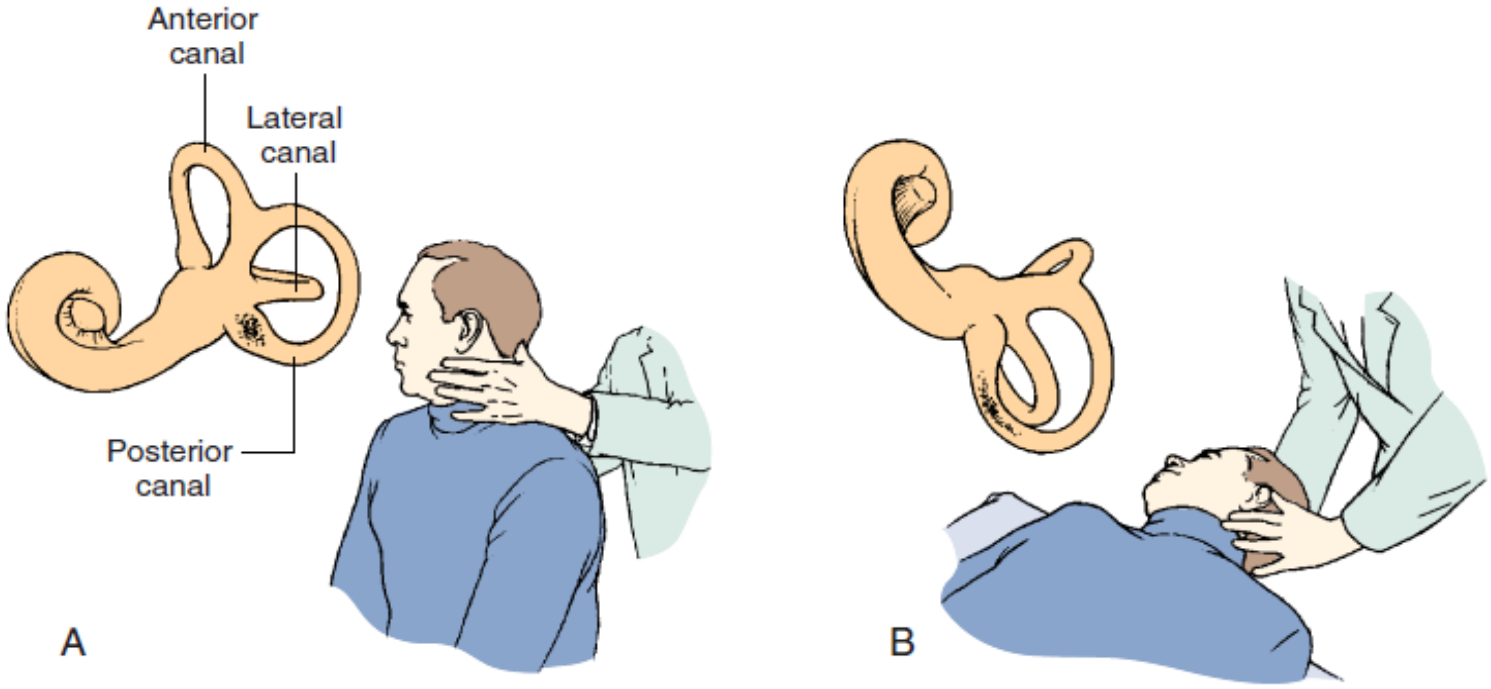


Normal leftward head impulse

Positive leftward head impulse

Positional tests

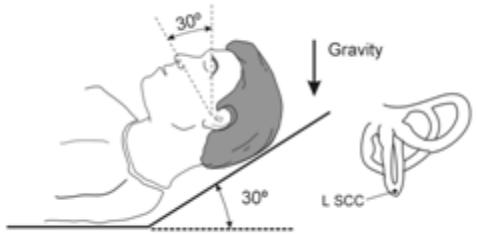
- 1. DIX HALLPIKE- Posterior semi circular canal



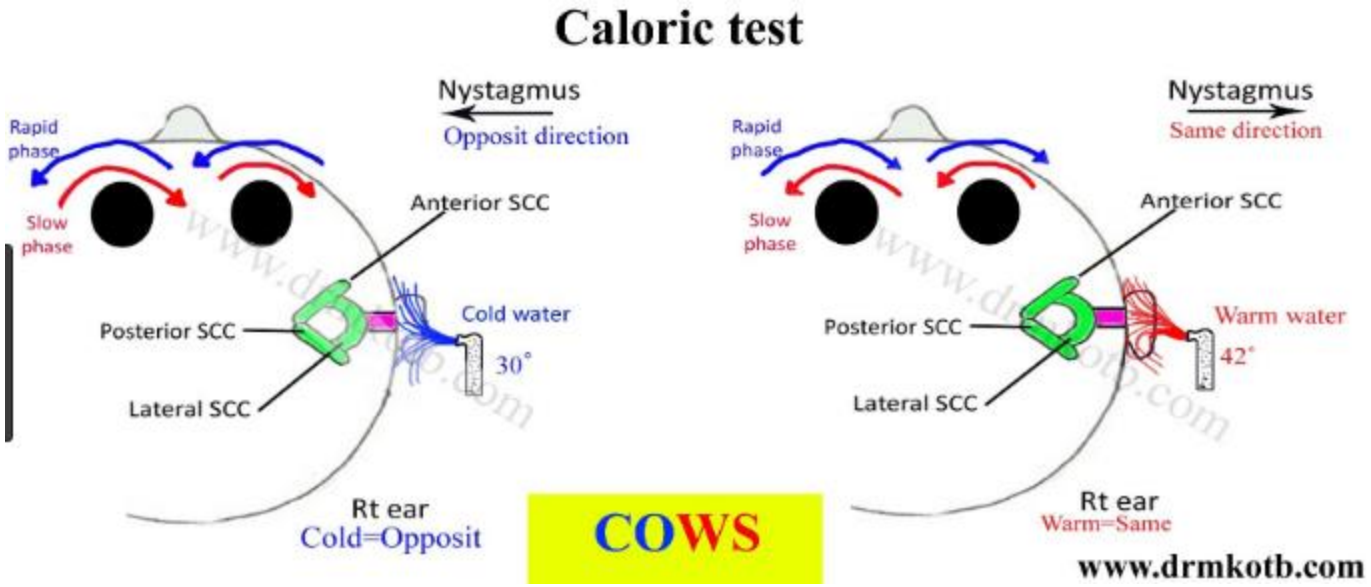
- Supine head roll test for Horizontal scc

Lowering the patient's head backwards and to the side allows debris in the posterior canal (A) to fall to its lowest position, activating the canal and causing eye movements and vertigo (B). (From Hullar TE, Minor LB. Vestibular physiology and disorders of the labyrinth. In: Glasscock ME, Gulya AJ, eds. Surgery of the Ear. 5th ed. Toronto: BC Decker; 2003.)

Caloric test



- warm irrigations (44 degrees C)
- cold irrigations (30 degrees C)
- in the order of left cold, right cold; left war, right warm



Interpretation

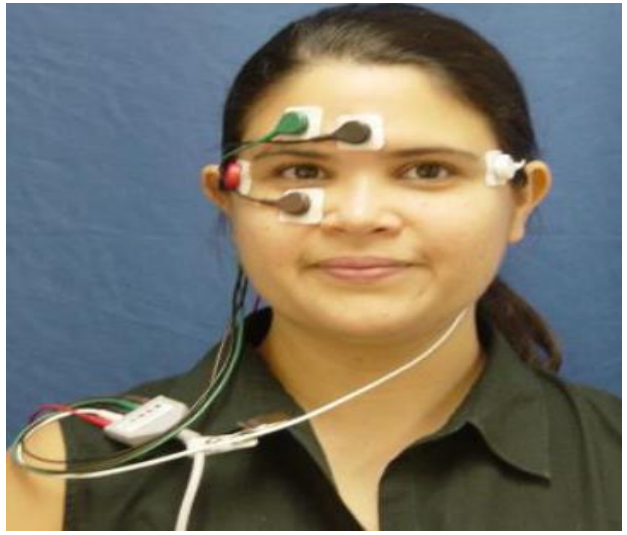
- B/L absence of caloric nystagmus: aminoglycoside toxicity, postmeningitis.
- U/L absence :U/L vestibular schwannoma, vestibular neuritis
- Caloric response decreased on side of lesion
- Perverted nystagmus: central pathology

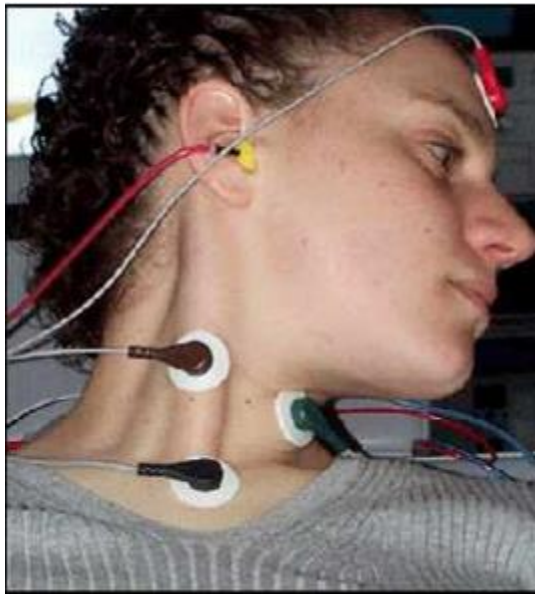
  - UW % > 25% (range 20% - 30 %)
  - DP % > 30% (range 25% - 50%)
- for eg left vestibular neuritis causes right directional preponderance ie right beating nystagmus is stronger than the left beating nystagmus.

Cummings CW et al; Cummings ORL – HNS text book, 5th Ed

Other tests

- Electronystamography
- Videonystamography
- Vestibular Evoked Myogenic Potentials (VEMP)
- Audiogram
- Imaging CT scan/ MRI





Cummings CW et al; Cummings ORL – HNS text book, 5th Ed

Imaging

- Indications:

1. Central Neurologic signs and symptoms
2. Risk factors for Cerebrovascular disease
3. Progressive unilateral hearing loss (14)

14. Hotson JR, Baloh RW. Acute vestibular syndrome. N Engl J Med 1998; 339:680.

- Magnetic resonance or conventional angiography of the posterior fossa vasculature may be useful in diagnosing vascular causes of vertigo such as vertebrobasilar insufficiency, thrombosis of the labyrinthine artery, anterior or posterior inferior cerebellar artery insufficiency, and subclavian steal syndrome.[26]
- Neuroimaging studies can be used to rule out extensive bacterial infections, neoplasms, or developmental abnormalities if other symptoms suggest one of those diagnoses.[26,2]
- (include motor deficits, particularly crossed hemiplegia; dysarthria or dysphagia; inability to walk; bidirectional or vertical nystagmus; and sings of cerebellar dysfunction) (include motor deficits, particularly crossed hemiplegia; dysarthria or dysphagia; inability to walk; bidirectional or vertical nystagmus; and sings of cerebellar dysfunction)

TREATMENT

- TREAT THE CAUSE
- REHABILITATE THE PATIENT

- 1. Cawthorne Cooksey exercises
- 2. Particle repositioning:
  - Epley’s manoeuvre
  - Semont’s Manoeuvre

Take home message

- Good history taking and thorough physical examination is mandatory
- Rule out life-threatening causes
- Differentiate between central and peripheral causes

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TINNITUS

SERAH NDEGWA

LECTURER

DEPT SURGERY- ENT THEMATIC UNIT

11TH APRIL 2019

OUTLINE

- Introduction
- Epidemiology
- Classification
- Etiology
- Pathogenesis
- Diagnosis
- Treatment
- Future and controversies
- References

INTRODUCTION

- *Tinnire* – to ring. Tinnitus - ringing in the ear.
- perception of sound in absence of stimulation /no external acoustic source.
- A Conscious experience of a sound that originates in the head of the owner.
- hissing, sizzling and buzzing, pulsatile
- can be persistent, intermittent
- Can be perceived in one or both ears

EPIDEMIOLOGY

- According to the American Tinnitus association 50M people in the US have chronic tinnitus(>6mon)[1]
- Can occur in children[2]
- prevalence increases with age and with hearing loss[3,4]
- Male > females[1]
- peak age 40-70 years[4]
- Whites > blacks[1]
- Greater risk in smokers and low SES[1]

TINNITUS IN CHILDREN

- Underestimated-no vocabulary to explain,may consider it normal,fear to disclose(withdrawal)
- Look for changes in attention,depression,poor school performance,insomnia.
- 6-13% of children with normal hearing on & off [2]
- 24-29% -with hearing difficulties.
- Causes-inborn, ME infections, wax, deafness, noise, meningitis,asprin ,ET dysfunction.

CLASSIFICATION

1. **OBJECTIVE**:
   - can be percieved by the patient and also by the examiner.
   - Is from somatosounds generated by sources adjascent to or within the ears or by structures that transmit sounds to the ear
   - Can be pulsatile or non pulsatile

2. **SUBJECTIVE**:
   - Perceived only by the patient
   - Can be pulsatile or non pulsatile

ETIOLOGY

OBJECTIVE	SUBJECTIVE
AV malformations	Meniers disease
Aberrant carotid artery	Otosclerosis
Persistent stapedial artery	Acoustic neuroma
Glomus tumours	Noise exposure
Dehiscent jugular bulb	Head trauma
Benign intracranial hypertension	Ototoxic drugs(aminoglycosides, loop diuretics, asprin, antimalarials,cisplatin, vincristine)
Venous hum	Presbycusis
Artherosclerosis	Middle ear effusions
Pagets disease	Menengitis
Palatal myoclonus	TMJ disorders
Tensor tympani/stapedial myoclonus	Syphillis
High output states: anaemia, pregnancy, thyrotoxicosis	Depression

PATHOGENESIS

- Tinnitus can be triggered anywhere along the auditory system
- Majority of patients have sensorineural dysfunction
- On the other hand somatosounds in the proximity of the cochlear can be perceived as tinnitus, usually the somatosounds are of vascular or musculoskeletal origin
- Various theories have been postulated for pathogenesis of tinnitus without somatic origin

PATHOGENESIS

a) Discordant dysfunction theory[5]:

- Tinnitus is thought to originate from damaged or dysfunctional outer hair cell and relatively normal inner hair cells
- Damage to outer hair cells leads to increased neuronal activity of the dorsal cochlear nucleus due to lack of inhibition from type II fibers
- Increased neuronal activity is perceived as tinnitus by higher centres

PATHOGENESIS

b) Neural plasticity (like "phantom pain") [6]
   - Deprevition of auditory input caused by hearing loss and exposure to loud noise causes hyperactivity in the nuclei of the auditory pathway
   - There also is unmasking of dormant synapses

c) Similar neurologic changes have been observed in chronic neuropathic pain and tinnitus sufferers that support proposal for similar mechanisms[7]

d) Auditory seizure[8]:
   - Tinnitus has been likened to an auditory seizure and some antiseizure medications in some patients has been found to have little success

PATHOGENESIS

e) Psychological disorders[9] :

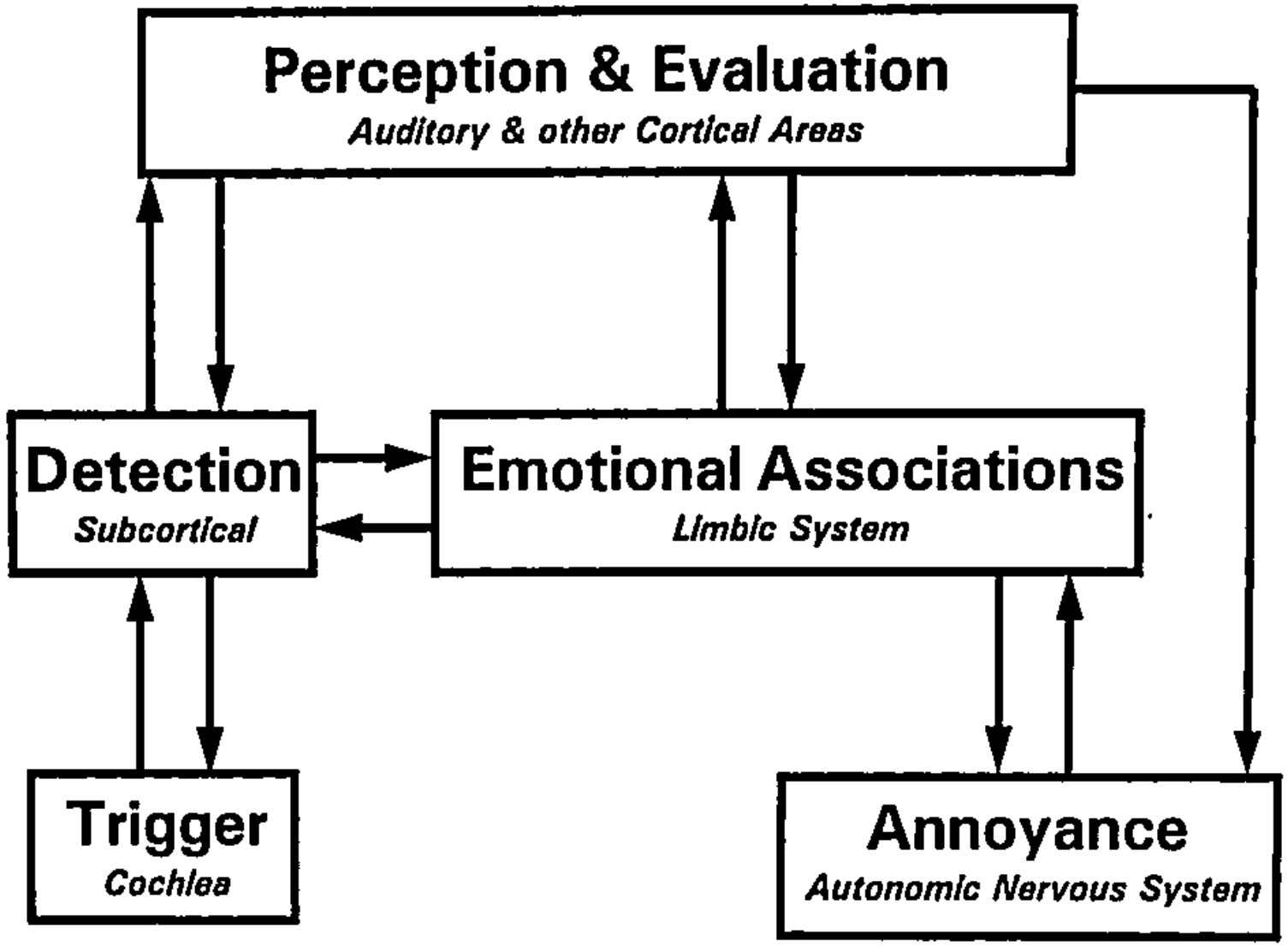
- Patients with tinnitus have been shown to have other associated psychological disorders e.g. depressive illness
- Endogenous endorphins ares released during stress which potentiate action of glutamate in the cochlear
- Patients with tinnitus have been found to have anxiety and depression with elevated serotonin and GABA levels[10]
- Serotonin and GABA receptors have been out the auditory system

PATHOGENESIS

f. Jastreboff neurophysiological model[11]:

- Principally,auditory pathway & several non auditory systems play essential role in tinnitus.
- Stresses non auditory system dominates in determining annoyance level.
- Proposes treatment by inducing & facilitating habituation to tinnitus signal.
- Goal-to reach level though patient percieves tinnitus as unchanged, they aren't aware of it &/or no annoyance occurs.

JASTREBOFF NEUROPHYSIOLGICAL MODEL[11]



DIAGNOSIS

- It is important to keep in mind that tinnitus is frequently a sign of hearing loss or other cochlear injury and may be the only complaint for a patient with a CNS lesion
- Impact of tinnitus to each individual is different, some "experience" tinnitus while others "suffer from it"
- Studies indicate that the degree of disability perceived by tinnitus patients and its impact on the patients QOL does not directly corelate with loudness, type of tinnitus or length of time with tinnitus

HISTORY

- Description of tinnitus : pulsatile, non-pulsatile, high pitch, low pitch, intermittent, constant
- Aggrevating or relieving factors
- Previous ear disease, noise exposure
- Hearing status
- Head injury
- TMJ dysfunction
- Current/ previous medication
- Medical conditions: hypertension, artherosclerosis, neurologic illness/surgery
- Ask specifically about: depression, anxiety, insomnia

PHYSICAL EXAMINATION

- ENT EXAM: otoscopy, TFT, EUM
- Complete CNS exam : cranial nerves
- Auscultation for bruits: over the neck, periauricular area, temple, orbit, mastoid
- If tinnitus is of venous origin it can be suppressed by careful pressure on the jugular vein

INVESTIGATIONS/EVALUATION

- FBC,ESR,U&Es,VDRL
- MRI: chiari malformation, multiple sclerosis, raised ICP, acoustic neuroma
- HRCT of temporal bone
- Suspected auditory system disorder: PTA, tympanometry, acoustic reflex testing, speech audiometry, OAE, ABR
- Suspected vascular aetiology: angiography(MR or CT)

INVESTIGATIONS/EVALUATION

- Impact of tinnitus on the patient be measured using various instruments:
  - Tinnitus handicap inventory
  - Tinnitus reaction questionnaire
  - Tinnitus severity scale
  - Tinnitus coping style questionnaire
  - Tinnitus functional index

TREATMENT

- Various treatments –most unsuccessful/unproven
- Targets in treatment include : Cochlea, NTs & receptors, ion channels.
- Treatment methods not able to reduce or eliminate the sensation on any consistent basis
- no drug that has been approved specifically for its treatment
- Counseling is key since most patients are frustrated and discouraged after being told by other health professionals "JUST LEARN TO LIVE WITH IT !" or "THERE IS NOTHING I CAN DO FOR YOU !"

TREATMENT

- Various modalities:
  a) Treatment of co-morbidities: depression, insomnia, drug toxicity, palatal myoclonus, glomus tumours e.t.c
  b) Sound treatments/technologies
     - Hearing Aids
     - Masking devices
     - Neuromonics Tinnitus Treatment[12] -combines acoustic stimulation with a structured program of counseling support by a clinician
  c) Surgical treatments : cochlear implants, microvascular decompression, cochlear nerve section

TREATMENT

d) Medical treatment
- Pharmacological Treatment
- Complementary and Alternative Medicine Therapies

e) Behavioural treatment:
- Cognitive Behavioral Therapy
- Tinnitus Retraining Therapy
- Biofeedback, Education, and Relaxation Therapies

f) Others :
- Transcranial magnetic stimulation[13]
- Transcutaneous electrical stimulation[14]
- Electrical stimulation with high grade pulse trains[15]
- Electrical stimulation and acupuncture

MEDICAL

- Glutamate receptor antagonist –caroverine, memantine, Acamprosate
- Antidepressants-amitriptyline, nortriptyline, trimipramine
- Anxiolytics-Alprazolam
- Anticonvulsants- Carbamazepine, Gabapentin
- Vasodilators/vasoactive substances- Prostaglandin E1
- Selective serotonin-reuptake inhibitors: fluoxetine and paroxetine
- Lidocaine IV/transtympanic

MEDICAL

- Complementary and Alternative Medicine Therapies
  - Ginkgo Biloba Extract - glutamate antagonist, strong anti-oxidant
  - melatonin
  - Acupuncture and hyperbaric oxygen
  - diet modifications eg avoid high-sodium foods, caffeine, chocolate, and other stimulants

COGNITIVE-BEHAVIORAL THERAPY

- Aim- to modify harmful behaviours & thoughts using “deconditioning” technique
- reduces arousal levels via relaxation therapy &changing –ve thoughts through cognitive therapy.
- Motivates the patient to alter their psychological response to their tinnitus by identifying and reinforcing coping strategies, distraction skills and relatation techniques

TINNITUS RETRAINING THERAPY

- Based on neurophysiological model.
- Goal-train CNS to interpret tinnitus as unimportant & ignore it.
- Has 2 components:-

  -intensive direct counselling.

  -sound therapy using sound generators which emit low level broad band noise.
- The patient reaches a point where they are unaware of the tinnitus unless they specifically and consciously focus on it

TINNITUS RETRAINING THERAPY

- Conditioned reflexes involving connections of auditory with limbic & ANS are retrained such that the subconscious part of auditory pathway blocks the tinnitus signal.
- Acoustic input with unimportant information is ignored(habituation)
- Inducing & sustaining habituation of conditioned reflexes removes –ve impact of tinnitus

BIOFEEDBACK AND RELAXATION THERAPIES

- control or habituate to the perceived ringing and the subsequent distress.
- Biofeedback therapy-listens to audio signal from EMG of frontalis muscle
- reduces perceived ringing & muscle tension
- strategies to self-manage their tinnitus.
- Relaxation therapies -focus pt's attention away from the sound; psychologically improving symptoms.

FUTURE

- PET and Transcranial magnetic stimulation are currently being used to help research in the mechanisms and hence treatment of tinnitus.[13]

CONCLUSION

- difficult to study and treat -no objective tools to quantify and measure.
- no therapeutically successful treatment in terms of medium or long term remission
- no universally accepted therapies for managing tinnitus
- interactions among the auditory, cognitive, affective, and mental health issues.
- Negative counseling can enhance preexisting tinnitus annoyance and anxiety levels and turn a subject who is just “experiencing” tinnitus to one who is “suffering from it”

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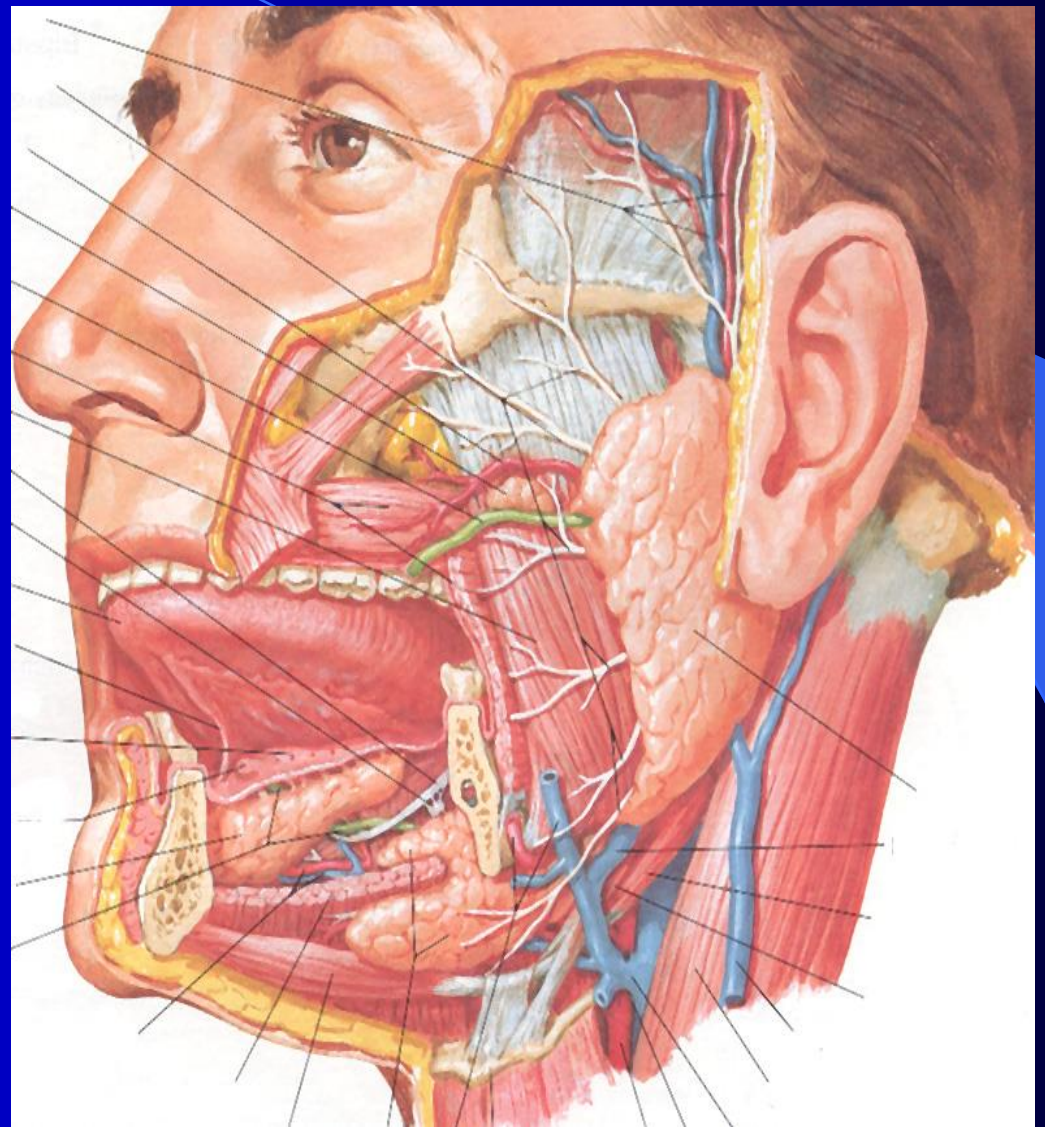
Nonneoplastic Diseases of the Salivary Glands

Dr mugwe

Introduction

- Two major categories: inflammatory and noninflammatory
- Usually involve major salivary glands
- Usually do not require extensive diagnostic workup



Inflammatory Diseases

- Mumps
  - Most common viral disorder of salivary glands
  - Peak age 4-6
  - 1 or both parotids after 2-3 week prodrome
  - Diagnosis: serology or urine
  - Complications: deafness, pancreatitis, meningitis, orchitis, Type I DM, chronic obstructive sialadenitis

Inflammatory Diseases

- Other Viruses
  - CMV, Coxsackievirus A, Echovirus, Influenza A, Lymphocytic choriomeningitis Virus
  - Treatment: symptomatic for all viral diseases
- Acute Suppurative Sialadenitis
  - Parotid most common site; peak age 50's-60's
  - 30-40% in post-op patients; most commonly GI procedures POD 3-5

Inflammatory Diseases

- Acute Suppurative Sialadenitis
  - Presentation: sudden, diffuse enlargement with associated induration and tenderness. Massage produces purulent saliva
  - 20% of cases bilateral
  - Pathogens: *Staph aureus most common;* Gram negatives, anaerobes also common
  - Treatment: hydration, improved oral hygiene, repeated massage of gland, IV abx, warm compresses, sialogogues

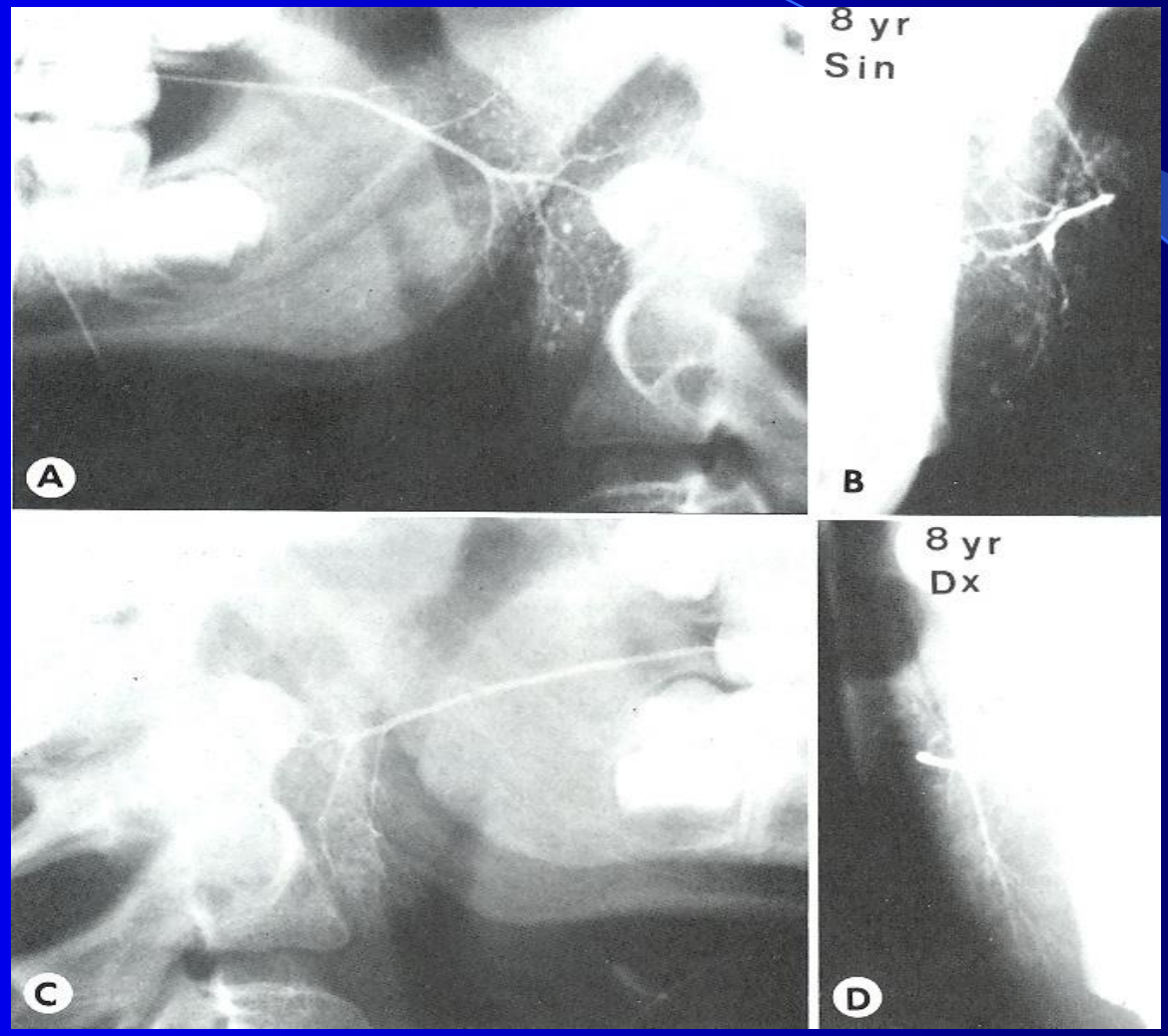
Inflammatory Diseases

- Acute Suppurative Sialadenitis
  - If no significant improvement in 24-48h, then proceed to incision & drainage *OR* image-guided needle aspiration
- Chronic Sialadenitis
  - Most commonly parotid
  - Usually from permanent damage during acute infection; occasionally from recurrent parotitis of childhood

Inflammatory Diseases

- Chronic Sialadenitis
  - Histologic changes: sialectasis, progressive acinar destruction, lymphocytic infiltrates
  - Saliva changes; returns to normal between attacks
  - Presentation: mild pain, recurrent parotid enlargement that worsens with eating; 80% develop xerostomia

Inflammatory Diseases



Inflammatory Diseases

- Chronic Sialadenitis
  - Treatment

    1) Underlying causes

    2) Sialogogues, massage, heat, hydration, abx during acute attacks

    3) Periodic ductal dilatation, duct ligation, total gland irradiation, tympanic neurectomy

    4) Excision

Inflammatory Diseases

- Recurrent Parotitis of Childhood
  - More common in males; peak age 5-7
  - ¾ give h/o Mumps; heredity plays no role
  - Presentation: Usually unilateral; when bilateral, one side worse

    Severe pain, fever, malaise during attacks

    Recurs

Inflammatory Diseases

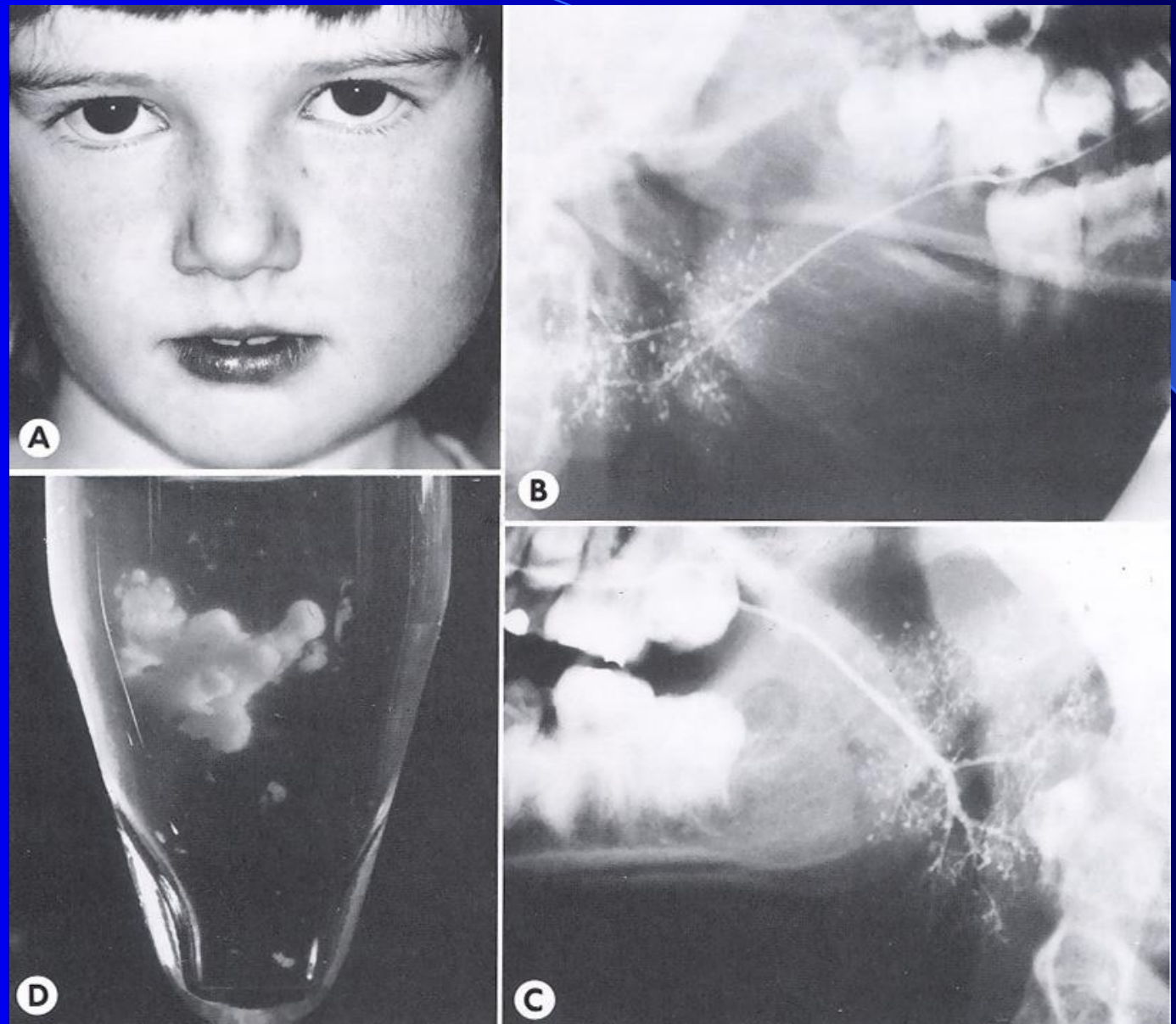
- Recurrent Parotitis of Childhood
  - Disease course (Ericson): onset age 3 months-16 years

    Exacerbations every 3-4 months

    55% of cases resolve with puberty

    25% no improvement with puberty
  - Histology: massive B-cell infiltration and dilated intraglandular ducts

Inflammatory Diseases

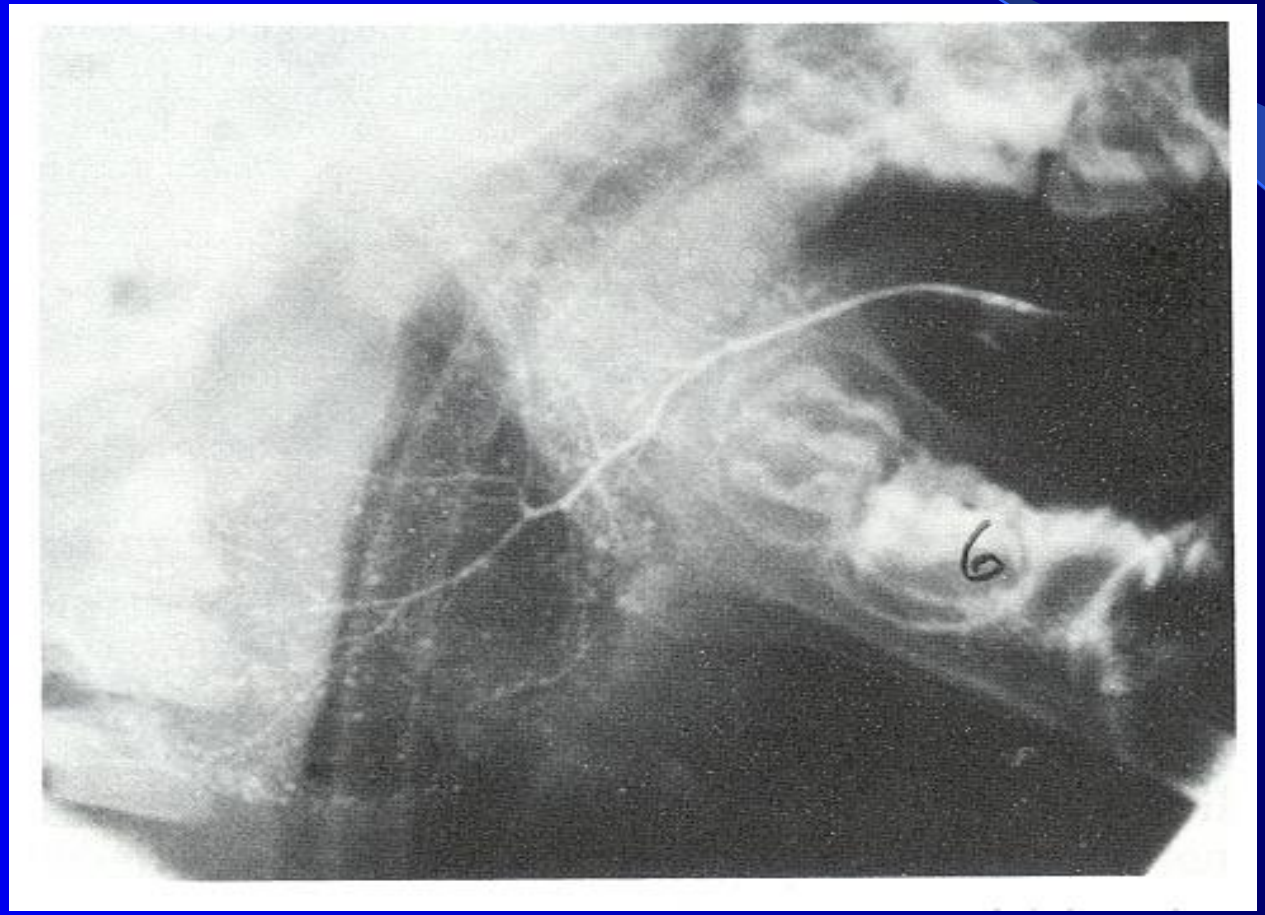


Inflammatory Diseases

- Recurrent Parotitis of Childhood
  - Sialogram: multiple peripheral sialectases 1-2 mm in diameter; changes persist w/ resolution of symptoms
  - Pathogens: flora ascend from oral cavity

    Balls of soft material common, but rarely yields frank pus
  - Treatment: Pen VK, warm massages, good oral hygiene, sialogogues, chewing gum

Inflammatory Diseases



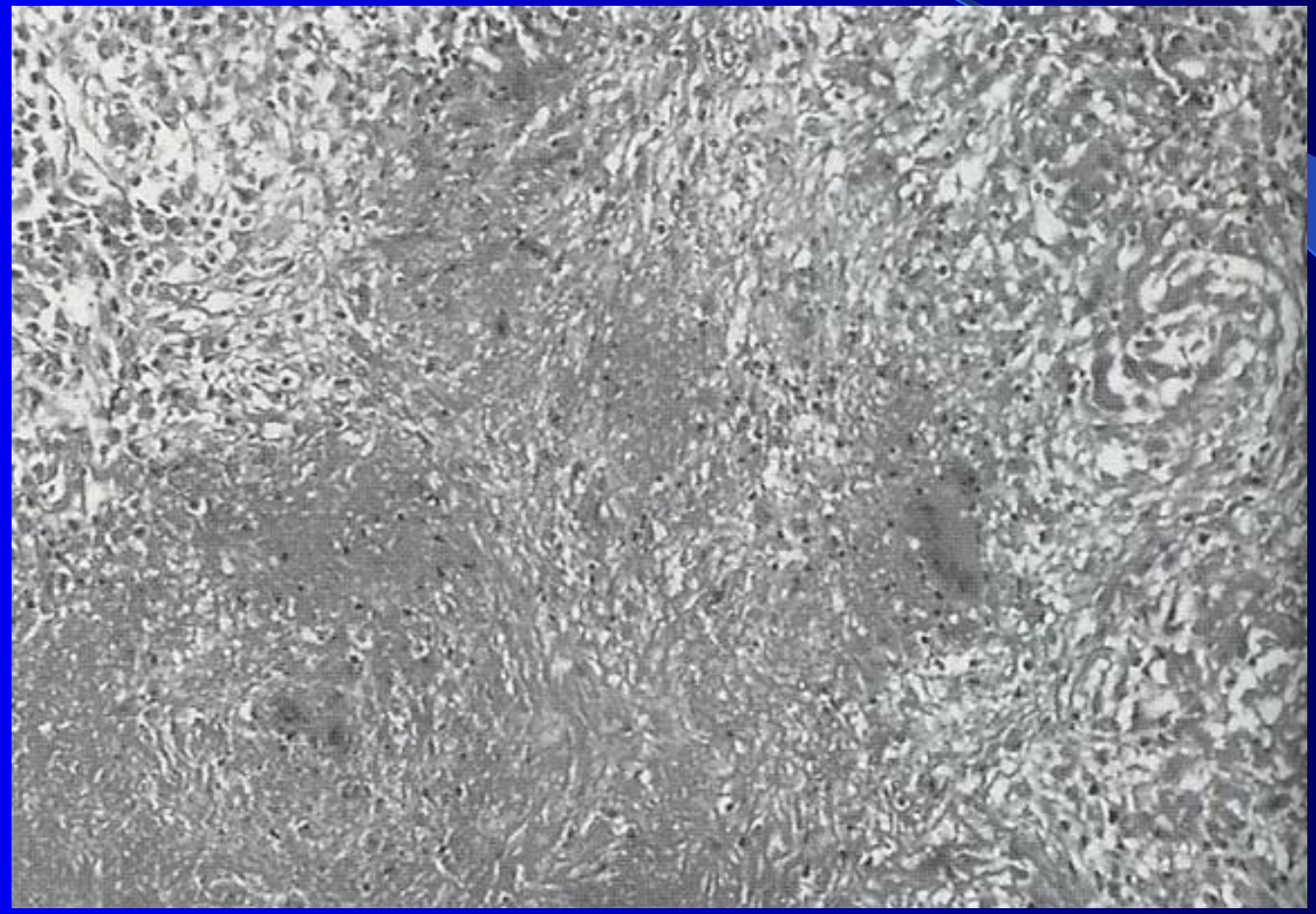
Inflammatory Diseases

- Benign Lymphoepithelial Lesion
  - Epimyoepithelial islands arise from
    1) lymphoreticular infiltrates
    2) acinar atrophy
    3) ductal metaplasia
  - Presentation: Asymptomatic enlargement of 1 gland
  - Risk of lymphoma, carcinoma, pseudolymphoma
  - No treatment necessary

Inflammatory Diseases

- Primary Tuberculosis
  - Presentation: Unilateral parotid
    May present as acute inflammatory lesion or as chronic tumorous lesion
  - Diagnosis: AFB stain of saliva AND PPD test
    FNA if tumorous lesion
  - Treatment: Anti-TB meds; excision if resistant
  - Secondary TB: systemic dz.; submandibular and sublingual glands more often involved

Inflammatory Diseases



Inflammatory Diseases

- Animal Scratch Disease
  - Typically attacks periparotid lymph nodes
  - Pathogens: *Bartonella henselae*, *Afipia felis*
  - Diagnostic Criteria (3/4):

    1) H/o contact w/ a cat and presence of scratch

    2) + skin test or + serology for *B henselae*

    3) + Gram stain and Cx

    4) Histology: stellate abscesses, pleomorphic intracellular bacilli, Warthin-Starry stain

Inflammatory Diseases

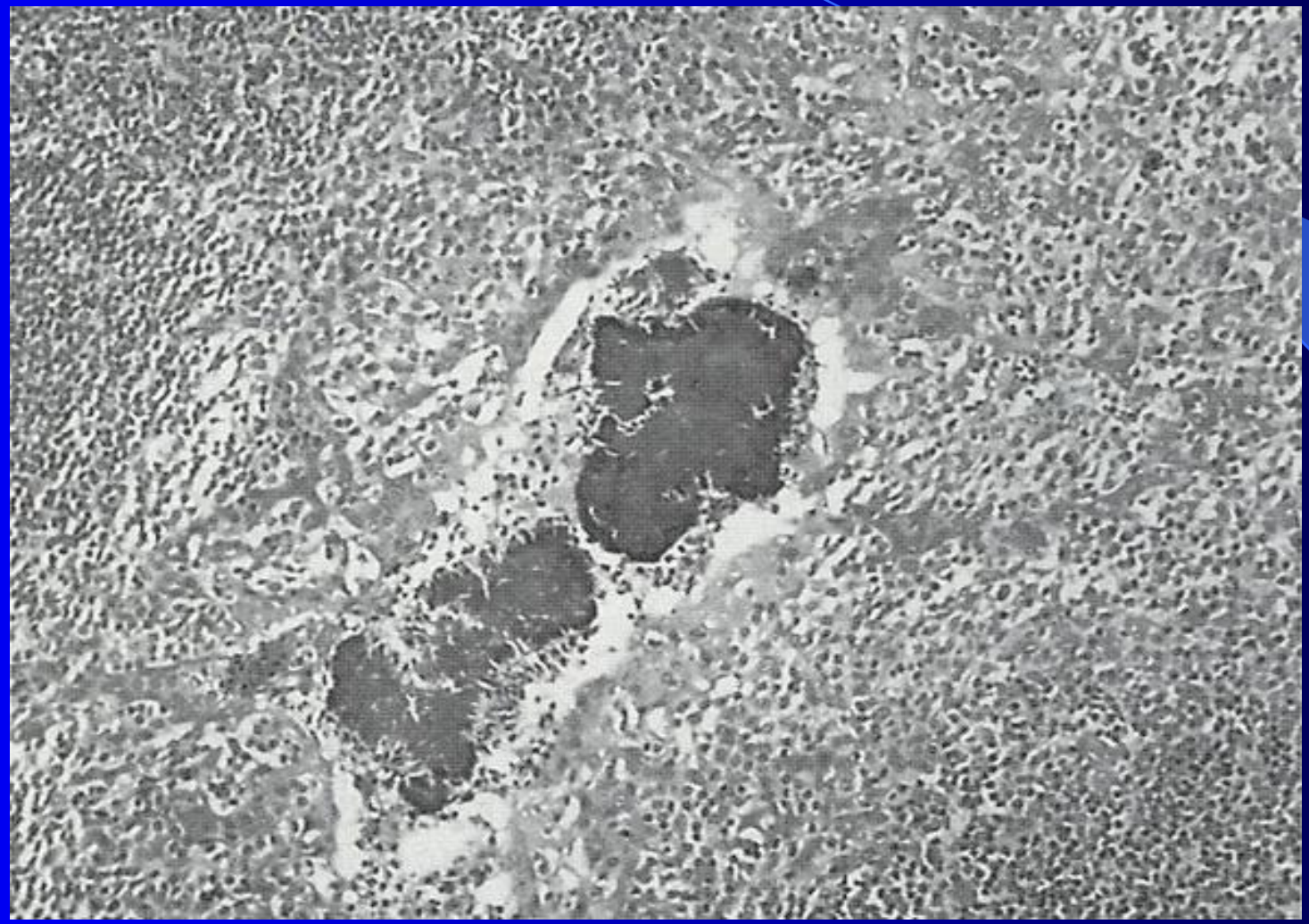
- Animal Scratch Disease
  - Should place PPD to r/o Tb
  - 96% resolve spontaneously within 2-6 months; close followup needed until adenopathy subsides
  - Treatment: Bactrim X 1 week, or Rifampin X 1-2 weeks

    IV Gentamicin in severe cases

Inflammatory Diseases

- Actinomycosis
  - Infection from tonsil or teeth
  - Presentation: 61% visible sinus tracts; 40% adenopathy; some have purplish skin discoloration
  - Histology: sulfur granules
  - Diagnosis: culture
  - Treatment: I&D, 2-6 weeks of IV Pen G

Inflammatory Diseases



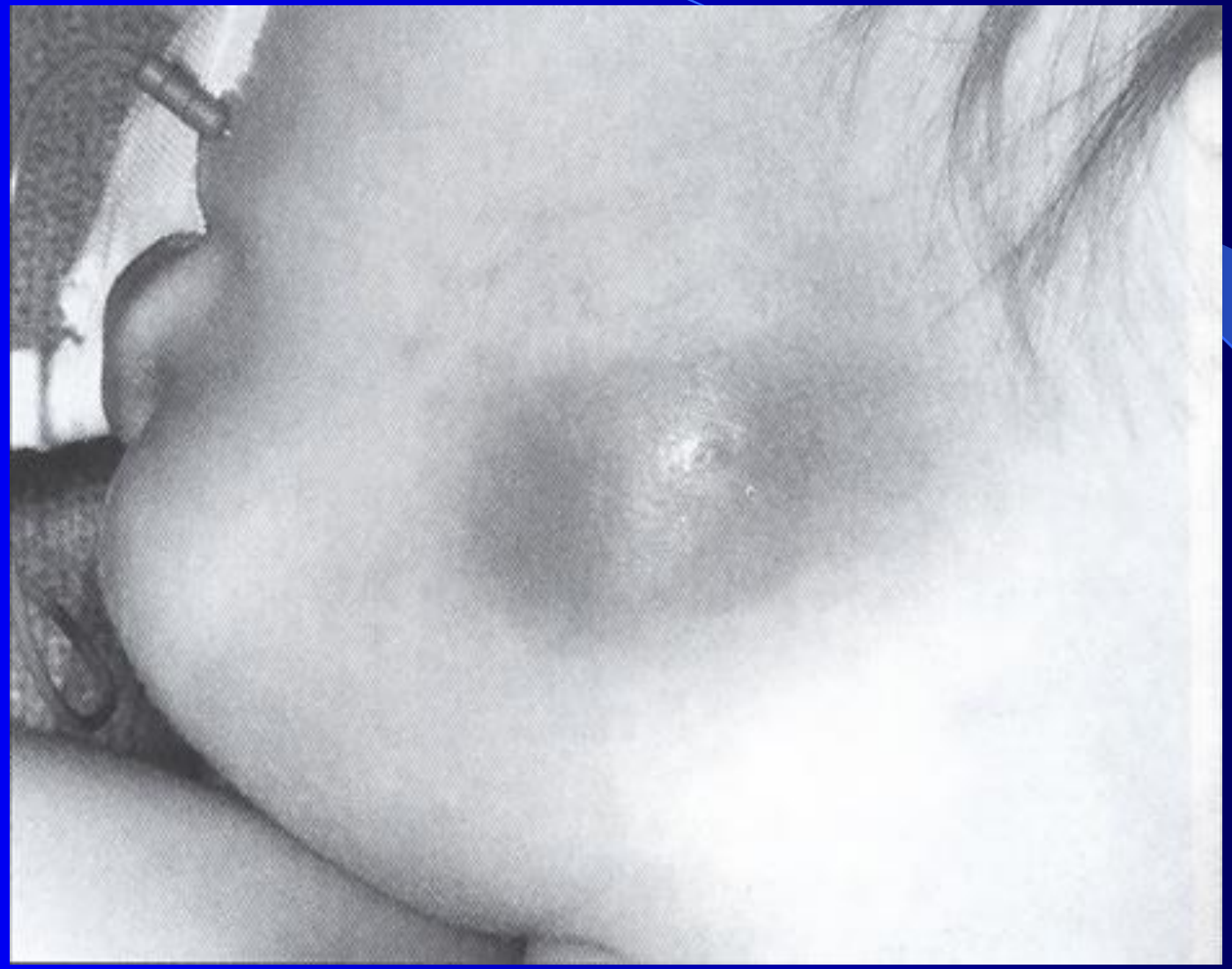
Inflammatory Diseases

- Atypical (Nontuberculous) Mycobacteria
  - Median age 28 months (usually ages 1-5)
  - Transmitted from soil to mouth/eyes
  - Presentation: focal swelling of face or neck (100%), change in overlying skin color (76%), necrosis or fistula of skin (52%); no systemic symptoms
  - Most common sites: submandibular area, parotid, upper neck, submental area

Inflammatory Diseases

- Atypical (Nontuberculous) Mycobacteria
  - Pathogen: *MAC* most common
  - Diagnosis: FNA diagnostic 87% of time; PPD's not helpful
    1) Culture from FNA, or
    2) + AFB stain from FNA, or
    3) Histology: granulomatous inflammation w/ caseating necrosis

Inflammatory Diseases



Inflammatory Diseases

- Atypical (Nontuberculous) Mycobacteria
  - Treatment: Curettage vs. Excision

    Curettage for lesions with extensive skin necrosis or fluctuant parotid lesions

    Surgical excision more effective

    Medications controversial; Macrolides may work for early disease

Inflammatory Diseases

- Sarcoidosis
  - 6% involve salivary glands clinically, 1/3 histologically
  - Heerfordt's syndrome (Uveoparotid fever):

    1) Uveitis

    2) Parotid enlargement

    3) CN VII paralysis

  Self-limited; uveitis can result in glaucoma – requires long term f/u

Inflammatory Diseases

- Sjogren's Syndrome: Background
  - Chronic, slowly progressive, benign; 2nd most common autoimmune disease behind RA
  - Lymphocyte-mediated destruction of exocrine glands producing keratoconjunctivitis sicca and xerostomia
  - 90% middle-aged women
  - 44% report PCN allergy

Inflammatory Diseases

- Sjogren's Syndrome: Background
  - Primary=exocrine glands only;
    Secondary=coexisting autoimmune disease

    Secondary form more common; salivary gland enlargement more common in primary form
  - Serology (similar pattern in SLE):

    1) ANA (50-80%)

    2) RF (75%)

    3) Ro/SS-A antibodies

    4) La/SS-B antibodies; 3 or 4 in up to 90%

Inflammatory Diseases

- Sjogren's Syndrome: Presentation
  - Xerostomia: most bothersome; difficulty swallowing dry food, difficulty speaking continuously, burning sensation, increased caries, problems wearing dentures; erythematous/sticky oral mucosa, atrophy of filiform papillae
  - Keratoconjunctivitis Sicca: gritty feeling under eyelids, blurred vision, burning sensation, thick strands at inner canthi, decreased tearing, redness/itching, photosensitivity; results from destruction of conjunctival epithelium

Inflammatory Diseases

- Sjogren's Syndrome: Presentation
  - Other exocrine gland involvement: dry nose, dry throat, xerotrachea, esophageal mucosal atrophy, atrophic gastritis, subclinical pancreatitis, vaginal dryness
  - 1/3 = fatigue, low grade fever, myalgias/arthralgias
  - Extraglandular involvement in ¼: Lungs, kidneys, vasculitis, nervous system

Inflammatory Diseases

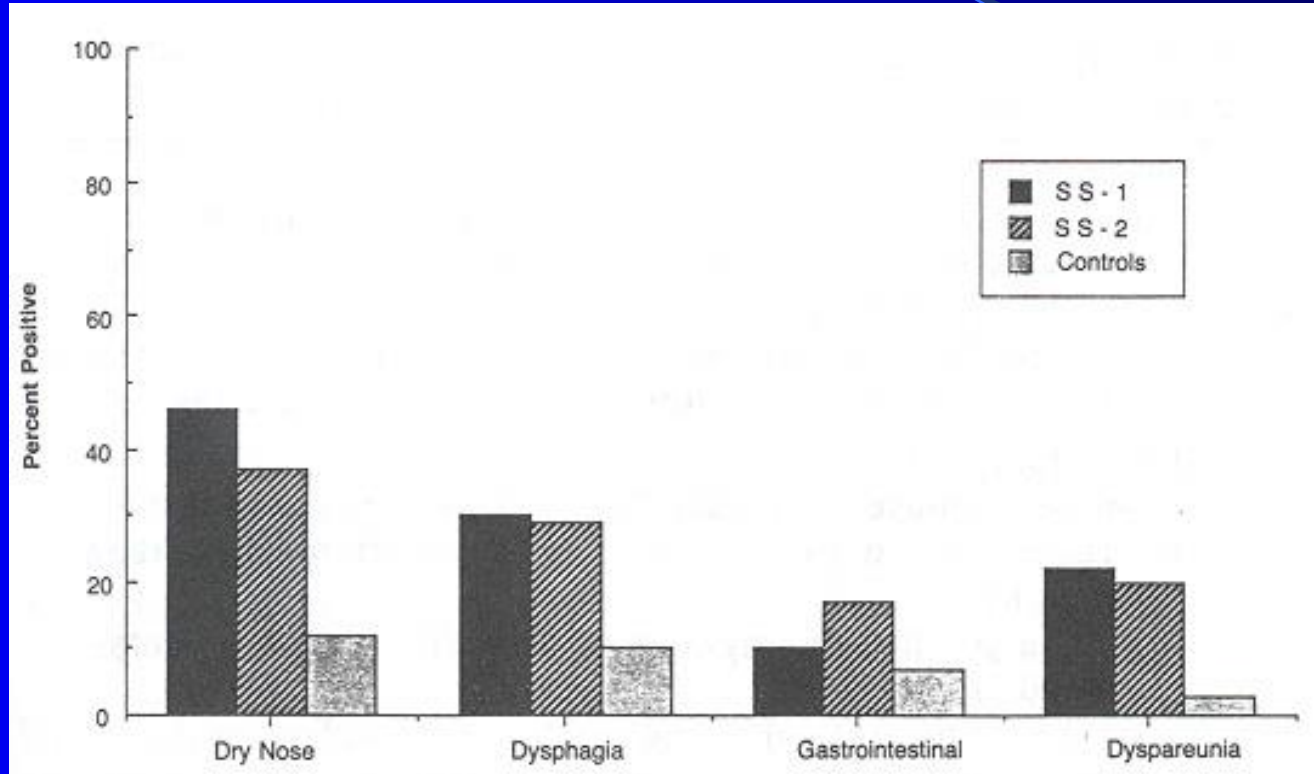


Fig. 15–3. Other glandular manifestations observed in Sjogren's syndrome patients from a multicenter study for the diagnostic criteria for Sjogren's syndrome, Pisa, 1992. (626 cases; 19 European centers)

Inflammatory Diseases

- Sjogren's Syndrome: Associated risks
  - Increased risk of
    1) NonHodgkin's Lymphoma (RR=44)
    2) Multiple Myeloma

Inflammatory Diseases

Table 15–4. *Preliminary criteria for the classification of Sjogren's syndrome (modified from Reference 9).*

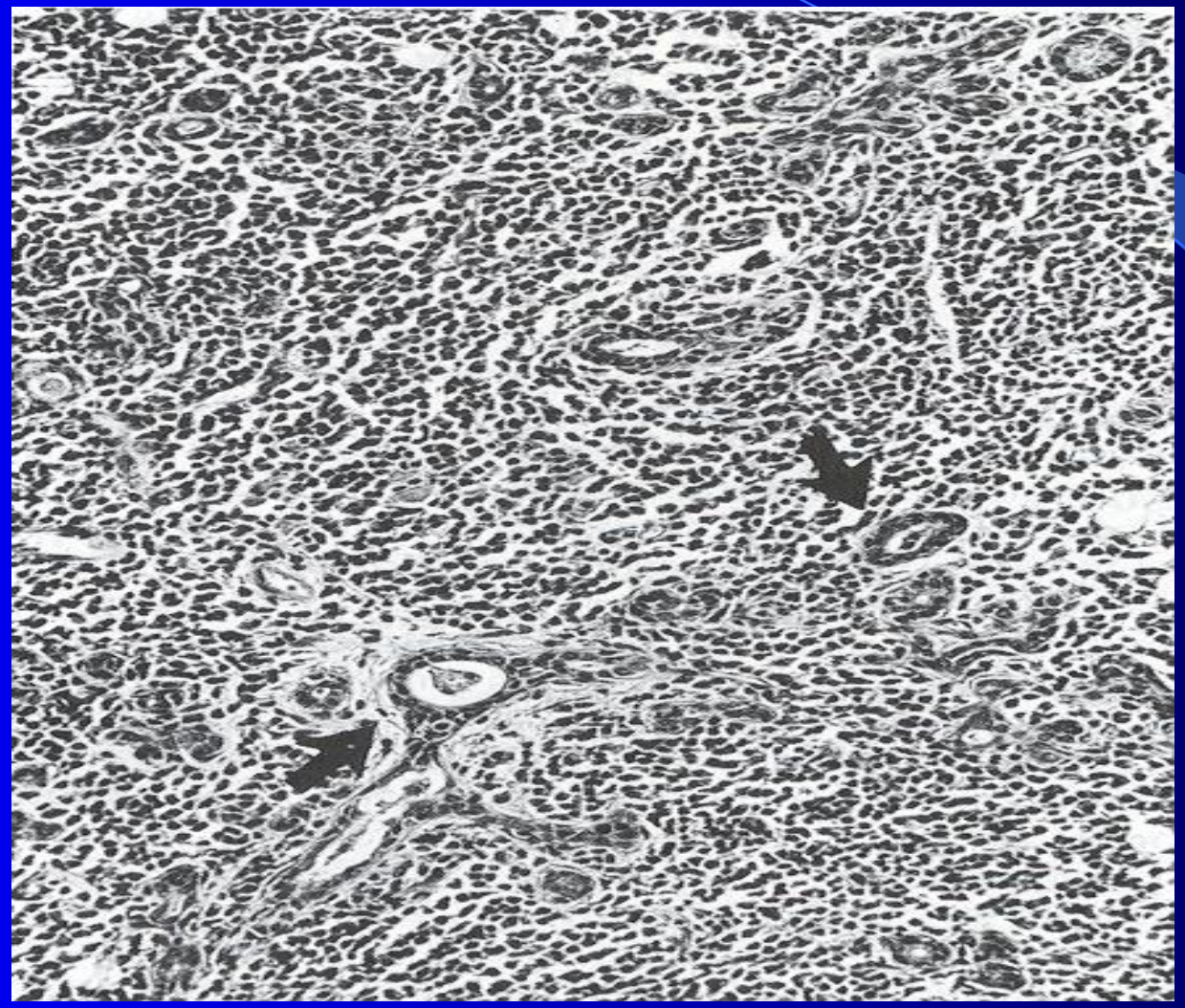
1. Ocular symptoms
   A positive response to at least 1 of the following 3 questions:
   (a) Have you had daily, persistent, troublesome dry eyes for more than 3 months?
   (b) Do you have a recurrent of sandy or gravel feeling in the eyes?
   (c) Do you use tear substitutes more than 3 times a day?
2. Oral symptoms
   A positive response to at least 1 of the following questions:
   (a) Have you had a daily feeling of dry mouth for more than 3 months?
   (b) Have you had recurrent or persistently swollen salivary glands as an adult?
   (c) Do you frequently drink liquids to aid in swallowing dry foods?
3. Ocular signs
   Objective evidence of ocular involvement determined on the basis of a positive result on at least 1 of the following 2 tests:
   (a) Schirmer-1 test ($\leq$5 mm in 5 minutes)
   (b) Rose bengal score ($\geq$4, according to the van Bijsterveld scoring system)
4. Salivary gland involvement
   Objective evidence of salivary gland involvement, determined on the basis of a positive result on at least 1 of the following 3 tests:
   (a) Salivary scintigraphy
   (b) Parotid sialography
   (c) Unstimulated salivary flow ($\leq$1.5 ml in 15 minutes)
5. Histopathologic findings
   Focus score $\geq$1 on minor salivary gland biopsy
   (focus defined as an agglomeration of at least 50 mononuclear cells, focus score defined as the number of foci/4mm^2 of glandular tissue)
6. Autoantibodies
   Presence of at least 1 of the following autoantibodies in the serum: Antibodies to Ro (SS-A) or La (SS-B) antigens or antinuclear antibodies or rheumatoid factor.

A patient is considered as having probable Sjogren's syndrome if 3 of 6 criteria are present, and as definite if 4 of 6 criteria are present

Inflammatory Diseases

- Sjogren's Syndrome: Histology
  - Severe lymphoid (T-cell) infiltrate can mimic lymphoma; heterogenous, lobular architecture preserved
  - Enlarged lymph nodes w/ pleomorphic infiltrates and frequent mitotic figures = "pseudolymphoma"
  - When biopsying, avoid epinephrine; send specimen in formalin

Inflammatory Diseases



Inflammatory Diseases

- Sjogren's Syndrome: Treatment
  - Incurable disease
  - Key=fluid replacement

    Artificial tears; eye patching, boric acid ointments for corneal ulceration

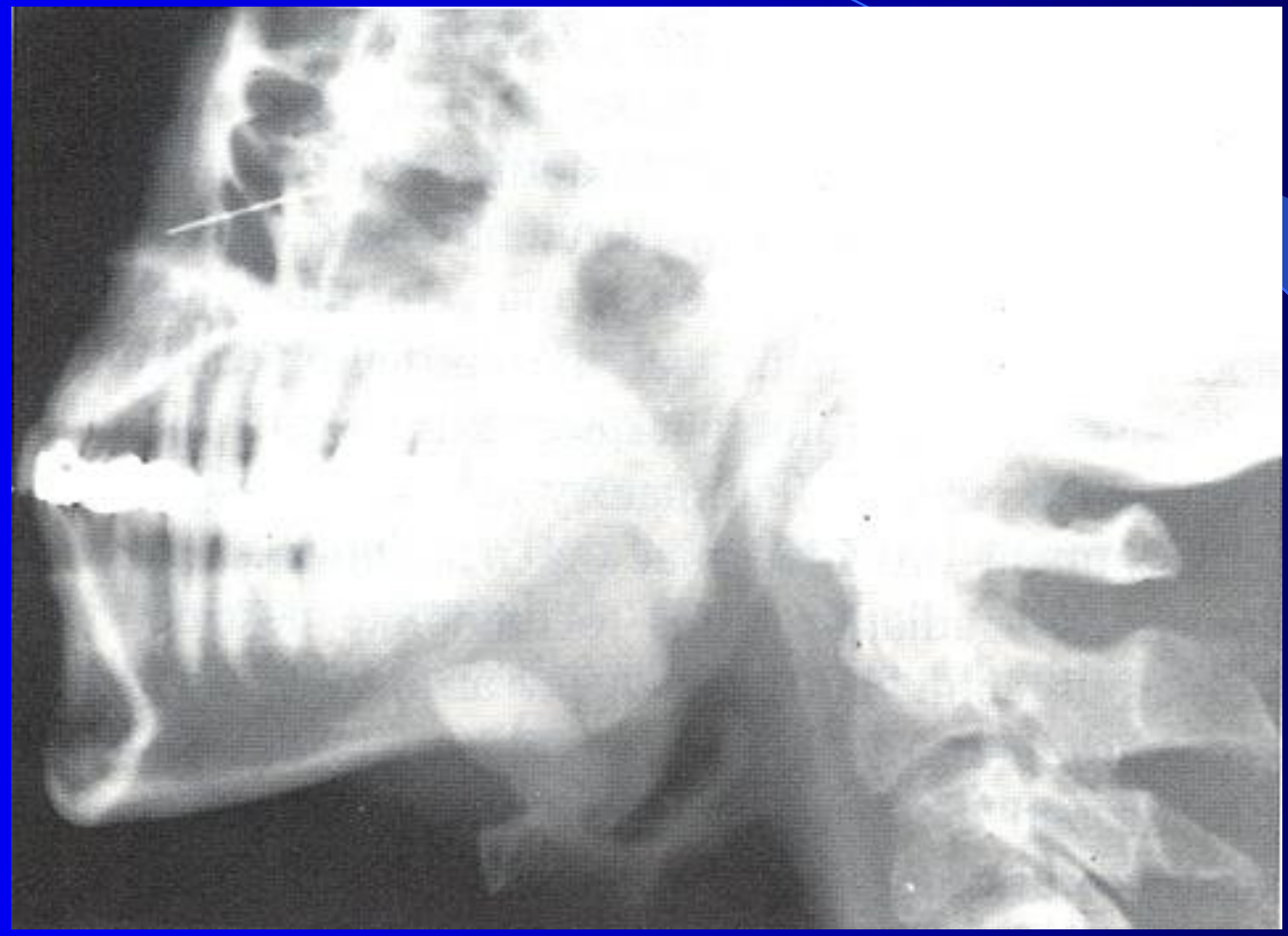
    Avoid diuretics, antihypertensives, antidepressants
  - Medications: Pilocarpine 5 mg TID; hydroxychloroquine; glucocorticoids 1 mg/kg/day

Noninflammatory Diseases

- Sialolithiasis
  - 80% submandibular gland (has more calcium concentration), 20% parotid
  - Only 1 stone in ¾ cases
  - 90% of submandibular stones radiopaque (high calcium content); 90% of parotid stones radiolucent
  - Presentation: recurrent swelling, pain worse with eating
  - Complications: sialadenitis, ductal ectasia, and stricture
  - Treatment: If near duct orifice, transoral removal of stone with marsupialization

    If near hilum, gland excision

Inflammatory Diseases



Noninflammatory Diseases

- Cysts
  - Mucoceles vs. Mucous cysts: minor salivary glands
  - 2-5% of all parotid lesions
  - Congenital: dermoid cysts, ductal cysts, 1st arch branchial cleft cysts
  - Acquired: BLL, trauma, parotitis, calculi, neoplasms

Noninflammatory Diseases

- Trauma
  - Identify the duct; can pass probe to ID distal duct; can milk gland to ID proximal duct
  - Transected duct: end-to-end anastomosis over polyurethane catheter with 9-0 suture; remove catheter after 2 weeks
  - Salivary-cutaneous fistula: repeat aspiration and pressure dressings; sialogram; excision if conservative treatment fails
  - Blunt trauma: drain large hematomas early

Noninflammatory Diseases

- Sialadenosis
  - Nonneoplastic, noninflammatory enlargement of salivary glands associated with systemic disorders
  - Usually asymptomatic
  - Causes=obesity, malnutrition, malabsorption, and alcoholic cirrhosis; very rarely does sialadenosis occur in nonalcoholic cirrhosis

Noninflammatory Diseases

- Cheilitis Glandularis
  - Enlargement of the labial salivary glands; clear, thick, sticky mucus; can result in lower lip eversion
  - Treatment: vermilionectomy
- Kussmaul's Disease (Dialodochitis Fibrinosa)
  - Mucous plug obstructing duct
  - Treatment: rehydration, gentle massage, sialogogues

Noninflammatory Diseases

- Necrotizing Sialometaplasia
  - Benign, self-healing process of unknown etiology
  - Presentation: usually hard palate, usually males; asymptomatic mucosal ulceration
  - Histology: easily mistaken for SCCA, mucoepidermoid CA; lobular necrosis + squamous metaplasia + preserved lobular architecture
  - Treatment: biopsy for diagnosis, but treatment unnecessary
  - Subacute necrotizing sialadenitis=painful, nodular variant

pseudoparotomegally

1. Hypertrophy masseter
2. Obesity
3. Malnutrition
4. Malabsorption states
5. Uraemia
6. Hypothyroidism
7. Myxedema
8. Testicular and ovarian hypertrophy
9. Pregnancy and lactation
10. Chronic relapsing pancreatitis
11. pneumoparotitis

SALIVARY GLAND NEOPLASMS

DR MUGWE

Salivary Gland Neoplasms

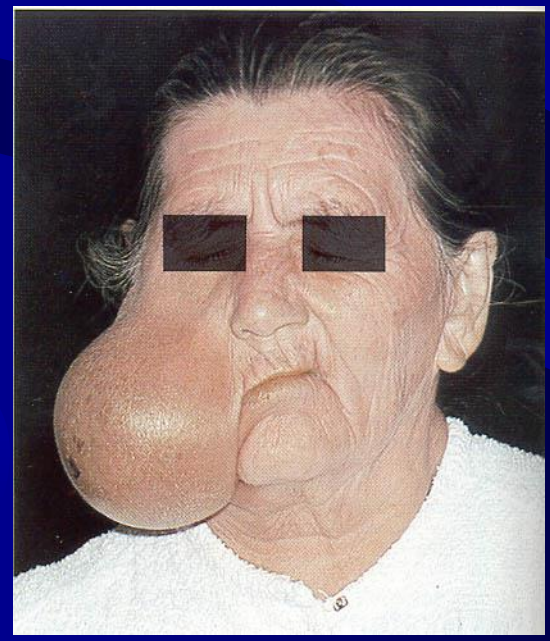
- Benign Neoplasms
- Malignant Neoplasms
- Controversial Issues

BENIGN
Salivary Gland Neoplasms

- Diverse histopathology
- Relatively uncommon
  - 2% of head and neck neoplasms
- Distribution
  - Parotid: 80% overall; 80% benign
  - Submandibular: 15% overall; 50% benign
  - Sublingual/Minor: 5% overall; 40% benign

Pleomorphic Adenoma

- Most common of all salivary gland neoplasms
  - 70% of parotid tumors
  - 50% of submandibular tumors
  - 45% of minor salivary gland tumors
  - 6% of sublingual tumors
- 4^{th}-6^{th} decades
- F:M = 3-4:1

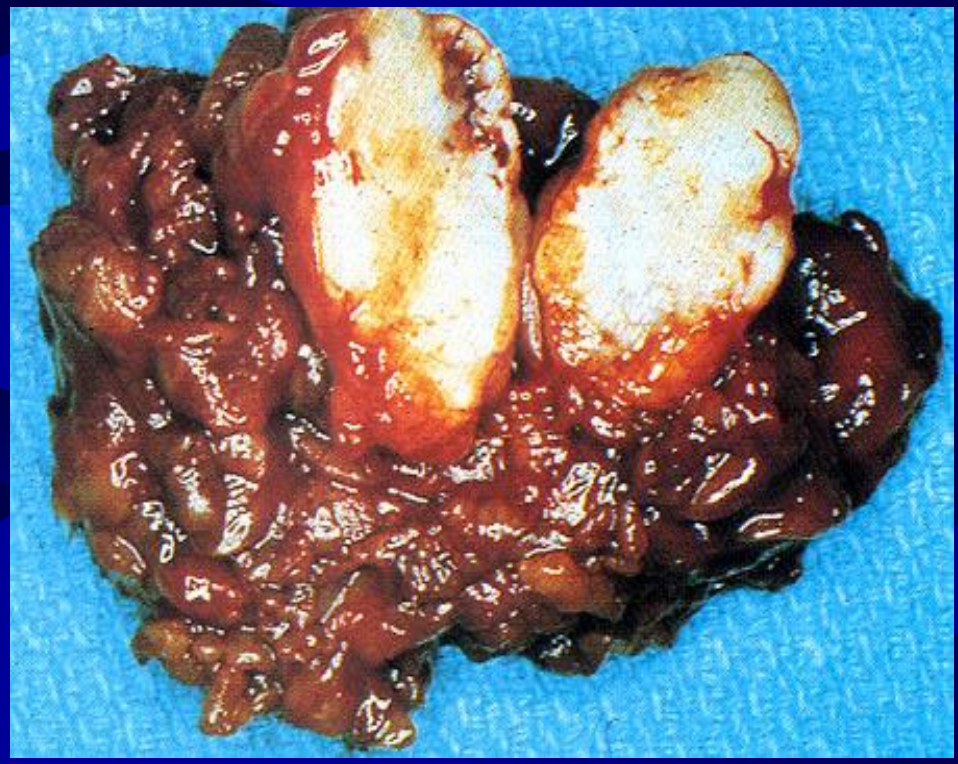


Pleomorphic Adenoma

- Slow-growing, painless mass
- Parotid: 90% in superficial lobe, most in tail of gland
- Minor salivary gland: lateral palate, submucosal mass
- Solitary vs. synchronous/metachronous neoplasms

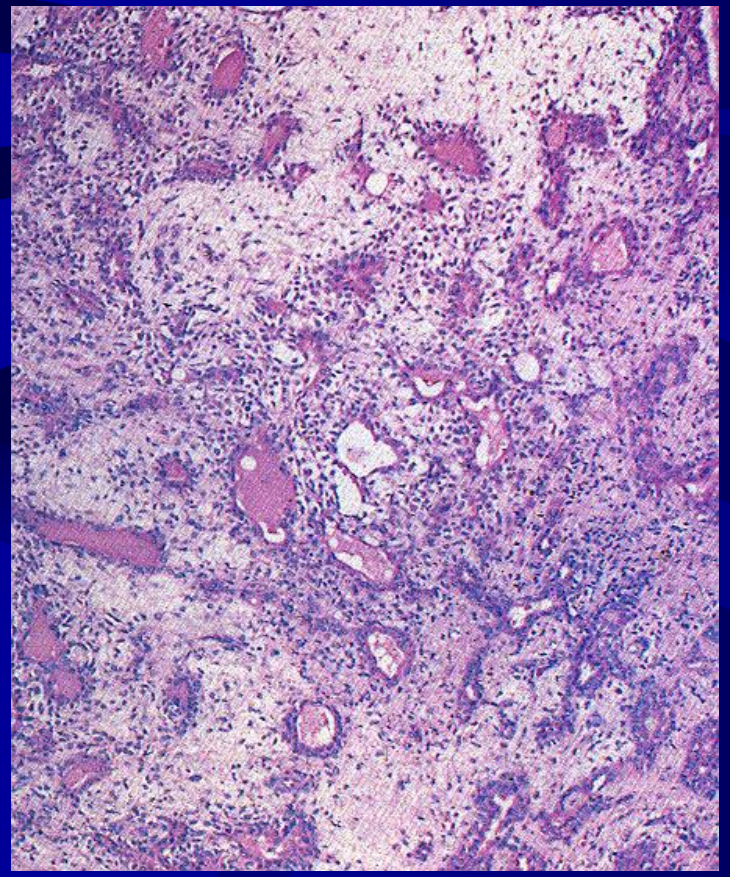
Pleomorphic Adenoma

- Gross pathology
  - Smooth
  - Well-demarcated
  - Solid
  - Cystic changes
  - Myxoid stroma

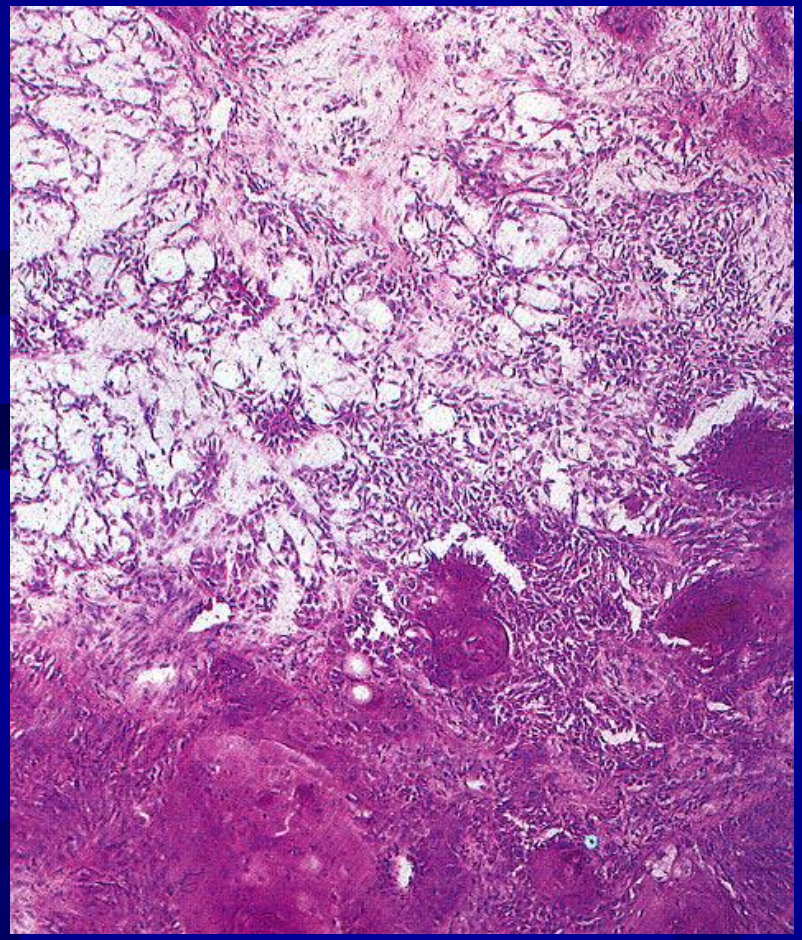


Pleomorphic Adenoma

- Histology
  - Mixture of epithelial, myopeithelial and stromal components
  - Epithelial cells: nests, sheets, ducts, trabeculae
  - Stroma: myxoid, chrondroid, fibroid, osteoid
  - No true capsule
  - Tumor pseudopods



Pleomorphic Adenoma



Pleomorphic Adenoma

- Treatment: complete surgical excision
  - Superficial Parotidectomy with facial nerve preservation
  - Submandibular gland excision
  - Wide local excision of minor salivary gland

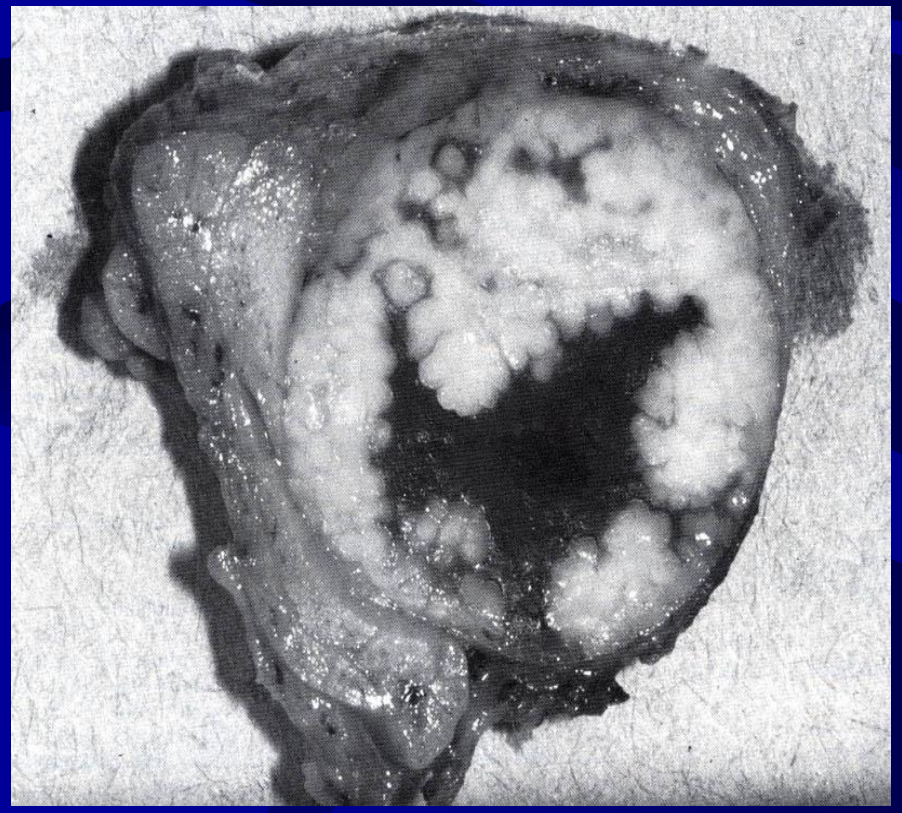
- Avoid enucleation and tumor spill

Warthin's Tumor

- AKA: papillary cystadenoma lymphomatosum
- 6-10% of parotid neoplasms
- Older, Caucasian, males
- 10% bilateral or multicentric
- 3% with associated neoplasms
- Presentation: slow-growing, painless mass

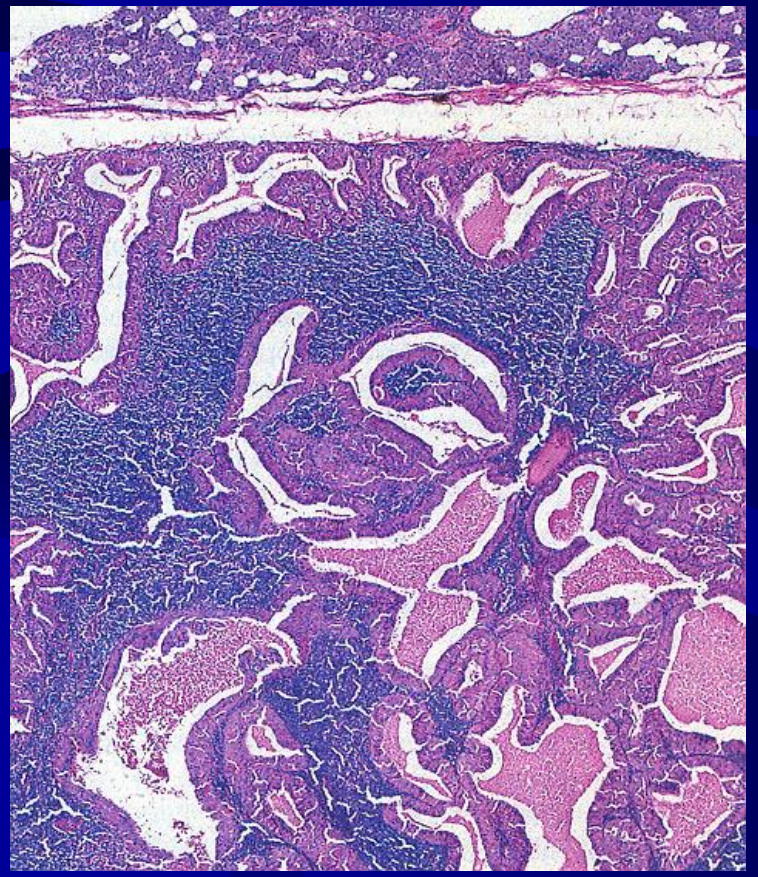
Warthin's Tumor

- Gross pathology
  - Encapsulated
  - Smooth/lobulated surface
  - Cystic spaces of variable size, with viscous fluid, shaggy epithelium. N.b- pleomorphic adenoma not cystic at all!
  - Solid areas with white nodules representing

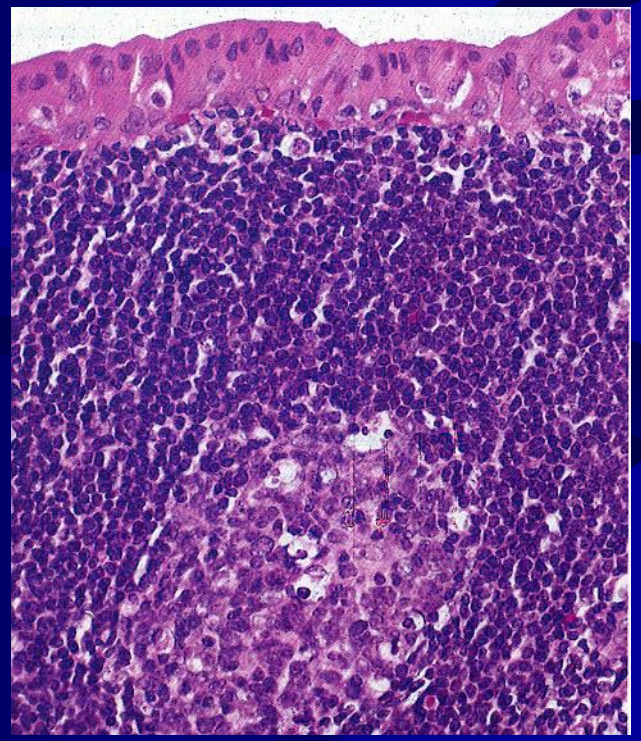


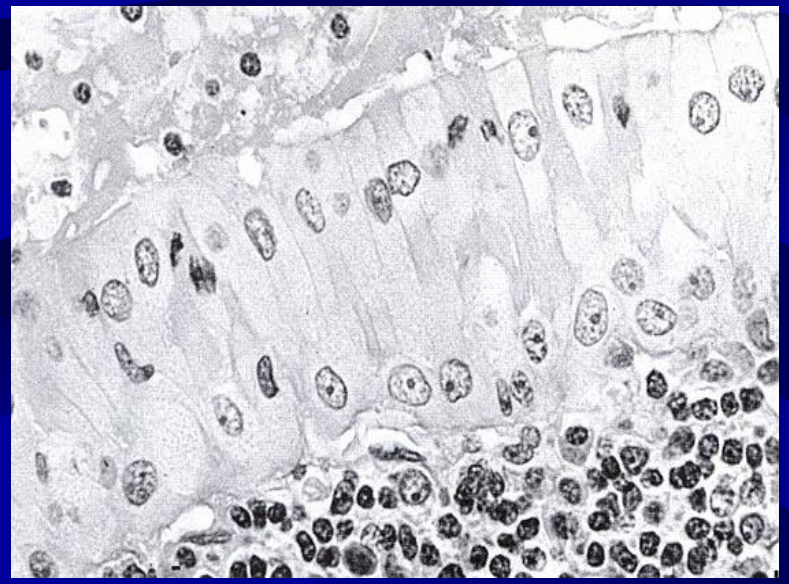
Warthin's Tumor

- Histology
  - Papillary projections into cystic spaces surrounded by lymphoid stroma
  - Epithelium: double cell layer
    - Luminal cells
    - Basal cells
  - Stroma: mature lymphoid follicles with germinal centers



Warthin's Tumor





Oncocytoma

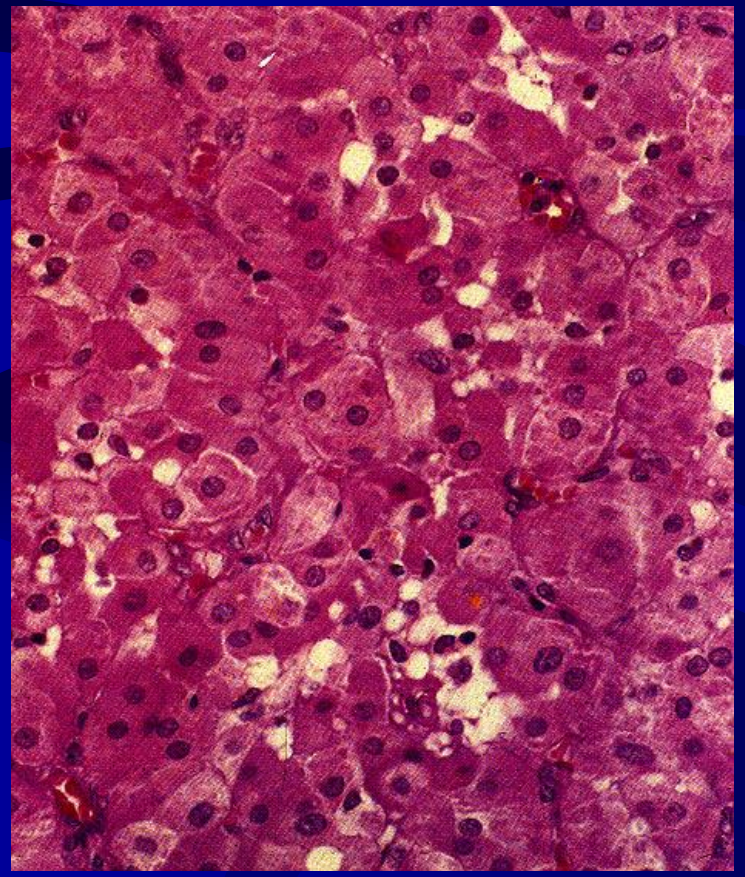
- Rare: 2.3% of benign salivary tumors
- 6^{th} decade
- M:F = 1:1
- Parotid: 78%
- Submandibular gland: 9%
- Minor salivary glands: palate, buccal mucosa, tongue

Oncocytoma

- Presentation
  - Enlarging, painless mass
- Technetium-99m pertechnetate scintigraphy
  - Mitochondrial hyperplasia

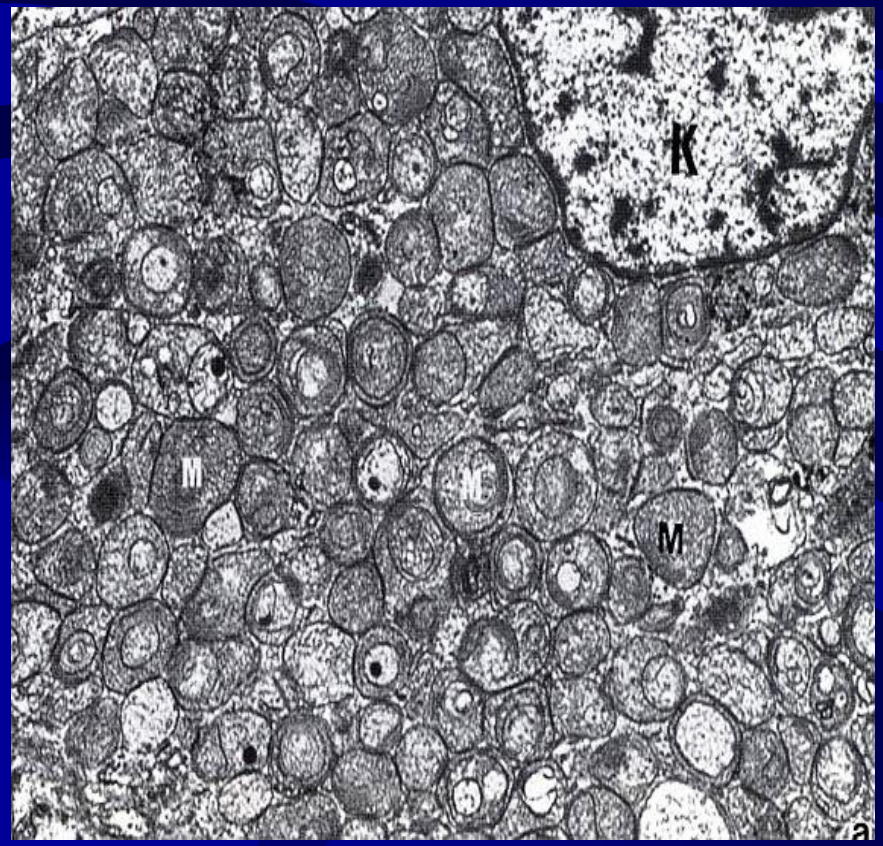
Oncocytoma

- Gross
  - Encapsulated
  - Homogeneous, smooth
  - Orange/rust color
- Histology
  - Cords of uniform cells and thin fibrous stroma
  - Large polyhedral cells
  - Distinct cell membrane
  - Granular, eosinophilic cytoplasm
  - Central, round, vesicular nucleus



Oncocytoma

- Electron microscopy:
  - Mitochondrial hyperplasia
  - 60% of cell volume
  - Most tumor diagnosis- FNA



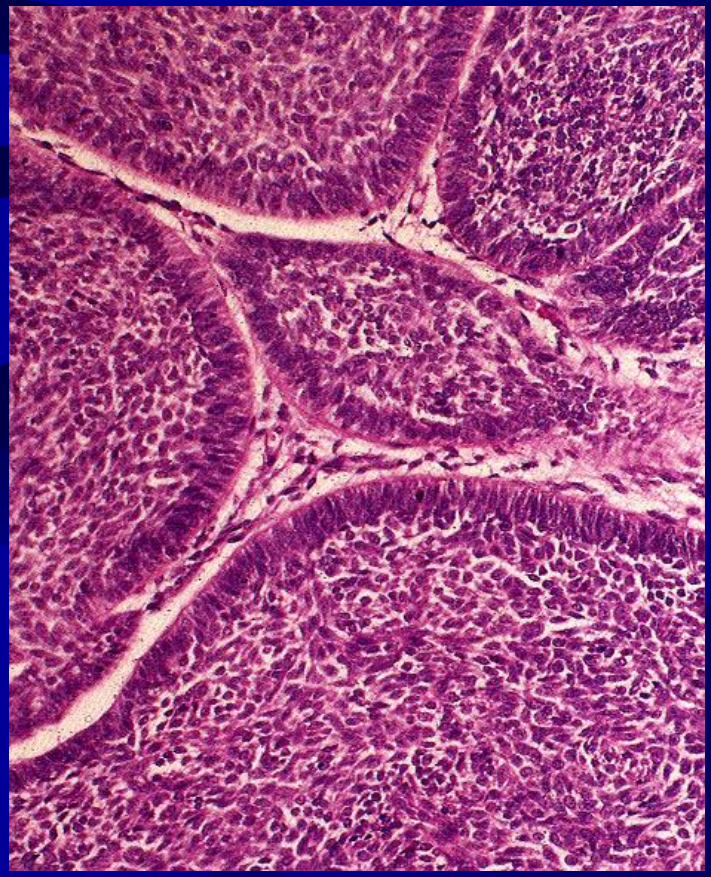
Monomorphic Adenomas

- Basal cell, canalicular, sebaceous, glycogen-rich, clear cell

- Basal cell is most common: 1.8% of benign epithelial salivary gland neoplasms
- 6^{th} decade
- M:F = approximately 1:1
- Caucasian > African American
- Most common in parotid

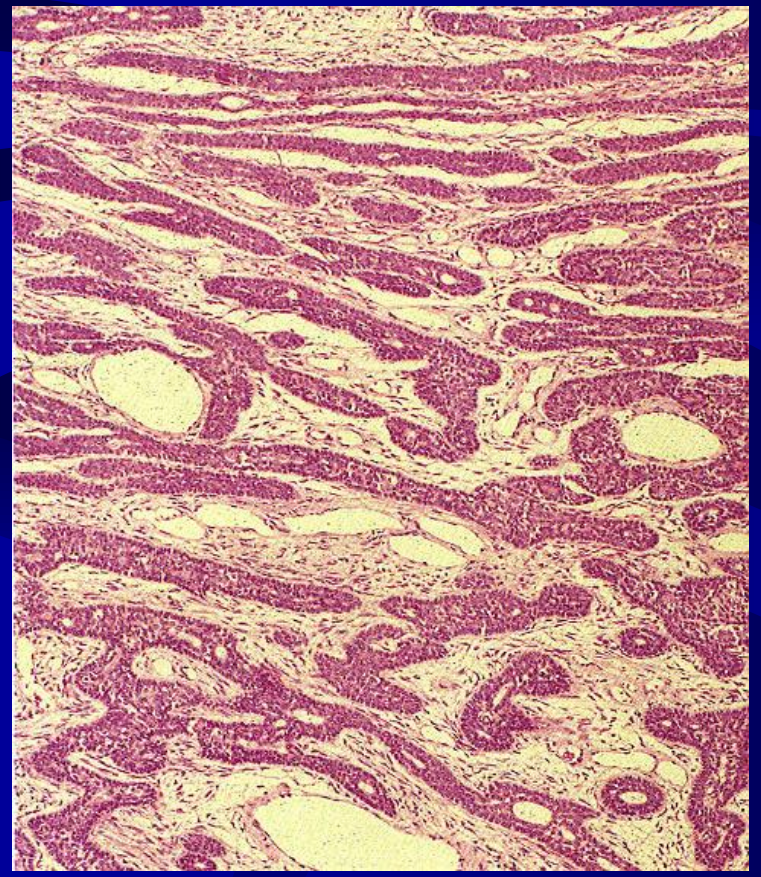
Basal Cell Adenoma

- Solid
  - Most common
  - Solid nests of tumor cells
  - Uniform, hyperchromatic, round nuclei, indistinct cytoplasm
  - Peripheral nuclear palisading
  - Scant stroma



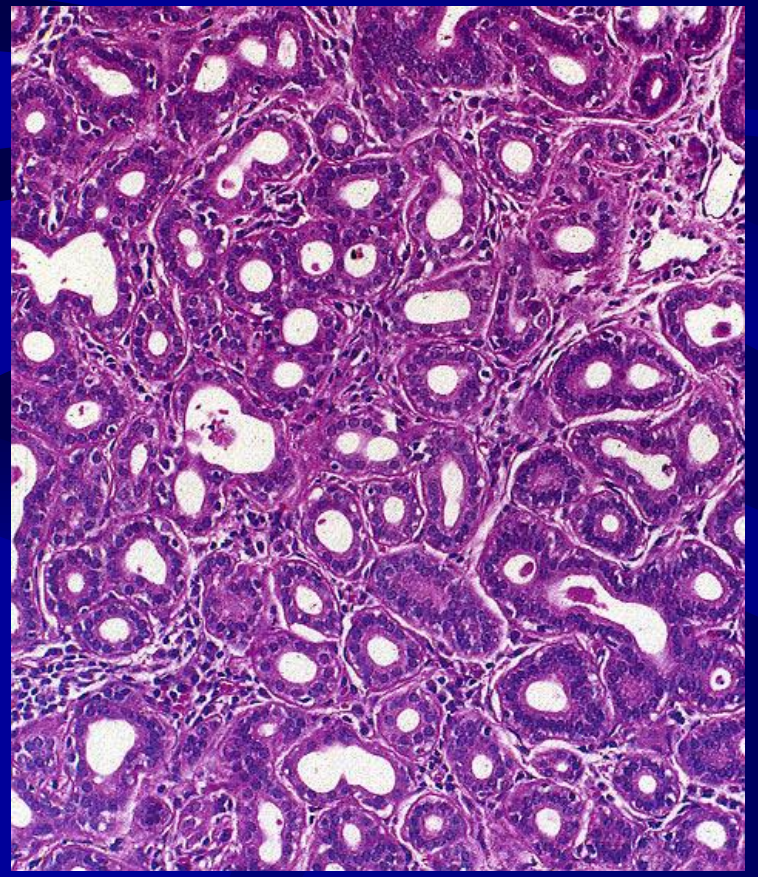
Basal Cell Adenoma

- Trabecular
  - Cells in elongated trabecular pattern
  - Vascular stroma



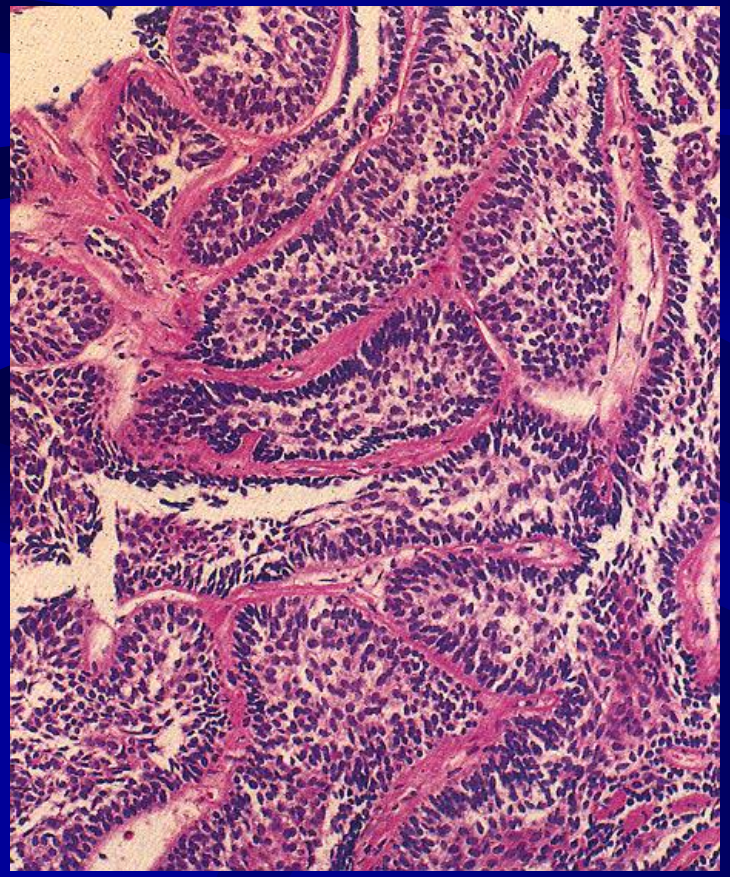
Basal Cell Adenoma

- Tubular
  - Multiple duct-like structures
  - Columnar cell lining
  - Vascular stroma



Basal Cell Adenoma

- Membranous
  - Thick eosinophilic hyaline membranes surrounding nests of tumor cells
  - “jigsaw-puzzle” appearance

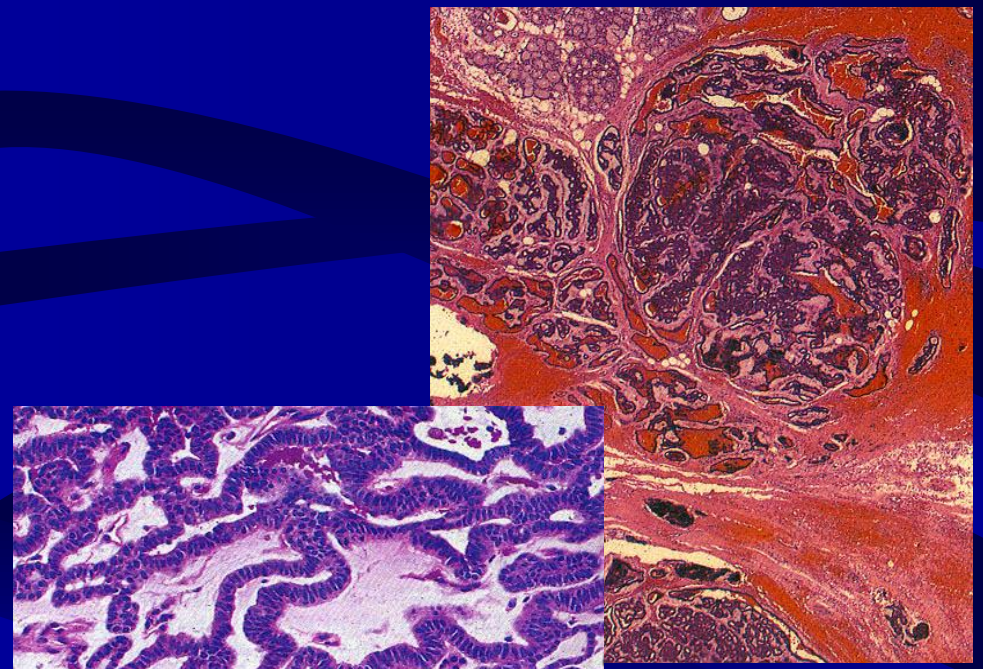


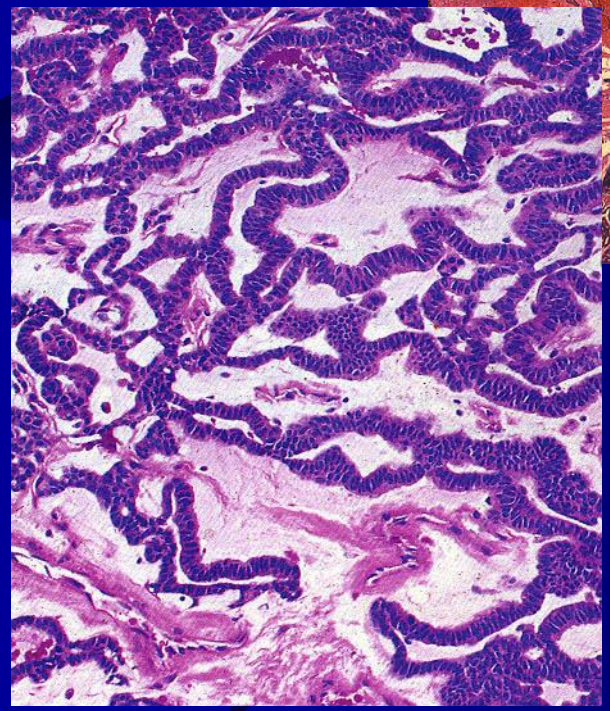
Monomorphic Adenomas

- Canalicular adenoma
  - 7^{th} decade
  - F:M – 1.8:1
  - Most common in minor salivary glands of the upper lip (74%)
  - Painless submucosal mass

Canalicular Adenoma

- Histology
  - Well-circumscribed
  - Multiple foci
  - Tubular structures line by columnar or cuboidal cells
  - Vascular stroma



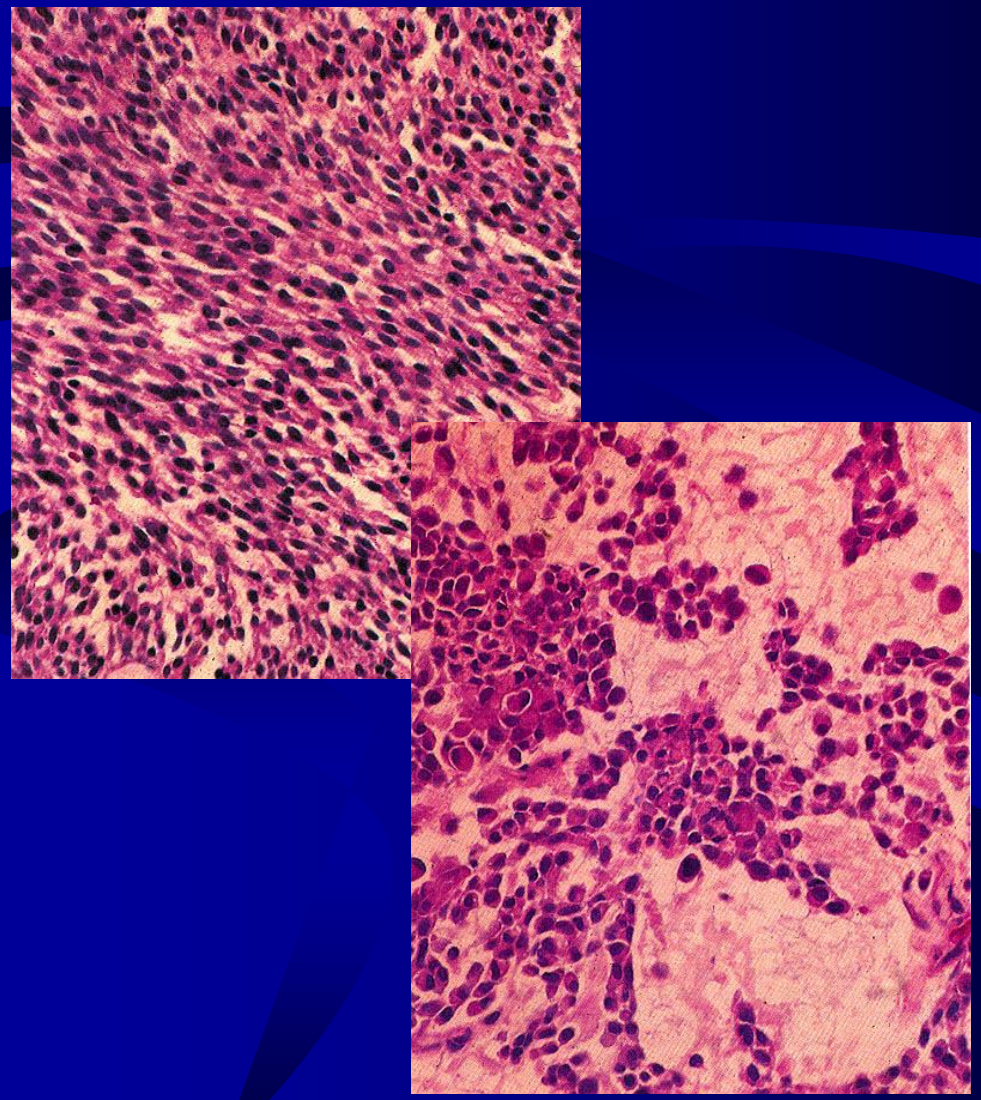


Myoepithelioma

- <1% of all salivary neoplasms
- 3rd-6th decades
- F>M
- Minor salivary glands > parotid > submandibular gland
- Presentation: asymptomatic mass

Myoepithelioma

- Histology
  - Spindle cell
    - More common
    - Parotid
    - Uniform, central nuclei
    - Eosinophilic granular or fibrillar cytoplasm
  - Plasmacytoid cell
    - Polygonal
    - Eccentric oval nuclei



MALIGNANT TUMORS

Mucoepidermoid Carcinoma

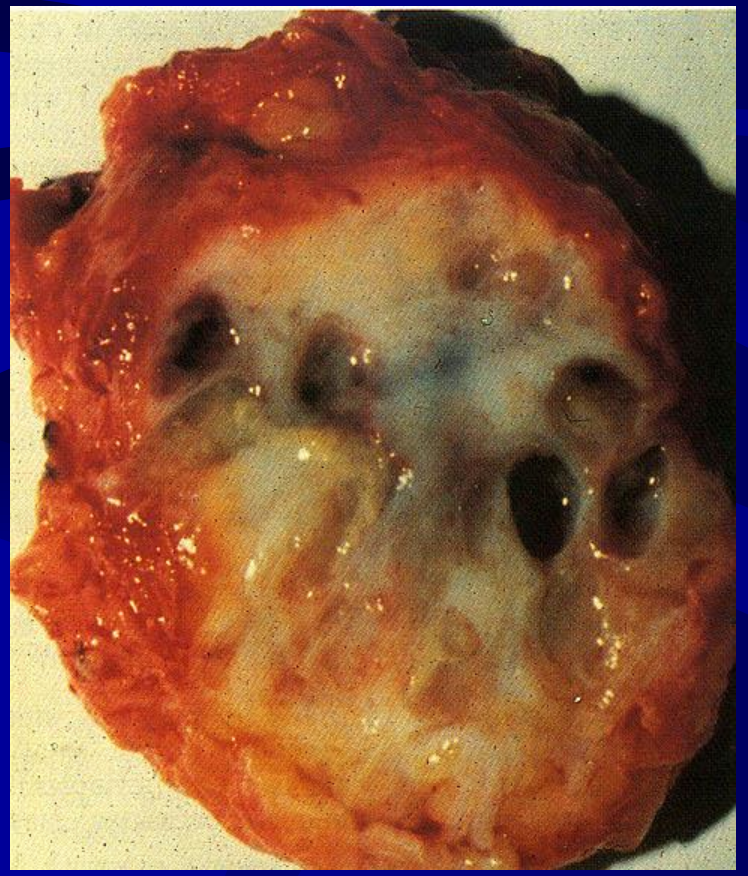
- Most common salivary gland malignancy in children
- 5-9% of salivary neoplasms
- Parotid 45-70% of cases
- Palate 18%
- 3rd-8th decades, peak in 5th decade
- F>M
- Caucasian > African American

Mucoepidermoid Carcinoma

- Presentation
  - Low-grade: slow growing, painless mass
  - High-grade: rapidly enlarging, +/- pain
  - **Minor salivary glands: may be mistaken for benign or inflammatory process
    - Hemangioma
    - Papilloma
    - Tori

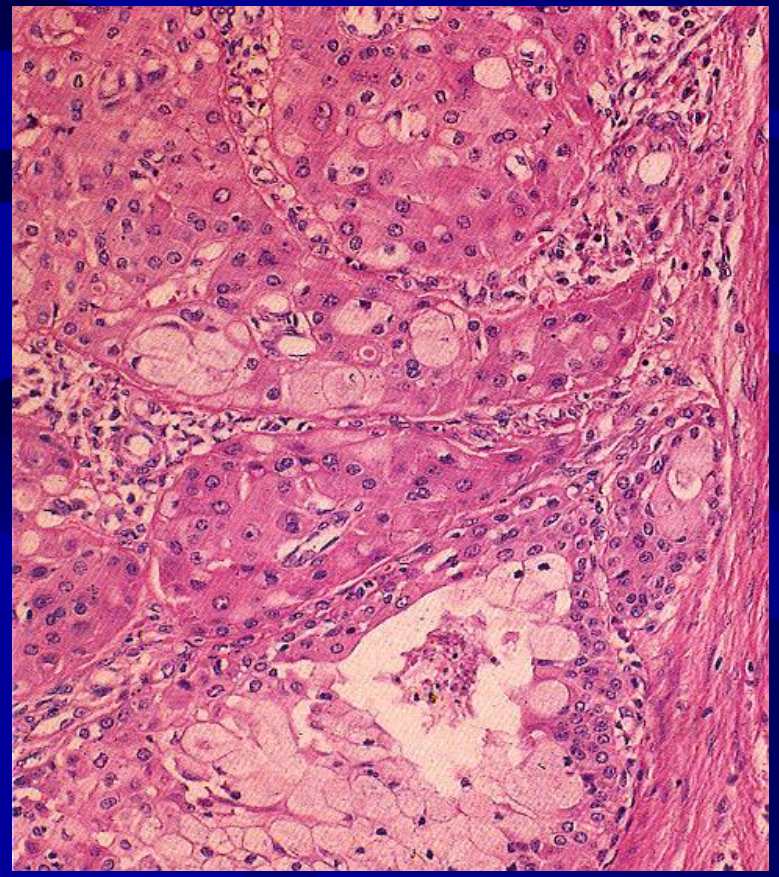
Mucoepidermoid Carcinoma

- Gross pathology
  - Well-circumscribed to partially encapsulated to unencapsulated
  - Solid tumor with cystic spaces



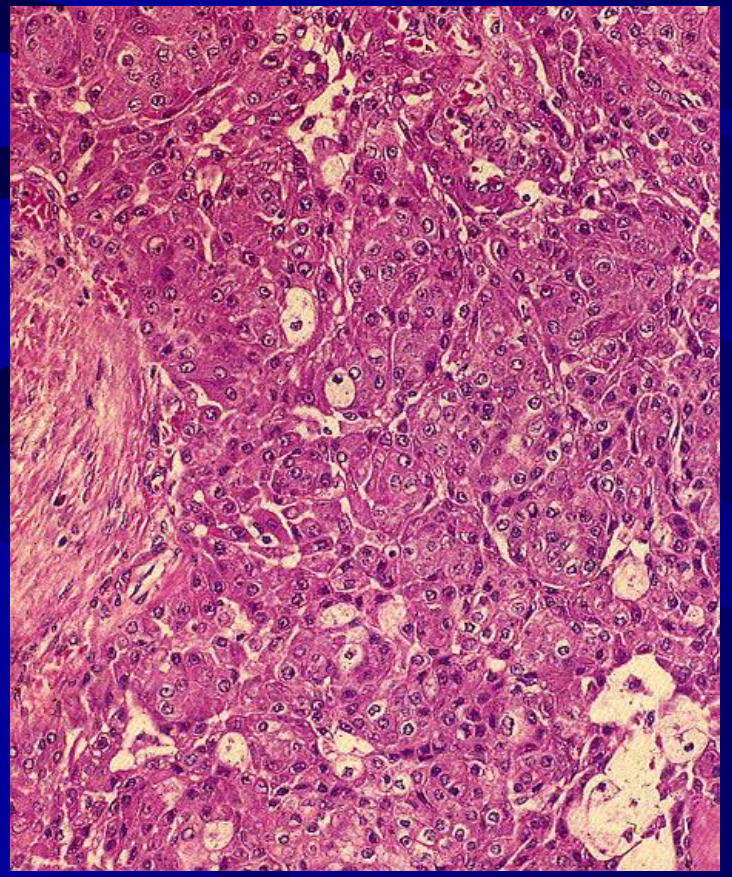
Mucoepidermoid Carcinoma

- Histology—Low-grade
  - Mucus cell > epidermoid cells
  - Prominent cysts
  - Mature cellular elements



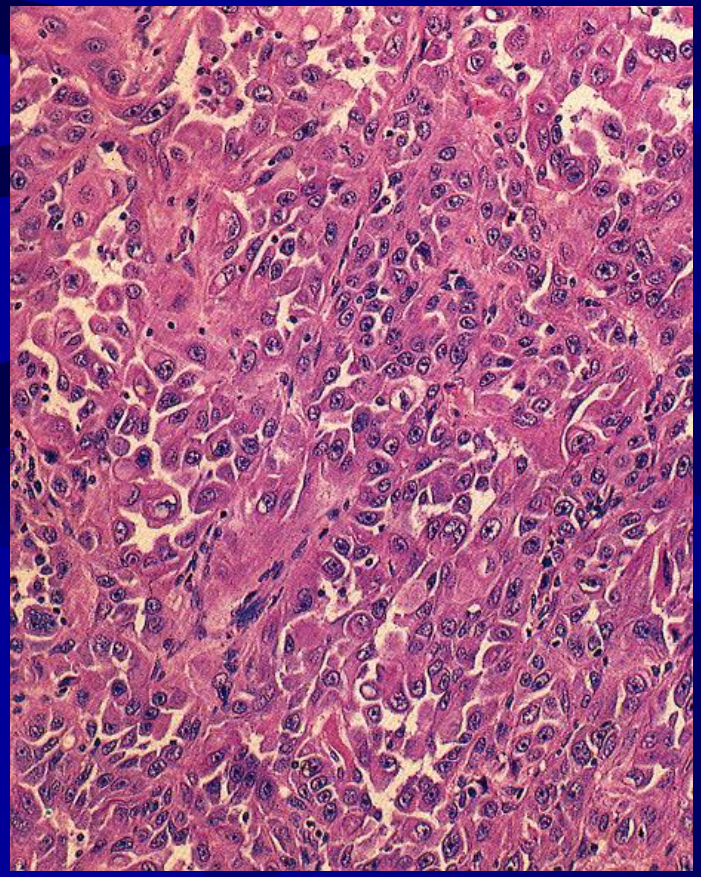
Mucoepidermoid Carcinoma

- Histology—Intermediate-grade
  - Mucus = epidermoid
  - Fewer and smaller cysts
  - Increasing pleomorphism and mitotic figures



Mucoepidermoid Carcinoma

- Histology—High-grade
  - Epidermoid > mucus
  - Solid tumor cell proliferation
  - Mistaken for SCCA
    - Mucin staining



Mucoepidermoid Carcinoma

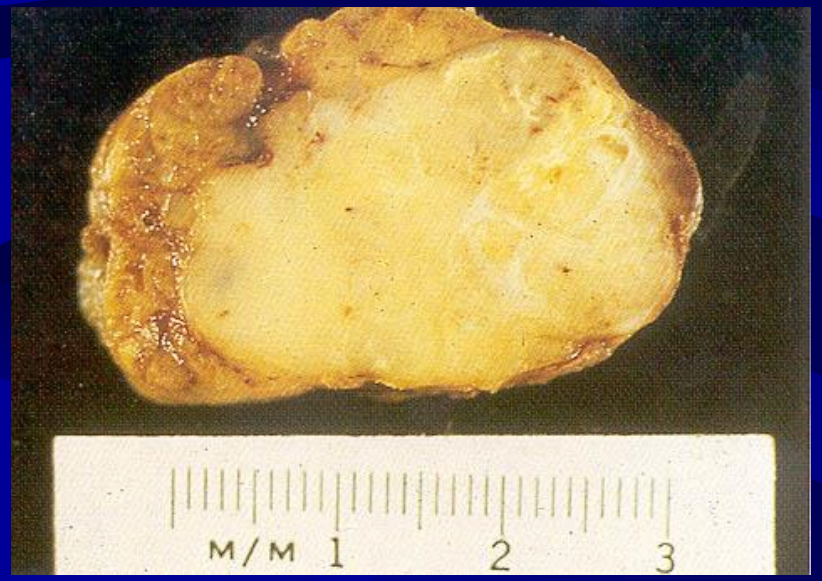
- Treatment
  - Influenced by site, stage, grade
  - Stage I & II
    - Wide local excision
  - Stage III & IV
    - Radical excision
    - +/- neck dissection
    - +/- postoperative radiation therapy

Adenoid Cystic Carcinoma

- Overall most common malignancy
- Most common in submandibular, sublingual and minor salivary glands
- M = F
- 5^{th} decade
- Presentation
  - Asymptomatic enlarging mass
  - Pain, paresthesias, facial weakness/paralysis

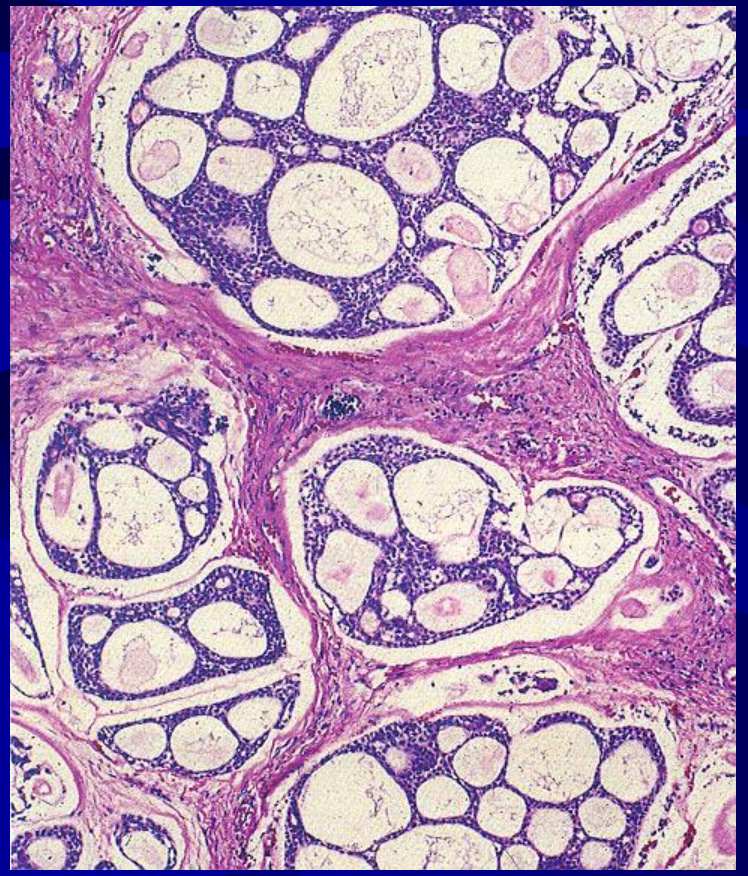
Adenoid Cystic Carcinoma

- Gross pathology
  - Well-circumscribed
  - Solid, rarely with cystic spaces
  - infiltrative



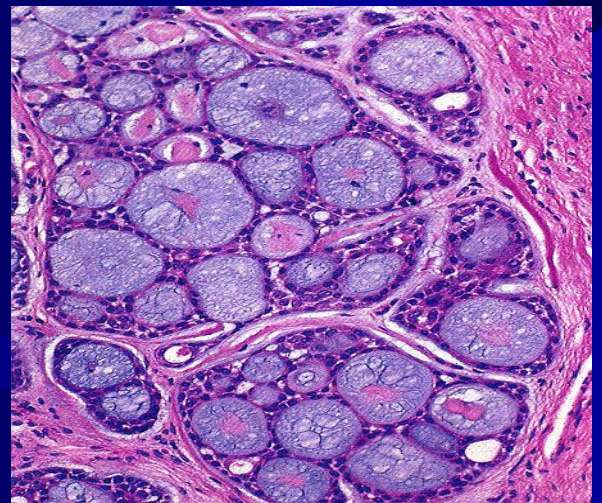
Adenoid Cystic Carcinoma

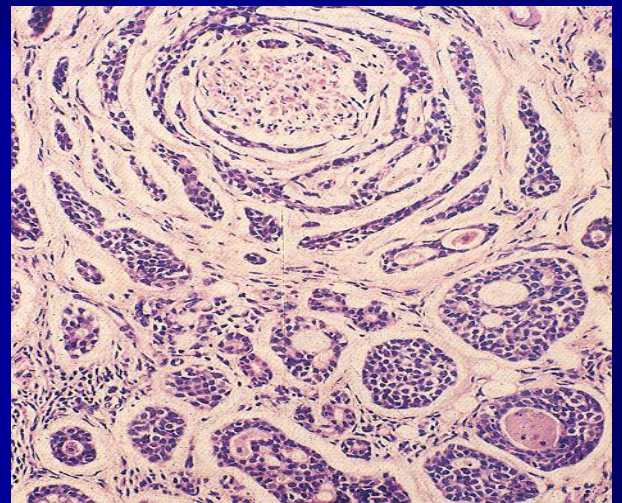
- Histology—cribriform pattern
  - Most common
  - “swiss cheese” appearance



Adenoid Cystic Carcinoma

- Histology—tubular pattern
  - Layered cells forming duct-like structures
  - Basophilic mucinous substance
- Histology—solid pattern
  - Solid nests of cells without cystic or tubular spaces





Adenoid Cystic Carcinoma

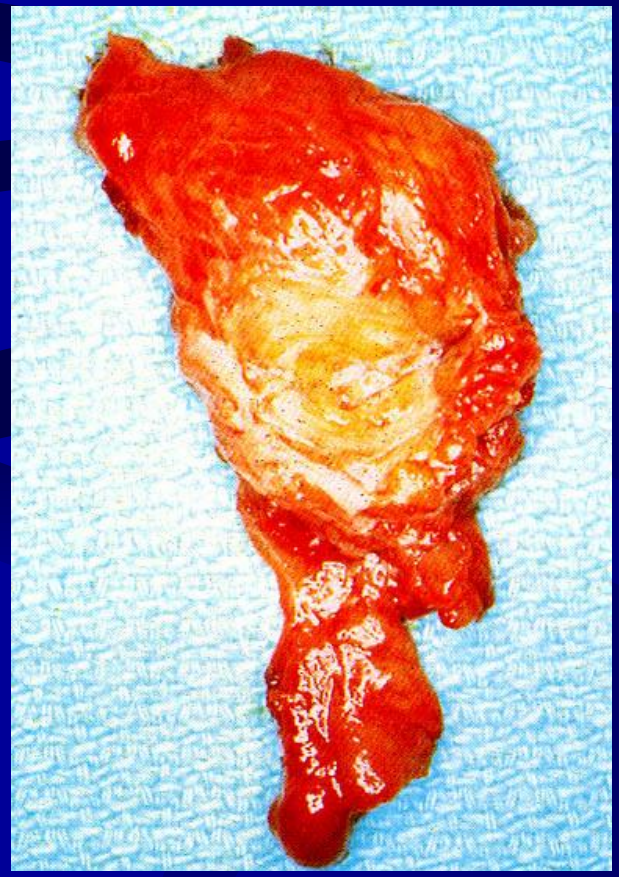
- Treatment
  - Complete local excision
  - Tendency for perineural invasion: facial nerve sacrifice
  - Postoperative XRT
- Prognosis
  - Local recurrence: 42%
  - Distant metastasis: lung
  - Indolent course: 5-year survival 75%, 20-year survival 13%

Acinic Cell Carcinoma

- 2nd most common parotid and pediatric malignancy
- 5th decade
- F>M
- Bilateral parotid disease in 3%
- Presentation
  - Solitary, slow-growing, often painless mass

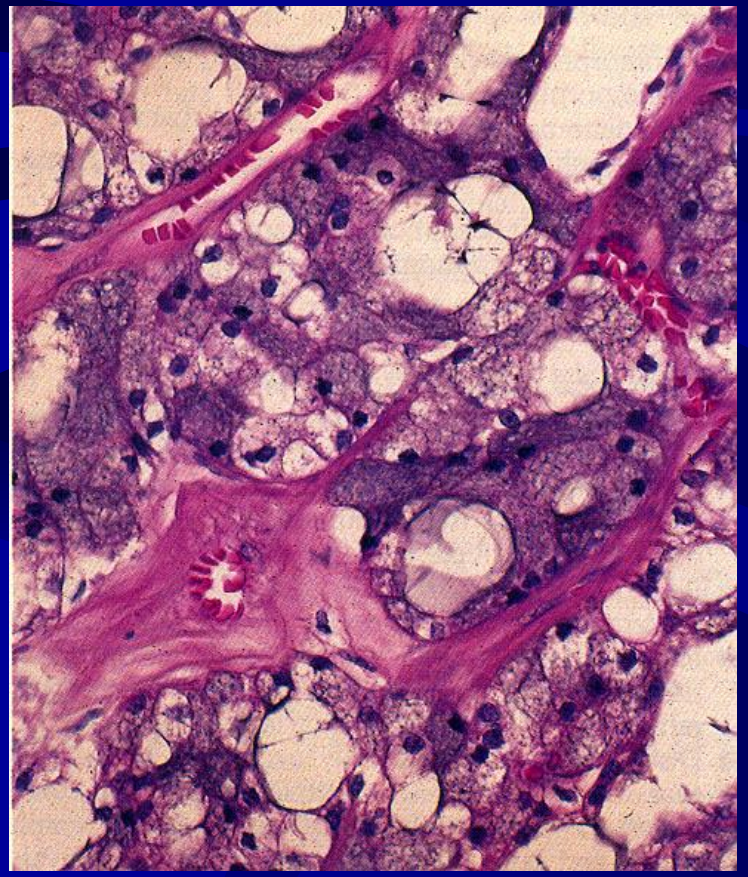
Acinic Cell Carcinoma

- Gross pathology
  - Well-demarcated
  - Most often homogeneous



Acinic Cell Carcinoma

- Histology
  - Solid and microcystic patterns
    - Most common
    - Solid sheets
    - Numerous small cysts
  - Polyhedral cells
  - Small, dark, eccentric nuclei
  - Basophilic granular cytoplasm



Acinic Cell Carcinoma

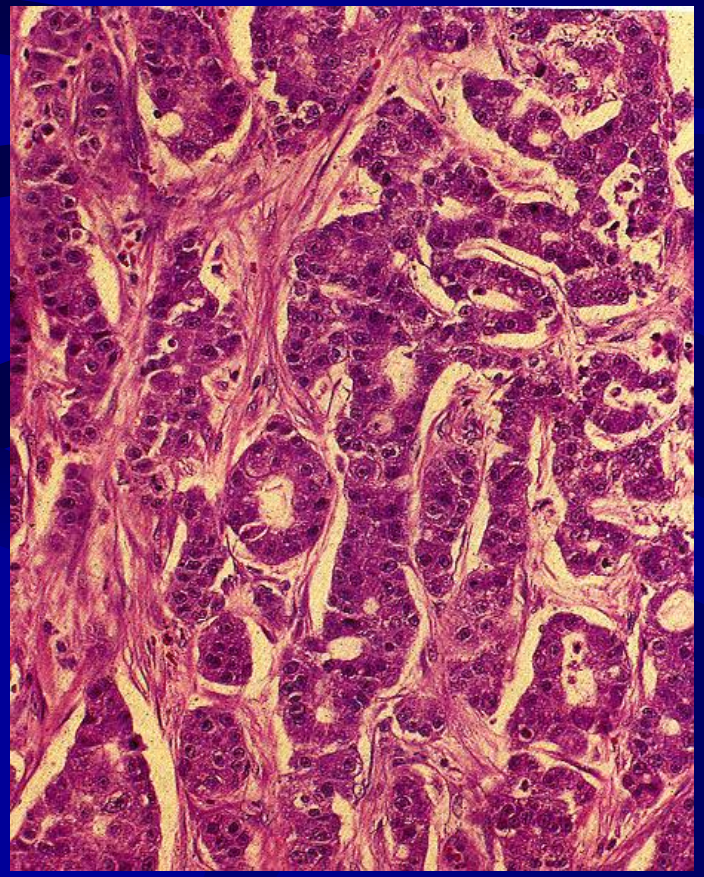
- Treatment
  - Complete local excision
  - +/- postoperative XRT
- Prognosis
  - 5-year survival: 82%
  - 10-year survival: 68%
  - 25-year survival: 50%

Adenocarcinoma

- Rare
- 5th to 8th decades
- F > M
- Parotid and minor salivary glands
- Presentation:
  - Enlarging mass
  - 25% with pain or facial weakness

Adenocarcinoma

- Histology
  - Heterogeneity
  - Presence of glandular structures and absence of epidermoid component
  - Grade I
  - Grade II
  - Grade III



Adenocarcinoma

- Treatment
  - Complete local excision
  - Neck dissection
  - Postoperative XRT
- Prognosis
  - Local recurrence: 51%
  - Regional metastasis: 27%
  - Distant metastasis: 26%
  - 15-year cure rate:
    - Stage I = 67%
    - Stage II = 35%
    - Stage III = 8%

Malignant Mixed Tumors

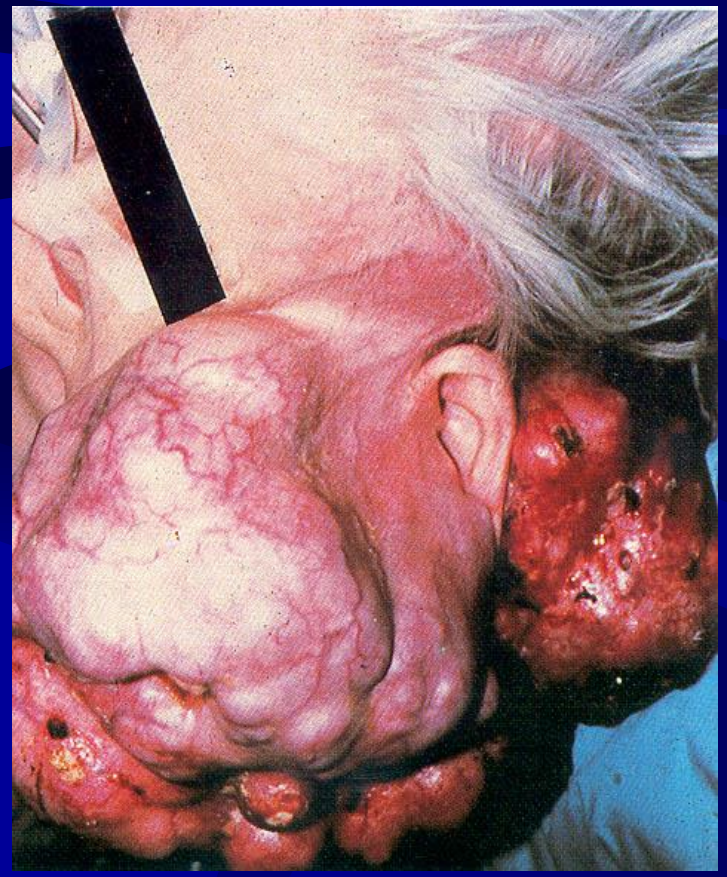
- Carcinoma ex-pleomorphic adenoma
  - Carcinoma developing in the epithelial component of preexisting pleomorphic adenoma
- Carcinosarcoma
  - True malignant mixed tumor—carcinomatous and sarcomatous components
- Metastatic mixed tumor
  - Metastatic deposits of otherwise typical pleomorphic adenoma

Carcinoma Ex-Pleomorphic Adenoma

- 2-4% of all salivary gland neoplasms
- 4-6% of mixed tumors
- 6^{th}-8^{th} decades
- Parotid > submandibular > palate
- Risk of malignant degeneration
  - 1.5% in first 5 years
  - 9.5% after 15 years
- Presentation
  - Longstanding painless mass that undergoes sudden enlargement and pain

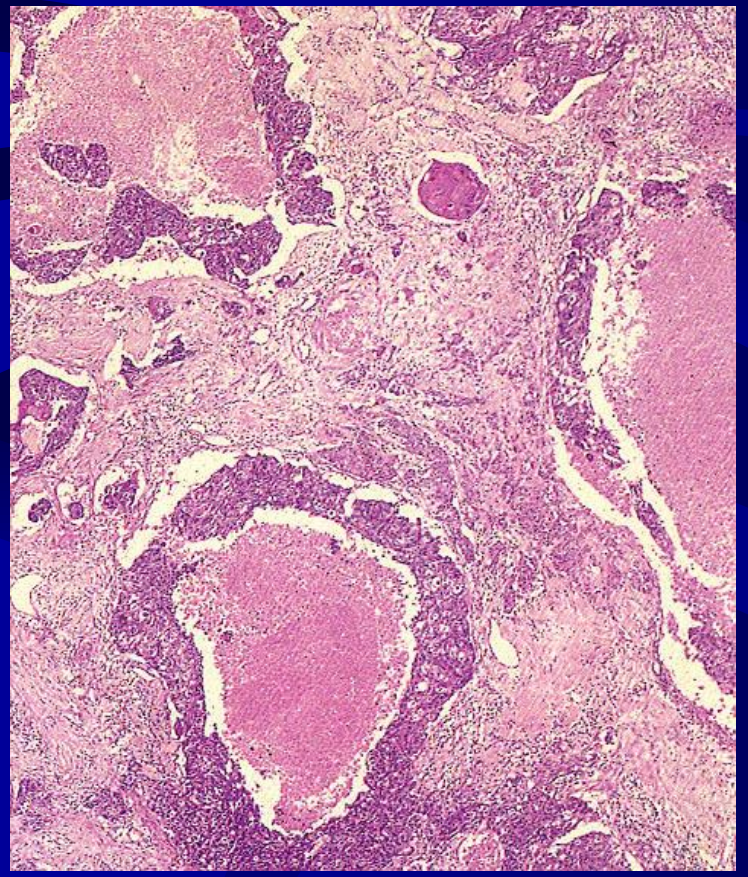
Carcinoma Ex-Pleomorphic Adenoma

- Gross pathology
  - Poorly circumscribed
  - Infiltrative
  - Hemorrhage and necrosis (grows too fast for its blood supply)



Carcinoma Ex-Pleomorphic Adenoma

- Histology
  - Malignant cellular change adjacent to typical pleomorphic adenoma
  - Carcinomatous component
    - Adenocarcinoma
    - Undifferentiated/ anaplastic



Carcinoma Ex-Pleomorphic Adenoma

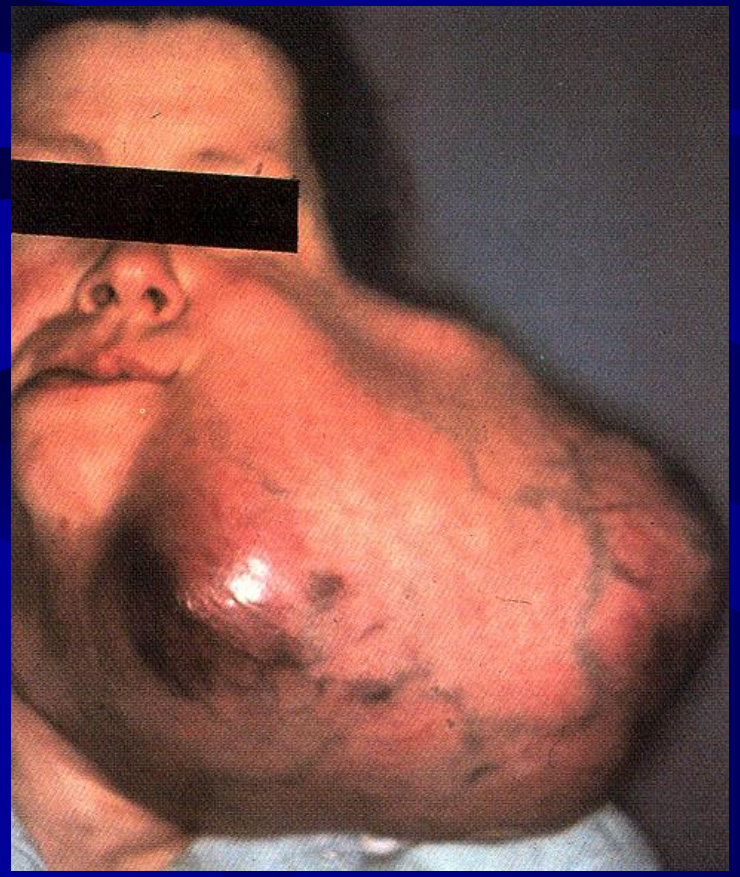
- Treatment
  - Radical excision
  - Neck dissection (25% with lymph node involvement at presentation)
  - Postoperative XRT
- Prognosis
  - Dependent upon stage and histology

Carcinosarcoma

- Rare: <.05% of salivary gland neoplasms
- 6th decade
- M = F
- Parotid
- History of previously excised pleomorphic adenoma, recurrent pleomorphic adenoma or recurring pleomorphic treated with XRT
- Presentation
- Clinical diagnosis

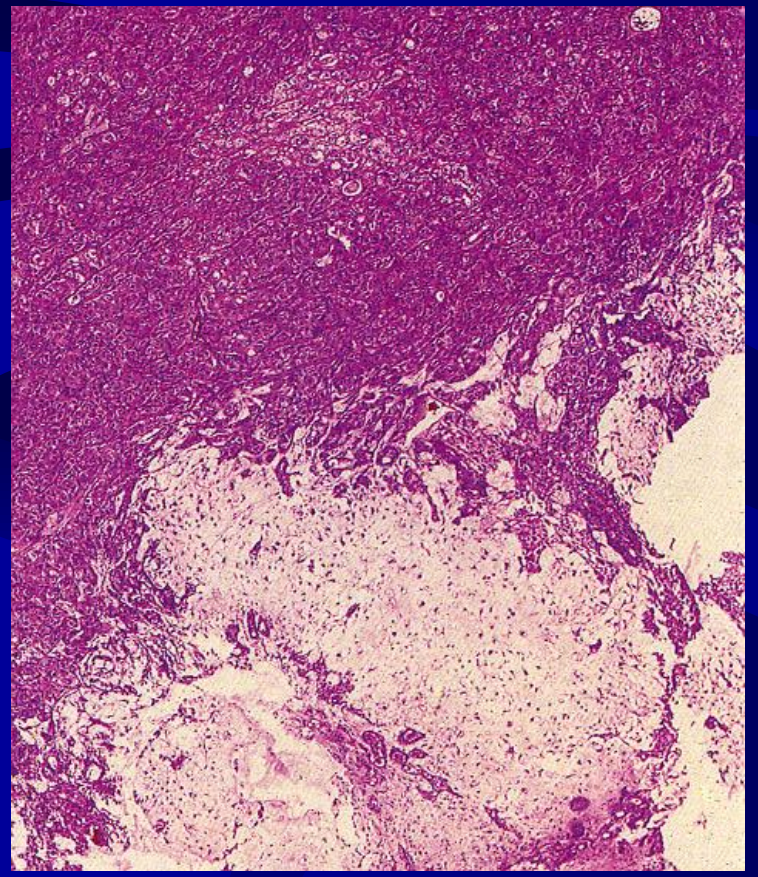
Carcinosarcoma

- Gross pathology
  - Poorly circumscribed
  - Infiltrative
  - Cystic areas
  - Hemorrhage, necrosis
  - Calcification
  - Usually infiltrated the mandible



Carcinosarcoma

- Histology
  - Biphasic appearance
  - Sarcomatous component
    - Dominant
    - chondrosarcoma
  - Carinomatous component
    - Moderately to poorly differentiated ductal carcinoma
    - Undifferentiated



Carcinosarcoma

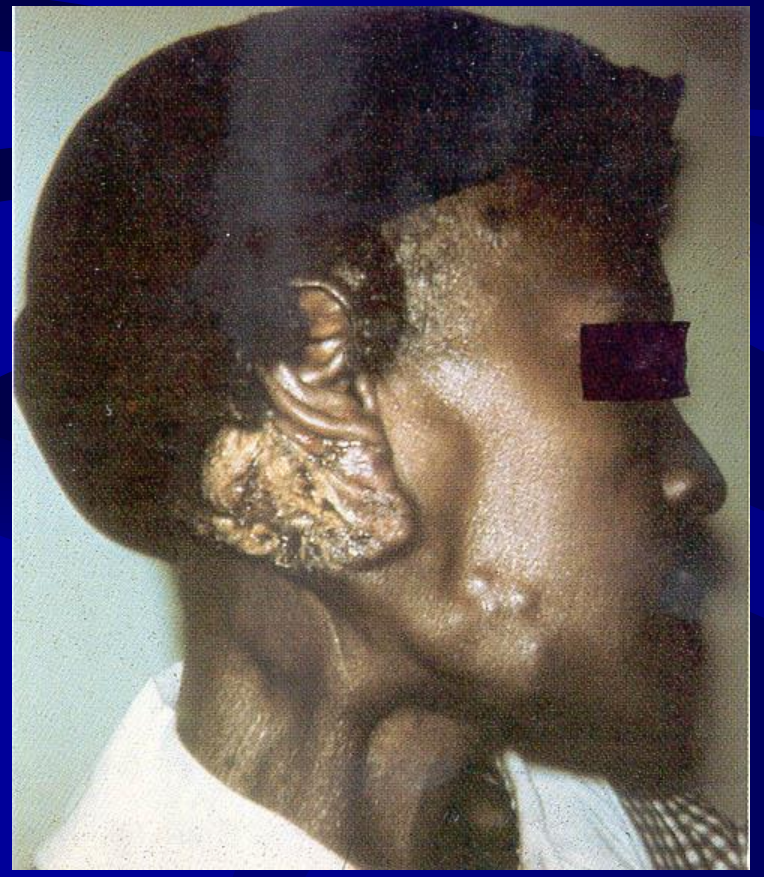
- Treatment
  - Radical excision
  - Neck dissection
  - Postoperative XRT
  - Chemotherapy (distant metastasis to lung, liver, bone, brain)
- Prognosis
  - Poor, average survival less than 2 ½ years

Squamous Cell Carcinoma

- 1.6% of salivary gland neoplasms
- 7^{th}-8^{th} decades
- M:F = 2:1
- MUST RULE OUT:
  - High-grade mucoepidermoid carcinoma
  - Metastatic SCCA to intraglandular nodes
  - Direct extension of SCCA

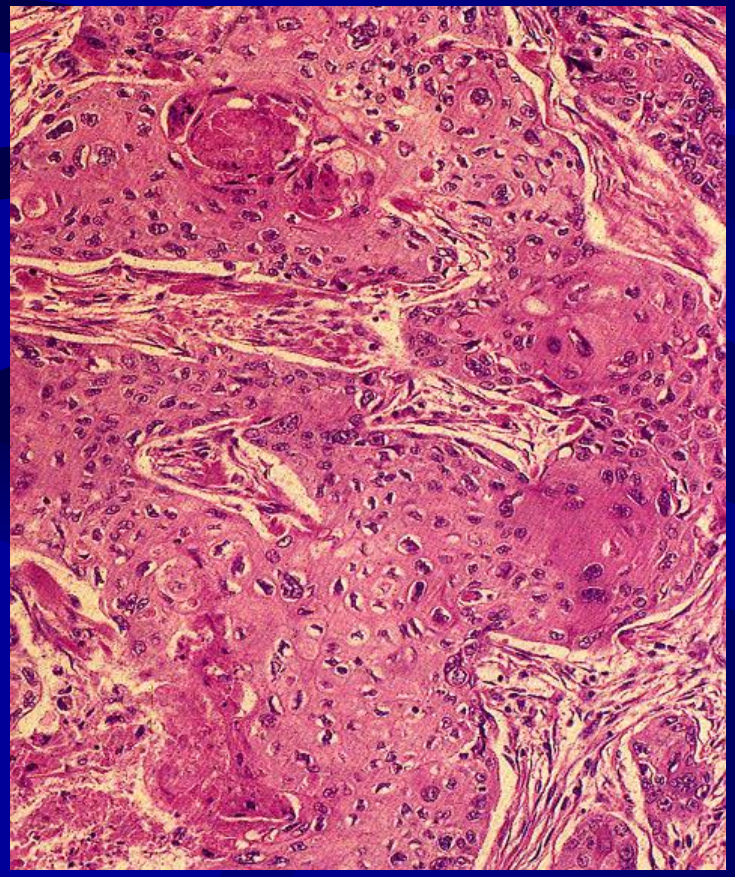
Squamous Cell Carcinoma

- Gross pathology
  - Unencapsulated
  - Ulcerated
  - fixed



Squamous Cell Carcinoma

- Histology
  - Infiltrating
  - Nests of tumor cells
  - Well differentiated
    - Keratinization
  - Moderately-well differentiated
  - Poorly differentiated
    - No keratinization

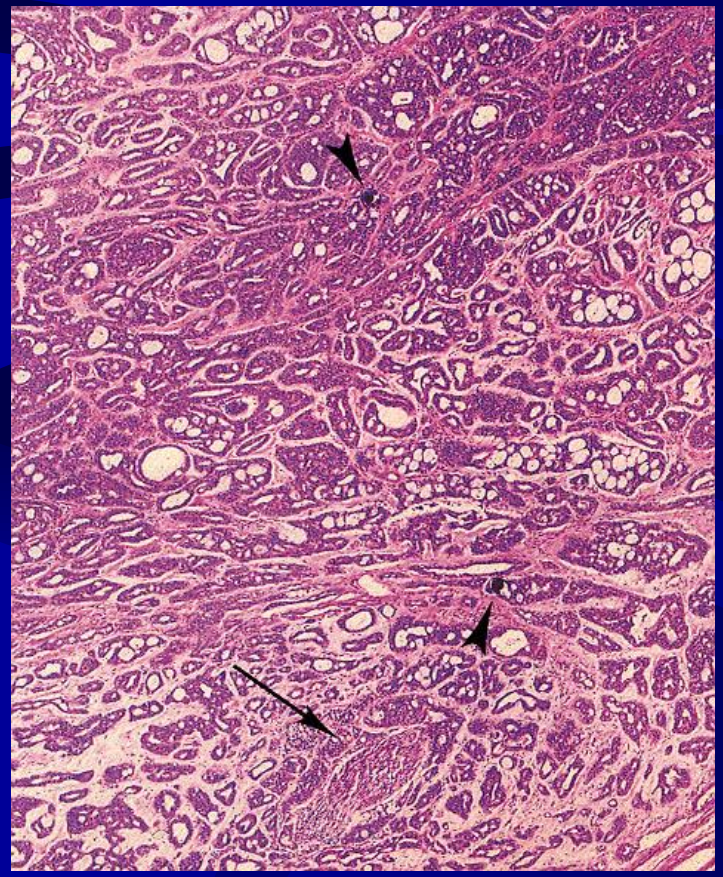


Squamous Cell Carcinoma

- Treatment
  - Radical excision
  - Neck dissection
  - Postoperative XRT
- Prognosis
  - 5-year survival: 24%
  - 10-year survival: 18%

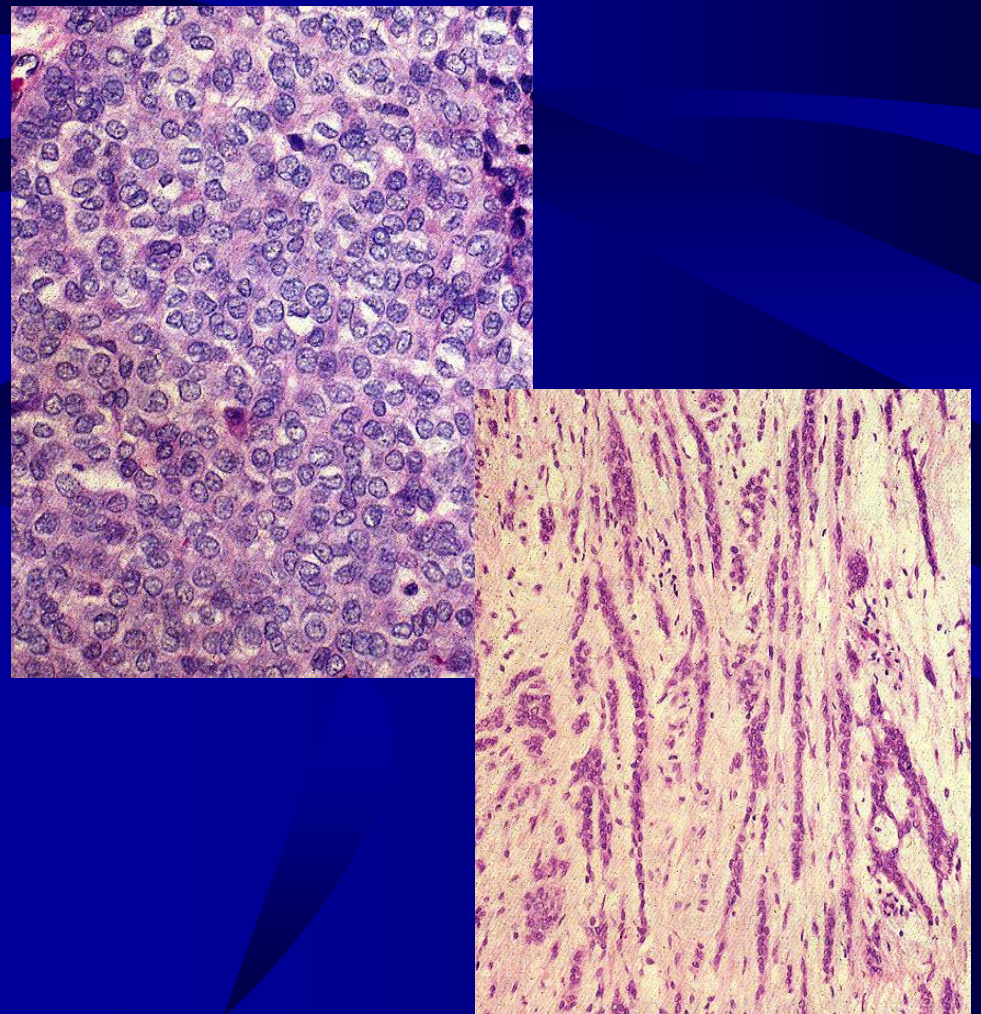
Polymorphous Low-Grade Adenocarcinoma

- 2nd most common malignancy in minor salivary glands
- 7th decade
- F > M
- Painless, submucosal mass
- Morphologic diversity
    - Solid, glandular, cribriform, ductular, tubular, trabecular, cystic



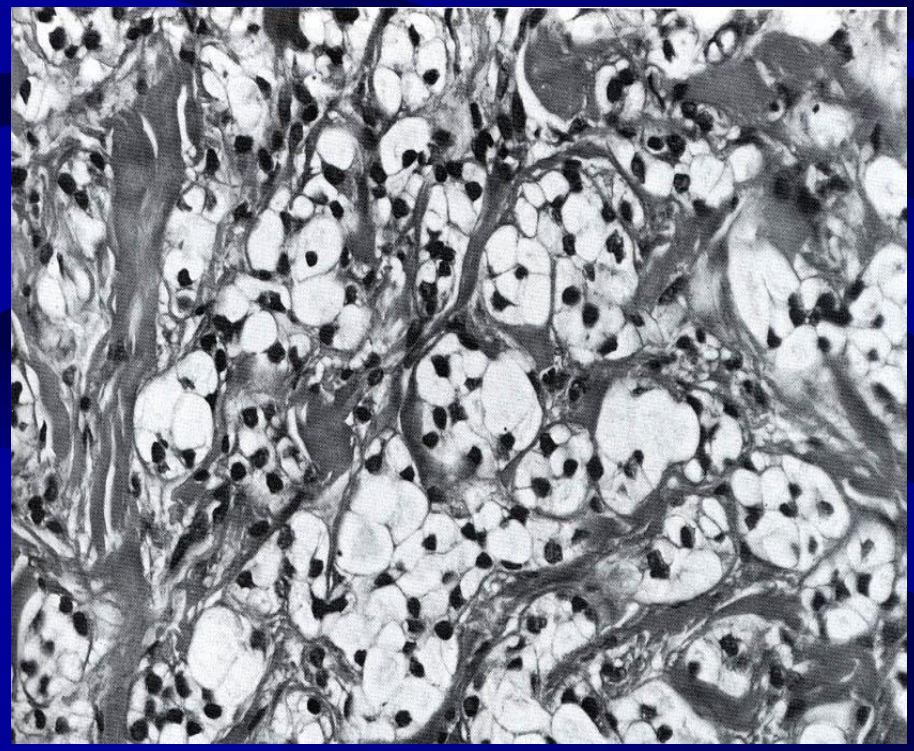
Polymorphous Low-Grade Adenocarcinoma

- Histology
  - Isomorphic cells, indistinct borders, uniform nuclei
  - Peripheral “Indian-file” pattern
- Treatment
  - Complete yet conservative excision



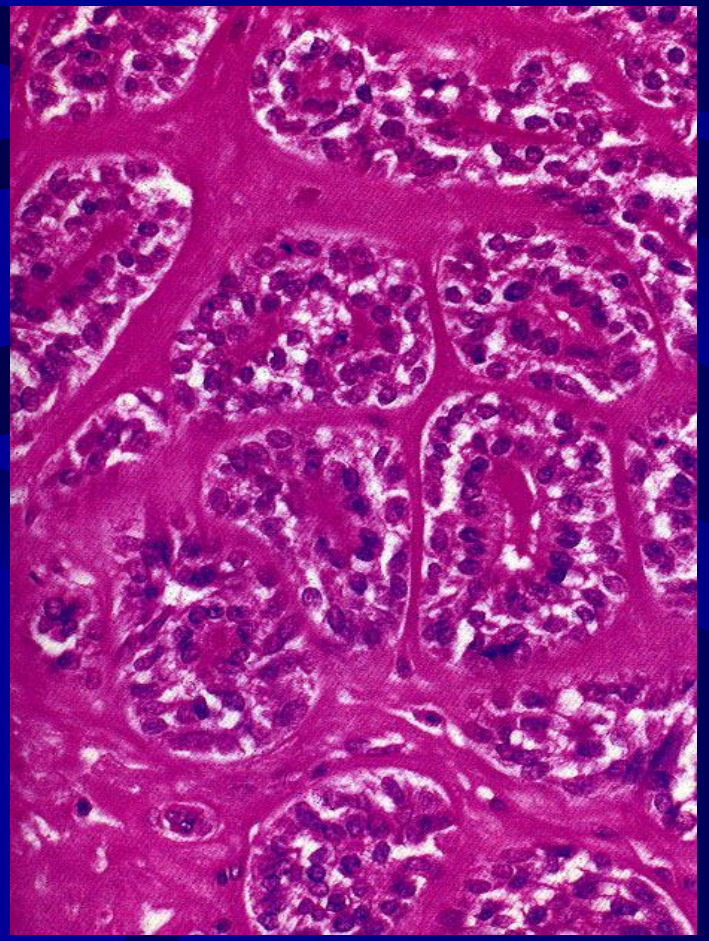
Clear Cell Carcinoma

- AKA glycogen-rich
- Palate and parotid
- 6^{th}-8^{th} decade
- M = F
- Histology
  - Uniform, round or polygonal cells
  - Peripheral dark nuclei
  - Clear cytoplasm
- Treatment
  - Complete local excision
  - Post surgical radiotherapy



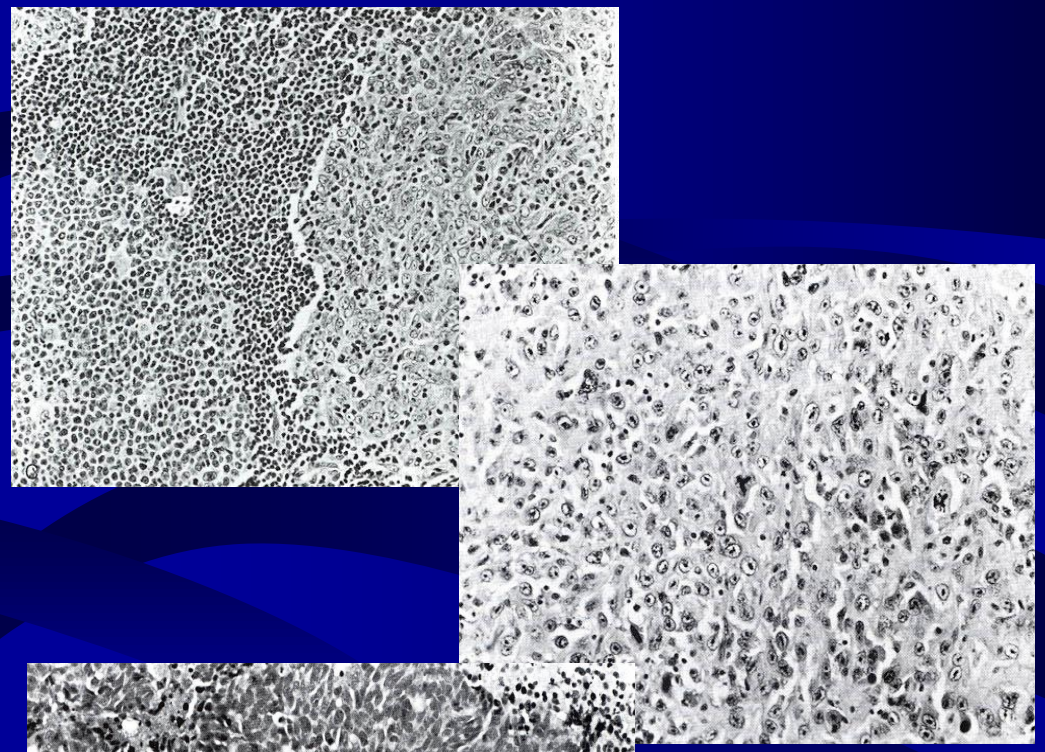
Epithelial-Myoepithelial Carcinoma

- < 1% of salivary neoplasms
- 6^{th}-7^{th} decades, F > M, parotid
- ? Increased risk for 2^{nd} primary
- Histology
  - Tumor cell nests
  - Two cell types
  - Thickened basement membrane
- Treatment
  - Surgical excision



Undifferentiated Carcinoma

- Lymphoepithelial
  - Eskimos: parotid, F > M, familial
  - Asian: submandibular, M > F
- Large-cell
  - Bimodal peaks
  - M > F
  - Parotid
- Small-cell
  - 6^{th}-7^{th} decades
  - M:F = 1.6:1
  - Parotid



Controversial Issues

- Management of the N0 Neck
  - Recurrence in the neck = low likelihood of salvage
  - Parotid: clinical neck disease, 16%
    - N- disease = 74% 5-year survival
    - N+ disease = 9% 5-year survival
  - Submandibular: clinical neck disease, 8%
    - N- disease = 41% 5-year survival
    - N+ disease = 9% 5-year survival
  - You'd like to do a neck dissection to be completely sure/ to determine aggressiveness of treatment

Management of the N0 Neck

- Increase risk of occult neck metastasis
  - **High-grade malignancies
  - **Advanced primary tumor stage (T3-T4)
  - High risk histology
    - Undifferentiated, SCCA, adenocarcinoma, high-grade mucoepidermoid, salivary duct carcinoma
  - Tumor size > 3cm
  - Patient > 54 years of age
  - Facial paralysis
  - Extracapsular, perilymphatic spread

Management of the N0 Neck

- Neck Dissection
  - Advantages
    - Pathologic staging
    - Improved counseling and prediction of prognosis
  - Disadvantages
    - Longer OR time, increase complications, increased cost
    - Functional deficits, cosmetic effects
  - Type
    - Parotid: levels II-IV
    - Submandibular: levels I-III

Management of the N0 Neck

- Radiation Therapy
  - Advantage
    - Avoids surgical sequelae
  - Disadvantages
    - Radiation effect on normal tissue
    - Radiation induced malignancies
  - Proponents argument: the same factors that increase the risk of occult neck disease also increase the risk for local recurrence and necessitate postoperative XRT to the primary so it is reasonable to treat the neck with XRT as well

Fine-Needle Aspiration Biopsy

- Efficacy is well established

- Accuracy = 84-97%
- Sensitivity = 54-95%
- Specificity = 86=100%

- Safe, well tolerated
- Plain biopsy will lead to upstaging the tumor

Fine-Needle Aspiration Biopsy

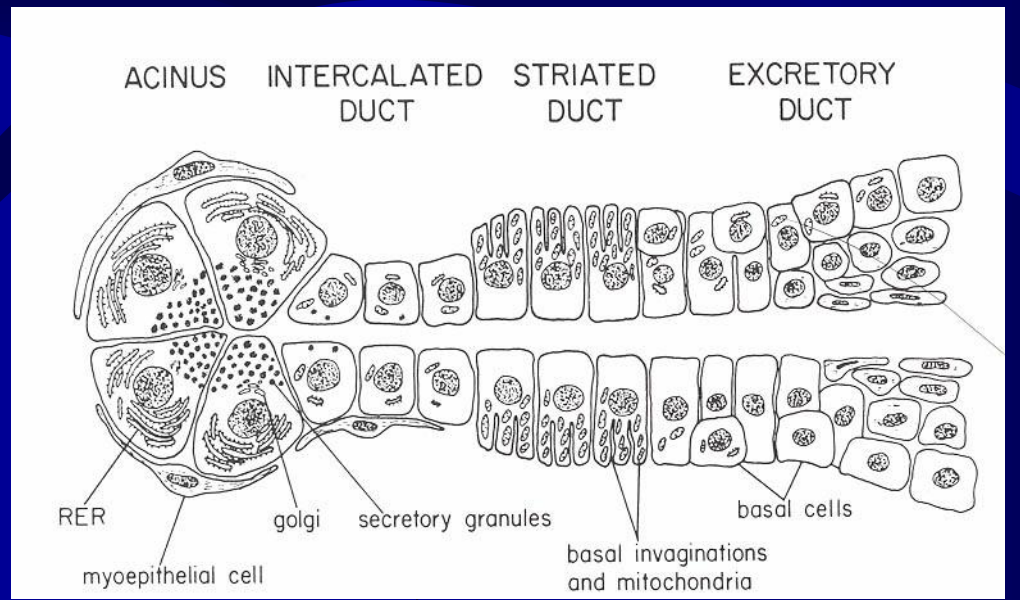
- Opponents argument:
  - Doesn’t change management
    - Surgery regardless of reported diagnosis
  - Obscuring final pathologic diagnosis
  - Frequency of “inadequate” sampling, requires multiple biopsies, prolongs course until definitive treatment, increases cost

Fine-Needle Aspiration Biopsy

- Proponent's argument:
  - Important to distinguish benign vs. malignant nature of neoplasm
  - Preoperative patient counseling
  - Surgical planning
  - Differentiate between neoplastic and non-neoplastic processes
    - Avoid surgery in large number of patients

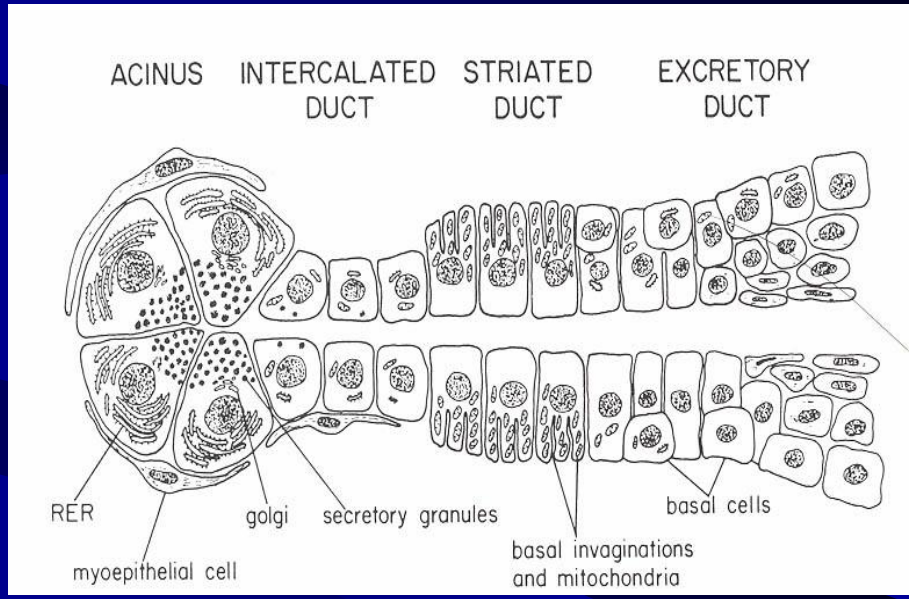
Bicellular Theory

- Intercalated Ducts
  - Pleomorphic adenoma
  - Warthin's tumor
  - Oncocytoma
  - Acinic cell
  - Adenoid cystic
- Excretory Ducts
  - Squamous cell
  - Mucoepidermoid



Multicellular Theory

- Striated duct—oncocytic tumors
- Acinar cells—acinic cell carcinoma
- Excretory Duct—squamous cell and mucoepidermoid carcinoma
- Intercalated duct and myoepithelial cells—pleomorphic tumors



Tumorigenesis

- Contradictory evidence:
  - Luminal cells are readily capable of replication
  - Acinar cells participate in gland regeneration
  - Immunohistochemical staining of S-100 protein
    - Present in many salivary gland neoplasms
    - Not present in normal ductal cells

Conclusions

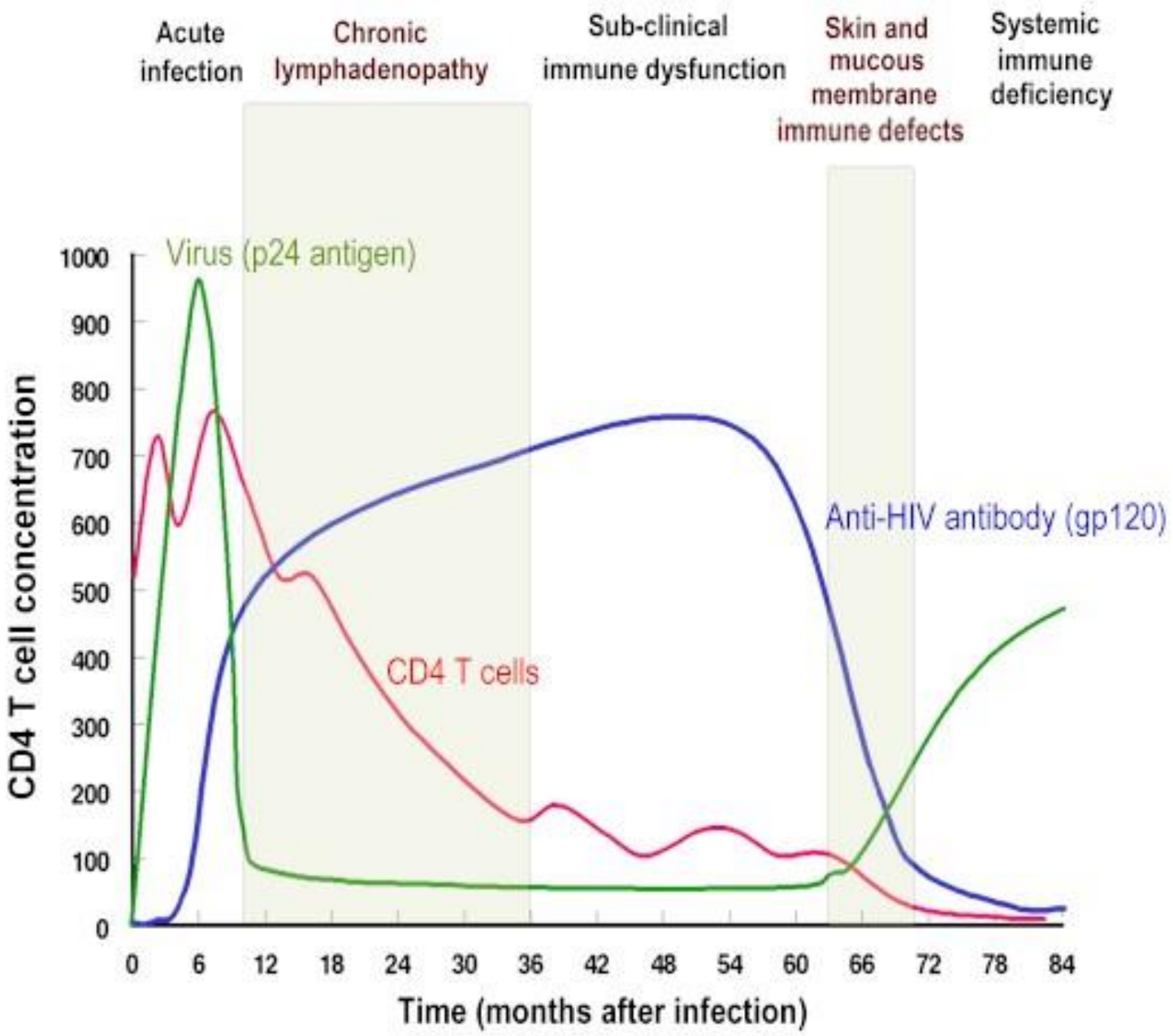
- Hugely diverse histopathology
- Accurate pathologic diagnosis does influence management
- Relatively rare malignancies
- Utilize preoperative studies when indicated
- Don’t believe everything you read!

Otolaryngologic Manifestations of HIV/ AIDS

OUTLINE

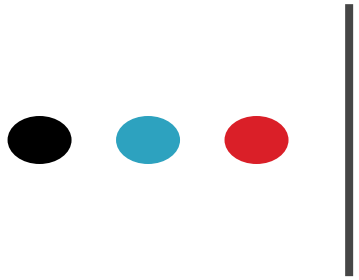
1. Otologic & Neurotologic.
2. Rhinologic
3. Oral Cavity
4. Pharynx
5. Larynx
6. Neck
7. Surgery

Natural hx of HIV infection



INTRODUCTION

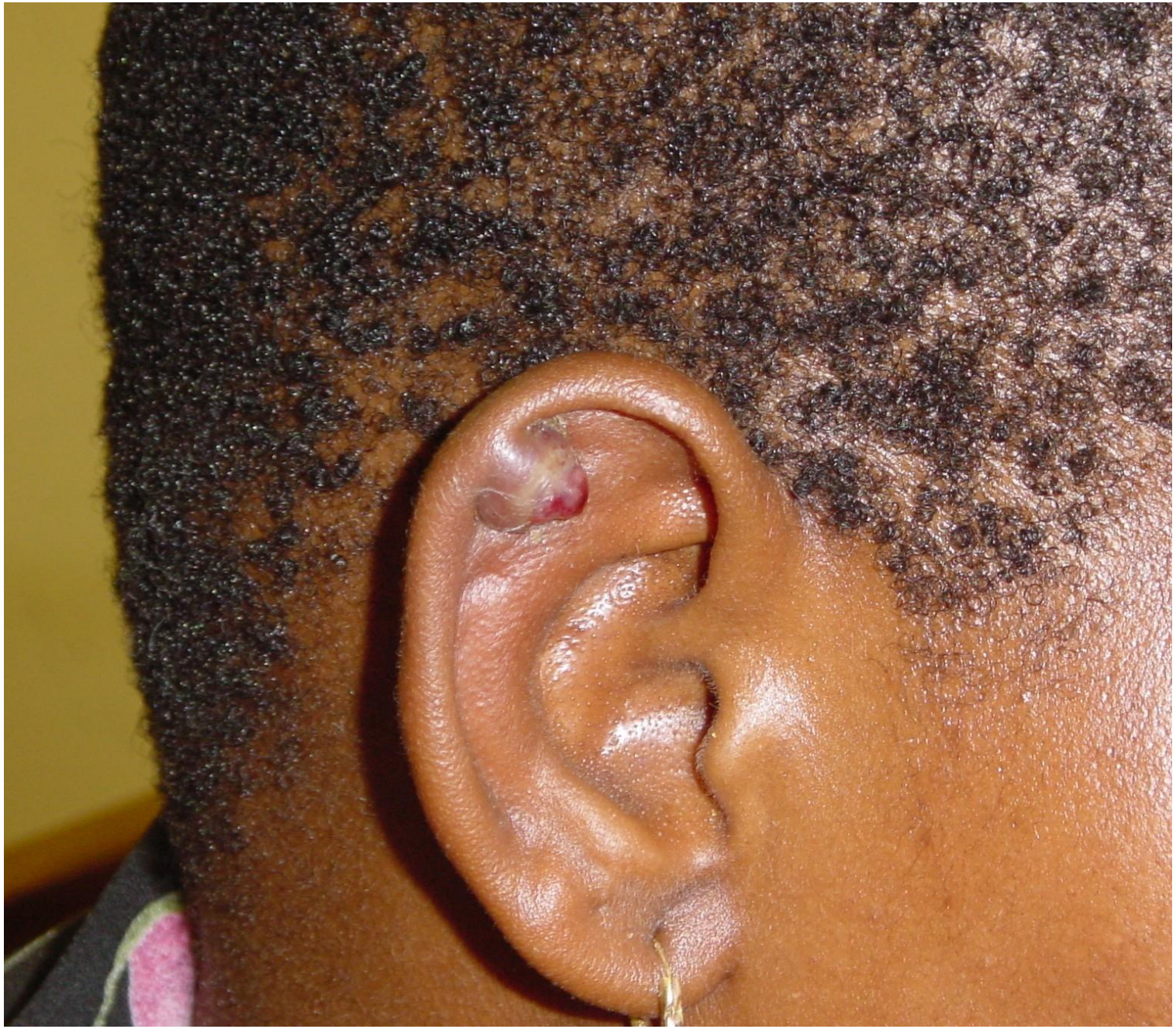
- Initially 41% of patients with AIDS –HN dse
- Now ~100% develop HN manifestations
  - Infections
  - Neoplasms
  - Primary neurological damage



OTOLOGY

External Ear

- Infections
  - Staph aureus - pinna cellulitis, furuncle
  - Pneumocystis -aural polyp
  - Mycobacterium tuberculosis
  - P. aeruginosa
  - Otomycosis – *Candida spp. & aspergilus spp*
- Eczema



Otitis Externa

- Predisposing factors
  - excessive irritation
  - mechanical trauma
- Hearing loss, otalgia and inflamed external auditory canal with purulent debris within
- Treatment
  - Prolonged suctioning of exudate
  - Topical antibiotic treatment

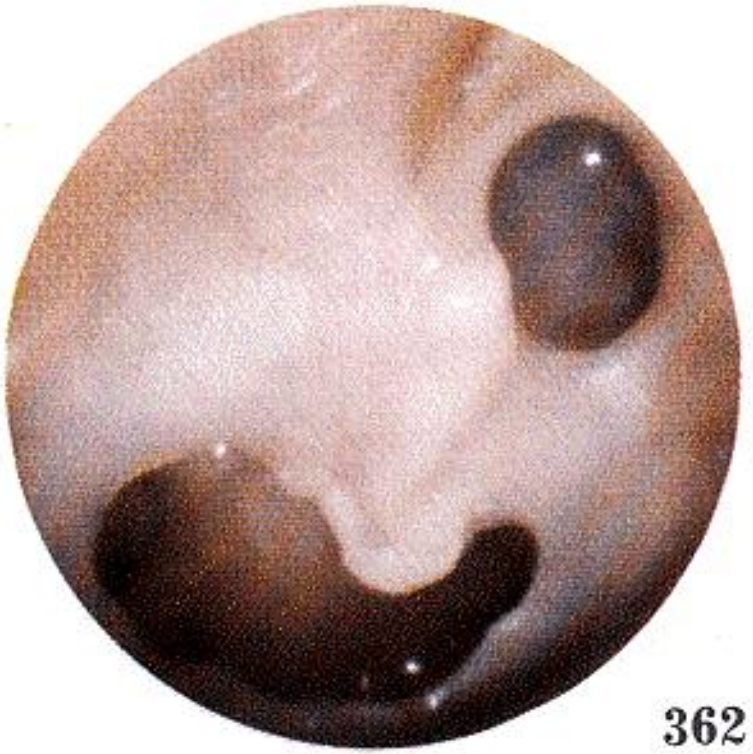
Malignant otitis externa

- Osteomyelitis of the skull base.
- Severe, progressive pain, fever and granulation tissue within the ear canal
  - *P. aeruginosa*
  - *Pneumocystis carinii,*
  - *M. tuberculosis and other common pathogens*
- Prolonged intravenous antibiotics and surgical debridement.

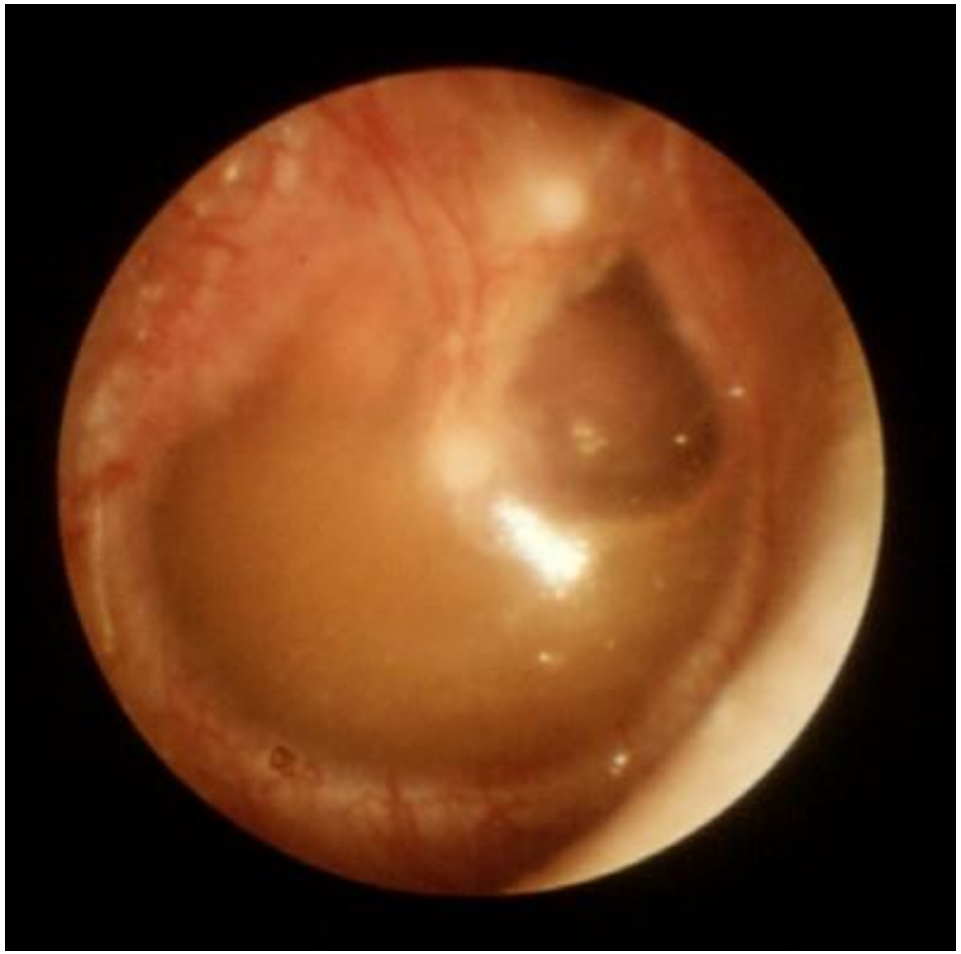
Middle Ear

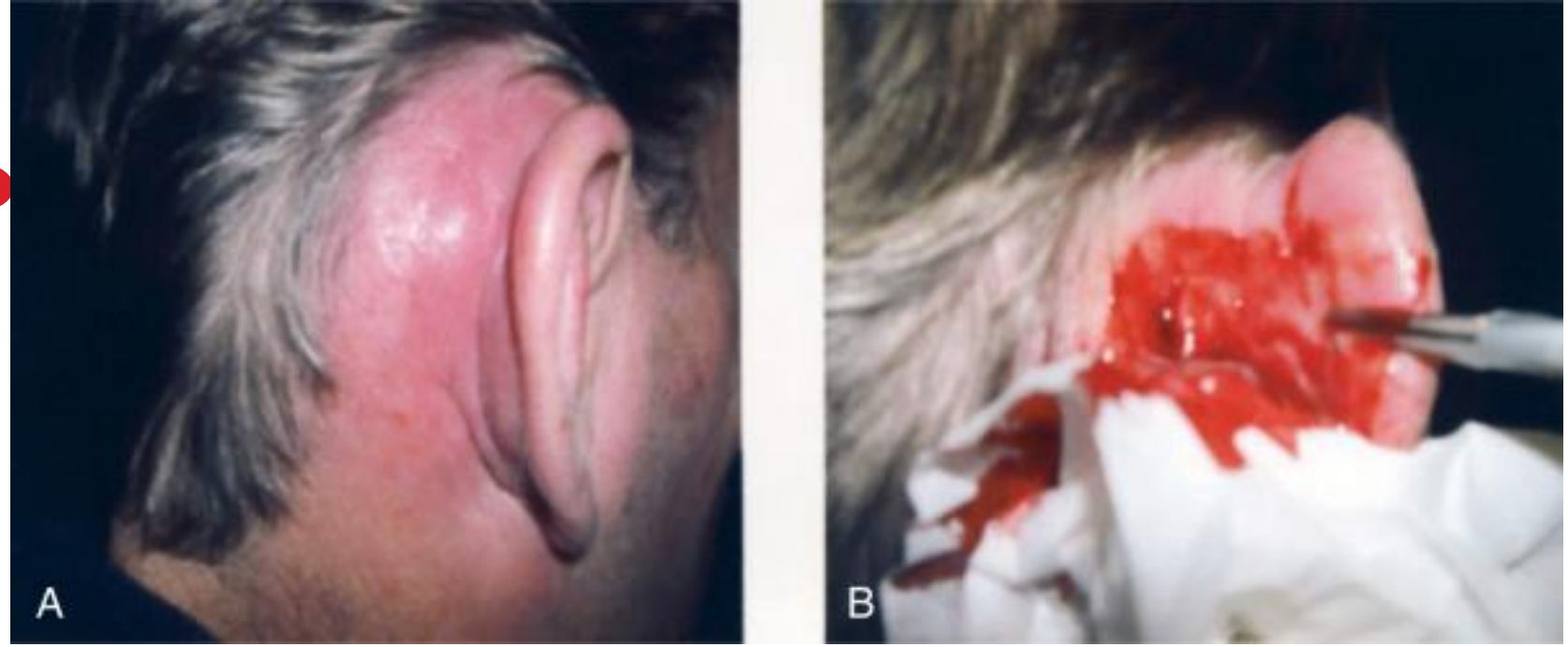
- Predisposing factors
  - Nasopharyngeal lymphoid hyperplasia,
  - Sinusitis & Allergies
  - Nasopharyngeal neoplasms or
  - Eustachian tube dysfuntion
- Most common -serous otitis media and recurrent acute otitis media (esp children)- OM and recurrent acute otitis media esp children.
- Adults - SOM

- Pathogens similar for both HIV+ & HIV-
  - Staphyloccocus
  - Pseudomonas
  - Pneumocystis carinii,
  - M. tuberculosis
  - Candida
  - Aspergilus
- Acute mastoiditis common
- In patients with nasopharyngeal lymphoid hyperplasia adenoidectomy will improve eustachian tube function



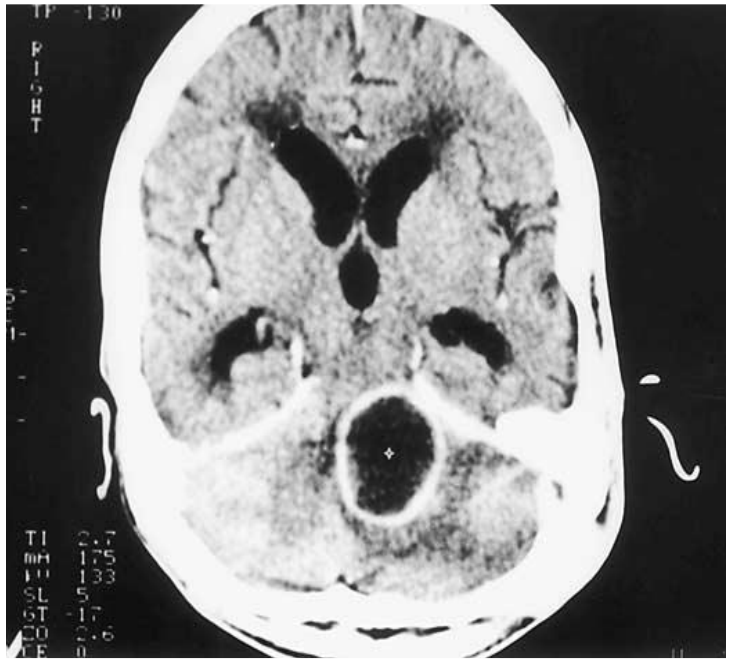
Otologic TB - CSOM, painless, odorless, watery otorrhea with multiple miniature tympanic membrane perforations





MASTOIDITIS

BRAIN ABSCESS SECONDARY TO MASTOIDITIS



SYPHILLIS

- TM perforation with CHL
- Temporal bone Gummas
- Endolymphatic hydrops/Menieres syndrome
- Tetrad: -fluctuating hearing loss
  - episodic vertigo
  - tinnitus (usually low-tone roaring)
  - aural fullness
- Rx: penicillin, ampicillin, tetracycline, erythromycin until serologic markers normalize
- Corticosteroids

Inner Ear- Sensorineural hearing loss

- Occurs in 20-50% of HIV+ patients.
- Increased latencies on ABR suggest central demyelination consistent with a viral infection.
- May be unilateral or bilateral
- Worsens steadily with increasing frequencies

- Etiology- primary infection by HIV of either CNS or peripheral auditory nerve, cryptococcal meningitis and idiopathic.
- Also
  - Tuberculosis
  - Cryptococcosis
  - Bacterial infections.
  - CNS tumours
  - Syphillis

Vertigo

- In HIV patients occurs together with other neurologic symptoms
- Frequently a symptom of subacute encephalitis or HIV dementia complex.

Neurotologic manifestations.

- Ramsay hunt syndrome
- Facial nerve palsy.
- Gradenigo's syndrome
- Opportunistic infections
  - Toxoplasmosis
  - Cryptococcal meningitis
  - Tuberculous meningitis
- Otosyphilis
  - fluctuating, asymmetric or sudden hearing loss
- Central nervous system
  - ataxic pursuit
  - optikinetic nystagmus
  - loss of caloric excitability

Causes of Hearing Impairment in the HIV/AIDS population

- Adenoids - Eustachian tube obstruction
- Opportunistic infections
- Direct effects of HIV
- Ototoxic medications
- Retrocochlear tumours

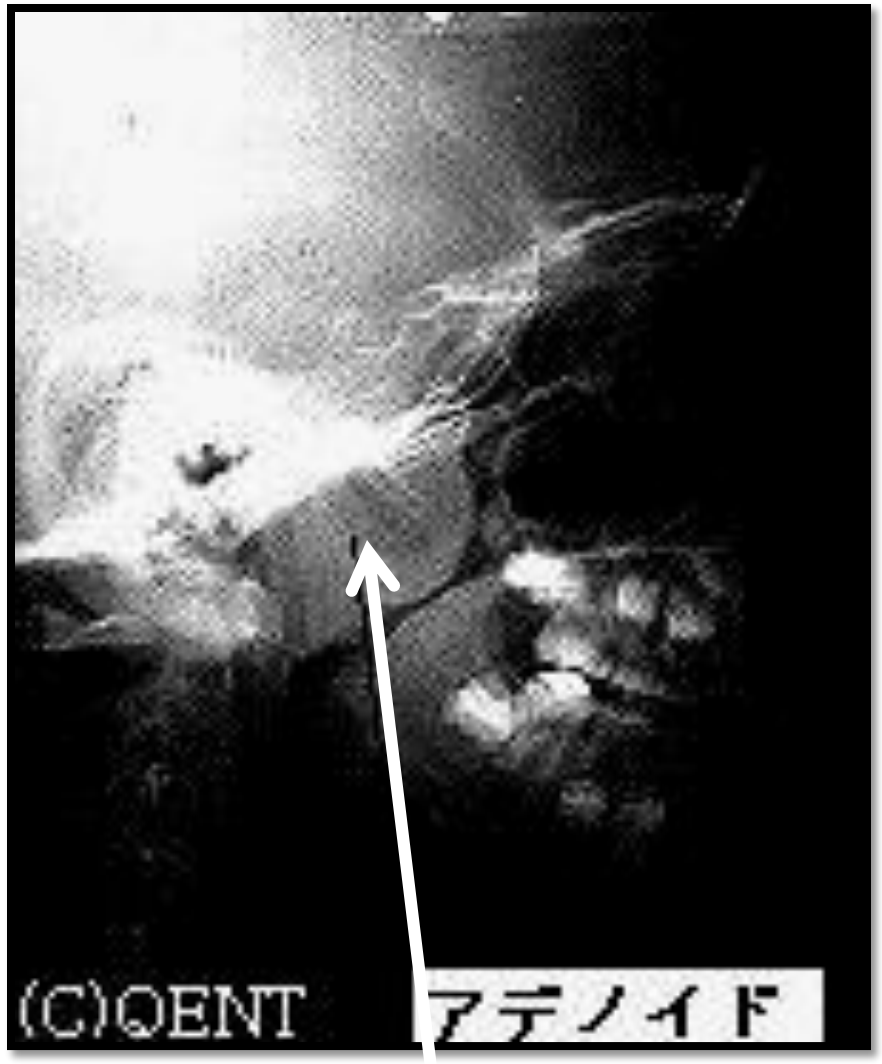
RHINOLOGY

- Loss of T-cells → polyclonal B-cell activation → both increase Ig secretion and hyperplasia of the lymphoid tissues → obstruction (ostia) & ↑allergy.

- Giant herpetic nasal ulcer.
  - > 3 cm
  - Begin in vestibule
  - Extend to the septum or face
- Sino-nasal lymphoma
  - CT shows bone destruction.
  - No dissemination.
  - Radiotherapy
- Kaposi's sarcoma
  - Nasal obstruction
  - Nasal discharge
  - Epistaxis

Rhinosinusitis

- Prevalence 20-70%
- Normal organisms as HIV-
- Atypical opportunistic
  - *Alternaria alternata, Aspergillus, Pseudallescheria boydii,Cryptococcus and Candida albicans*
  - CMV
- Medical therapy effective
- Surgery indicated to facilitate to obtain tissue specimens to diagnose other infections and malignancies



Nasopharyngeal lymphoid hyperplasia,

Nasal Allergy

- B-cell activation - increased production of circulating immune complexes and IgA, IgG and IgE.
- IgE- associated with increased allergies (AR)
- The intensity may suggest chronic persistent bacterial rhinosinusitis.
- Topical nasal steroid sprays + systemic antihistamines are
- Allergen avoidance

ORAL CAVITY

- Fungal infections
  - candida species - 90%
  - cryptococcus
  - histoplasma.
- Gingivitis
- Herpes simplex –all areas, larger 3cm, deeper, persists for longer
- Malignancy of the oral cavity, oropharynx

Candidiasis

- The most common oral condition in HIV /AIDS
- Occurs even in CD4 counts 200-500 mm3.
- Milky pseudomembrane forms or leukoplakia
- Less typical
  - Atrophic candidiasis -erythematous patches in the buccal mucosa, palate, or tongue.
  - chronic hypertrophic form is also often
- Angular cheilitis -nonhealing fissure at oral commissure
- KOH preparation of scrapings from lesions
- Topical therapy with nystatin or clotrimazole
- Systemic therapy with ketoconazole, fluconazole, Amphotericin B and prophylactic antifungal may be indicated in severe cases of immunosuppression

Herpes simplex

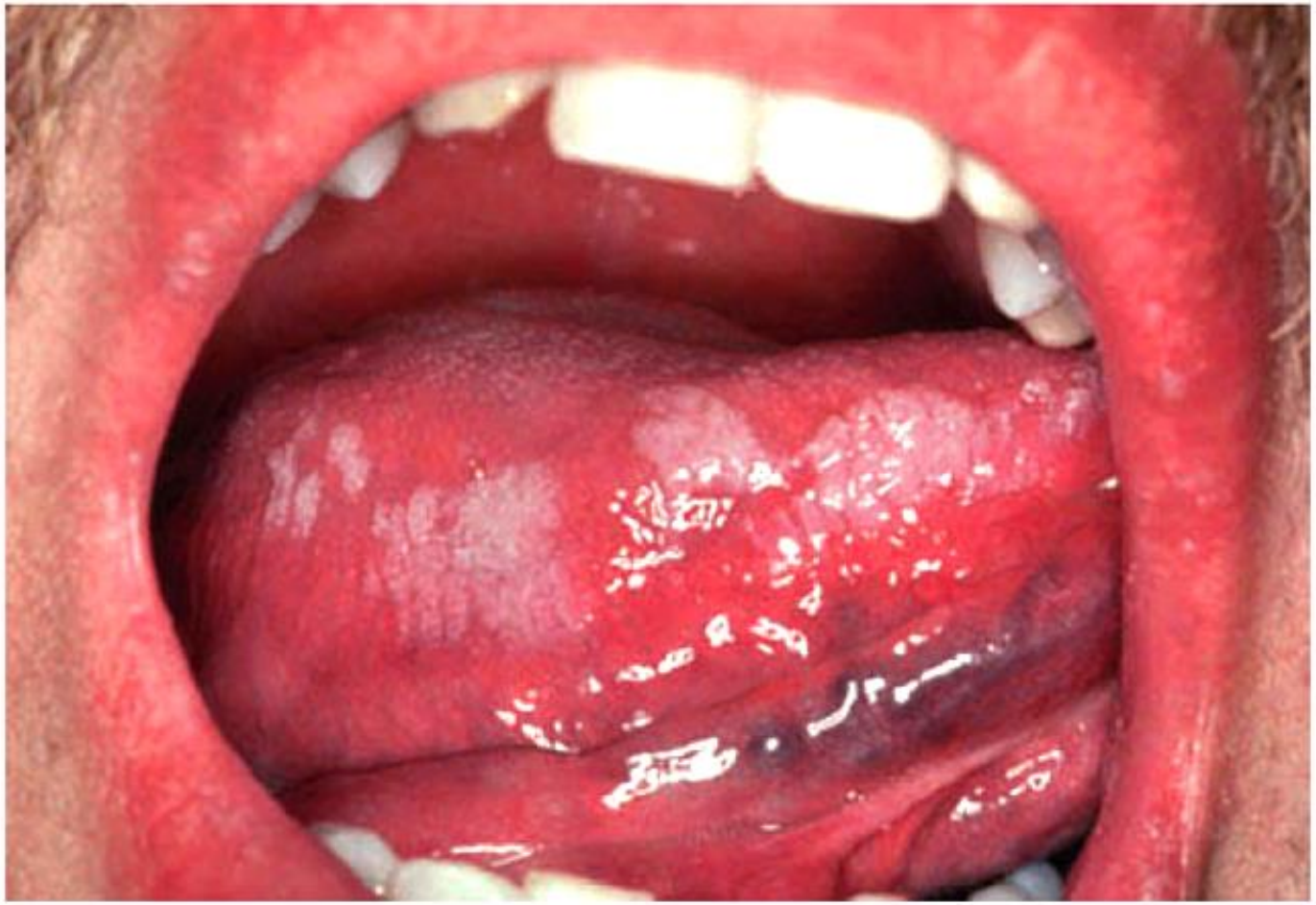
- Herpes labialis
  - crops of fever blisters on the palate, gingiva, or other oral mucosal surfaces.
- Tend to be larger and more numerous, recur more frequently, and often persist longer
- Also extend onto adjacent skin and coalesce to form giant herpetic lesions

- Treatment not necessary if
  - lesions are small
  - relatively asymptomatic
  - Lesions beginning to heal.
- Oral acyclovir for large or symptomatic lesions

EBV

- Hairy leukoplakia - EBV–induced benign epithelial hyperplasia.
- White, vertically corrugated lesion along the anterior lateral border of the tongue.
- Exclusive to HIV
- More rapid progression to the full-blown AIDS.
- Primarily asymptomatic
- Treatment unnecessary

Fig. 4.16 Oral hairy leukoplakia



Typical whitish patches on the border of the tongue.

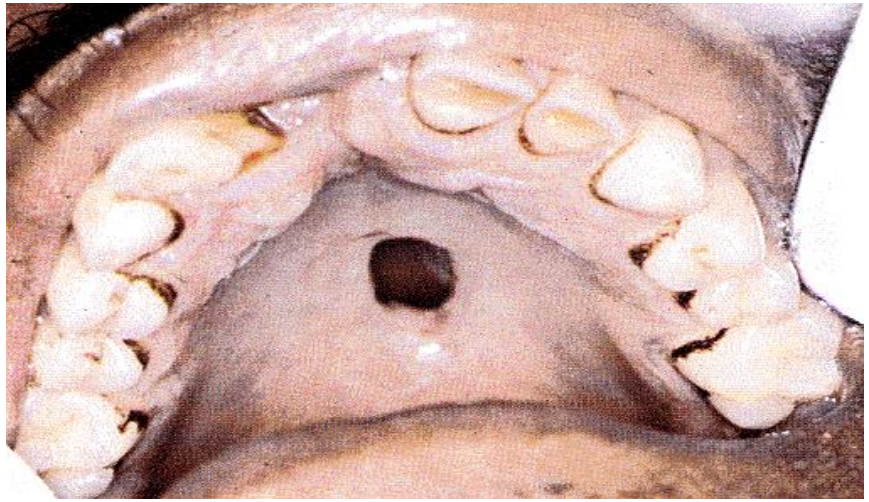
Recurrent Aphthous Ulceration

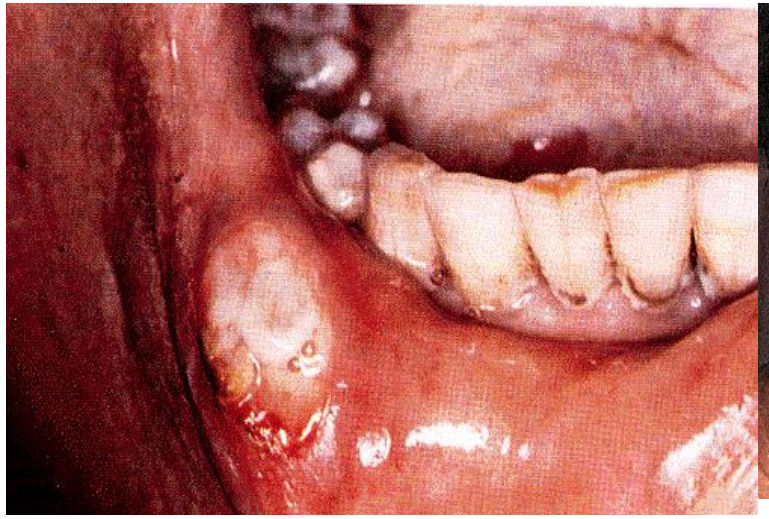
- Coalescing of smaller lesions into large ulcers
- Can present anywhere in the oral cavity or pharynx
- Associated with severe odynophagia
- Worsened by secondary infection
- Surgical or laser excision of these lesions is the treatment of choice

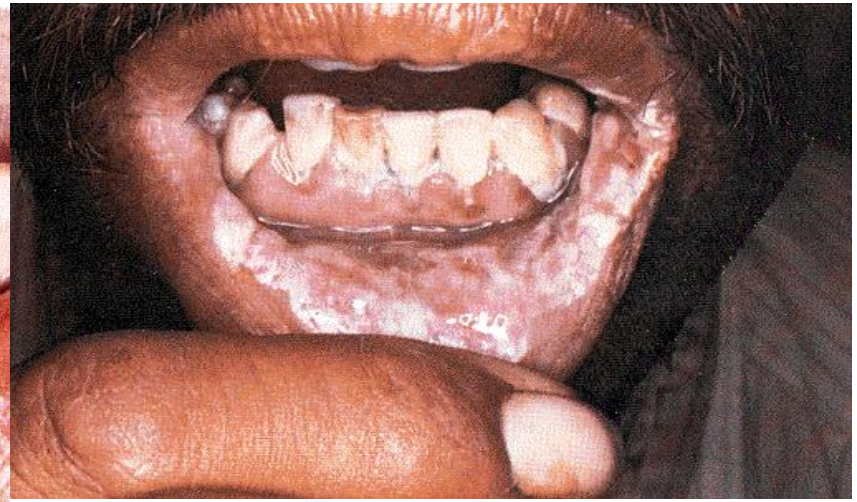
Kaposi's sarcoma of the oral cavity

- Most common site
- 95% are found on the palate then gingiva, buccal mucosa, and soft palate
- Initially asymptomatic, flat or raised red-blue lesion.
- It may become painful if ulcerated or superinfected.
- Very large tumors may become lobulated.
- Oral KS associated with increased ISS and lower CD4+ counts (mean, 66.6 cells/ml)

SYPHILLIS







LARYNX

- Supraglottitis
  - Particularly aggressive in some cases.
  - Presentation similar but conservative treatment mostly fails and airway intervention required
- Laryngeal tuberculosis
  - The vocal folds most common site (50 – 70%)
  - False cords, posterior larynx
- KS lesions
- Candidiasis

PHARYNX

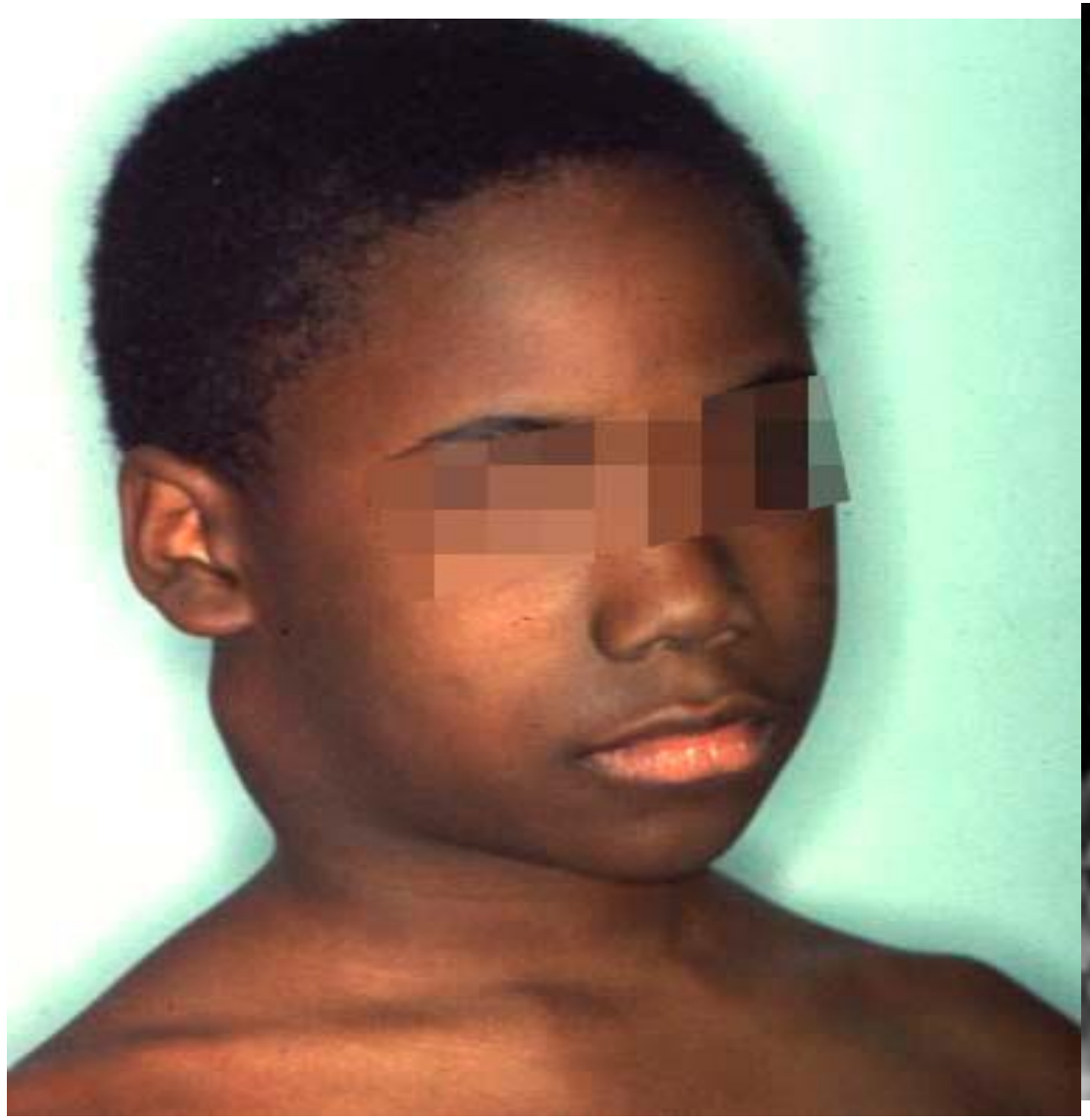
- Candidiasis
- Lymhoma
- KS
- PGL (Waldeyer’s Ring)
- Tonsillar Hypertrophy
- Odynophagia, Dysphagia

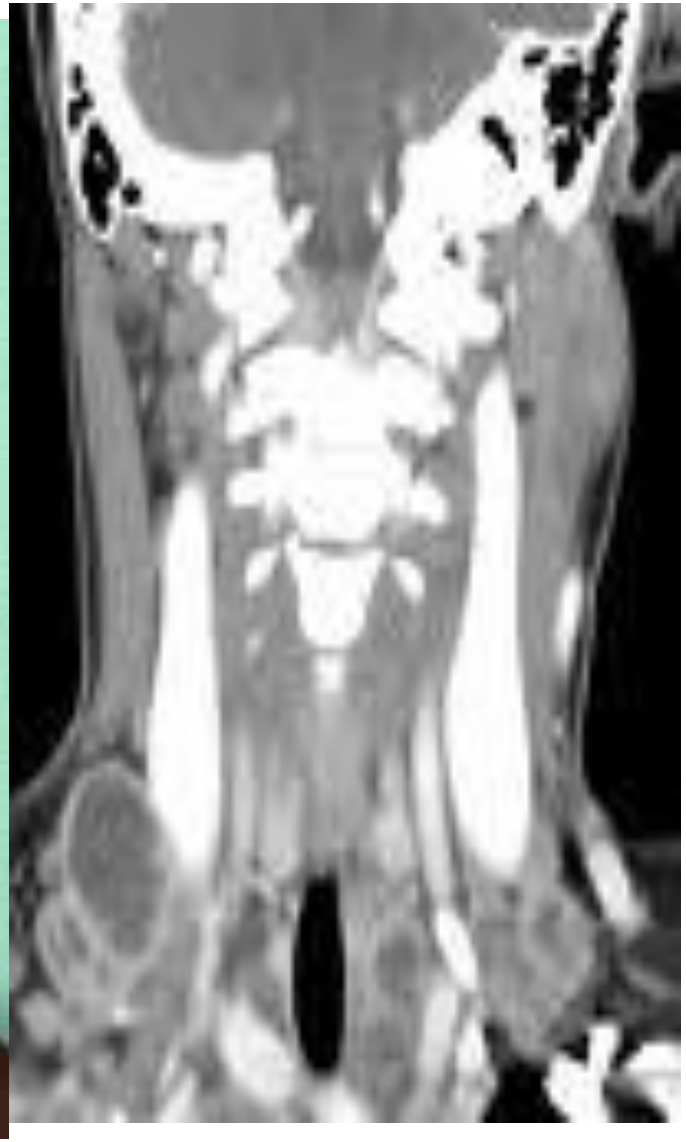
SALIVARY GLAND

- Xerostomia - common complaint in AIDS (10%)
- Cause unknown (? CMV)
- Treatment
  - Frequent saline rinses
  - Sialogogues
  - Topical fluoride applications.
- Cystic parotid enlargement
  - Nontender, unilateral or bilateral.
  - Rx is aspiration but cosmetically deforming lesions or intractable lesions- doxycycline sclerosis, low-dose DXT, parotidectomy.
- Benign & malignant proliferation in the LN – PGL and lymphomas
- Surgery avoided- refractoriness of lesion

NECK

- Persistent generalised lymphadenopathy
- KS
- Lymphoma
- TB adenopathy
- CMV adenopathy
- EBV adenopathy





Cervical HIV Lymphadenopathy

- Unexplained generalized lymphadenopathy involving >2 extra-inguinal sites and lasting more than 3months
- The axilla and neck are the most common sites.
- Only symptom is neck swelling.
- Indications for biopsy include recent weight loss and rapid increase in size.

Tuberculosis

- 50-70% Extrapulmonary disease
- Common sites
  - cervical nodes
  - larynx
- Only symptom an enlarging neck mass which is usually firm and nontender +/- inflammation
- *Mycobacterium avium complex (MAC) infection* most common mycobacterial infection.
- Response of atypical mycobacterial infections to traditional antimycobacterial drugsis often poor
- Azithromycin or clarithromycin are effective

Extrapulmonary Pneumocystis

- Cervical nodes- respond to medical therapy
- Thyroid gland
  - Diffuse goitre like swelling
  - Hypothyroidism

Mycosis

- Cryptococcosis, histoplasmosis, and coccidioidomycosis
- A cervical mass
- Cryptococcus neoformans the most prevalent cause
- The typical granulomatous lesions of fungal infections may be absent
- Therapy with amphotericin B is moderately effective.

Surgery and HIV

- Risk of seroconversion at about 0.3% per parenteral exposure to HIV positive blood
- HIV testing for all patients undergoing major surgery?
- Major surgery- CD4 counts >350
- Exception- I & D, Tracheostomy, Mastoid abscess (ER)

Malignancy

- Most common malignancy -Kaposi Sarcoma
- Incidence - 47%
- Oral cavity, gingival surfaces of the oropharynx, ear, larynx and nose.
- May be exophytic and ulcerated.
- Secondary infection - severe, increasing pain, difficulty with mastication and swallowing, and difficulty maintaining good oral hygiene

KS Classification

- Idiopathic multiple sarcoma of the skin.
- Classic/European
- Endemic/african
- Lymphadenopathic
- Transplant
- HIV associated

KS TREATMENT - Local therapy

- Oral and epiglottic KS -Intralesional vinblastine
- Cryotherapy (superficial and limited KS)
- Radiotherapy (regional disease of the HN)
- Low-dose DXT >90% control of cutaneous lesions.
- Mucositis- complication of DXT of mucosal dse
- Mucositis reduced using low-dose fractions, and an accumulative dose less than 30 Gy.
- Carbon dioxide and argon laser (excision of canalicular or tympanic membrane lesions)

Systemic chemotherapy

- Single drug or combinations
- Include
  - Vincristine
  - Vinblastine
  - Bleomycin
  - Taxanes
  - Adriamycin

Lymphoma

- Up to 10% develop some lymphoma.
- Usually high-grade, aggressive, B-cell tumors (Non-Hodgkins)
- Predisposition for extranodal sites -bone marrow, oral cavity, sinuses, skin, liver, GI tract and CNS

Non-Hodgkins Lymphoma

- 2nd most common HIV-associated malignancy.
- Commonly presents as a nontender, rapidly enlarging neck mass.
- Majority are high grade
- In HIV NHL, Hodgkin's disease & SCC tend to be more aggressive and less responsive to treatment
- Surgery and chemotherapy
- HIV patients tolerate radiotherapy very poorly

DO'S AND DON'T'S IN SURGERY

Surgical outcome in general seems to correlate with CD4 count

- Reinforce universal precautions by regular teaching, practicing, and testing.
- Wear impermeable gowns.
- Wear protective eyewear and masks for all procedures.
- Cover head and both ears completely with OR hat.
- Double gloves. Glove micro perforations occur in as many as 67% of head and neck cases. Double gloving has reduced this risk to 7-10% in several studies.
- Maintain high level of concentration by all staff involved in the procedure.
- Consider taking breaks during long procedures to help improve concentration.
- Minimize the number of personnel involved in the procedure, especially on high- risk patients.

DOS AND DONTS

- Limit handling of sharp instruments to the most experienced operators and assistants.
- Minimize the number of instruments on the procedure table.
- Change gloves every few hours during long procedures, even if the glove is believed not to be penetrated.
- Pass every sharp instrument to a “neutral” zone such as a metal basin instead of hand to hand.
- Use of blunt suture needles (i.e., Ethiguard). Blunt point on electrocautery.
- Use of blunt tip retractors.
- Use of electrocautery for cutting instead of the scalpel.
- Never recap needles.

MANAGEMENT OF CERVICAL MASSES

HO OBURRA

ANATOMICAL ORIGINS

- Skin and Appendages
- Subcutaneous Tissues
- Muscles
- Neurovascular bundles
- Lymphatic Tissue
- Salivary glands
- Visceral Epithelium
- Cervical Vertebra

CLINICOPATHOLOGICAL CLASSIFICATION

- Solid and Cystic Masses

CYSTIC MASSES

CONGENITAL

- Thyroglossal cysts
- Branchial cysts
- Dermoid inclusion cysts
- Cystic Hygroma

ACQUIRED

- sebaceous
- Hydatid Cyst
- Abscesses

Management of Cystic Masses

- Surgical Excision for congenital cysts

Injection of sclerosing agents or simple aspiration confer temporary remissions

Incision and drainage and Biopsy for abscesses

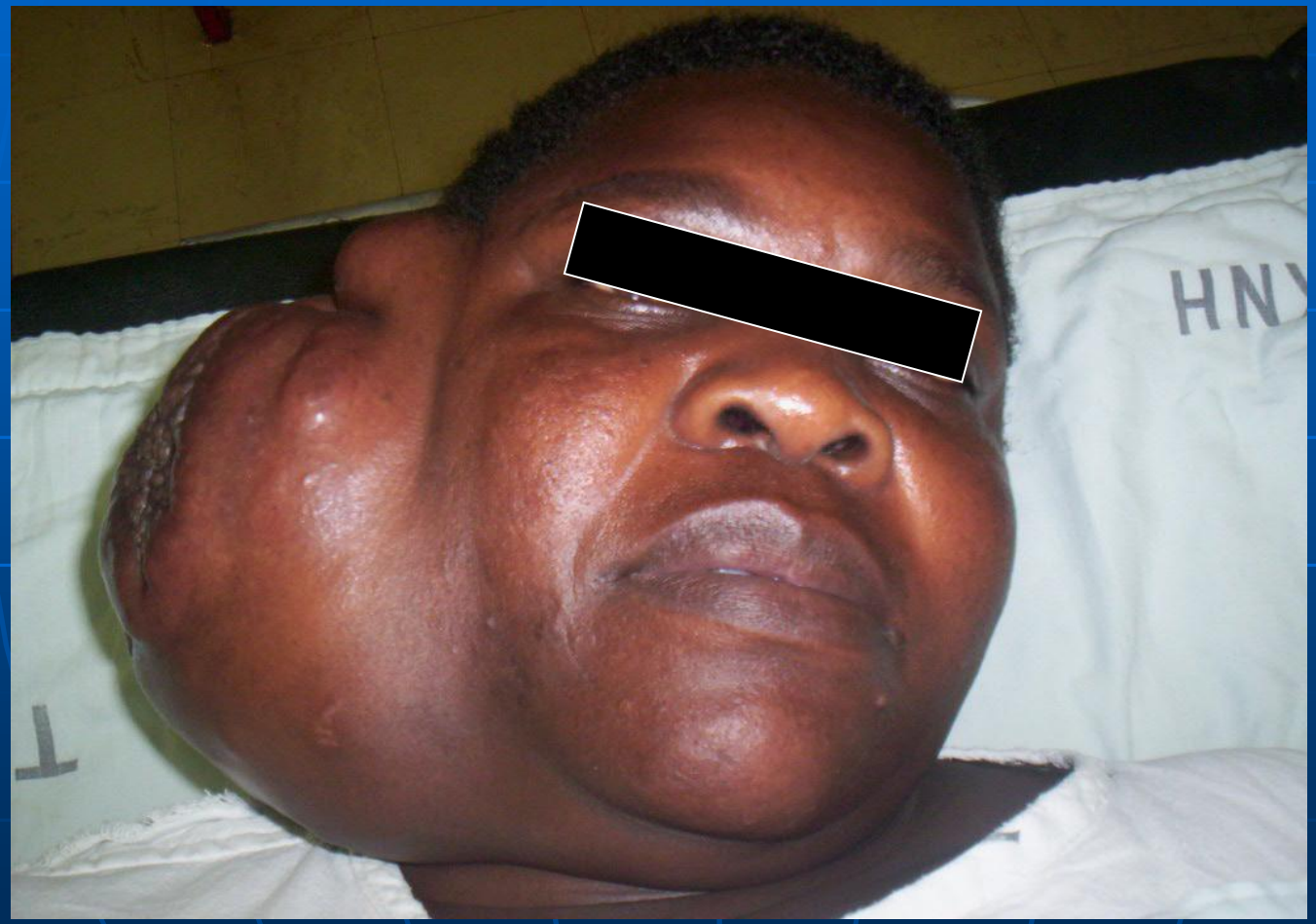
Fine needle aspiration and skin tests for hydatid cyst

Cystic parotid mass- HIV- Sclerose with 90% Ethanol

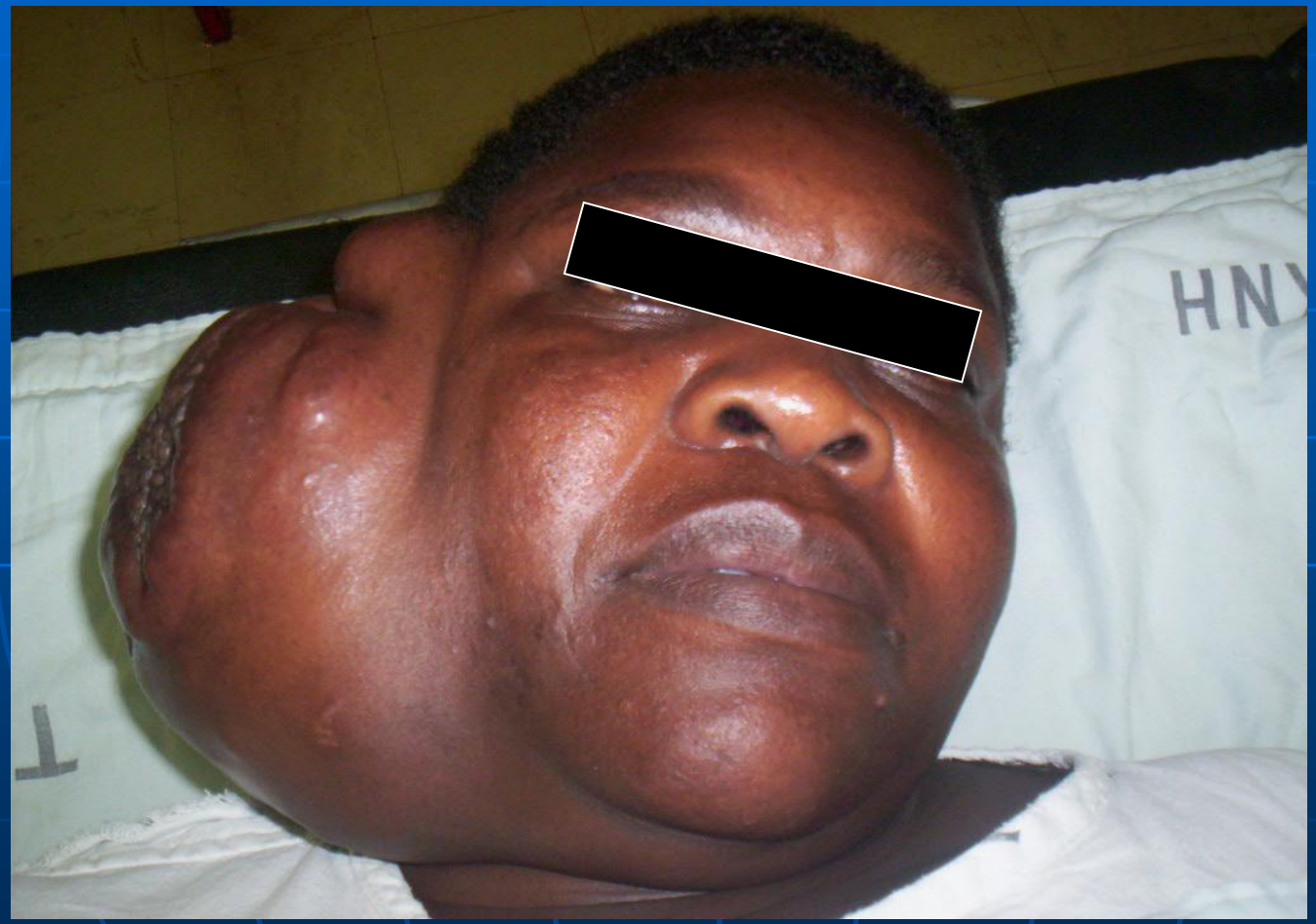
SOLID MASSES

1. **Inflammatory Masses**
   - Infective and autoimmune Lymphadenitis
2. **Neoplastic Masses**
   - Primary and secondary lymphatic neoplasms, Rhabdomyosarcoma, salivary gland tumours (and masses), thyroid tumours, glomus tumour, carotid body tumour
3. **Endocrine Masses-**Thyroid goitre, Thyroiditis
4. **Traumatic masses**

Recurrent Parotid tumour Treated by Surgical Excision and DXT



Recurrent Parotid tumour Treated by Surgical Excision and DXT



5. Vascular masses-

- Aneurysms

6. Idiopathic Masses

- Hypertrophy of Masseters
- Miscellaneous: Prominent transverse process of the cervical vertebra.

ASSESSMENT OF SOLID CERVICAL MASSES HISTORY

- Onset
- Symptoms- cough, blocked nose, dysphagia, ear problems, git problems, chest symptoms
- Duration
- Rate of growth
- Associated illnesses- breast disease, lung disease, kidney disease, past git surgery, risky sexual behaviour (HIV).
- Occupation

- Ingestion of alcohol and tobacco use.
- Family History of similar illness.

EXAMINATION
Clinical Officer Paramedic

EXAMINATION

Inspect

Stand infront or behind patient

Assess Mass visually

Note the site

Palpate

Consistency

Discrete

Tender

Size

Mobility/Fixity

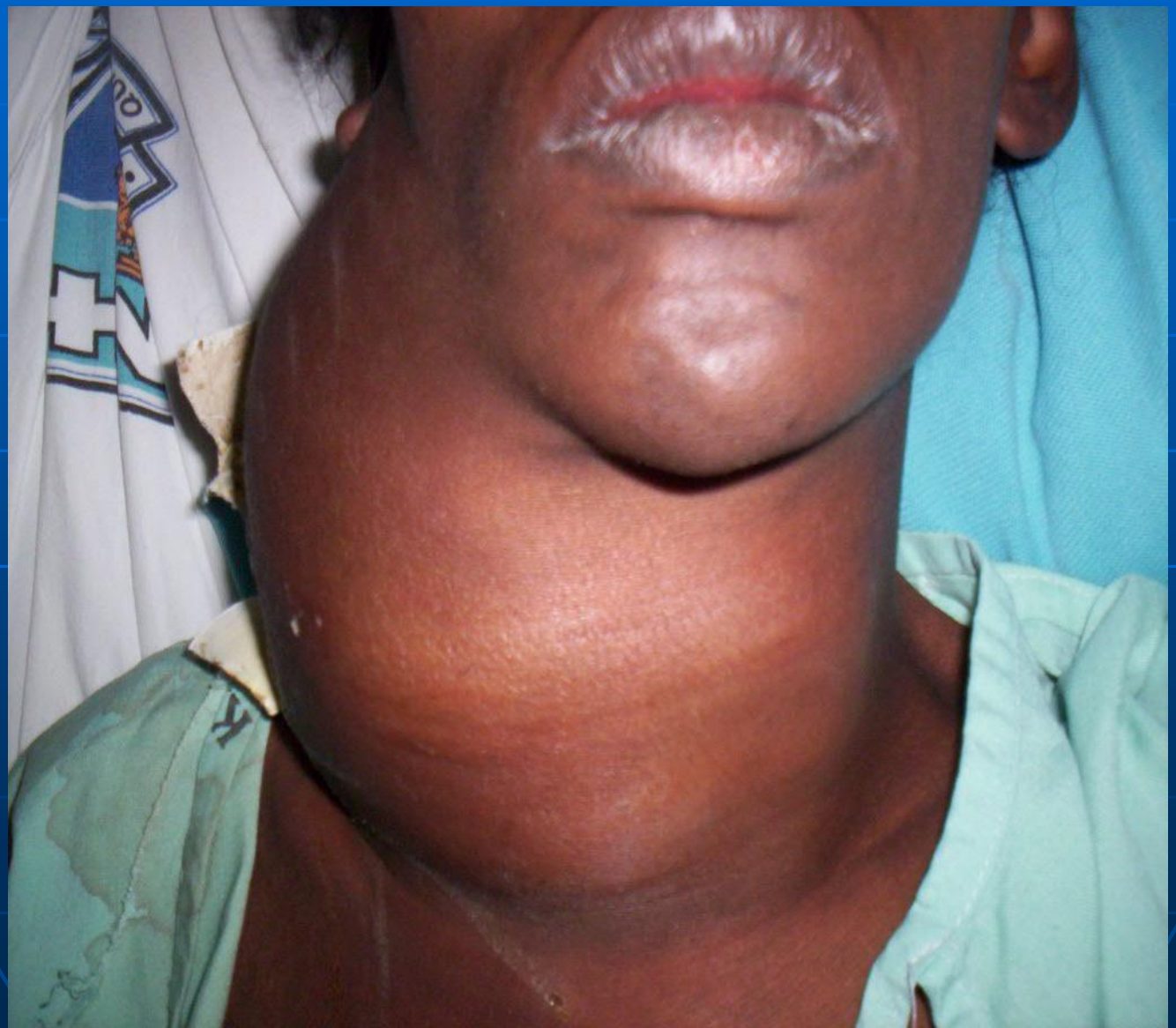
Special features to appreciate

- Pulsatile- Aneurysm, carotid body tumour
- Parotid swelling-check facial nerve integrity
- Midline Cervical swelling- Thyrotoxicosis or Addison’s disease features. Moves with swallowing.

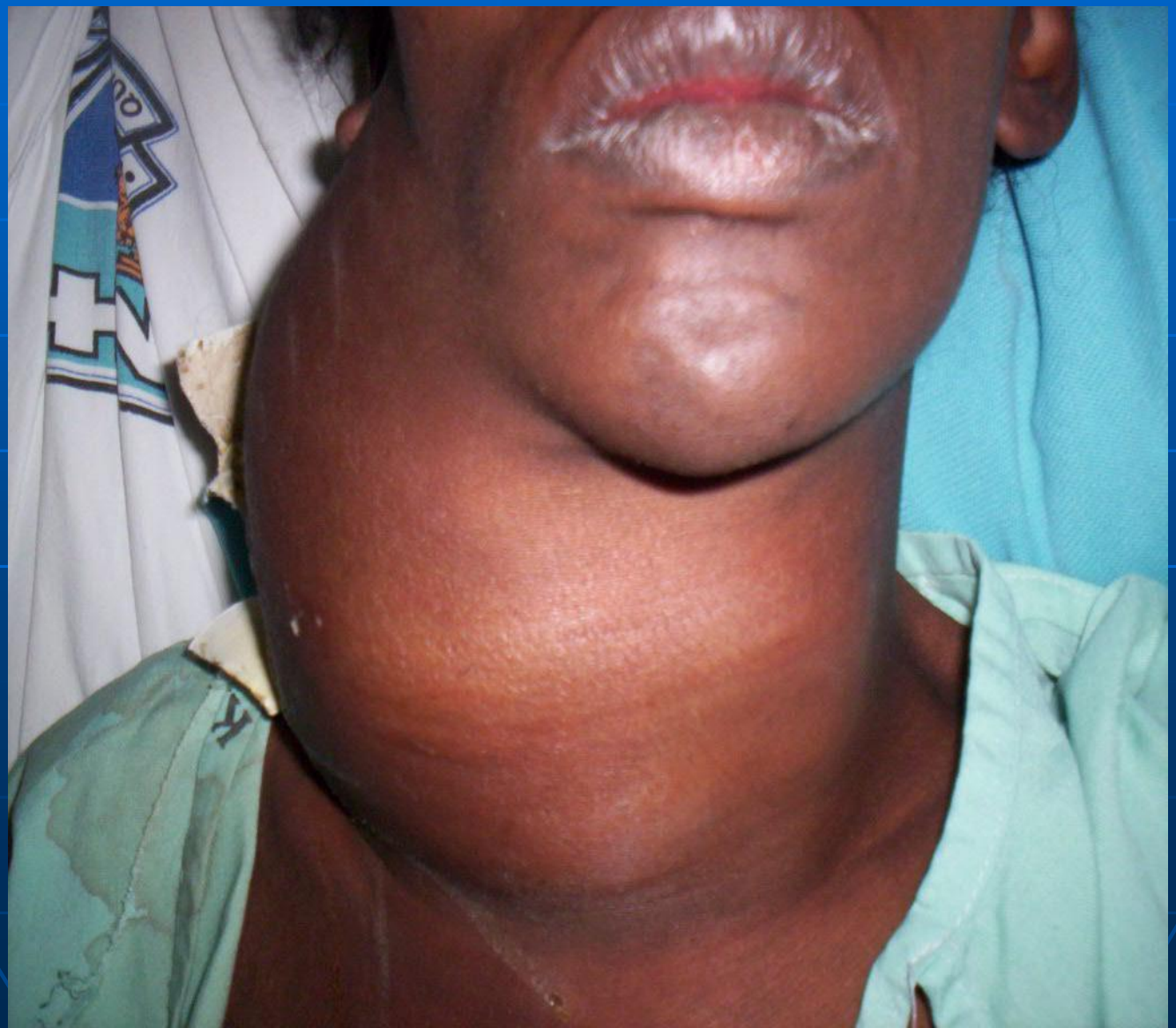
- Probability of Malignant Neoplasm-Full examination of patient for metastases mandatory.

Advanced Nasopharyngeal Carcinoma

Recurrent Cervical soft Tissue Sarcoma



Recurrent Cervical soft Tissue Sarcoma



INVESTIGATIONS

GUIDED

Inflammatory- Appropriate procedure-FNA, C/S, Biopsy, Special stains (ZN stain)

- Neoplastic- Imaging, FNA, Biopsy, IHC, PCR
- Endocrine- Hormonal Profiles, Imaging
- Traumatic- Imaging, assessment of functions etc
- Vascular-Imaging (scans etc).

FOR METASTASES AND MULTIPLICITY

- CXR
- Abdominal ultrasound
- LFTs- Alk PO_4se
- Radionuclide Scans
- PET scans
- ETC ETC

MANAGEMENT

- Depends on cause
- ***A SLOW ONSET, SLOW GROWING, DISCRETE OR DIFFUSE, NON TENDER, FIRM, FIXED OR MOBILE CERVICAL MASS IS NEOPLASTIC UNTIL PROVED OTHERWISE.***

STEPS OF MANAGEMENT

- Note Basic Characteristics
- Examine whole patient with emphasis on head and neck
- Do FNA

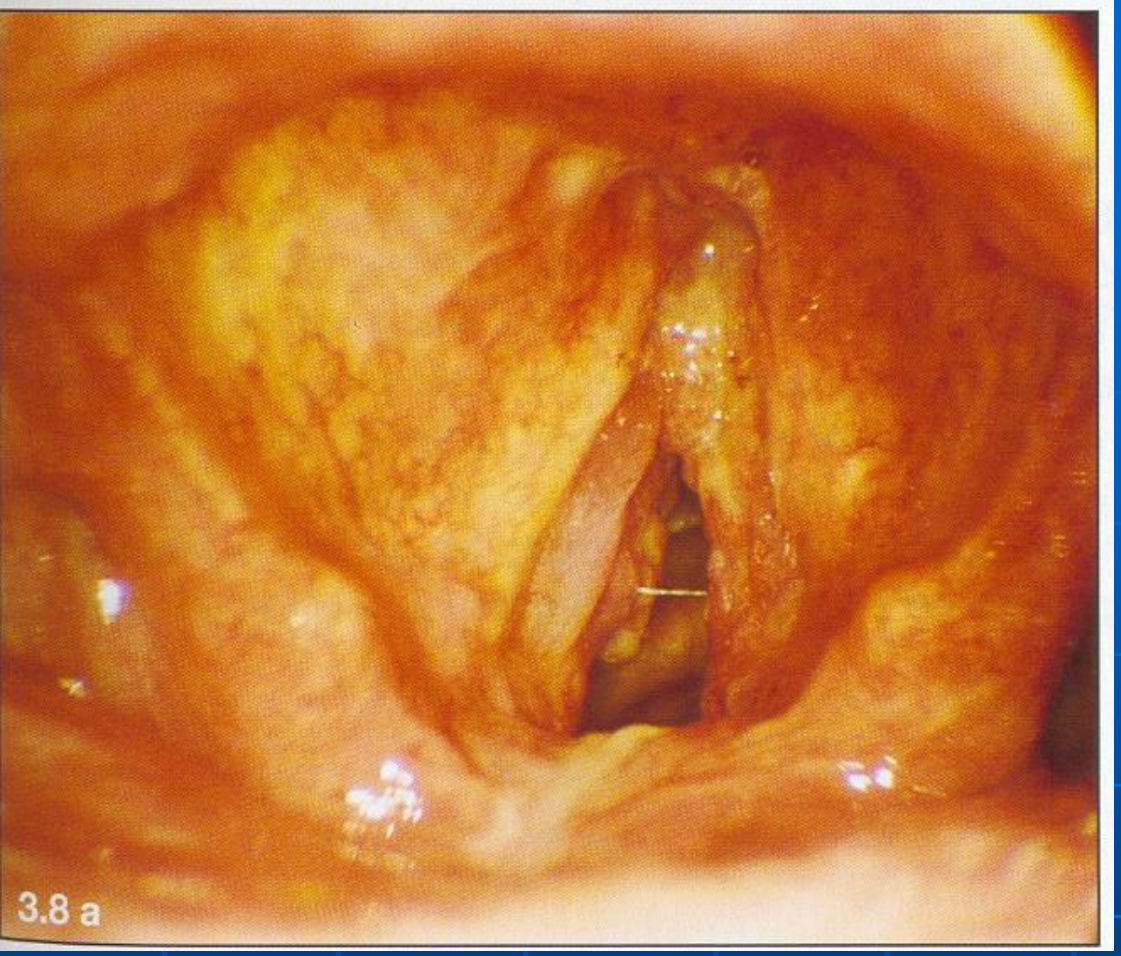
Look for Possible Primary!!!!!!!!!!

- Direct examination oral cavity/throat.
- Do rigid (or flexible) endoscopy/Biopsy or examination under relevant anaesthesia whichever appropriate in UADT, Bronchus, Breast, git or kidney neoplasm.

Tongue Cancer

MOST COMMON SITES IN ADULTS

- Nasopharynx
- Tongue base
- Hypo pharynx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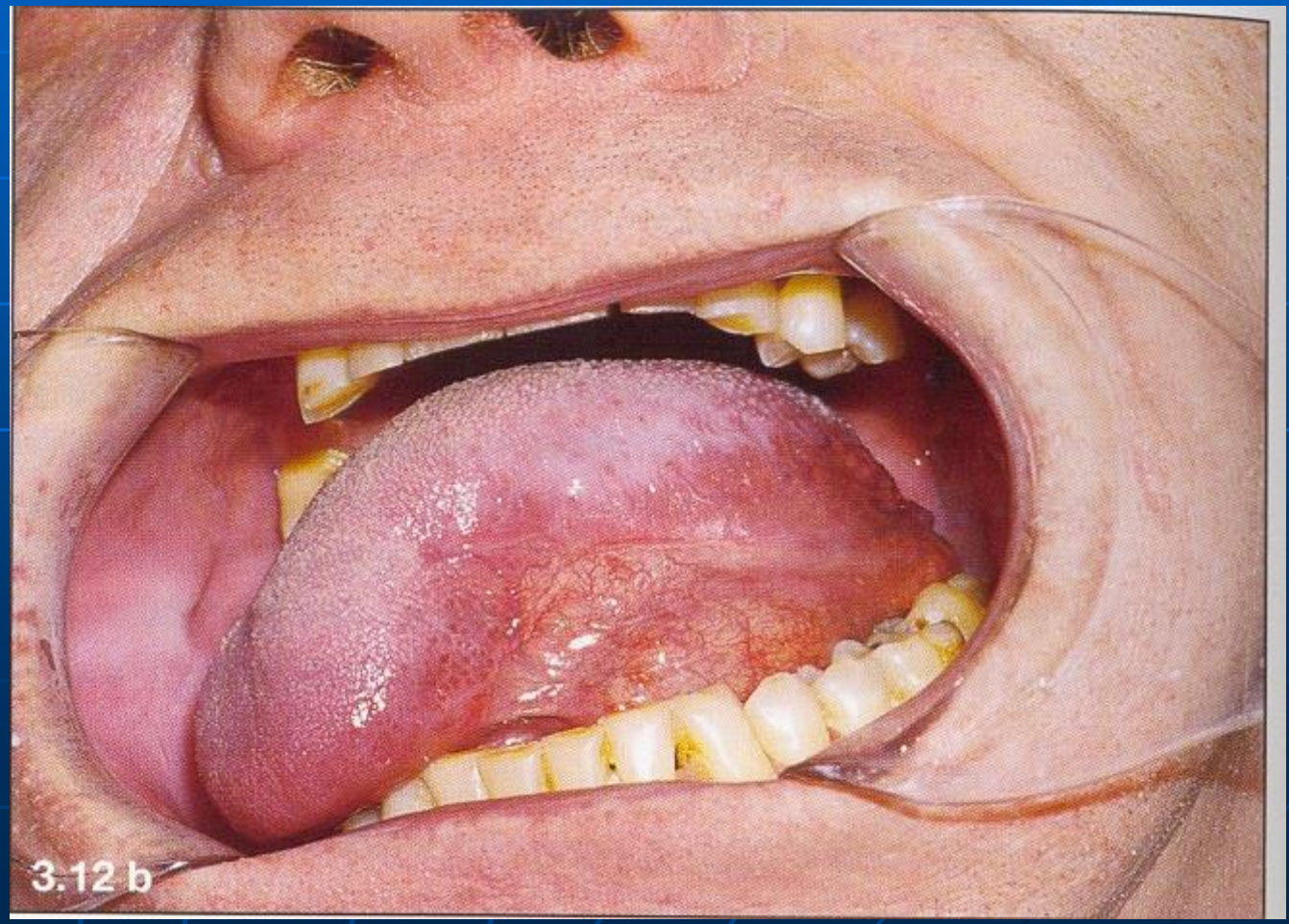
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- NO GROSS PRIMARY DETECTED?
- Take Guided Biopsies in all the three sites
- Wait for Histology

NEGATIVE HISTOLOGY???

- Excisional Biopsy of the mass with safe margins.

Squamous cell carcinoma?

- Radiate the neck and wait for primary to appear.

  Options-Latent Membrane proteins (EB-Virus)

Lymphoma?

- CXR, haemogram (WBC)Total body scan, bone marrow, paedal lymphangiography,

TIPS

- NO OBVIOUS MACROSCOPIC PRIMARY.
- 40% will reveal primary in guided biopsy
- 40% the primary will always appear
- 20%- No primary detected- cancer probably appearing from inclusion Dermoid, branchial cyst etc.

TIPS Contd

1.Solitary supraclavicular mass

- The primary is most likely below the clavicle- git, breast, kidney, bronchus, thoracic oesophagus

2. Posterior triangle single matted node Probably tuberculous

3. Non Hodgkin's lymphoma- HIV screen

- GOODDAY, GOD BLESS YOU
- AU REVOIR, QUE DIEU VOUS BENISE
- ALAMSIKI, NA MUNGU AWABARIKI
- INSHALLAH
- ADIOS
- THIE NAWEGA, NGAI AMUADHIME
- OLINDUE, NYASAE AKHULINDE
- KWAHERI NGAI AMUATHIME
- MWENDE MANA, MWENDE NA SERE
- ORITI, ROUTH OKONYU

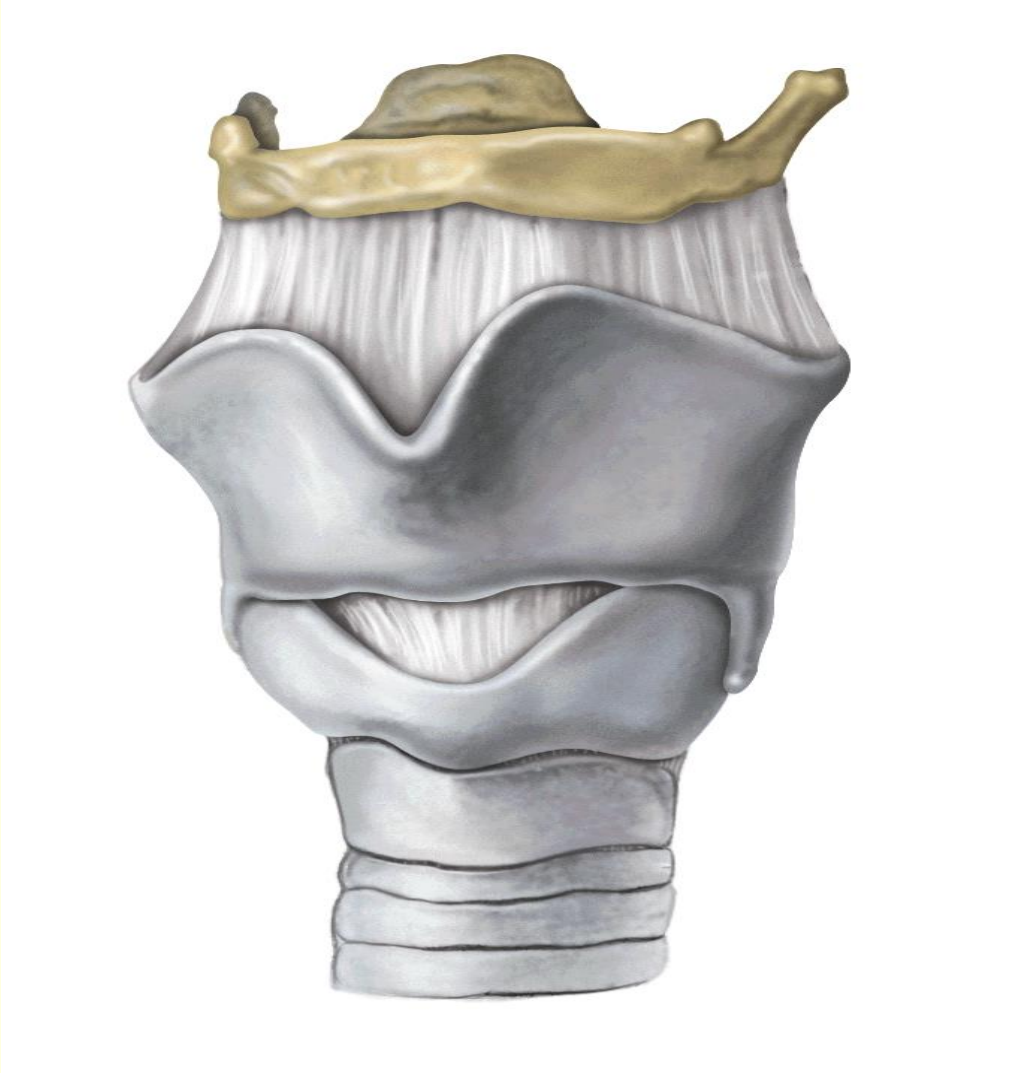
CARCINOMA OF THE LARYNX

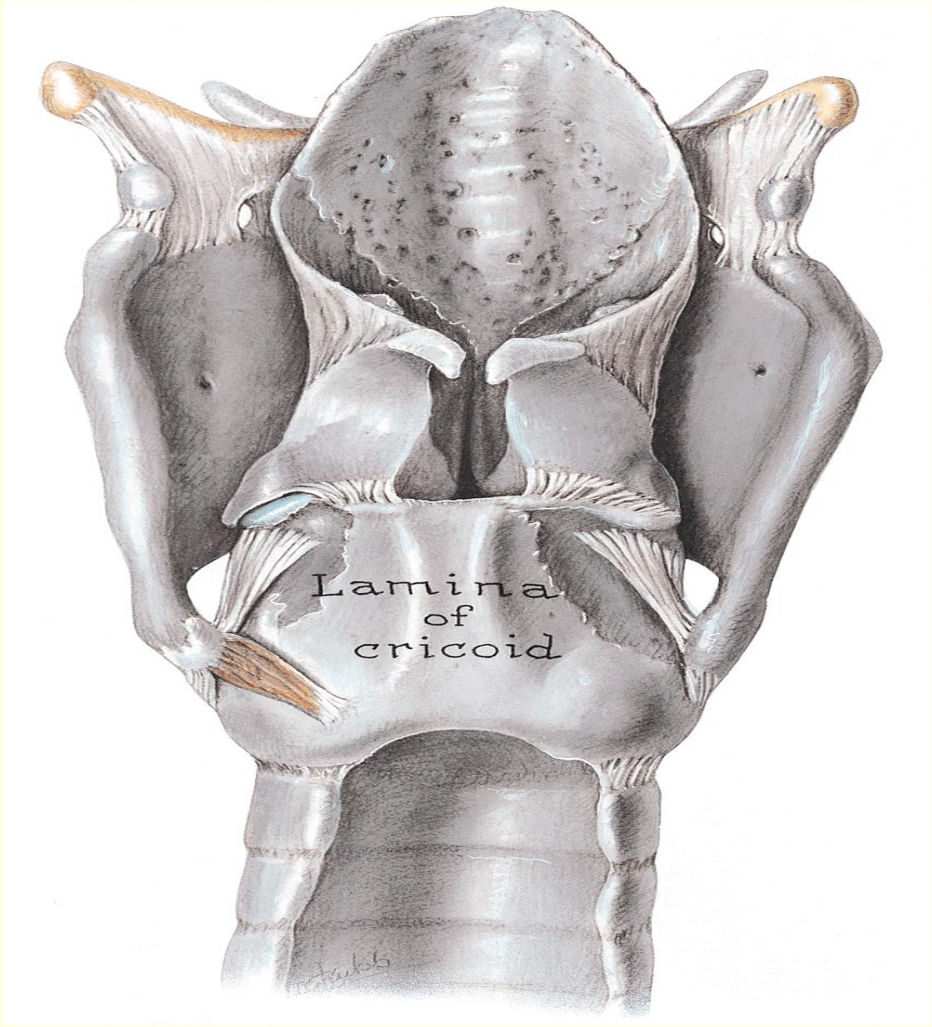
MBChB V

Introduction

- 1 – 5% of all malignancies
- 25% of all head & neck cancers i.e., upper aerodigestive tract, sinonasal & salivary gland tumors
- It is the 2nd MC site for head & neck malignancy.
- Incidence worldwide varies from 3 to 10 per 100,000 thousand people.
- Male: female ratio of 4:1 (the ratio was higher but with more females practicing smoking & alcoholism, the ratio between M:F has decreased).
- It is more prevalent in low socio – economic classes.

Anatomy





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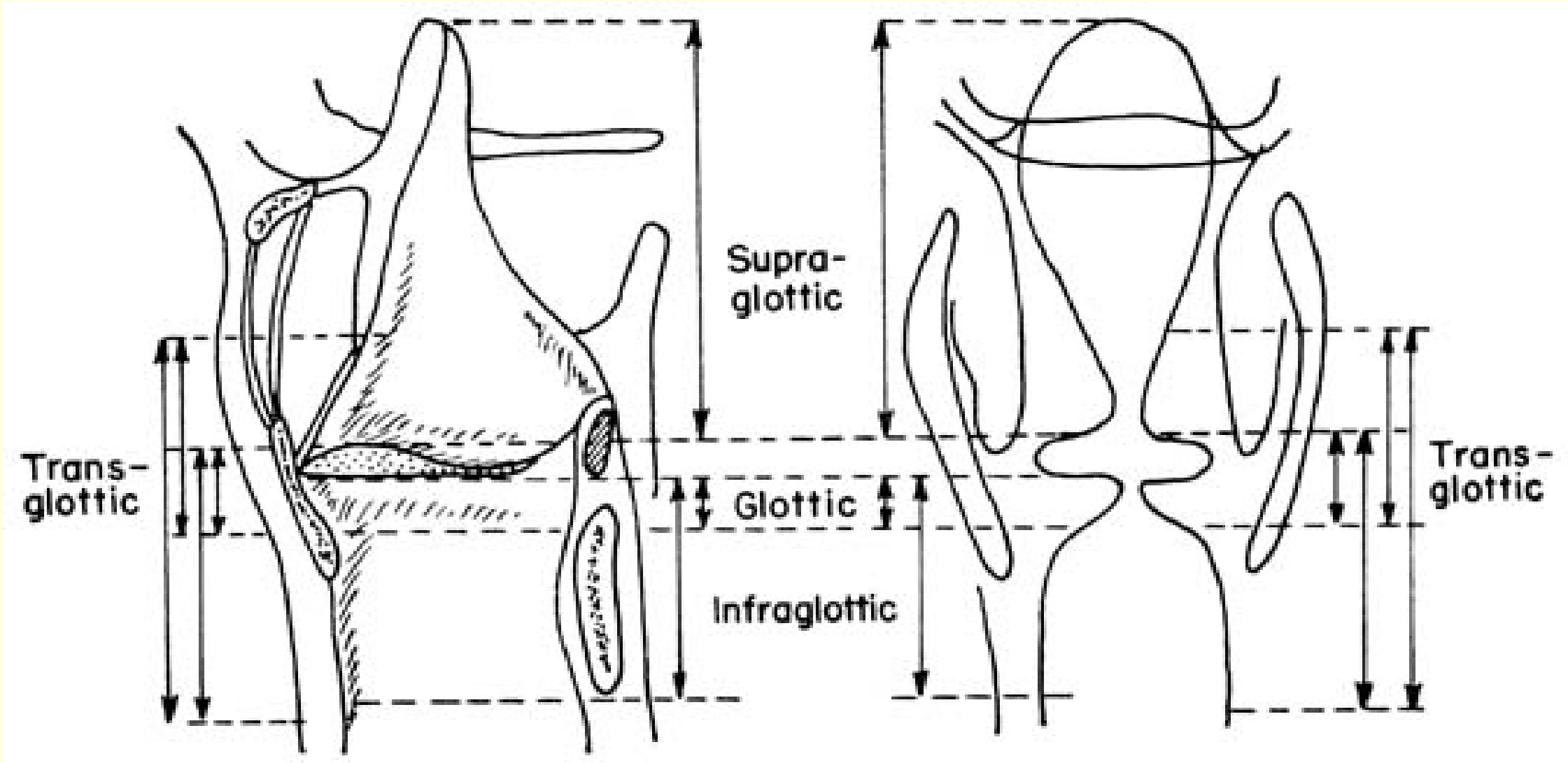

Supra-
glottic
Trans-
glottic
Glottic
Infraglottic
Trans-
glottic

LARYNGEAL REGIONS/SITES

- Supraglottis:
  - Epiglottis tip & laryngeal surface
  - Aryepiglottic folds
  - Arytenoids
  - False vocal cords or ventricular bands
  - Laryngeal ventricles
- Glottis:
  - Free margins & upper surface of both vocal cords
  - Anterior commissure
  - Posterior commissure
- Subglottis: From undersurface of vocal cords to inferior border of cricoid cartilage

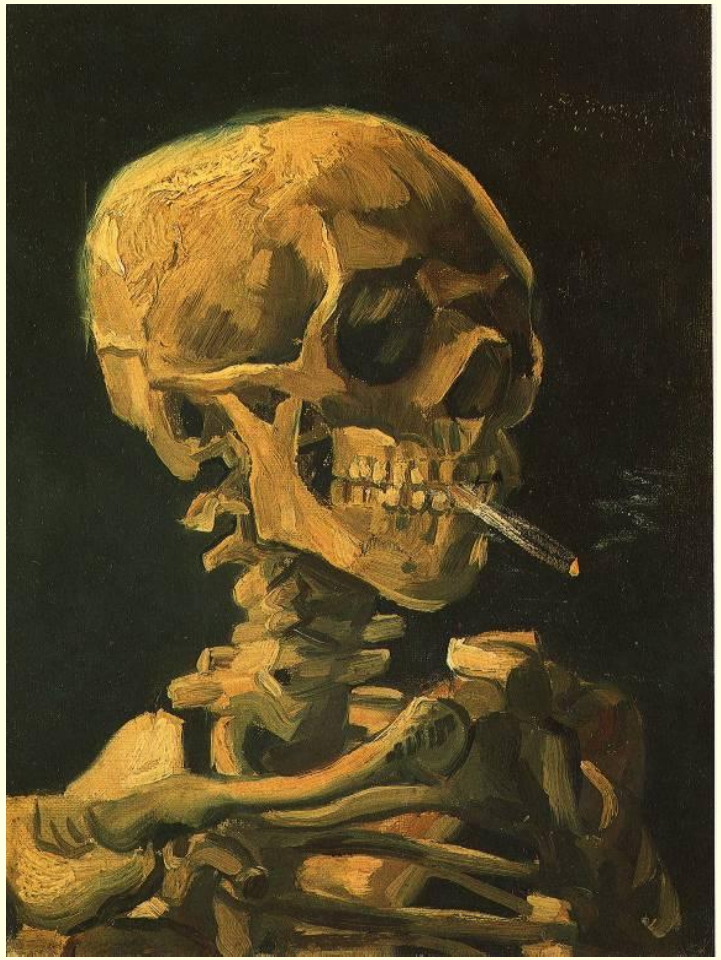
Subtypes

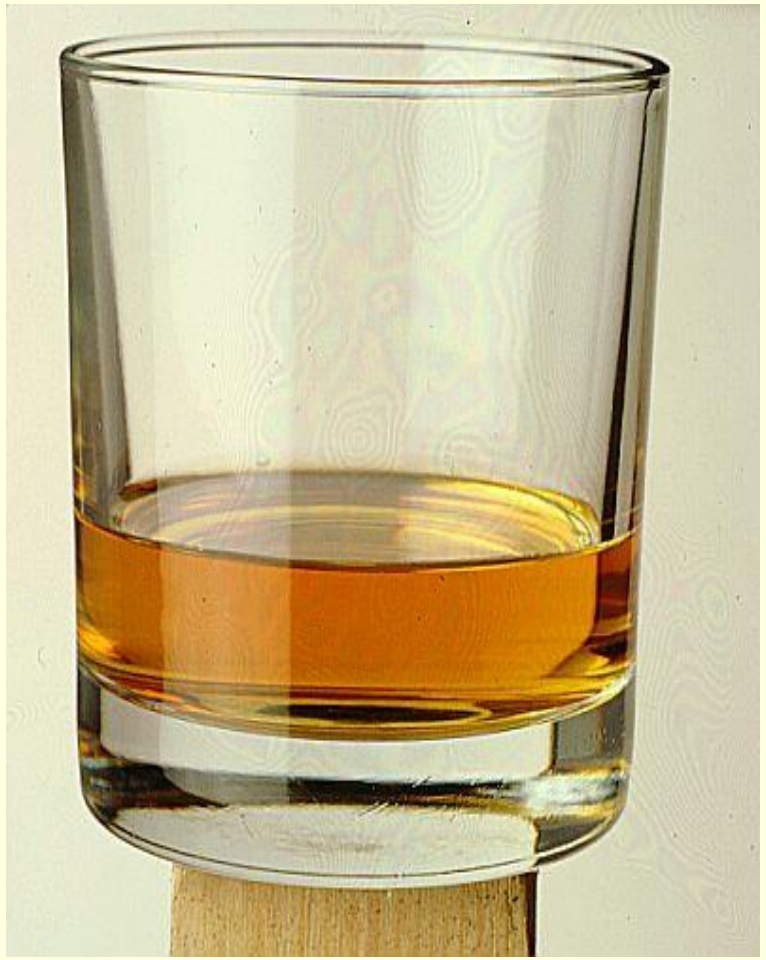
- Glottic Cancer: 50 – 75%
- Supraglottic Cancer: 30 – 40%
- Subglottic Cancer: 1%
  - Most subglottic masses are extension from glottic carcinomas

RISK FACTORS

- Smoking & alcohol have synergistic effects
- Previous radiation up to 10 yrs. Ago
- Environmental pollutants e.g. asbestos, paint, wood dust etc.
- Laryngopharyngeal reflux in GERD.
- HPV types 16 & 18
- Plummer – Vinson Syndrome
- Low SES
- (?) racial predilection

Cont.





CLINICAL PRESENTATION

- Progressive & unremitting dysphonia or hoarseness esp. in glottic cancers
  - If > 2 – 3 weeks do laryngoscopy
- Globus sensation
- Aspiration
- Neck swelling
- Dysphagia/ odynophagia: if the tumor grows behind towards the pharynx
- Cough & irritation
- Hemoptysis if the tumor ulcerates
- Stridor
- Dyspnea
- Otalgia
- Anorexia, cachexia & fetor oris

HISTORY

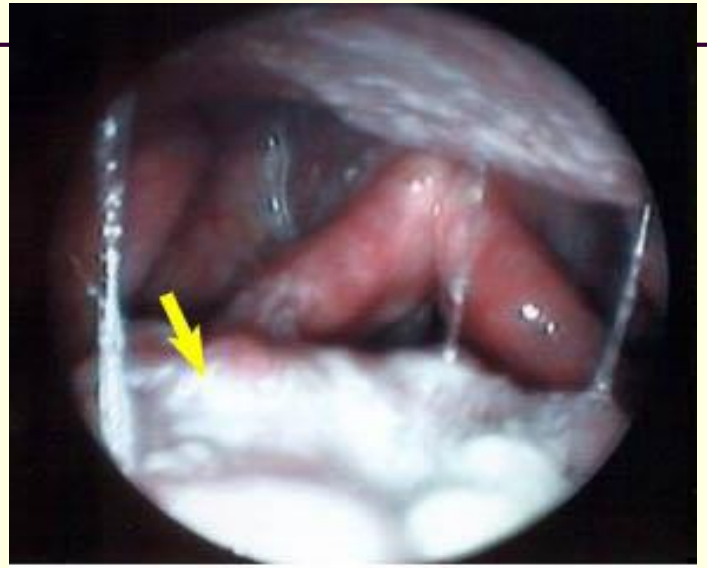
- Smoking & alcohol history
- Hemoptysis
- Familial history of malignancy

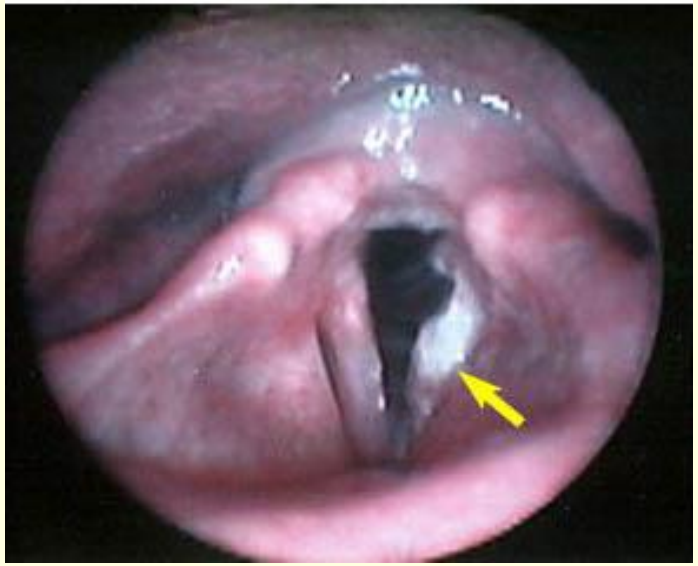
PHYSICAL EXAMINATION

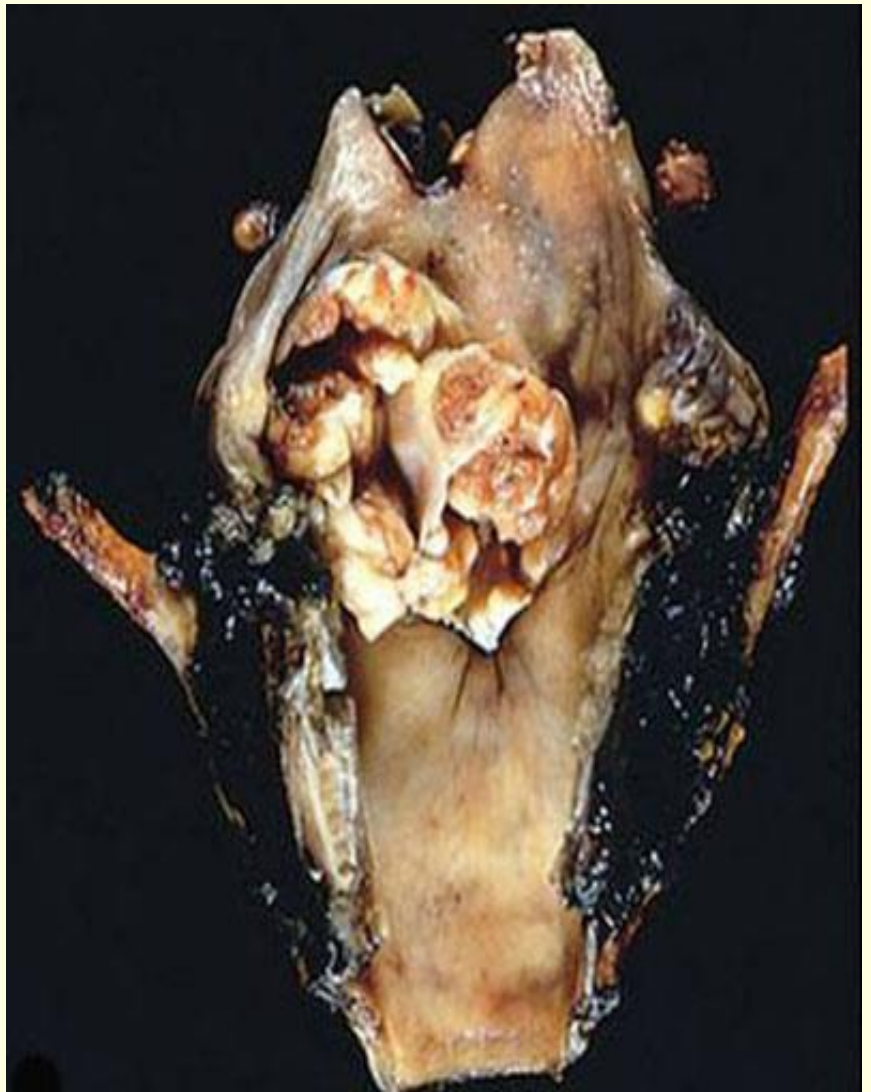
- Throat exam including indirect laryngoscopy
- Larynx
  - Indirect laryngoscopy
  - Flexible fibreoptic laryngoscopy
- Neck:
  - Neck mass
    - Supraglottic tumors are more likely to spread to the lymph nodes
    - Glottic tumors are unlikely to spread to the lymph nodes
  - Laryngeal swelling, widening/ splaying out of the thyroid cartilage & absence of crepitus on moving the thyroid cartilage as the tumor fixes it to the underlying structures
  - Palpate thyroid to exclude invasion
  - Nodes: number, level, size, mobility

Cont.

- Nose
- Ears
- General exam
- Respiratory exam: rhonchi, features of aspiration pneumonitis







Radiological investigations

- CT scan of the neck is the gold standard
  - Consider MRI
- CXR: to r/o metastases into the neck
- U/S of the abdomen: to r/o metastases to the liver or any of the abdominal structures
  - If you have a CT scan of the chest, you won't need an U/S
- ?PET Scan

Clinical investigations

- Hemogram
- Biochemical profile: U/E/Cr. LFTs
- Serology: VDRL, HIV
- ECG if above 50
- Pulmonary Function Tests if planning to do laryngectomy

Examination under anesthesia

- Is useful for:-
  - Staging
  - Obtaining biopsy specimen
  - Assess operability of tumor
  - To rule out field cancerization
- Includes:
  - Direct laryngoscopy
  - Hypopharyngoscopy
  - Esophagoscopy
  - Bronchoscopy

Histological examination

- Biopsy examined for:
  - Definitive diagnosis of malignancy
  - Identification of tumor type
  - Differentiation:
    - CIS
    - Well differentiated
    - Moderately differentiated
    - Poorly differentiated
    - Undifferentiated

Pathology

- Squamous Cell Carcinoma is the MC type: 85 – 95%
- Verrucous carcinoma: 1 – 2%
  - Doesn’t respond to radiotherapy hence treatment indicated is surgical.
- Others:
  - Sarcomas,
  - Salivary gland tumors,
  - Neuroendocrine tumors,
  - Metastatic tumors etc.

TNM staging - AJCC

- T – primary tumor
- N – Regional nodes
- M – Distant metastasis

Staging – Primary tumor (T)

TX	Minimum requirements to assess primary tumor cannot be met
T0	No evidence of primary tumor
Tis	Carcinoma in situ

Staging- Supraglottis

T1	Tumor limited to one subsite of supraglottis with normal vocal cord mobility
T2	Tumor involves mucosa of more than one adjacent subsite of supraglottis or glottis, or region outside the supraglottis (e.g. mucosa of base of the tongue, vallecula, medial wall of piriform sinus) without fixation
T3	Tumor limited to larynx with vocal cord fixation and or invades any of the following: postcricoid area, preepiglottic tissue, paraglottic space, and/or minor thyroid cartilage erosion (e.g. inner cortex)
T4a	Tumor invades through the thyroid cartilage and/or invades tissue beyond the larynx (e.g. trachea, soft tissues of neck including deep extrinsic muscles of the tongue, strap muscles, thyroid, or esophagus)
T4b	Tumor invades prevertebral space, encases carotid artery, or invades mediastinal structures

Staging- Glottis

T1	**Tumor limited to the vocal cord (s) (may involve anterior or posterior commissure) with normal mobilty**
T1a	**Tumor limited to one vocal cord**
T1b	**Tumor involves both vocal cords**
T2	**Tumor extends to supraglottis and/or subglottis, and/or with impaired vocal cord mobility**
T3	**Tumor limited to the larynx with vocal cord fixation and/or invades paraglottic space, and/or minor thyroid cartilage erosion (e.g. inner cortex)**
T4a	**Tumor invades through the thyroid cartilage, and/or invades tissues beyond the larynx (e.g. trachea, soft tissues of the neck including deep extrinsic muscles of the tongue, strap muscles, thyroid, or esophagus**
T4b	**Tumor invades prevertebral space, encases carotid artery, or invades mediastinal structures**

Staging- Subglottis

T1	Tumor limited to the subglottis
T2	Tumor extends to vocal cord (s) with normal or impaired mobility
T3	Tumor limited the larynx with vocal cord fixation
T4a	Tumor invades cricoid or thyroid cartilage and/or invades tissues beyond larynx (e.g. trachea, soft tissues of the neck including deep extrinsic muscles of the tongue, strap muscles, thyroid, or esophagus)
T4b	Tumor invades prevertebral space, encases carotid artery, or invades mediastinal structures

Staging- Nodes

N0	No cervical lymph nodes positive
N1	Single ipsilateral lymph node ≤ 3cm
N2a	Single ipsilateral node > 3cm and ≤6cm
N2b	Multiple ipsilateral lymph nodes, each ≤ 6cm
N2c	Bilateral or contralateral lymph nodes, each ≤6cm
N3	Single or multiple lymph nodes > 6cm

Staging- Metastasis

M0	No distant metastases
M1	Distant metastases present

Stage Groupings

STAGE	T	N	M
0	Tis	N0	M0
I	T1	N0	M0
II	T2	N0	M0
III	T3	N0	M0
	T1-3	N1	M0
IVA	T4a	N0-2	M0
	T1-4a	N2	M0
IVB	T4b	Any N	M0
	Any T	N3	M0
IVC	Any T	Any N	M1

Spread of Glottic Tumor

- Lymphatic spread only in advanced stages
- Anterior commissure involvement associated with thyroid cartilage invasion
- Lateral spread to muscles leads to impaired or loss of vocal cord mobility
- May spread to supra- or subglottis

Spread of Supraglottic Tumor

- Early lymphatic spread to upper and/or middle cervical nodes
- May be unilateral or bilateral
- Early pre – epiglottic spread

Spread of Subglottic Tumours

- Lymphatic spread to tracheal nodes and onwards into upper mediastinal nodes

Factors to consider when choosing a treatment modality

- Available structures
- Patient structures
- Patient's expectations
- Disease stage
- Comorbidities: will the patient withstand long hours under gA

Treatment

- Treatment goals:
  - Cure
  - Palliation
  - Rehabilitation

Premalignant lesions

- Includes carcinoma in situ
- Can be treated **surgically** by stripping the entire lesion.
- Some advocate the use of a **CO_2 laser** to accomplish this but there are concerns about accuracy of review of the pathology.
- **Open surgery** may be used.

Early – stage laryngeal cancer (T1 & T2)

- Can be treated with either **radiation therapy** or **surgery** alone.
- Offer about the same (85 – 95%) cure rate.
- Advantages of surgery:
  - Shorter treatment & hospitalization period
  - Spares option of radiation for recurrence
- Disadvantages: may have worse voice outcomes.
- Radiotherapy is given for 6 – 7 weeks, avoids surgical risks, but does have complications:
  - Mucositis, odynophagia, laryngeal edema, xerostomia, esophageal stricture, laryngeal fibrosis, radionecrosis, hypothyroidism etc.

Advanced stage cancer (T3 & T4)

- Treated with a combination of surgery and radiotherapy.
- Start with surgery then follow with radiation because after radiation, tissues become firm and the planes are lost.
- Chemo – radiation and salvage surgery
  - Chemotherapy (cisplatin & 5 – FU)

Treatment of the neck

- Early primary tumors treated with **radiotherapy** receive radiotherapy to the neck.
- Glottic tumors without neck nodes treated with **surgery** do not need any neck dissection.
- Any primary tumors of the supraglottis or subglottis treated by surgery require neck dissection.
- Any tumor associated with nodal metastases requires neck dissection.

Cont.

- Chemotherapy can be used in addition to irradiation in advanced stage cancers
- Two agents used are Cisplatin and 5 – fluorouracil
- Cisplatin thought to sensitize cancer cells to XRT enhancing its effectiveness when used concurrently.
- Studies have shown similar survival rates as compared to total laryngectomy with adjuvant radiation but with voice preservation.
- Role in treatment still under investigation

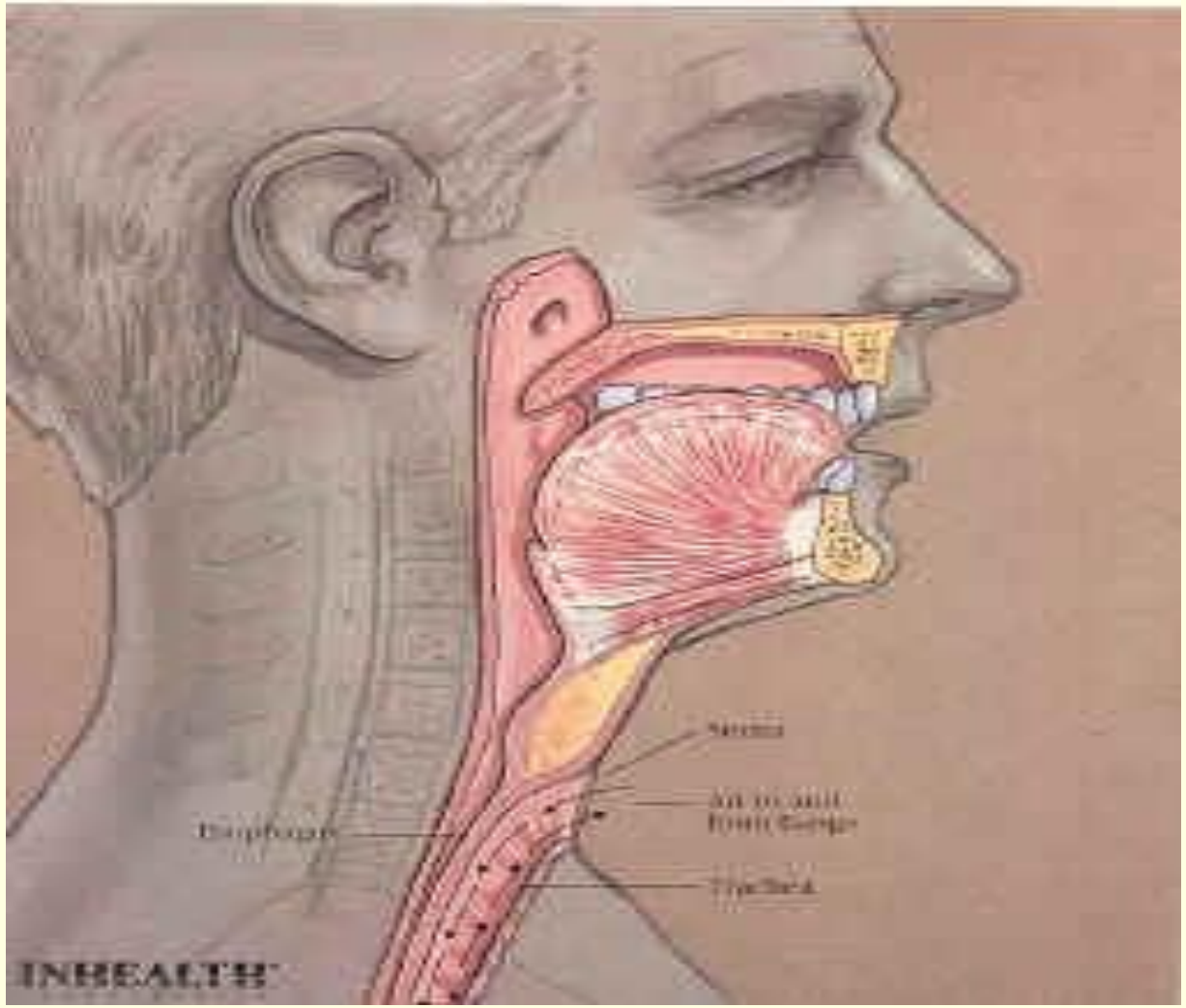
Types of laryngectomy

- Total for advanced disease
- Partial (option that enables organ & perhaps function preservation)
  - Cordectomy: if confined to one cord
  - Supraglottic/ horizontal laryngectomy
  - Hemilaryngectomy
  - Supracricoid
- Near – total laryngectomy: almost ¾ of the larynx is removed and some units are left for voice production.

Complications of total laryngectomy

- Hematoma/seroma
- Wound infection
- Embolism
- Stomal recurrence
- Tracheal crusting
- Pharyngocutaneous fistula
- Hypocalcaemia
- Hypothyroidism
- Pharyngeal stenosis
- Failure to acquire speech

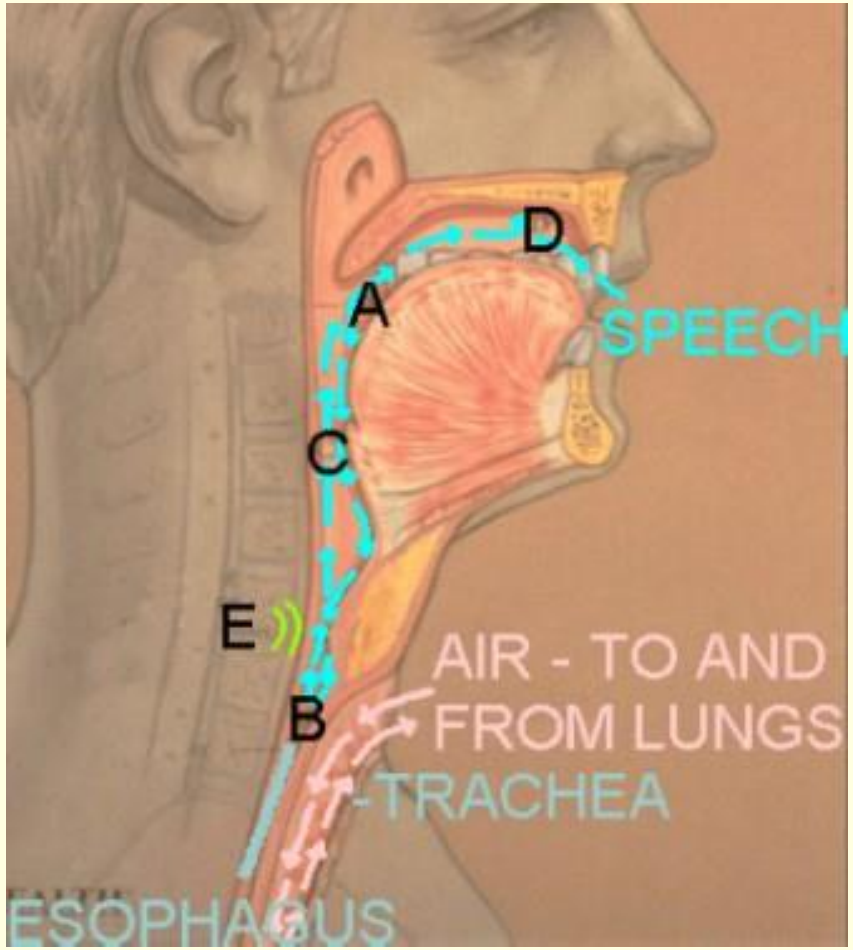
Total Laryngectomy



Rehabilitation

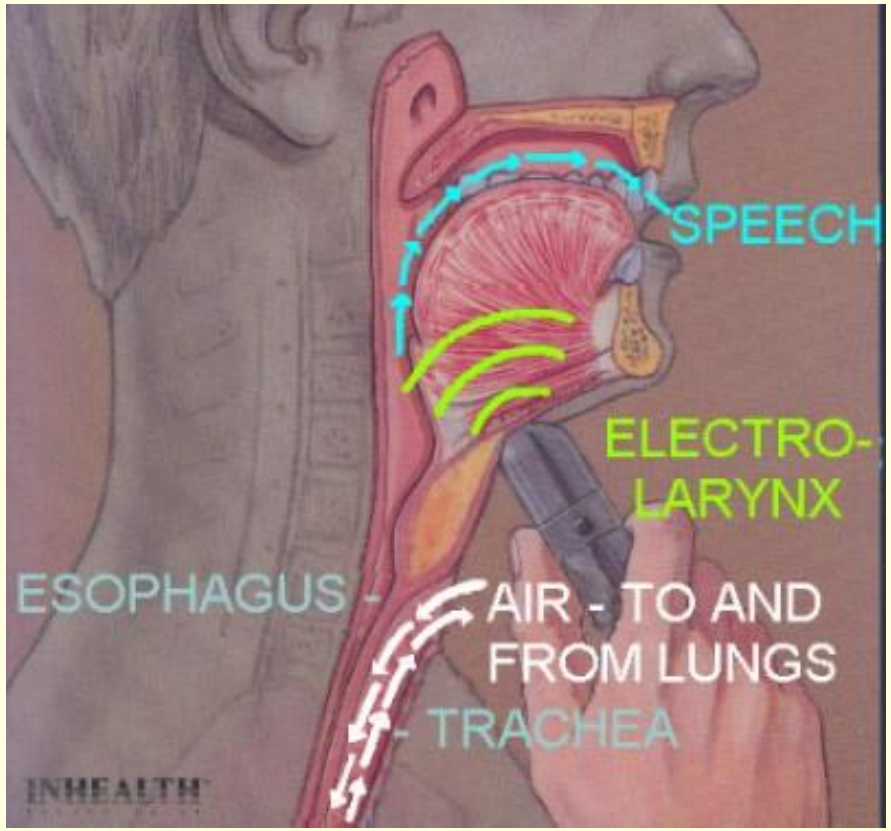
- Speech
  - Tracheostomal prosthesis
  - Electrolarynx
  - Pure esophageal speech
- Swallowing
  - If too difficult settle for a PEG/ gastrotomy

Esophageal speech



A. Tongue press to inject air into esophagus.
B. Air enters esophagus.
C. Air released from esophagus to produce sound.
D. Sound shaped into speech.
E. Location of tissue vibration for sound.

Electrolarynx



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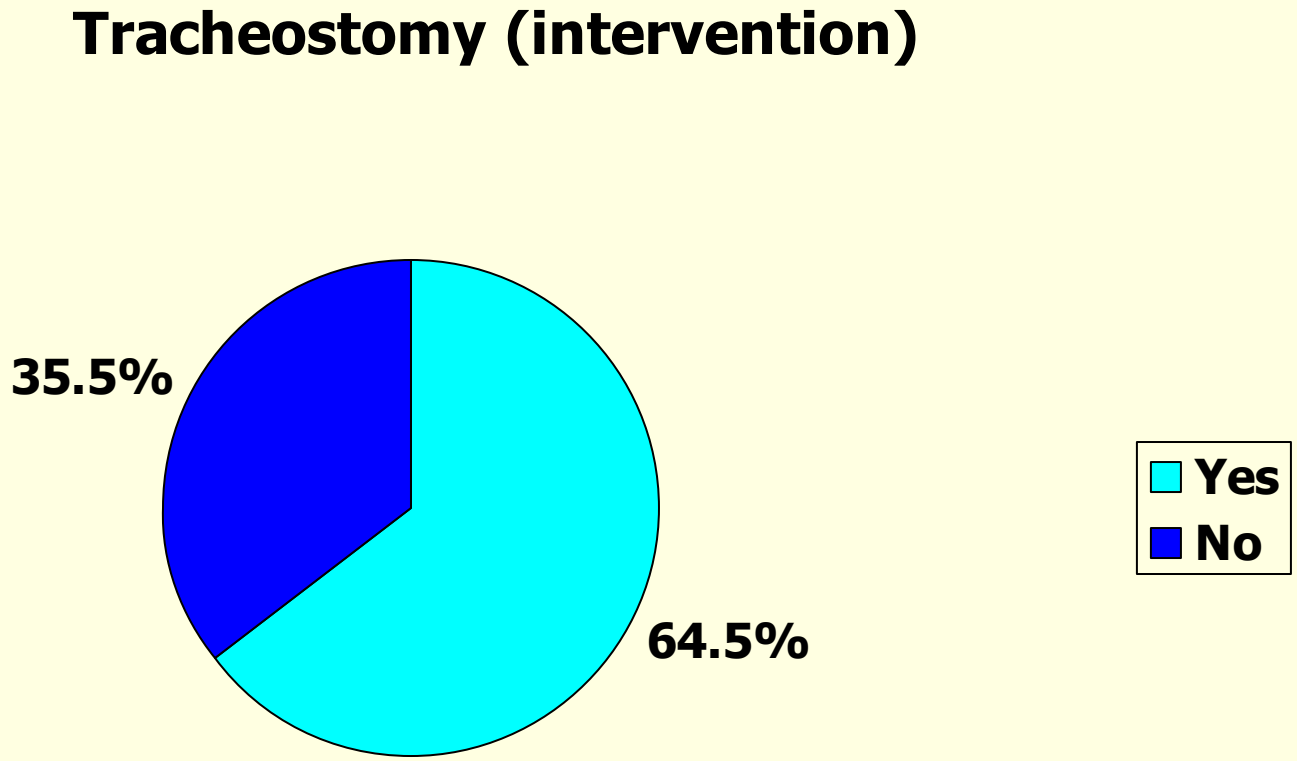
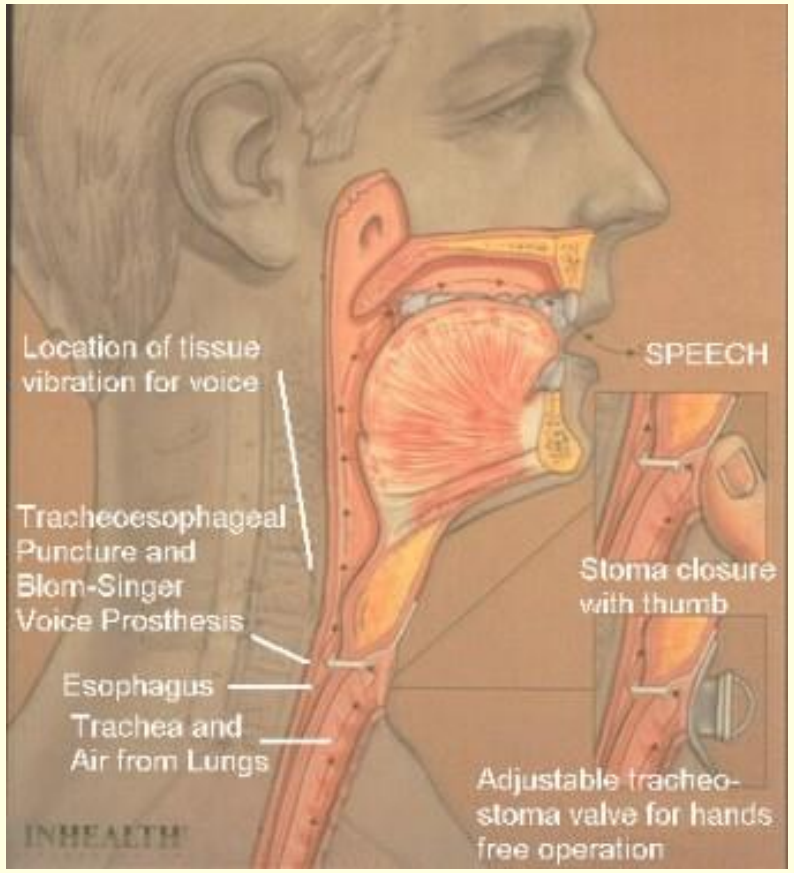
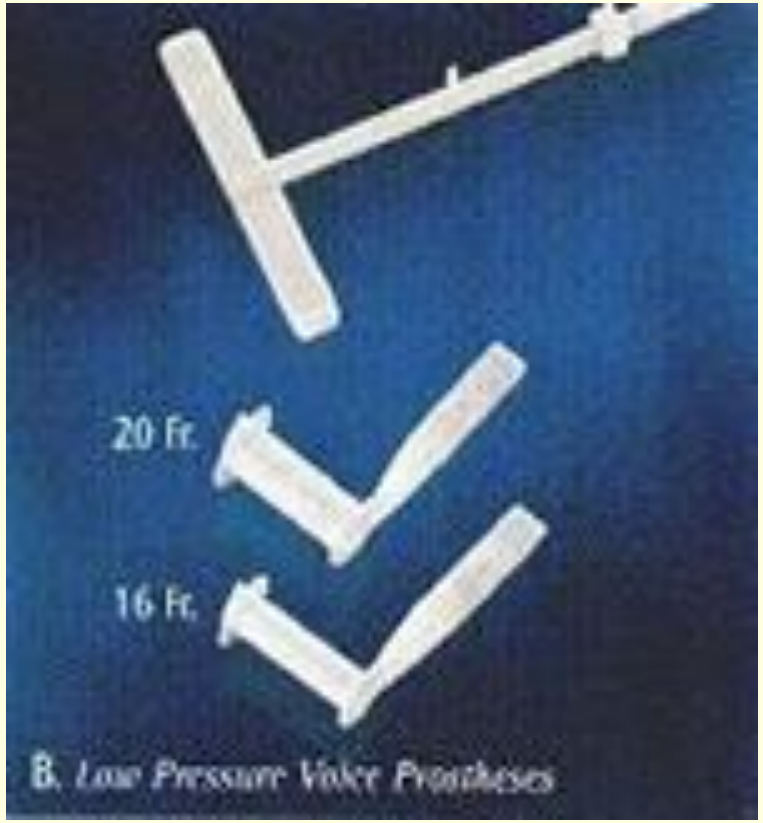


Tracheo – esophageal prosthesis





Prognosis

- Better than that of other neck cancers owing to:
  - Early diagnosis (hoarseness)
  - Most are glottic carcinomas & have a low rate of spread.
- Five year survival for:
  - Stage I → 95%
  - Stage II → 85 – 90%
  - Stage III → 70 – 80%
  - Stage IV → 50 – 60%

Follow up

- For life
- To detect residual disease, recurrence or second primaries
- Seen at 4 – 6 week intervals in first year, every 2 months in second year, every 3 months in 3rd and 4th years and annually thereafter
- Patients are considered cured after five years disease free since most cancers recur in the first two years

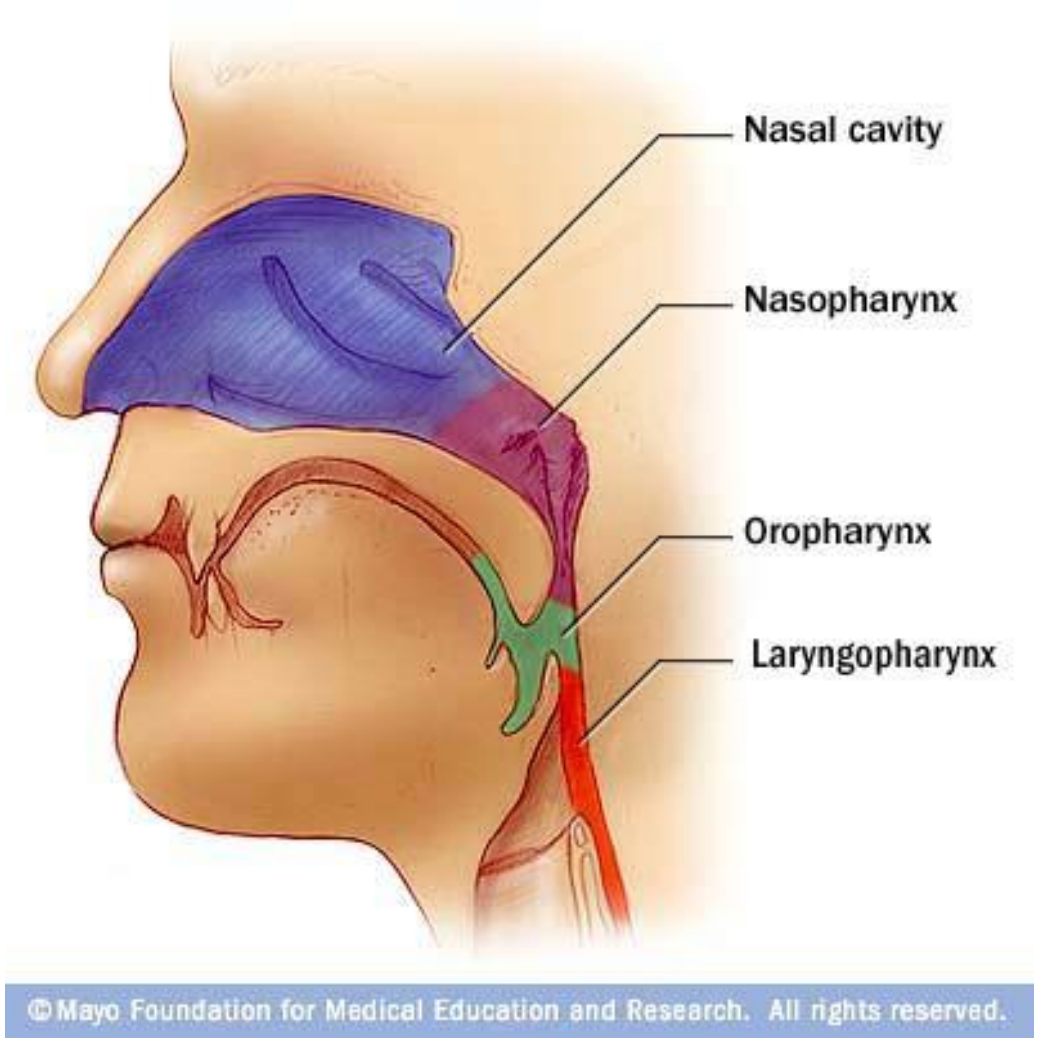
NASOPHARYNGEAL CARCINOMA (NPC)

BY: DR. ASWANI

MBChB VI

2019

NASOPHARYNX

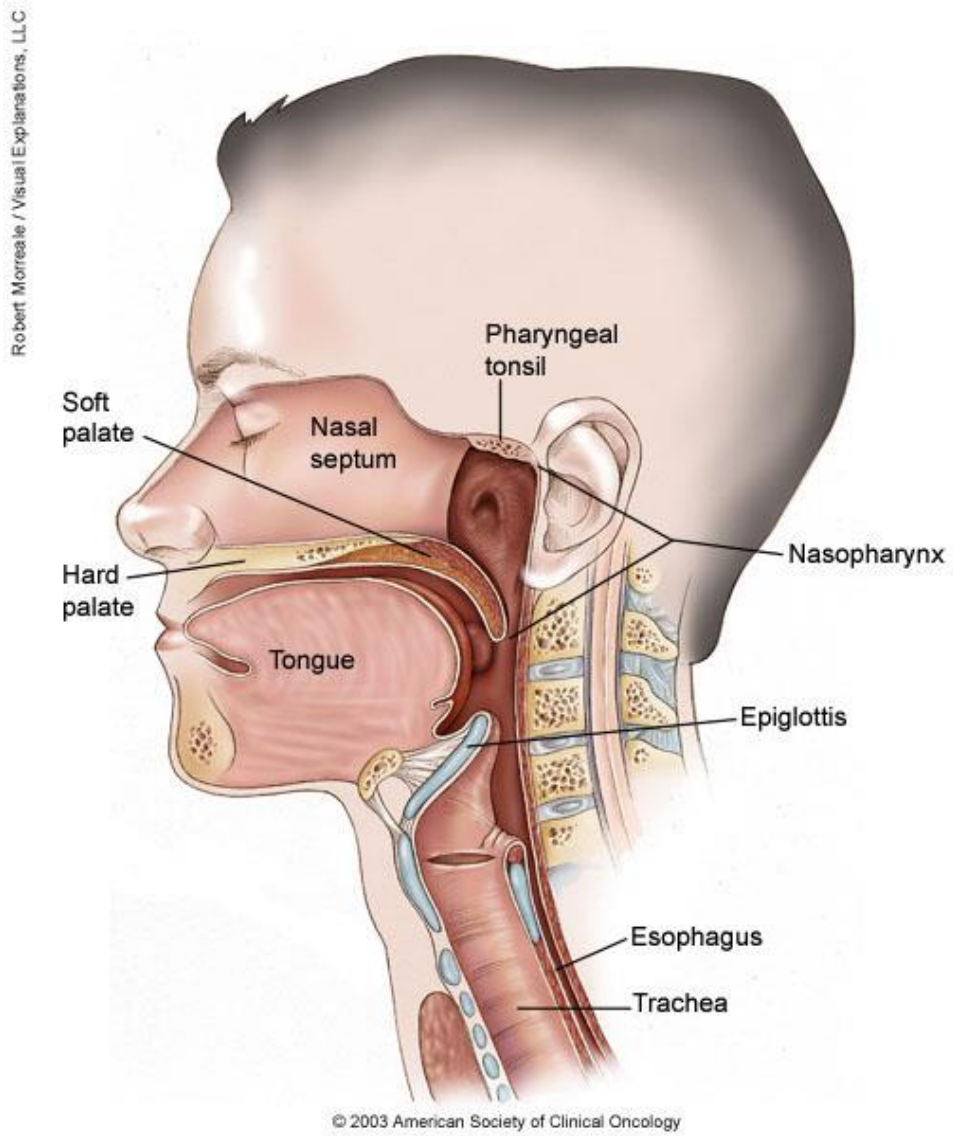


Pharynx starts at the choana where the nose ends & ends where the esophagus begins.

Post – nasal space (PNS) / nasopharynx – Surgical Anatomy

- Space with rigid walls 4cm high, 4 cm wide & 2cm deep.
- Epithelium in infancy is **columnar ciliated** but in adults it undergoes squamous metaplasia except in ***Fossa of Rosenmuller***
- Submucosa has abundant lymphoid stroma (adenoids).
  - Atrophy with growth.

The Nasopharynx



SURGICAL ANATOMY - WALLS

- Anterior – choana & nasal septum
- Floor/ inferiorly – soft palate & opening of oropharynx
- Lateral – Eustachian tube openings & fossae of Rosenmuller
- Roof/ superior – base of skull inferior to body of sphenoid
- Posterior – bodies of C1 & C2. It is continuous with roof

EPIDEMIOLOGY

- Dependent on interplay of genetic & environmental factors
- **High incidence** in Southern China – Kwang Tung province
- **Intermediate incidence** in Kenya & parts of N. Africa
- **Low incidence** as in N. America.

Cont.

- High incidence areas have one age peak: 35 – 50 yrs.
- ~~Low~~ Intermediate incidence areas have two age peaks (Bimodal): 9 – 15 & 35 – 50 yrs.
- 2nd commonest head & neck cancer in Kenya
  - Following cancer of the oral cavity
  - Followed by laryngeal carcinoma
- Male: female → 3:1 to 3:2

Aetiology

- Epstein – Barr Virus (EBV) → commonest aetiology in high incidence areas.
- Genetic factors
- Dietary habits
- Environmental factors
- Others: Human papillomavirus (HPV), smoking.

EBV

- Only undifferentiated or poorly differentiated forms are consistently associated with EBV. Common in the Chinese populations.
- NPC arises **many years** after peak incidence of EBV infection
- Evidence:
  - EBV markers have been found in NPC cells e.g. viral capsid antigen (VCA), early antigen (EA), antibody dependent cell mediated cytotoxicity (ADCC) & the nuclear antigen (EBNA)

Dietary habits

- Nitrosamines in salted fish → 90% of NPC among young Hong Kong Chinese attributable to consumption of ***salted fish***.
- Other preserved foods e.g. dried fish, salted duck eggs, fermented soya bean paste etc.

Environmental factors

- Thought to influence regional distribution hence static high incidence among indigenous Chinese populations compared to decline among Chinese born in N. America
- ?Highland areas in Kenya: people in cold areas stay indoors and get exposed to smoke?

Genetic factors

- HLA typing among Singapore Chinese has shown high prevalence with certain HLA groups.

Others

- Polycyclic hydrocarbons
- Chronic nasal infection
- Poor hygiene
- Poor ventilation
- Smoke
- Tobacco smoking: associated with the well differentiated types & mostly in the low incidence areas like in America.
- HPV
- ? Hormones: testosterone, *Dehyndrepiandrosterone*

Clinical presentation

- Nasal symptoms (***initially unilateral***):
  - Obstruction: if the tumour grows anteriorly into the nasal cavity
  - Epistaxis
  - Rhinorrhoea: blockage as mucus is not draining posteriorly as it should.
  - Nasal mass in advanced cases
- Neck mass (due to metastases to the cervical lymph nodes)
  - Most common presentation.
  - Occurs in 70%
  - Usually painless

Cont.

- Aural (initially unilateral): due to growth of the tumor laterally where the openings of the Eustachian tubes are
  - Second most common (50%)
  - Ear blockage
  - Aural fullness
  - Otitis media with effusion: due to negative pressure in the middle ear causing fluids to accumulate in the middle ear
  - Conductive hearing loss
  - Otalgia
- Trismus if the tumor grows laterally involving the muscles of mastication.

Cont.

- Oropharyngeal: due to growth of the tumor inferiorly towards the oropharynx
  - Mass
  - Displaced palate
  - Postnasal drip
  - Hyponasal speech
  - Dysphagia/odynophagia
  - Airway obstruction

Cont.

- Orbital: if the tumor grows superolaterally towards the orbit
  - Advanced disease
  - Proptosis
  - Visual disturbance
  - Immobile EOMs.

CNS INVOLVEMENT

- Xerophthalmia → greater sup. petrosal n
- Facial pain → Trigeminal n.
- Diplopia → CN VI
- Ophthalmoplegia → CN III, IV & VI
  - Cavernous sinus or superior orbital fissure
- Horner's syndrome → cervical sympathetic involvement
- Cranial nerves in extensive base of skull involvement: IX, X, XI, XII
- Headache
- Convulsions

Tumor Spread

- Direct submucosal extension
- Lymphatic spread: nodes of ***Rouviere***
- Haematogenous: to liver, lungs, bones

Distant Metastases

- Rare (<3%) to:-
  - Lungs
  - Liver
  - Bones:
    - Spine
    - Pelvis
    - Ribs

DIAGNOSTIC WORK UP

- History:
- Clinical Examination
- Investigations

History

- ENT:-
  - Otological/ aural symptoms
  - Nasal symptoms
  - Throat symptoms
- Local extension:
  - Orbital
  - CNS
  - Trismus
- Metastases:
  - Nodal status
  - Chest/abdominal/skeletal
- Query about risk factors

Examination

- Focus:
  - Anterior rhinoscopy
  - Otoscopy & tuning fork tests (Weber & Rinne's test)
  - Throat examination: abnormal post – nasal drip, displace of palate
  - Cervical lymphadenopathy
  - Orbital: proptosis, mobility of eyeball
  - Cranial nerves
- Examine other systems

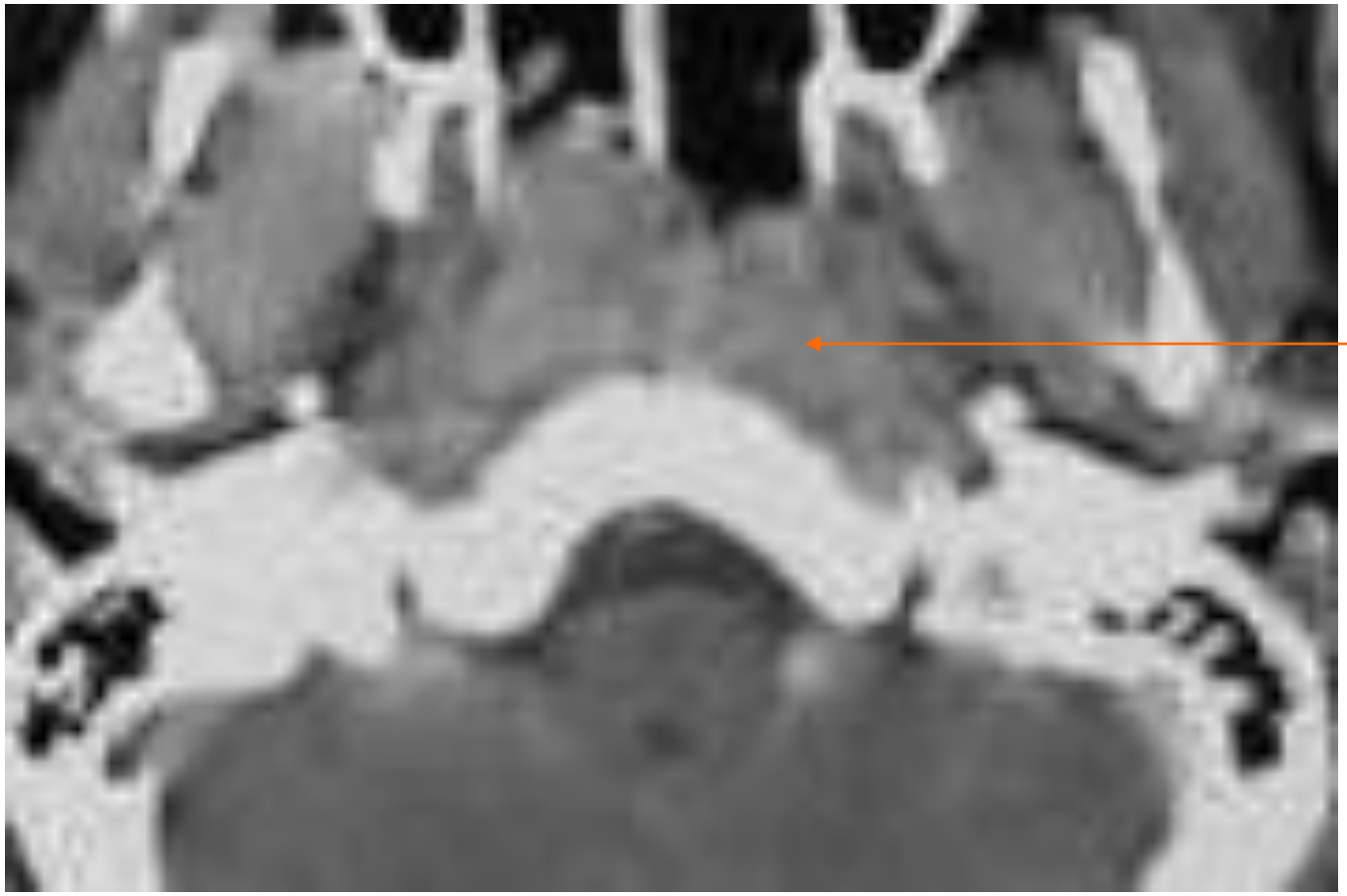
Laboratory studies

- FBC:
  - Low Hb due to bleeding; fit for treatment
- Urea, creatinine & electrolytes
- Liver function tests
- Audiometry: confirms hearing loss
- EBV – related antigens are not done routinely - research & screening
  - Fall & rise with the resolution or recurrence of the tumor.

IMAGING STUDIES

- CT scan of the neck: investigation of choice for extent of disease
- Metastases:
  - Chest → CXR
  - Abdomen → Ultrasound
  - Bone → skeletal scan (when applicable)

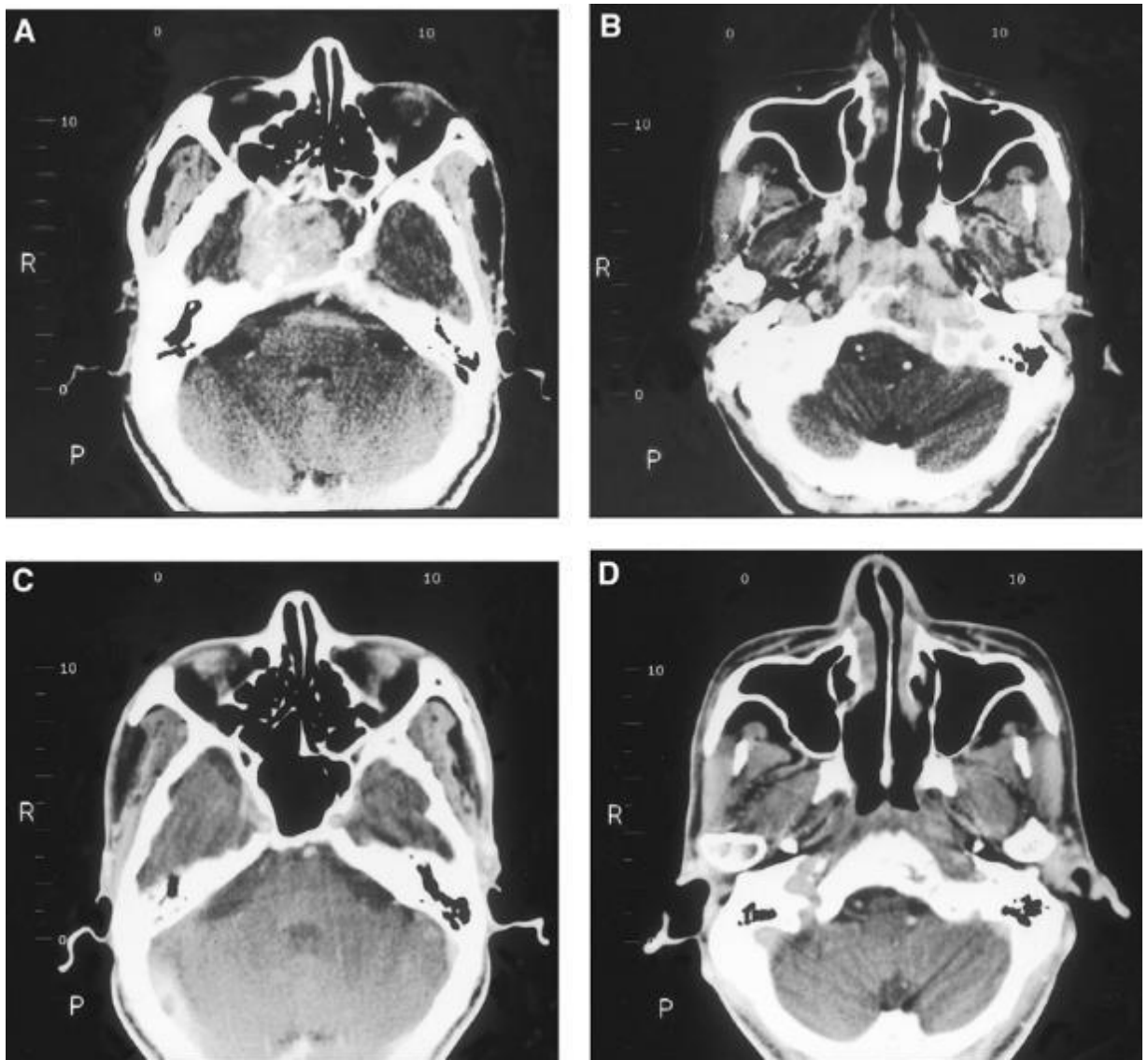
NPC CT SCAN



Mass in the nasopharynx obstructing the choana

NPC – CT SCAN

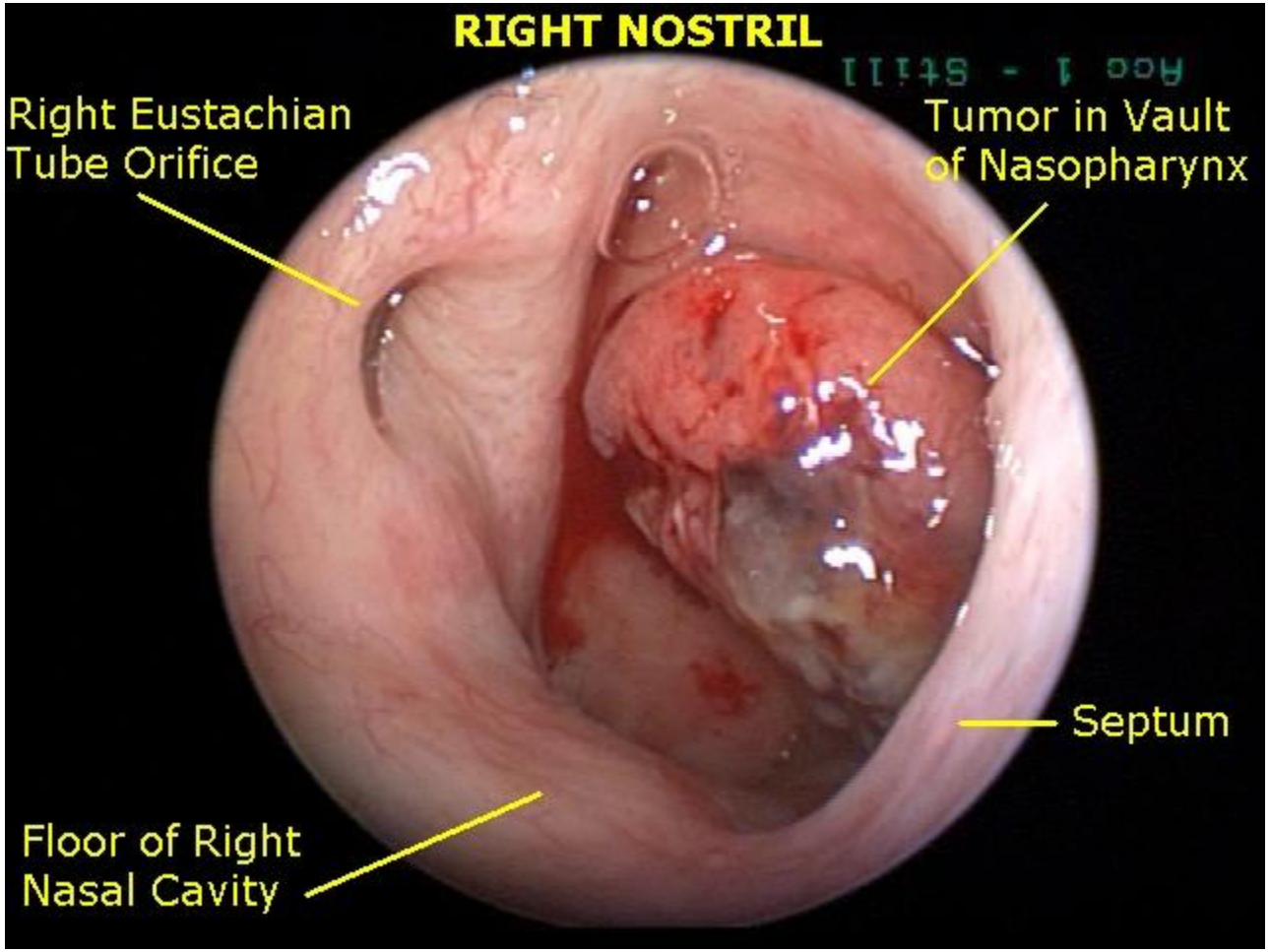
Opacification of mastoid air cells



Biopsy

- Obtained from nasopharynx under vision using endoscope under GA or LA (xylocaine)
- FNAC from neck nodes – if indicated
- Biopsy necessary for histological diagnosis and/or immunohistochemistry

Endoscopic view of NPC



WHO Classification

1. WHO TYPE I: Keratinising SCC (well differentiated)
   - Associated with smoking in low incidence areas
2. WHO TYPE II: Non – keratinising SCC
3. WHO TYPE III: Undifferentiated SCC
   - Types II & III are associated with EBV infection in high incidence areas

TNM Staging

- Different staging systems employed
- AJCC

T – Primary Tumor

- Tx: Minimum requirements for assessment of primary tumor not met
- T0: No primary tumor
- Tis: carcinoma in situ (has not gone below the basement membrane)
- T1: Primary tumor confined to nasopharynx or extends to oropharynx and/or nasal cavity without parapharyngeal extension

T – Primary Tumor

- T2: Primary tumor with parapharyngeal extension
- T3:Primary tumor involves bony structures of skull base and/or paranasal sinuses
- T4: Primary tumor with involvement of intracranium, cranial nerves, hypopharynx , orbit, infratemporal fossa or masticator space

N – Regional Nodes

- Nx: Regional lymph nodes cannot be assessed
- N0: No regional lymph node metastases
- N1: Unilateral nodes ≤ 6cm above supraclavicular fossa
- N2: Bilateral nodes ≤ 6cm above supraclavicular fossa
- N3: Nodes :
  - N3a: greater than 6cm
  - N3b: in supraclavicular fossa

Distant Metastases

- MX: Distant metastases cannot be assessed
- M0: No distant metastases
- M1: Distant metastases

Treatment

- Early tumors (T1 – 2): Radiotherapy to primary site and bilateral neck
- Advanced tumors (T3 – 4): Chemoradiation

Role of surgery

- Limited to:
  - Biopsy
    - REMEMBER: opening a lymph node with metastases upstages the tumor.
  - Neck dissection for residual neck disease when primary disease is cured
  - Salvage surgery for small primary residual or recurrent tumor

Prognosis

- Dependent on stage of disease. 5 year survival rates are:
  - Stage I – 90%
  - Stage II – 70%
  - Stage III – 60%
  - Stage IV :
    - Without distant metastases → 40%
    - With distant metastases → 0%

TAKE HOME MESSAGE

NPC is easily misdiagnosed as allergic rhinitis (nasal blockage & post – nasal drip). Try & catch these patients early enough before the tumor spreads.

END

A stone is broken by the last stroke of the hammer.

This doesn't mean that the 1st stroke is useless.

~ Persistence ~

#Jesus_Is_Lord☺