4 YEAR ENT NOTES

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HISTORY AND PHYSICAL EXAMINATION

The Ear

- Symptoms relevant to hearing
  - Pain: Otalgia
  - Discharge: Otorrhoea
  - Deafness
  - Tinnitus

SYMPTOMS RELEVANT TO BALANCE

- Dizziness
- Vertigo → 'things seem to be moving round'
- Unsteady gait
- Oscillopsia → 'when I am walking I see people going up and down'
- Nausea/vomiting

EXAMINATION OF THE EAR

- Inspection of pinna and conchus
- Inspection of the external meatus and the ear drum using an otoscope
  - Hold the otoscope like a pen.

Features:

- Swelling
- Reddening → inflammation
- Fungi
- Otorrhoea
- Perforation of the ear drum

Equipment:

- Otoscope
- Ear speculum
- Microscope
- Otoendoscope

TESTING OF HEARING

- Speech
- Tuning forks: conductive hearing loss vs. SNHL (Sensorineural hearing loss arises from the labyrinth)

- Audiometers: Shows how much one is hearing and what frequency it is.
- Tympanometers: Tell the pressures in the middle ear where the ossicles are
- BSER

EXAMINATION AND TESTS OF THE BALACE SYSTEM

- Fistula test
- Gait
- Romberg test
  - Close the eyes, patient sways
- Nystagmus: spontaneous or induced
- Past-pointing
- Dysdiadochokinesis
- Video-nystagmography

THE NOSE AND PARANASAL SINUSES SYMPTOMS

- Nasal sinuses drain into the nose.
- They include:
  - Maxillary, Ethmoid, Sphenoid and Frontal
- Patients may complain of:
  - Nasal blockage
  - Rhinorrhoea
  - Loss of smell sensation (Anosmia)
  - Epistaxis
  - Cacosmia or Halitosis
  - Headaches
    - ANY PROBLEM IN THE NOSE AND SINUSES PRESENTS WITH HEADACHES (REFERRED HEADACHE)

Inspection and examination:

- Nasal profile
- Anterior & posterior rhinoscopy
- Naso-endoscopy
- Flexible nasopharyngoscope

SIGNS IN PARANASAL SINUS DISEASE

- Blood
- Obstructive turbinates
  - In allergic people
  - Cause blockage
- Polyps

  - Seen through a naso-endoscope
- Neoplasms
- Deviated nasal spasm
- Pus

INVESTIGATION TECHNIQUES

- Lateral soft tissue cervical plain X rays
- CT scans, MRI scans
- Naso-endoscopy and biopsy
- Rhino-manometry
- Allergic tests

ORAL CAVITY AND THROAT SYMPTOMS

- Dysphagia
- Odynophagia
- Lump in throat sensation
- Snoring
- Pain in mouth
- Ulcer in mouth

SIGNS

- Ulcer
- Stomatitis
- Oral/pharyngeal thrush
- Papilloma (warts)
- Infected/enlarged tonsils
- Redundant uvula

LARYNX AND CERVICAL TRACHEA SYMPTOMS

- Hoarseness
  - Chronic laryngitis is a differential in chronic hoarseness
- Respiratory stridor

EXAMINATION TECHNIQUE

- Indirect laryngoscopy
  - Use a mirror
  - Spray LA to prevent gagging and retching in the patient
- Naso-pharyngo-laryngoscope
- Rigid direct laryngo-pharyngoscopy
- Fibre-Optic laryngo-pharyngoscopy (with camera and monitor)
- Stroboscopy

CERVICOFACIAL AREA

- That area of the head and neck excluding the eyes, the brain, the spine and the great vessels.

SYMPTOMS

- Swelling
- Ulcers
- Sinuses

EXAMINATION

- Ask patient to identify site
- Inspect → walk around patients
- Palpate the swelling:
  - Solid or cystic
  - Mobile or fixed
    - A mobile mass is easy to remove as it has not attached to adjacent structures.
    - If fixed → infiltrating cancer probably
  - Measure it
  - Discrete or diffuse or lobulated
  - Sinuses → fungal infection or tuberculous infection
  - Pulsatile → either a very vascular mass, it is overlying a blood vessel, it is an aneurysm or a carotid body tumour.
    - DO NOT BIOPSY A PULSATILE MASS!
  - Painful → inflammation; painless → tumour
- Stand behind the patient and palpate the mass plus the whole neck and head
- If probably neoplastic (most probably of lymphoid origin), look for the primary site
- If no primary, do FNA first

EPISTAXIS

Definition

Acute haemorrhage from the nostril, nasal cavity, or nasopharynx

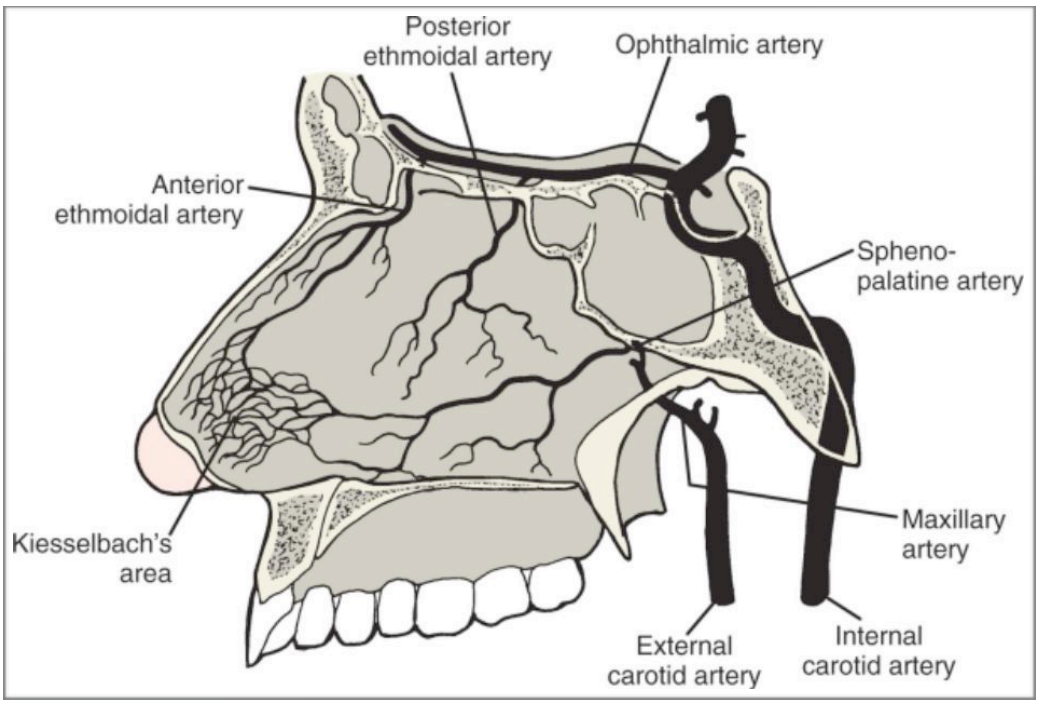
Introduction

- One of the most common ENT emergency
- Male to female ratio 1.6:1
- Higher incidence in older patients
- Clinically — bleeding either from the lateral nasal wall or from the septum.
- Minor epistaxis usually originates from the anterior nasal septum
- Is often the result of minor trauma to the septal mucosa.
- Children — A result of nose picking
- Adults — A result of desiccation of the mucosa

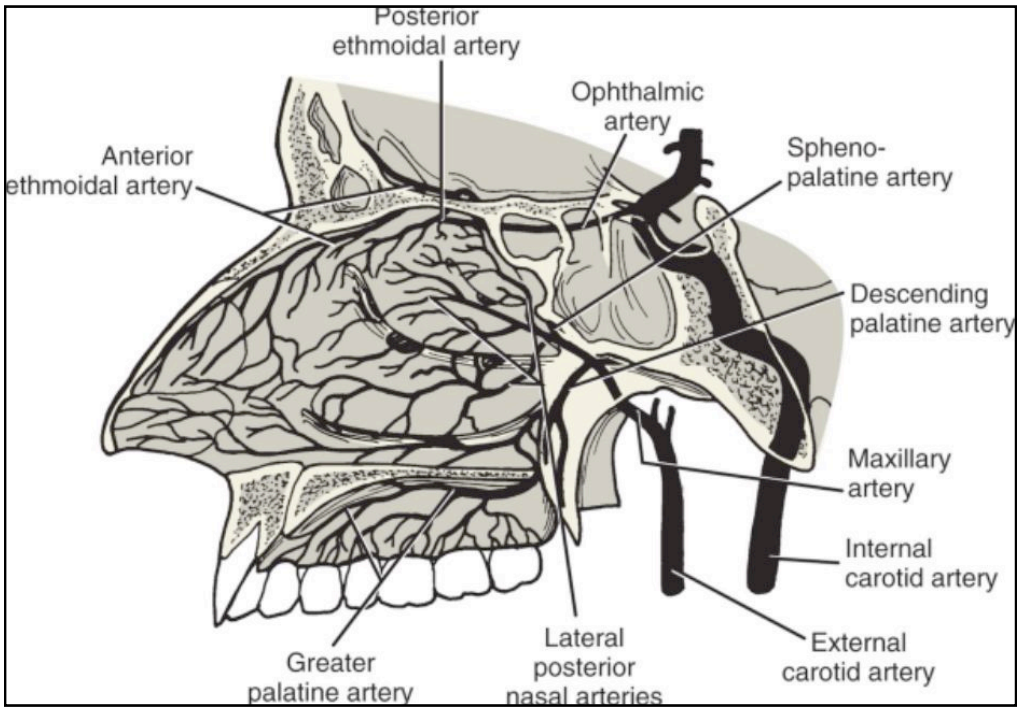
Vascular supply of the nose

- The nasal mucosa has a rich arborising network of submucosal vessels.
- Arterial blood supply from internal and external carotid arteries.
- Confluence of the two systems occurs particularly at the caudal end of the septum.
- A number of arteries anastomose with each other → Little's area.
- The anterior septal plexus is termed Little's area or Kiesselbach's plexus
- Is a confluence of
  - Septal branch of sphenopalatine (Ext. Carotid artery)
  - Septal branch of superior labial artery (Ext. Carotid artery)
  - Greater palatine artery (Ext. Carotid artery)
  - Anterior ethmoidal (Int. Carotid artery)
- This is the site of most anterior epistaxis
- **Retrocollumellar vein** runs 2 mm parallel and behind the columella
  - Is superficial
  - Is a common reason for venous bleeding in children
- Venous epistaxis from retrocollumellar vein tends to occur in subjects <35yrs
- Venous epistaxis usually short lived
- **Woodruffs plexus** is a plexus of vessels lying inferior to the posterior end of the inferior turbinate
  - It is a frequent site of adult epistaxis.
  - It causes a venous posterior bleed.
- 70% of the bleeding occurs from the septum

Vascular supply of the nasal septum



Vascular supply of the lateral nasal wall



Aetiology:

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

Local:

1. Trauma
   - Nose picking, facial trauma, RTA, fracture base of skull.
2. Idiopathic (from Little's area)
3. Inflammatory
   - Rhinitis (infective, allergic), Sinusitis, Specific nasal infections (bacterial, fungal, TB)
4. Anatomical/structural deformities of the nose
   - congenital or acquired
   - deviated nasal septum
   - Nasal spur
   - Hypertrophied or rotated turbinates (paradoxical) drying, crusting, bleeding
5. Neoplastic (Benign or malignant)
   - in the nose or paranasal sinuses and postnasal space tumours
   - Juvenile angiofibroma (exclusively in the adolescent males, recurrent and severe episodes of epistaxis. Never biopsy since patient will bleed excessively)
   - Aneurysms of internal carotid artery
6. Environmental;
   - high altitude
   - air conditioning
   - toxic or chemical irritant
   - Cold winter weather
7. Foreign bodies;
   - Unilateral, purulent nasal discharge and bleeding.
   - Usually in children or the mentally retarded
8. Iatrogenic;
   - excessive prescription of intranasal topical steroids
   - can lead to changes in mucosa and bleeding
   - After nasal surgery. (septoplasty, FESS)

General causes

9. Hypertension - associated with local factors
   - elderly; arteriosclerotic vessels do not contract well and the nasal mucosa becomes atrophic hence dries up and cracks easily and vessels may rupture especially during a hypertensive episodes
10. Blood dycrasias
    - vary in ability to cause epistaxis
    - Usually diagnosed in early life by excessive bleeding after minor trauma.
    - Deficiency of factors VIII (Haemophilia A), (Haemophilia B). Factor IX
    - Von Willebrand factor VII impaired PLT adhesiveness.
    - Leukaemia, lymphomas, Idiopathic thrombocytopenia purpura ITP, ossler Rendeu weber syndrome
11. Alcohol abuse — poor diet especially Vitamin C, K deficiency
12. Parenchymal liver damage (deceased fibrinogen and prothrombin
13. Pregnancy especially folic acid deficiency (decreased platelets)
14. Drugs (anticoagulants, aspirin, NSAIDs, CAF, carbenicillin).
15. Systemic toxic agents-phosphorous, mercury.
16. Infectious diseases (Typhoid, rheumatic fever, whooping cough
17. Cardiovascular disorders (MS, CHD, CCF, COA)
18. Immunosuppression decompensated (HIV)
19. Allergic diseases
20. Malnutrition

Clinical presentation

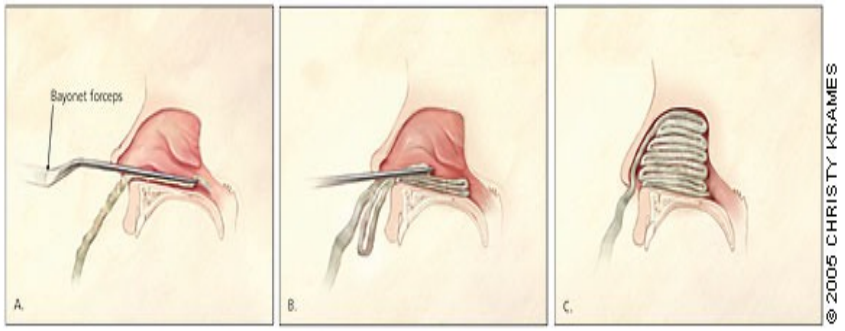
- Sudden onset
- Occasionally preceding headache
- May be unilateral,
- Smell of blood in the throat, trickling in the throat
- Swallow and vomit fresh blood
- Anxiety (increased PR, BP) increased bleeding
- Elderly decompensated very fast (hypovolaemic shock)

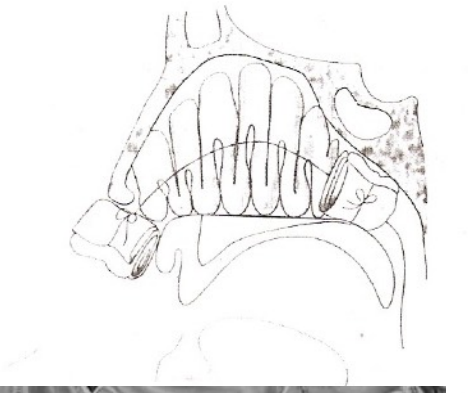
Management

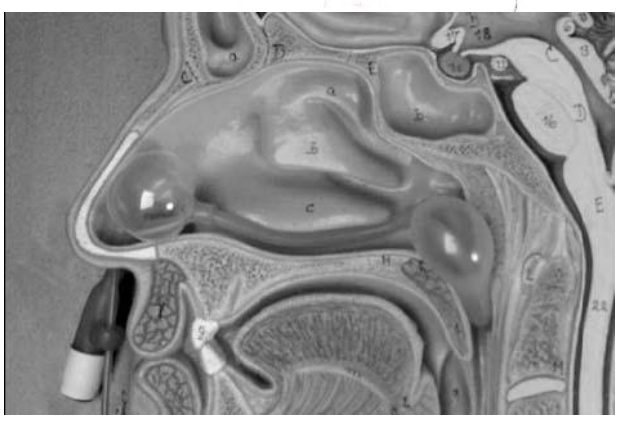
1. Medical history
2. Physical examination
3. Laboratory investigations
4. Radiological investigations
5. EUA or endoscopy

General management

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



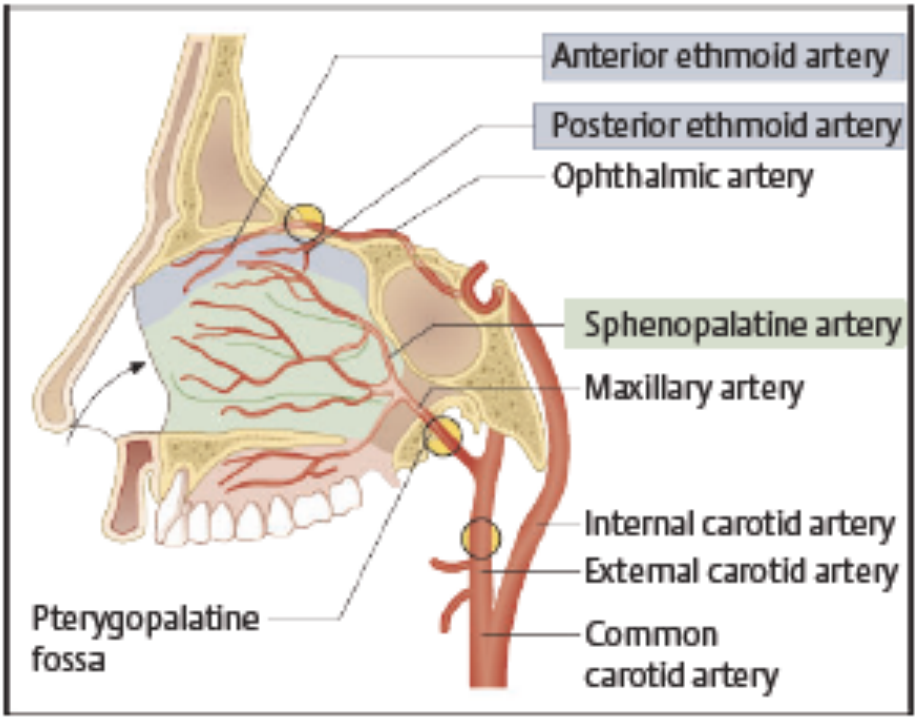




Assessment of blood loss

- Classify
  - Class I 10-15% of total blood volume (minimal blood loss <700 ml)
  - Class II 15-30%
  - Class III 30-40%
  - Class IV >40% (>2000 ml)
- Rules of fluid replacement:
  - Crystalloid fluid= 3:1
  - Colloids fluids= 1:1

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.

- The patient is evaluated in the seated position,
- Adequate light suction anaesthetic solution,
- Packing materials and cautery.
- Topical vasoconstrictor and anaesthetic agent. (oxymetazoline and xylocaine)
- Most bleeding sites are anterior and accessible to local treatment.
- Bleeding sites that are not visible on anterior rhinoscopy most likely from posterior (sphenopalatine artery)
- Trivial haemorrhage — first aid measures.
- Mild-moderate (patient may develop shock)
- Main aim is to stop haemorrhage.
- Firm pressure to the nostrils 5-10 mins seated upright, head facing downwards.
- Advice the patient to breath through the mouth.
- Arrange for good light, nasal cannula and suction machine.
- anaesthetic agent + vasoconstrictor in solution

Non-surgical management

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

- Posterior packing

Nose packing

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

Surgical management

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

TRACHEOSTOMY

The Upper Airway

- The nose plays a very important role in the upper airway. As air enters the nostrils large particles of dust and dirt are filtered. The mucous membranes of the nasopharynx further filter this air, warm or cool the inspired air, and humidify it.
- The column of inspired air travels through the oral pharynx to the laryngopharynx. Here it passes through the larynx where the vocal cords are located. The larynx is located at the top of the trachea. When a person breathes in, the vocal cords open, allowing air to pass freely into the trachea.

The Larynx

- The larynx is composed of nine cartilage structures
- Three large Single Cartilages
  - Epiglottis
  - Thyroid
  - Cricoid
- Three Paired Cartilage
  - Arytenoids
  - Cuneiforms
  - Corniculates
- The cricoid cartilage is the only circumferential cartilage of the trachea. This is an important landmark used during tracheostomy.

Trachea

- The trachea is a tubular structure. It extends from the larynx through the neck to the thorax (10 – 14 cm in length).
- The trachea terminates at the carina and bifurcates into the left and right main stem bronchi.
- Within the thorax the trachea lies in the mediastinum, its lower position is directly behind the heart and large vessels.

Indications for Tracheostomy

There are usually four main goals. The procedure may be done to achieve a combination of the following goals.

1. Patent airway
2. Protects lungs from obstruction or aspiration
3. Removal of secretions
4. Long term ventilatory support

- When compared to an endotracheal tube, tracheostomy tubes are considered more beneficial in that they:
  - Prevent further laryngeal injury from translaryngeal tube
  - Improve patient comfort
  - Decrease work of breathing
  - Provision of speech mechanism
  - Increase patient mobility

Procedure:

- An incision is usually made at the 2nd, 3rd or 4th tracheal cartilage.
- During insertion of the Tracheostomy tube, the obturator replaces the inner cannula. Its smooth surface protrudes from the outer cannula, minimising tracheal trauma. Once the tracheostomy tube is inserted, the obturator is removed and replaced with an inner cannula. Make sure the inner cannula is locked in place.
- The obturator should be placed in a plastic bag and kept at the bedside in case the tube needs to be reinserted immediately.
- Usually tracheostomy is performed as a surgical procedure. However, there is a procedure known as Percutaneous tracheostomy that is performed at the bedside. This procedure consists of passing a J-tipped guide wire and placing the tracheostomy tube.

Post-Tracheostomy Complications

There are two ways to categorise complications. Immediate surgical complications and late complications:

1. Haemorrhage

   Massive bleeding may occur a few days to several weeks post–op. Delayed haemorrhage may be due to erosion of the tracheal wall. The tip of the tube may then perforate a major blood vessel usually the innominate artery.

2. Wound Infections

   Infection at the tracheostomy site may be minor and respond to local treatment. Serious mediastinitis can result from an untreated tracheostomy wound infection.

3. Tracheitis

   A dry tracheitis will develop if humidification of the airway is inadequate. Secondary infections may require treatment with antibiotics.

4. Pneumonia

   Aseptic technique during surgery including suctioning can prevent pneumonia or lung abscess. Please use sterile suction catheter or closed suction system to avoid bacterial invasion of respiratory system.

5. Sub-glottic oedema

   Usually associated with infants and children when the incision is made via the 1st or 2nd tracheal cartilage. Oedema of the sub-glottic area may develop. The swelling of the mucosa will restrict the airway above the tube and can be difficult to decannulate the patient.

6. Tracheal stenosis

This is the narrowing of the trachea by stenotic grannula tissue (scar tissue). This narrowing can occur at the:

- Tracheal opening
- Cuff site
- Position of tube tip

7. Tracheoesophageal Fistula

   The posterior wall of the trachea erodes into the underlying oesophagus. This complication is rare but if it occurs it is potentially fatal.

- Monitoring cuff volumes and having a properly sized tracheal tube helps to decrease this incident.
- Tracheal stenosis at the cuff site can be avoided by using a tracheal tube with a low volume cuff and monitoring the cuff pressure.
- Erosion of the tip of the wall can be decreased by tube used (proper length, size and curvature).
- Many tubes now soften slightly with the patient's body temperature, which allows the tube to conform to the patient's trachea.
- Movable neck plates also help in the process for the tube to conform to the trachea

RHINOSINUSITIS

Introduction:

- Defined as inflammation of the nose and paranasal sinuses for 12 weeks or less with 2 or more of the following symptoms:
  - Blockage/congestion
  - Discharge (anterior or posterior nasal drip)
  - Facial pain or pressure
  - Reduced or loss of smell
- Increasing incidence and prevalence
- Affects 14% of general population
- 5-10% of viral URTI progress to bacterial rhino sinusitis
- Difficult to separate affections of nose and sinuses
- Mucus blanket of sinuses continuous with that of nasal cavities
- Viral URTIs involve both nose and sinuses but bacterial infections of sinuses do not involve the nose

Definition:

- Condition manifested by an inflammatory response involving mucous membrane of nasal cavities and paranasal sinuses and the fluid within these cavities and/or underlying bone

Embryology:

SINUS	Intrauterine Development	X-Ray	Adult	Adult	
Maxillary					
Ethmoid					
Frontal					
Sphenoid					

Anatomy of the Sinus

Osteomeatal complex

- The osteomeatal complex (or unit), sometimes less correctly spelled as osteomeatal complex, is a common channel that links the frontal sinus, anterior and middle ethmoid sinuses and the maxillary sinus to the middle meatus that allows air flow and mucociliary drainage.
- Gross anatomy
  - The osteomeatal complex is composed of five structures:
    1. maxillary ostium: drainage channel of the maxillary sinus

2. infundibulum: common channel that drains the ostia of the maxillary and ethmoid sinuses to the hiatus semilunaris
3. ethmoidal bulla: usually a single air cell that projects inferomedially over the hiatus semilunaris
4. uncinate process: hook-like process that arises from the posteromedial aspect of the nasolacrimal duct and forms the anterior boundary of the hiatus semilunaris
5. hiatus semilunaris: final drainage passage; a region between the ethmoid bulla superiorly and free-edge of the uncinate process

- Functional concept,
- not anatomic structure → No rigid boundaries

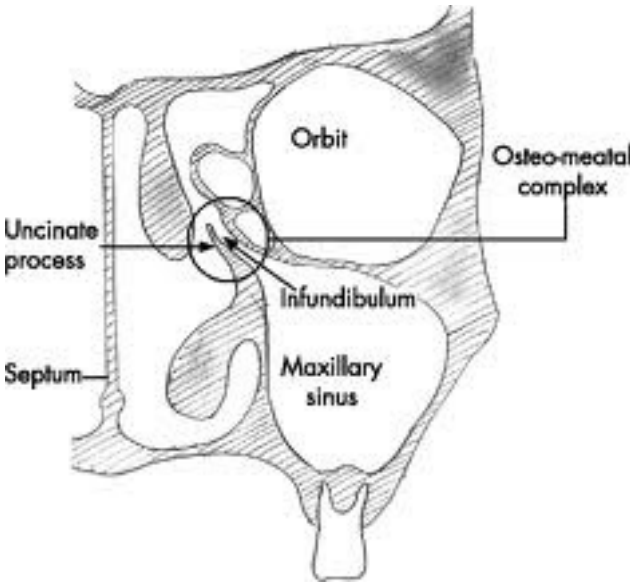
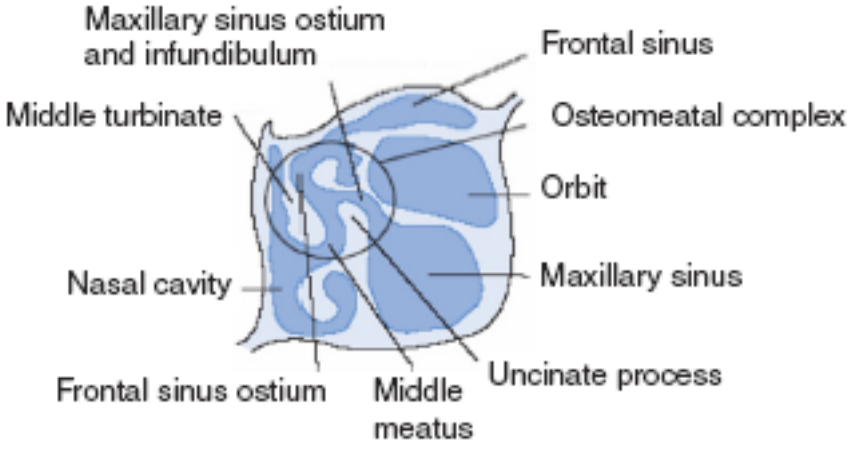


Figure 2. Coronal View Of The Osteomeatal Complex



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Pathophysiology

- Normal function:
  - Patent Ostia
  - Normal cilia
  - Normal mucous secretion
- Primary abnormality
  - Obstruction of osteomeatal complex by oedema or mechanical obstruction
- Inflammation is the primary mechanism responsible for cardinal symptoms of acute rhinosinusitis
- Underlying inflammation leads to increased vascular permeability → impaired mucociliary function → stasis

Associated risk factors:

1. Allergic rhinitis — in 60% of patients with CRS
2. GERD — extra-oesophageal manifestation of GERD
3. Defects in mucocillary clearance
4. Viral infections — repeated
5. Systemic diseases — Wegener's
6. Anatomic abnormalities eg. Deviated nasal septum, concha bullosa
7. Both Asthma and AERD — Aspirin Exacerbated Respiratory Disease are associate with CRS)
   - Samter's triad*
     - Aspirin hypersensitivity
     - Bronchial asthma
     - Nasal polyposis

Allergies in CRS

- Most common predisposing factors in adults
- In children, it is second to URTIs

Rhinosinusitis and nasal polyposis

- CRS without nasal polyposis >60%
- CRS with is 20-33%

Aetiology

- Viral
- Fungal → foreground glass thickening
  - Allergic fungal sinusitis
  - Invasive fungal sinusitis — immunocompromised invaded the CNS

- Bacterial
  - S. pneumoniae — 30%
  - H. influenza — 20%
  - S. pneumoniae — 5%
  - Anaerobic:
    - Peptostreptococcus — 56% etc
  - Acute RS and CRS have different bacteriology
    - Commonest organisms in acute RS
      - S. pneumoniae
      - H influenza
      - M. catarrhalis
      - S. aureus
      - Enterobacteria
    - CRS
      - Mixture of both aerobic and anaerobic (commonest: bordetella and peptostreptococcus)
      - Specimen 0s multi-bacterial
      - S. aureus
      - S. pyogenes

Classification

- Acute < 4 weeks
- Sub-acute 4 – 12 weeks
- Chronic > 12 weeks

Symptoms

1. Facial pain or pressure
   - Tightness in the face esp. when they bend
2. Reduction or loss of smell
3. Nasal congestion or blockage
4. Anterior discharge or postnasal drip — mucopurulent
5. Headaches
   - Frontal — ethmoid and frontal sinus involved — or occipital — sphenoid sinus

Diagnosis:

1. History
2. Clinical examination
   - Rhinoscopy
   - Transillumination not useful due to non-specificity
   - Sinonasal endoscopy

- Rigid or flexible endoscope to see mucopurulent material draining from the osteomeatal complex and nasal polyps

3. Imaging
   - Plain X-rays — water view
     - Mucous membrane thickening >5 mm
     - Complete opacification of 1 or more sinuses
     - *Fluid levels in the sinuses → infective process*
     - *Ground glass appearance*
   - CT scans:
     - Isolated or diffuse mucosal thickening
     - bone changes
     - Fluid levels
   - MRI — sensitive but lacks specificity; when soft tissue pathology is being suspected e.g. malignancy

Management:

- Medical management
  - Improve drainage and aeration
  - Improve mucociliary clearance
  - Treat bacterial infection if present
  - Treat underlying cause — allergy
- Surgical management:
  - Failed medical management
  - Correct structural abnormalities

Complications:

- Intracranial
  - Meningitis, subdural, epidural and cerebral abscesses
- Orbital
  - Pre and postseptal infections
  - Orbital cellulite
  - Orbital abscess
  - Cavernous sinus thrombosis
  - Sphenoidal-ocular syndrome
- Bone
  - Osteomyelitis through thrombophlebitis spread or direct extension
  - Pressure related bone erosion
  - Extensive mucocele
  - Secondary obstruction of the sinus ostium (esp. frontal sinus)
- Extensive mucocoele

ALLERGIC RHINITIS

There has been an increase in the prevalence of AR

Allergic rhinitis basics:

- Rhinitis refers to inflammation of the mucosa of nasal passages.
  - A condition that results from a type I hypersensitivity reaction towards airborne allergens.
- Brief episodes of rhinitis are usually causes by viruses such as the common cold.

Table – Classification of rhinitis[1,10,11]

Type	Subtype	Characteristics
Allergic rhinitis	Seasonal, perennial, or episodic	IgE to specific allergens produced; when allergen detected by mast cells, cytokine mediators of allergic symptoms released
Nonallergic rhinitis	Vasomotor	Triggered by irritants (perfumes, chlorine, cold air, exercise)
	Infectious	Consider chronic sinusitis as the cause or as a complicating factor in refractory rhinitis (obtain sinus CT scan)
	Hormonally induced	Nonallergic rhinitis in association with pregnancy or the menstrual cycle
	Gustatory	Profuse rhinorrhea associated with eating
	Granulomatous	Seen in granulomatous disorders such as Wegener granulomatosis, sarcoidosis, midline granuloma, and granulomatous infections
	Drug-induced	Causative agents include phosphodiesterase inhibitors, oral contraceptives, antihypertensives, aspirin, NSAIDs, and intranasal decongestants (rhinitis medicamentosa)
Occupational rhinitis		Can be allergic (exposure to a protein causing allergy in the workplace) or nonallergic (eg, irritant)
Mixed rhinitis		Has both IgE-mediated and nonallergic triggers

- Allergic rhinitis is caused by a nasal reaction to small airborne particles called allergens.
  - Allergic rhinitis can be seasonal (occurring during a specific season) or perennial (occurring year round).
  - The allergens that most commonly cause seasonal allergic rhinitis include pollens from trees, grasses, and weeds, as well as spores from fungi and moulds.
  - The allergens that most commonly cause perennial allergic rhinitis are dust mites, cockroaches, animal dander, and fungi or moulds.
  - Perennial allergic rhinitis tends to be more difficult to treat.
- Acquired potential for developing adverse reactions
  - Immunologically mediated to substances harmless to most of us
- A rapidly developing immunologic reaction occurring within minutes after combination of an antigen with an antibody bound to mast cells in an individual previously sensitised to that antigen.

Epidemiology

- Rhinitis is a common disorder that affects up to **40% of the population**.
- Allergic rhinitis is the most common type of chronic rhinitis, affecting 10% to 20% of the population, and evidence suggests that the prevalence of the disorder is increasing.

Predisposing factor:

1. Genetics —
   - 60% IF BOTH PARENTS
   - 40% IF ONE PARENT
   - 20% NONE
2. Environmental — Hygiene theory
   - The hygiene hypothesis is a hypothesis that states a lack of early childhood exposure to infectious agents, symbiotic microorganisms (such as the gut flora or probiotics), and parasites increases susceptibility to allergic diseases by suppressing the natural development of the immune system. In particular, the lack of exposure is thought to lead to defects in the establishment of immune tolerance.
   - The hygiene hypothesis has also been called the "biome depletion theory" and the "lost friends theory".

Pathogenesis:

- Initial sensitisation
- Re-exposure to very small amounts
  - Allergic reaction is developed since mediators are preformed

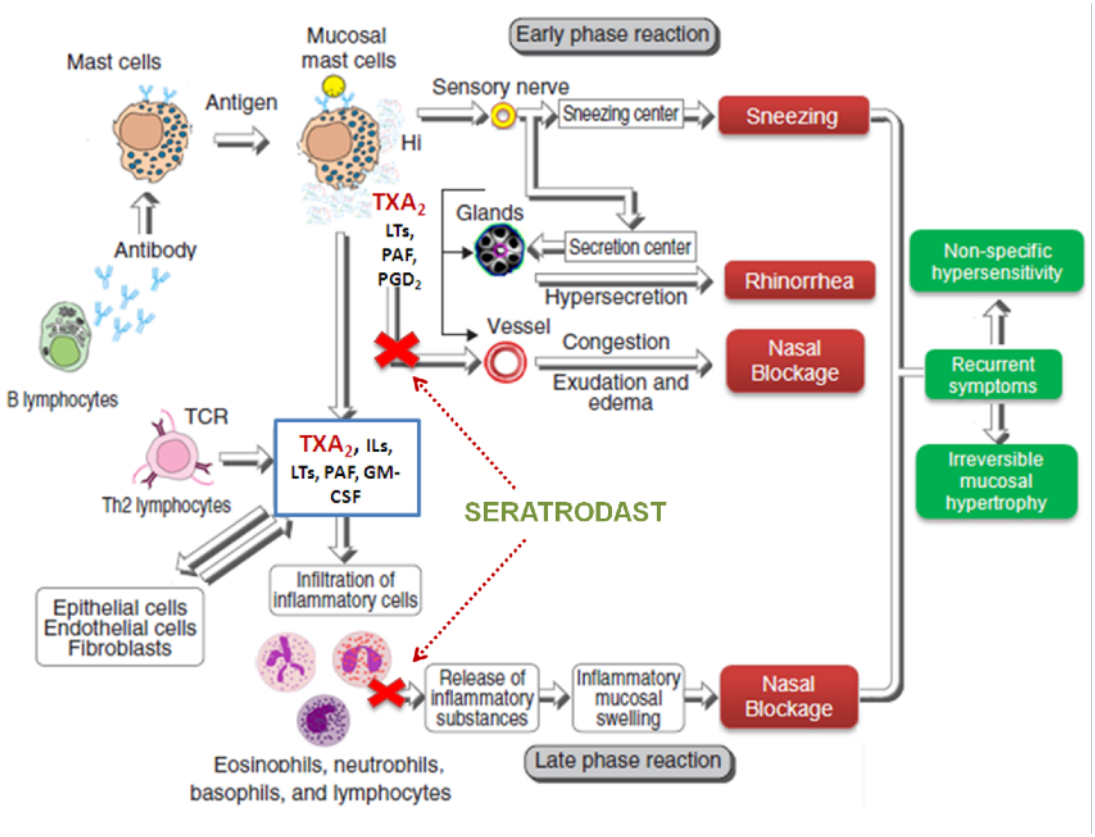
Starts with the sensitisation phase that does not have clinical signs and symptoms. An Antigen Presenting Cell presents an antigen together with MHC-II to a naïve CD4+ cell which matures into a Th2 cell. The Th2 cell elaborates cytokines (IL-2,-4,-5-6,-13) which promote recruitment, differentiation and proliferation of eosinophils and B cells.

Th2 cells interact with B cells so that they become plasma cells which produce antibodies (IgE) that specifically bind with the antigen. These antibodies then bind to mast cells and basophils by their Fc receptors.

On re-exposure to the same antigen, the IgE antibodies on mast cells and basophils undergo cross linkage by the antigen resulting in transduction of a signal into the cytosol of those cells which leads to:

(a) An immediate response which is degranulation of the cytosol to release potent biogenic substances (e.g. histamine, proteolytic enzymes) which contribute to the inflammatory reaction.(within minutes to hours)

(b) The delayed phase which involves production of prostaglandins and leukotrienes which cause increased vascular permeability and contraction of smooth muscles of bronchial and nasal glands. (within hours to days)



Classification — ARIA:

- Based on duration and is subdivided into intermittent or persistent
- Is based on severity and is subdivided into mild or moderate to severe
- Treatment is based on this classification

ARIA

- Intermittent
  - < 4 days per week or > 4 weeks in a year
- Moderate
  - > 4 days in a week an> 4 week in a year
- Severe
- Mild

Symptoms:

- **Nose:** watery nasal discharge, congestion, sneezing, itching, post-nasal drip, loss of taste, facial pressure or pain
- **Eyes:** itchy red eyes, feeling of grittiness in the eyes, swelling and blueness of the skin below the eyes (allergic shiners)
- **Throat and ears:** sore throat, hoarse voice, congestion or popping of the ears, itching of the throat or ears
- **Sleep:** mouth breathing, frequent awakening, daytime fatigue

Signs:

- Allergic shines
- Allergic salute
- Supra-tip crease line
- Nasal discharge
  - Pale nasal mucosa
  - Hypertrophied turbinates

DDx of non-allergic & allergic rhinitis		
Non-allergic	**Allergic**	
Temporal pattern of symptoms	perennial	seasonal or perennial with seasonal exacerbations
Type of symptoms	congestion, rhinorrhea, posterior drainage, sinus pressure	sneezing, pruritus, congestion, rhinorrhea, posterior drainage, sinus pressure
Age of onset	70% are older than 20	70% are younger than 20
Precipitating factors	nonspecific irritants	specific antigens± nonspecific irritants
Other atopic disease	not present	frequently present
Family history of rhinitis	not frequent	frequent

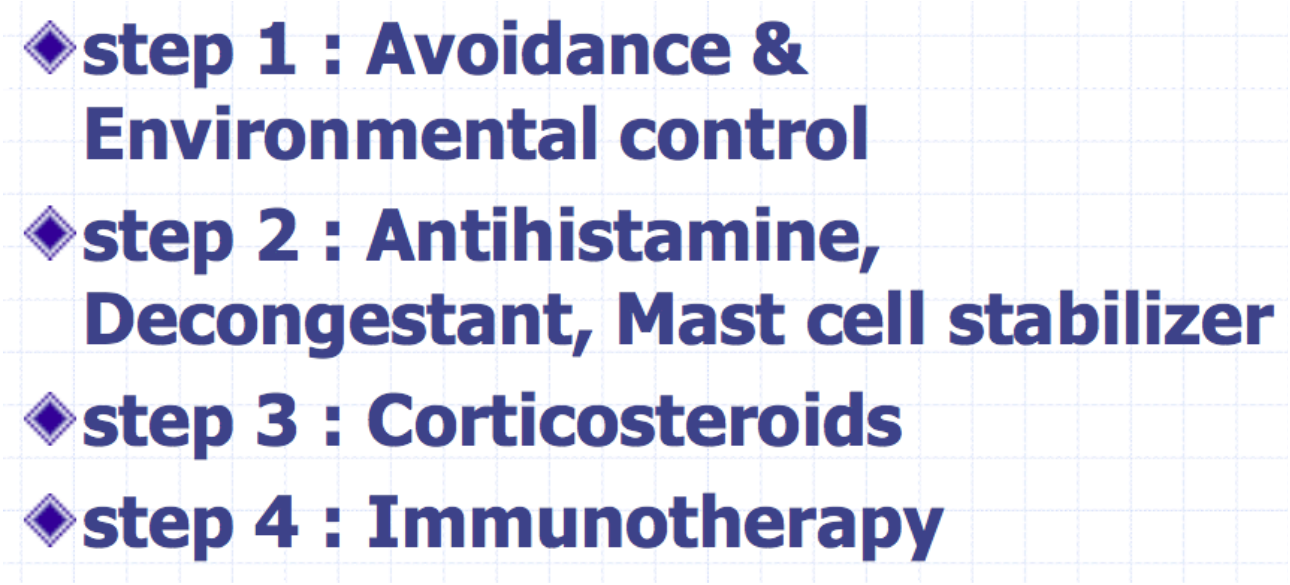
Diagnosis:

- Hx and PE
- In vitro tests
- In vivo tests
  - Sub-cut injection of allergen wheal
- ELISA test
- RAS test
- Nasal provocation test

Complications

- Recurrent sinusitis
- Formation of nasal polyps
- Otitis media
- Orthodontic problems
- Increased risk of developing asthma and eczema.

Treatment:



- Environmental — also preventive
- Immunology
- Pharmacotherapy
  - **Nasal irrigation:** Rinsing the nose with salt-water (saline) solution, such as saline nasal spray or irrigation kits like the Neti pot, to remove allergens and irritants
  - **Nasal Glucocorticoids**: Steroids delivered by a nasal spray
    - Nasal glucocorticoids available by prescription: Fluticasone
    - Maximal effectiveness may not be noticeable for days to weeks; therefore these medications are most successful when used daily
  - **Antihistamines:** Antihistamines do not relieve nasal congestion and are often combined with nasal steroids or decongestants to provide greater symptom relief
    - Antihistamines: Diphenhydramine, Chlorpheniramine, Loratadine, Desloratadine, Cetirizine, Levocetrizine, Fexofenadine. Diphenhydramine & Chlorpheniramine may causes sedation/drowsiness.
    - Nasal Antihistamines: Azelastine and Olpatadine
  - **Decongestants:** Decongestants are used to relieve nasal swelling. Oral decongestants elevate blood pressure and are not appropriate for people with high blood pressure or certain heart conditions.
    - Decongestants available OTC: Pseudoephedrine.

- Nasal Decongestants: Oxymetazoline) and Phenylephrine. *Nasal decongestant sprays should not be used for more than 2-3 days at a time.*

Special groups

- Children
- Allergic rhinitis of pregnancy

Prevention:

- Avoidance of allergens — *Reduce exposure to triggers when possible*

NASAL POLYPOSIS

Definition

- Polyp is derived from Greek, meaning many footed (*poly, many; pous, footed*) but a polyp has only one foot (stalk). Finally , a disease characterised by the occurrence of multiple polyps is most correctly named nasal polyposis & strictly speaking, it is not a nasal but a **sinonasal disease**.
- A polyp presents in the nasal cavity with a grape-like appearance, having a body& a stalk. The surface is smooth& the colour is more yellow than the pink mucous membrane.
- Nasal polyp originate around the openings of the sinuses.
  - The polyps protrude into nasal cavity from the middle& superior meatus, resulting in nasal blockage& abolishing airflow to the olfactory region.

Epidemiology

- Initial trigger unknown
- Associated with
  - Asthma
  - Aspirin sensitivity
- The prevalence rate of nasal polyposis is about 2%. It increases with age, reaching a peak in those aged 50years & older. The male: female ration is about 2:1. The disease is more frequent in nonallergic than in allergic patient with rhinitis& asthma.
- Nasal polyposis occurs with a high frequency in groups of patients having specific airway disease. (nonallergic rhinitis, nonallergic asthma, allergic fungal rhinitis, cystic fibrosis, primary ciliary dyskinesia, Churg-Strauss syndrome)

Aetiology

- **Aspirin triad:** A triad of nasal polyposis asthma , asthma& aspirin intolerance represent the most aggressive form of the disease.
- **Allergic fungal sinusitis**: Nasal polyp occur in almost all patients with allergic fungal rhinitis. In this disease , inflammation is typically eosinophil-dominated as in aspirin triad.
- **Allergy:** As most polyp are characterised by tissue eosinophilia, it has been the belief for decades that allergy is a significant cause of nasal polyposis. However this view has been challenged because most studies have failed to show higher occurrence of positive skin tests to inhaled allergens in patients with polyps than in the general population.
- **Keith** were unable to show any deterioration of nasal symptom or eosinophilia during the pollen season in patient having a positive skin test to pollen.Thus , it appears that allergy is not a well-documented cause or aggravating factor in nasal polyposis.
- **Cystic fibrosis:** Nasal endoscopic demonstrated polyps in 45% of adult with cystic fibrosis.
- **Primary ciliary dysfunction(Kartagener's syndrome):** Absent mucociliary clearance & recurrent bacterial infections result in nasal polyposis in about 40% of the patient.
- **Young's syndrome :**It consists of chronic rhinosinusitis, nasal polyps, bronchiectasis& azoospermia.

(It is more correct to say allergic-like inflammation but cause is unknown.)

Pathology & pathogenesis

1. **Site of polyp formation:**Nasal polyps are mainly situated in the middle meatus & that polyps originate from the mucous membrane of the outlets (ostia, clefts recesses) from the paranasal sinuses.This area is so critical for sinus pathology is also referred to as the osteomeatal complex.
2. **Localisation factor for polyps:**
   - **Touching mucous membrane** in the narrow osteomeatal complex results in the release of pro-inflammatory cytokines from epithelial cells.
   - Special airflow, air current & pressure in the upper part of the nose.
   - **Nerve endings** near the border between the nose & paranasal sinuses are thin & may easily damaged by cytotoxic proteins, release by eosinophils.
3. **Surface epithelium:** The major part of surface epithelium is covered by ciliated pseudo-stratified epithelium, but in addition transitional & squamous epithelium are found.
   - Cytokines from eosinophils can damage the respiratory epithelium & induce hypersensitivity in nasal mucosa.
   - Disturbance of the epithelium may occur on exposure to chemical, physical & immunological stimuli can lead to the release of pro-inflammatory cytokines.
4. **Innervations:** Nasal polyps develop in the area where the lining of the nasal mucosa join that of the sinus & **these marginal zone** contain thin nerve fascicles which may have increase sensitivity to cytokines (loss of innervations is an important factor in the formation of polyps).
5. **Goblet cells:** Average density of goblets cells is much lower than in the nasal mucosa.
6. **Submucosal glands:** The density of mucosal gland is more than 10 times less than in the nasal mucosa. All glands in the polyps are abnormal & cystic degeneration with stagnation of mucus.
7. **Blood vessels:** The vascularity of polyps is minimal as compared to the normal nasal mucosa.
8. **Inflammation:** Nasal polyposis is the ultimate form of inflammation of nasal airway, preferentially develops in subtypes of inflammatory diseases. Although , IgE-mediated allergy is not an important etiological factors but recent data indicate that both mast cells& histamine are involved in the inflammation & pathogenesis of polyps.

Clinical presentation

- Patients as a rule have suffered from perennial nonallergic rhinitis(idiopathic rhinitis) with profuse watery rhinorrhoea for some years, then nasal blockage gradually develops& becomes persistent. Simultaneously, secretions become more viscous & are removed by noisy sniffing as postnasal drip.
- Impaired airflow in the upper part of the nose reduces or abolishes sense of smell & with that taste. It mars the pleasure of eating & drinking.
- When polyps develop in children, it can cause widening of the ethmoidal cells,& flattening & broadening of the nasal bridge (frog nose).

Diagnosis & staging

Rhinoscopy, endoscopy:

- Endoscopy with a rigid scope is the preferred examination as it can diagnosis a small polyp in the middle meatus . The examination is performed after simple spray of the nose with a local anaesthesia & a vasoconstrictor.

Endoscopic staging of nasal polyposis

ENDOSCOPIC APPEARANCE	SCORE
No polyps	0
Restricted to middle meatus	1
Below middle turbinate	2
Massive polyposis	3

Imaging

- Plain x-ray is not helpful.
- A CT scan of the nose & paranasal sinus gives an excellent demonstration of the anatomy& pathology.

Treatment

- Medical treatment consists of intranasal & systemic steroids.
- Possibly leukotrienes antagonists may have an additional effect in selected patients.

Intranasal steroids

Intranasal steroids are the by far the best documented type of treatment for nasal polyposis.

1. Responsiveness to nasal corticosteroids: Some patients do not response to topical steroids. This may be due to inadequate intranasal distribution of the spray in a very blocked nose. Responsiveness can be achieved after a short course of steroids.
2. Polyp size: Intranasal steroids will not eliminate polyps but the treatment clearly reduces their size.
3. Nasal airway patency: A significant effect on nasal blockage.A patent airway is not necessarily a normal airway.Pressure from polyps may have changed the normal slit-like airway to a wide tube in the lower part of the nasal cavity. At the same time there may have considerable pathology & blockage in the upper part of the nasal cavity.
4. Rhinitis symptoms: like running, sneezing have been varied effects. Overall symptoms reduction 50%.(blockage is not rhinitis symptoms).
5. Sense of smell: The effect of topical steroids in contrast to systemic administration is poor. This probably due to spray do not reach the olfactory mucosa.
6. Recurrence of polyps: Topical steroids can delay the recurrence of polyps after surgery.

7. Sinus pathology: No effect on sinus pathology.
8. Safety: Extensively used for 30 years without any report of serious adverse events.

Systemic steroids

- Systemic steroid can be given as orally or as a depot-injection.
- Total dose of glucocorticoid in a depot-injection corresponds to about 100 mg prednisolone. A depot-injection releases glucocorticoid for 2 to 3 weeks & orally given similar period of time.
- When given in orally, a higher total dose is probably necessary e.g. 25 mg prednisolone daily for 10-14days.
- A short course of systemic steroid is usually equally effective as simple polypectomy with a snare& it may serve as a **medical polypectomy.** In severe cases, requiring endoscopic surgery, preoperative use of a systemic steroid will considerably facilitate surgery.

Surgical treatment

- As an inflammatory disease of the mucous membrane, surgery cannot be expected to cure the disease.
- A mild case presenting for first time with large polyps, polypectomy can have a long-lasting effect.
- In more severe cases with persistent symptoms ,surgery is added to medical treatment in order to reduce the amount of inflammatory disease, open up the nasal airway& improve the ventilation of the paranasal sinuses.
- The authors concluded that medical treatment is sufficient to treat most of the cases of nasal polyposis. If nasal obstruction remains the main problem after medical treatment then surgery is indicated.

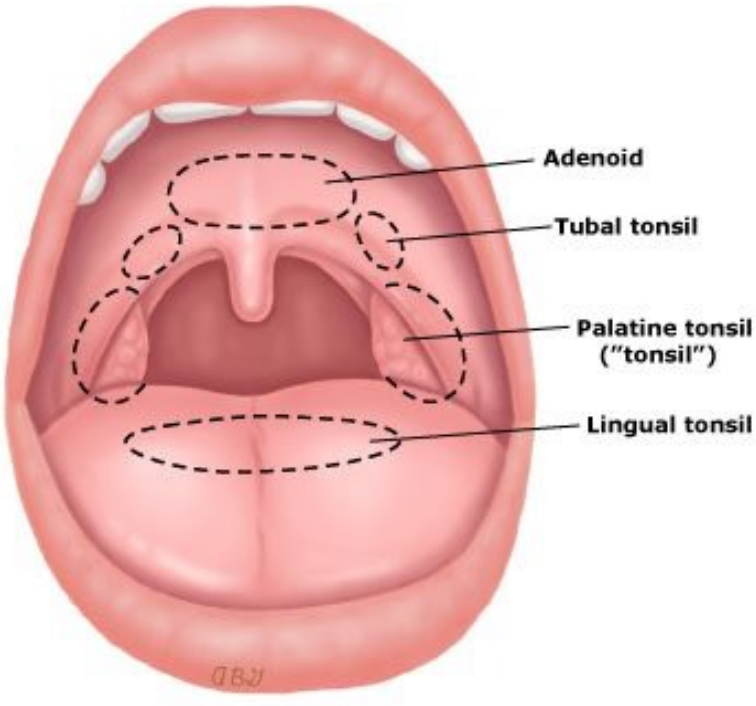
TONSILS AND ADENOIDS

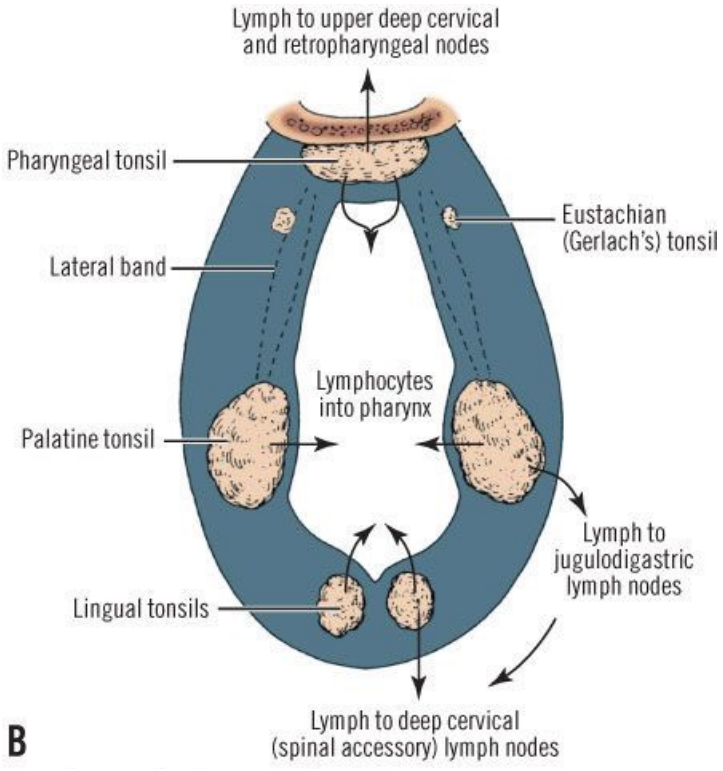
Physiology

- Tonsil and adenoid infection are very common in **childhood.**
- **Adenoid - Tonsil Surgery (ATS)** is the most commonly performed procedure in the history of surgery.
- For airborne antigens, the adenoids and tonsils are the 1st site of immunological contact.
- The tonsils are most immunologically active at **4 – 10 years** whereas, the adenoids are at **2 – 5 years.**

Anatomy

- A tonsil is any collection of lymphoid tissue with **no afferent vessels** but has efferent vessels, unlike a lymph node.
- There are 3 main groups of lymphatic tissue in the head & neck:
  1. **WALDEYER'S RING**
  2. **TRANSITIONAL LYMPHATICS**
  3. **CERVICAL LYMPH NODES**





Waldeyer's ring:

- This is a **ring of lymphoid tissue** that is integral to immunoglobulin immunity.
- Composed of:

- **Adenoids/Pharyngeal tonsils**
  - Are found on the posterior wall & roof of the nasopharynx.
  - These **CANNOT** be seen from the oral cavity since the palate covers them. They can only be seen through the nasal cavity using endoscopy.
- **Tubal/ Eustachian tonsils**
  - Are found at the pharyngeal opening of the ET.
- **Palatine tonsils**
  - Embedded in the lateral wall of the oral pharynx on either side between the pillars of the fauces.
- **Lingual tonsils**
  - Lie at the base of the tongue just anterior to the epiglottis

Bacteria and Viruses commonly cultured from Tonsils and Adenoids

- **BACTERIA:**
  - Aerobic
    - Group A β haemolytic *Streptococci*
    - Groups B, C & G (especially in the first year of life)
    - H. influenza, S. pneumoniae, M. catarrhalis, Staphylococci, Neisseria, Mycobacteria.
  - Aerobic
    - Peptostreptococci & Actinomyces

- NOTE: THE RATIO OF ANAEROBIC TO AEROBIC MICRO-ORGANISMS IS 10: 1. THE LATTER ARE HOWEVER NOT AS INFECTIOUS AS THE FORMER.

- **VIRUSES:**
  - EBV
  - Adenovirus
  - Influenza A & B
  - Herpes simplex virus

PHARYNGITIS

- Inflammation of the oropharyngeal mucosa which is composed of:
- Soft palate
- Palatine tonsils
- Base of tongue
- Posterior pharyngeal wall
- 70% of infections are **viral** and 30% are bacterial.

CLINICAL PRESENTATION:

- Odynophagia
- Dysphagia
- Tonsillar enlargement
- Exudates & petechiae
- Fever
- Lymphadenopathy

ADENOTONSILLAR DISEASE INCLUDES:

1. *Adenoid infections*
2. *Adenoid hypertrophy*
3. *Tonsillar infections*
4. *Tonsillar hypertrophy*
5. *Neoplasia*

ADENOID INFECTIONS & HYPERTROPHY

A. Adenoiditis

(I) Acute adenoiditis

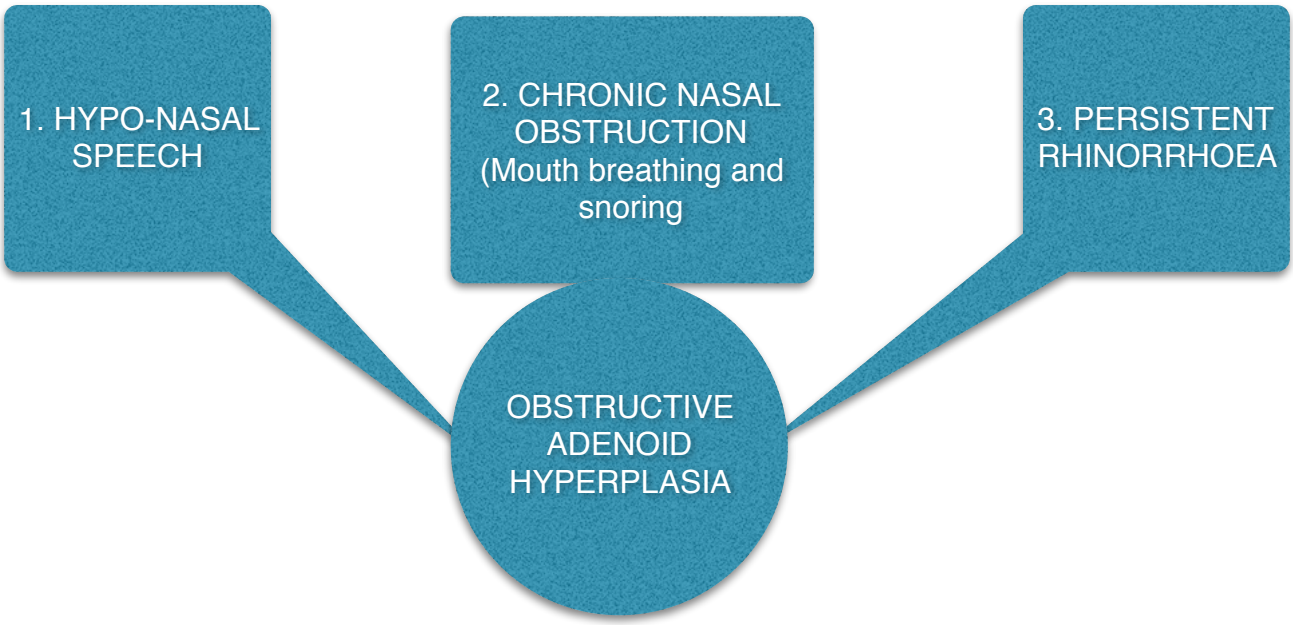
- Characterised by
- Purulent rhinorrhoea
- Nasal obstruction & mouth breathing
- Snoring
- Fever
- Recurrent otitis media (ROM)
- Rhinosinusitis

- Consider GERD in a child < 2 years with recurrent infection

(II) Chronic adenoiditis

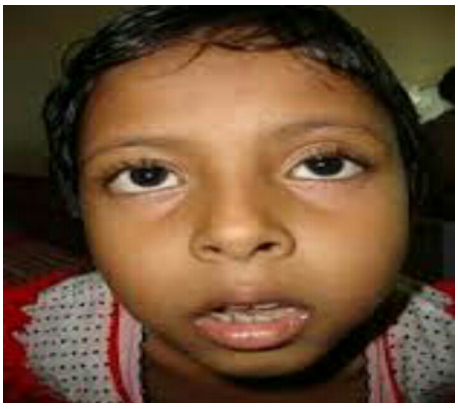
- This occurs if symptoms persist for **> 14 days.**
- It is characterised by
  - Persistent rhinorrhoea
  - Post-nasal drip
  - Malodorous breath/halitosis
  - Persistent or recurrent otitis media (OM)

B. OBSTRUCTIVE ADENOID HYPERPLASIA (TRIAD)



ADENOID FACIES

- Long, open — mouthed dull face
  - Hyper-somnolence
  - Mouth breathing
- Under - developed nostrils
  - Since the child becomes an obligate mouth breather
- Hypo - gnathic maxilla
  - High arched palate
  - Short upper lip
  - Prominent crowded upper teeth



PATHOLOGICAL EFFECTS OF ADENOID HYPERTROPHY

- Snoring
- Craniofacial malformations
- Rhinorrhoea (sinusitis)
- Recurrent otitis media
- Otitis media with effusion (OME)
  - Affects hearing
- Recurrent URTIs
  - Change in child's play habit

UPPER AIRWAY OBSTRUCTION (UAO) MANIFESTATION

- Snoring and mouth breathing
- Sleep apnea
- Daytime hyper — somnolence
- Behavioural disturbances
- Pulmonary hypertension and Cor Pulmonale
- Failure to thrive
  - Due to poor appetite and resulting delayed milestones
- Enuresis
  - Sleep too deeply when they get to sleep
- Hyper-nasal/hypo-nasal speech

TONSILLAR INFECTIONS & HYPERTROPHY

Acute Tonsillitis

- This acute inflammation of the **palatine tonsils** usually due to ***Streptococcal*** or less commonly, to **viral infection**

- CLINICAL PRESENTATION
  - **Sore throat** & **pain** most marked when swallowing (dysphagia) & referred to the ears (tympanic branch of the glossopharyngeal nerve)
  - High fever, malaise & headache
- O/E
  - Tender lymphadenopathy
  - Erythema with a purulent exudate
  - White membrane thin, non — confluent & confined to the tonsil (peels away **without bleeding**)
  - Compare with diphtheria*

Peri-tonsillar abscess

- Swelling superior to the tonsil accompanied by tender lymphadenopathy
- The uvula is displaced to opposite side

- **MANAGEMENT:**
  - Needle aspiration 90%
    - If it is a frank abscess
  - Incision & drainage
    - If recurrence occurs after needle aspiration
- Hydration
- Antibiotics
- Steroids

- **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.

UNILATERAL TONSILLAR ENLARGEMENT

- It is often due to asymmetric anatomic position of tonsils.
- Atypical infections involved:
  - Mycobacteria
  - Fungi
  - Chronic actinomycosis
- R/O neoplastic disease in the following cases:
  - Change in voice (hot potato voice)
  - New – onset snoring
  - Dysphagia
  - Neck – lymphadenopathy
- Excision biopsy (tonsillectomy)
  - Histology
  - Cultures for aerobic, anaerobic and fungal organisms
- Imaging (before surgery)
  - Extension beyond tonsillar capsule
- If neoplastic disease, it may probably be: **Squamous Cell Carcinoma** or **Lymphoma**.

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 cooperation
- Tonsillectomy indicated if recurrent, anaemic or not responsive to local control
- Rule out bleeding diathesis.

Chronic Tonsillitis

- Chronic sore throat
- Malodorous breath
- Excessive tonsillar debris (tonsilloliths)
- Peri – tonsillar erythema
- Persistent tender cervical lymphadenopathy
- Common in: GERD, allergic pharyngitis

OBSTRUCTIVE TONSILLAR DISEASE

- **CLINICAL FEATURES**
  - Muffled voice
  - Hyper - nasality
  - Dysphagia
  - Snoring / sleep disturbance
  - Changes in craniofacial skeleton
-
- **GRADING OF THE SIZE OF TONSILS**
  - Grade A → Tonsils in fossa (0)
  - Grade B → Tonsils less than 25% (+1)
  - Grade C → Tonsils less than 50% (+2)
  - Grade D → Tonsils less than 75% (+3)
  - Grade E → Tonsils greater than 75% (+4)

NEOPLASMS OR MASSES

- CONGENITAL
  - Teratoma
  - Haemangioma
  - Lymphangioma
  - Cystic hygroma

- BENIGN
  - Lipoma
  - Fibroma
- MALIGNANT
  - Unilateral, fast enlargement, cervical LN+
  - Most common**: lymphoma**
  - Metastases from other primaries e.g. melanoma, lung.

COMPLICATIONS OF TONSILLITIS

LOCAL

- Laryngeal oedema UAO
- Abscesses
  - Peri-tonsillar abscess
  - Para-pharyngeal abscess
  - Retro-pharyngeal abscess
- Suppurative adenitis
- Acute Otitis Media (AOM)
- Chronic tonsillitis

GENERAL
- Septicaemia (the tonsils are a very vascular area)
- Febrile convulsions (absolute indication for surgery)
- Meningitis
- AGN
- ARF
- Guttate psoriasis

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
    - *Oral cavity and oropharynx* → tonsillitis, PTA
    - *Nose* → rhinorrhoea, 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 and 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 routinely
- Serologic tests
  - If an immunodeficient syndrome is suspected e.g. Hypogammaglobulinaemia.

NOTE: In SCD, adenotonsillar disease should be treated as an emergency. This is because, infection can predispose the patient to dehydration, acidosis and hypoxia, all of which can precipitate a sickling crisis.

Differential diagnosis:

- Scarlet fever
- Diphtheria
  - The membrane is dirty grey, thick & tough; it **bleeds if peeled away**.
  - Diphtheria is uncommon due to immunisation
- Infectious mononucleosis
- This is associated with micropetechiae of the soft palate; atypical lymphocytes on smear and a **positive mono-spot test**.
- Leukaemia
- Agranulocytosis
- Atypical mycobacteria, fungi, actinomycosis
- Vincent's angina/ Trench mouth
  - Characterises by superficial, painful ulcers with erythematous borders
- Malignancies
- Fungi, syphilitic gumma, TB, leprosy, leishmaniasis
- Crohn's disease

SEQUELAE

1. Upper airway obstruction (UAO)
2. Para - nasal sinus disease
3. Craniofacial malformations
4. Otologic disease (Otitis)
5. Cardiac disease

A. UAO

- Increased PCO2 → Decreased PO2 → Reflex VC in pulmonary circulation in response to relative hypoxia → Pulmonary HTN → **Cor pulmonale.**
- Adenotonsillar disease is common in children after otitis media
- In children presenting for adenotonsillar surgery, Pulmonary HTN is most common.

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 adenotonsillar disease)
  - Resistant cases → consider clindamycin, ciprofloxacin +/-metronidazole (anaerobic)
- **Surgical (recurrent disease)**
  - Tonsillectomy & Adenoidectomy (T & A)
  - High risk groups after surgery — admit and monitor for 24 hours post-operation
    - < 3 years old
    - Severe Obstructive Sleep Apnea (OSA)
    - Bleeding disorders e.g. haemophilia, 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

B. 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

C. 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

D. 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 or more months in 6 months or for 4 or more months in 1 year
  - Otitis Media with Effusion (OME)
    - Painless hearing loss and intermittent purulent ear drainage that follows AOM or arises without prior to AOM
  - Chronic OME
    - Persistent otorrhoea present for > 6 weeks

OTITIS MEDIA WITH EFFUSION

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

E. CARDIAC DISEASE ECHOCARDIOGRAM OF OSA

- Hypertrophy of right side of heart
- Engorged vasculature of pulmonary
- Pulmonary HTN

TONSILLECTOMY & ADENOIDECTOMY

RECURRENT ACUTE TONSILLITIS: PARADISE CRITERIA FOR TONSILLECTOMY

1. MINIMUM FREQUENCY OF SORE THROAT EPISODES:
   - At least 7 separate episodes in 1 year
   - At least 5 separate episodes/yr in 2 years
   - At least 3 separate episodes/yr in 3 years
2. CLINICAL FEATURES
   - Sore throat plus 1 of the following:
     - Fever > 38.4°C
     - Cervical adenopathy (tender LN)
     - Tonsillar/pharyngeal exudate
     - Culture positive for GAS
3. TREATMENT
   - Antibiotics administered in the conventional dosage for proved or suspected *streptococcal* episodes.

- NOTE: REDNESS IN THE PHARYNX MEANS NOTHING IN A CHILD IN THE ABSENCE OF AN EXUDATE! DO NOT PRESCRIBE AN ANTIBIOTIC.

AMERICAN ACADEMY OF OTOLARYNGOLOGY GUIDELINE — HNS GUIDELINES

- Absolute indications for surgery:
  - Enlarged tonsils
  - Repeat peritonsillar abscess
  - Tonsillitis with febrile convulsions
  - Tonsils requiring abscess
- Relative indications
  - > 3 infections in a year
  - Persistent halitosis
  - Chronic or recurrent tonsillitis in a streptococcal carrier not responding to β lactamase resistant antibiotic
  - Unilateral tonsillar hypertrophy

Methods used in T & A

- MOST COMMON
  - Cold knife
  - Electro – cautery

- OTHERS
  - Coblation
  - Harmonic scalpel
  - Laser

COLD KNIFE:

- GOLD STANDARD
  - Less post-operative pain
  - Post-tonsillectomy haemorrhage less common than electro-cautery
  - Least expensive
  - More intra-operative blood loss
  - Cause bacteraemia
  - Takes longer

Electrocautery

- Types:
  - Mono - polar cautery
  - Bipolar cautery

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

Contraindications:

- Acute tonsillitis
  - If elective, postpone until acute tonsillitis resolves.
  - This prevents super-infection of the surgical wound
- Short palate
  - 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**
- Bleeding disorder

Complications of adenoidectomy

- Fever
- Vomiting
- Dehydration
- Airway obstruction
- Pulmonary oedema
- Haemorrhage
- Hyper - nasal speech
- Velo-pharyngeal incompetence
- Dental injury
- Burns
- Nasopharyngeal stenosis
- Atlanto-axial subluxations (Down's syndrome)

Complications of tonsillectomy

- Haemorrhage
- Hyper–nasal speech
- Dehydration
- Laryngeal oedema

OTITIS MEDIA

- **Definition:** Presence of a middle ear infection:
  - Inflammation of the tympanic cavity, mastoid, petrous apex and peri-labyrinthine air cells
- **Acute Otitis Media:** occurrence of bacterial infection within the middle ear cavity
- **Otitis Media with Effusion:** presence of non-purulent fluid within the middle ear cavity
- OM is the second most common clinical problem in childhood after upper respiratory infection.
- Bacteria and viruses enter the middle ear through the eustachian tube.
- The resulting infection causes the middle ear to fill with pus and other fluids.
- Pressure from this buildup pushes on the eardrum, causing pain.
- Because the eardrum cannot vibrate, there may be temporary hearing loss.
- Otitis may lead to Mastoiditis.

Epidemiology

- Peak incidence in the first two years of life (esp. 6-12 months)
- Predominantly childhood infection (<5 years) because the child's eustachian tube is more horizontal (15 degrees) hence is not drain as much as the adults (45 degrees)
- Boys more affected girls
- 1st peak → < 2 years
- 2nd peak 5th decade
- By 3 years → 75% of children develop AOM
- Prevalence of >4% CSOM → major disease
  - Highest prevalence in Inuits, Aborigines, Apache, Navajo
  - High prevalence (>4%) Sierra Leone, Gambia, Kenya, Tanzania
- 50% of children 1 yr of age will have at least 1 episode.
- 1/3 of children will have 3 or more infections by age 3
- 90% of children will have at least one infection by age 6.
- Occurs more frequently in the winter months

Manifestations:

1. headache, dizziness & vertigo
2. crackling sounds & tinnitus
3. feeling of fullness in ear
4. hearing loss
5. tympanic membrane may be retracted, thickened, dilated vessels apparent drainage

Types:

- **Acute Suppurative Otitis Media (AOM)**
  - Sudden abrupt onset of infection
  - Usually bacterial infection accompanied by viral URTI with an abrupt onset of signs & symptoms involving the middle ear cleft.

- Short in duration (< 6 weeks)
- Recurrent AOM (rAOM) →
  - 3 or more months of AOM in 6 months
  - 4 or more months of AOM in 1 year
- S. Pneumonia, H. Influenza, Moraxella catarrhalis

- **Chronic Otitis Media with Effusion (OME)**
  - Repeated infection that causes drainage and perforation
  - Persistence of in the middle ear cleft fluid for >12 weeks
  - Results in painless hearing loss and intermittent purulent ear drainage that follows AOM or arises without prior AOM
  - Usually due to gram negative organisms — Pseudomonas, S. aureus, E. Coli
  - Can lead to tympanic membrane retraction or necrosis of tympanic membrane or ossicles leading to hearing loss
  - May develop a cholesteatoma behind the TM.
- **Serous Otitis Media/Chronic Suppurative Otitis Media (CSOM)**
  - Fluid forms in middle ear *without* infection
  - The fluid is formed when a vacuum is developed in the middle ear caused by blocked eustachian tube
  - Usually the microbe are: S. Pneumonia, H. Influenza, Moraxella catarrhalis, Adenovirus
  - WHO — Tympanic membrane perforation with persistent otorrhoea for more than 2 weeks
  - The fluid is too thick to drain resulting in feelings of congestion, hearing loss.
  - **Treatment:** possible myringotomy; small doses of corticosteroids to decrease oedema in Eustachian tubes.

Risk factors:

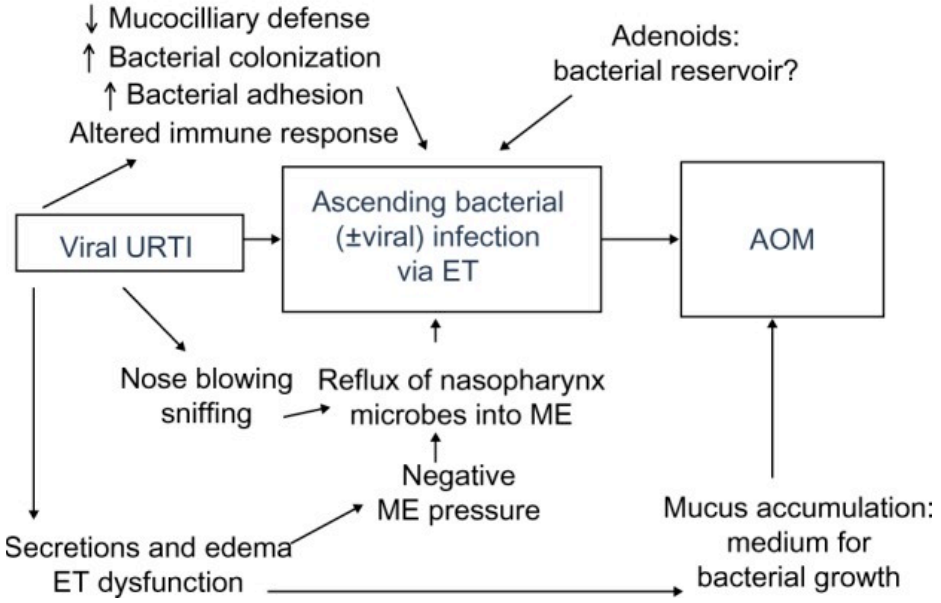
1. Upper Respiratory Infections
2. Allergies
3. Craniofacial abnormalities (cleft palate)
4. Down's Syndrome
5. Passive smoking

Pathogenesis

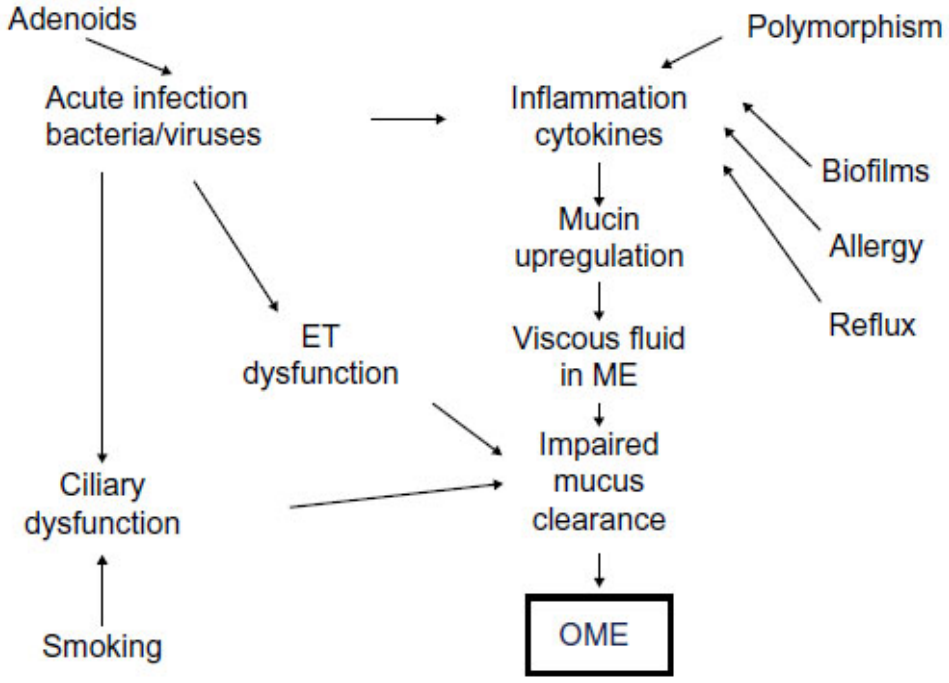
- This problem mainly deals with Eustachian tube dysfunction.
- Otitis Media usually follows an URI in which there is oedema of the Eustachian tube, leading to blockage.
- Stasis of these middle ear secretions lead to infection and irritation
- *Other factors*: allergic rhinitis; nasal polyps; adenoidal hypertrophy; Reflux or aspiration from nasopharynx;
  - **Infection:** viral and bacterial URTI
  - **Anatomic dysfunction:** Cleft palate, eustachian tube dysfunction
  - **Host factors:** Immature immunity, familial, method of feeding, sex (m>f), race
  - **Environment:** smoking, day-care attendance

- Hallmark is tympanic membrane perforation and/or otorrhoea
- Intense inflammation in the middle ear
  - Granulation
  - Cholesteatoma

Pathogenesis of AOM



Pathophysiology of Otitis Media with Effusion



Pathophysiology of Chronic Suppurative Otitis Media

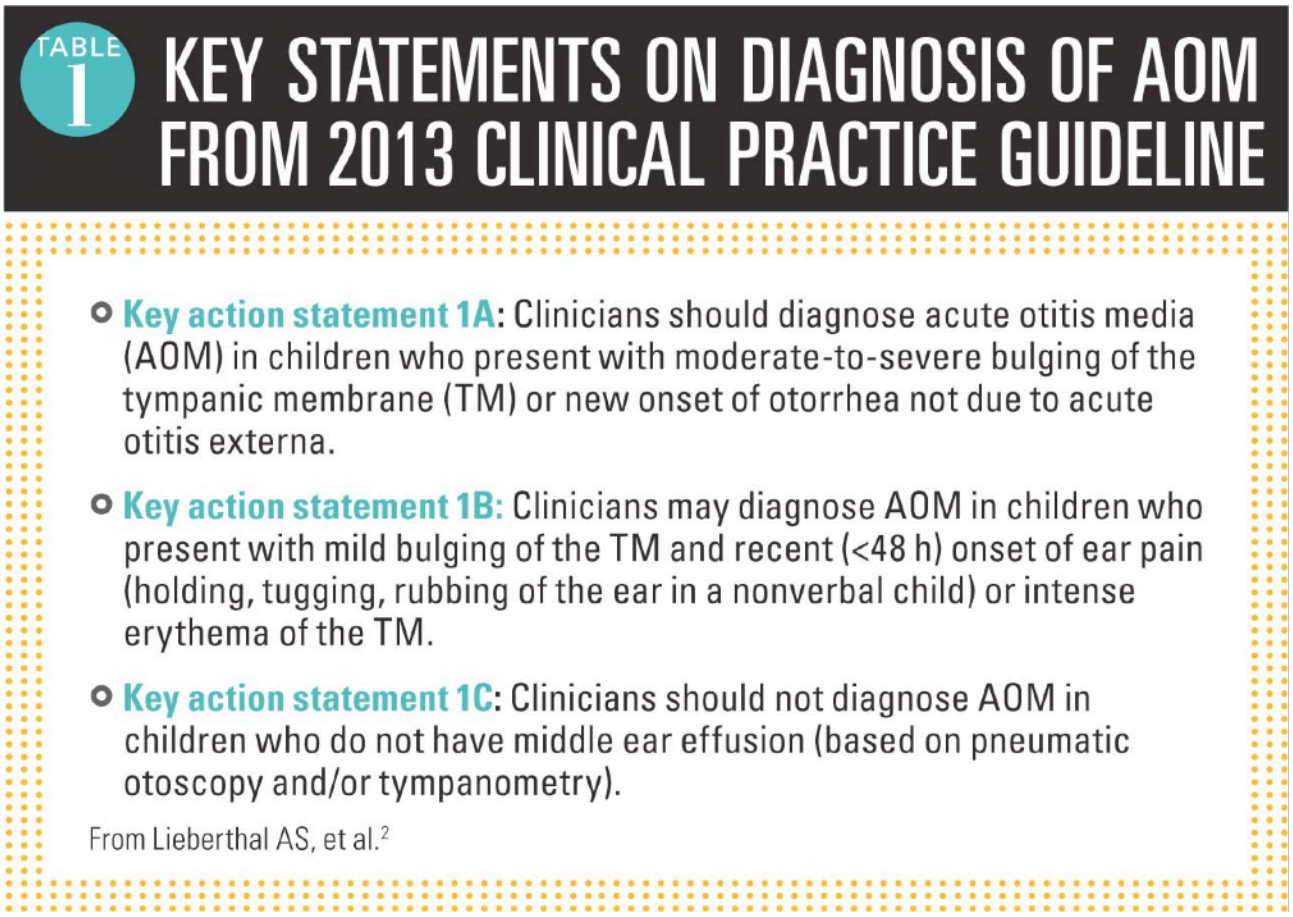
- Usually a sequelae of AOM or OME

Signs and Symptoms

- Neonates/Infants: change in behaviour, irritability, tugging at ears, decreased appetite, vomiting.
- Children(2-4): otalgia, fever, noises in ears, cannot hear properly, changes in personality
- Children (>4): complain of ear pain, changes I personality
- The classic description for Otitis Media is an erythaematic, opaque, bulging tympanic membrane with loss of anatomic landmarks including a dull/absent light reflex.
- Pneumatic Otoscopy: decreased tympanic membrane mobility

Diagnosis

- American Academy of Family Physicians 2015 guidelines for diagnosis AOM



- American Academy of Family Physicians 2015 guidelines for diagnosis OME
  - Pneumatic Otoscopy: standard tool for diagnosis; to demonstrate the presence of fluid within the middle ear cleft
  - **Impedance Tympanometry: useful for MEE.** Measures the resonance of the ear canal for a fixed sound as the air pressure is varied. Used if Pneumatic otoscopy not certain.
  - Do not intervene for 12 weeks unless hearing loss is present
  - Corticosteroids, antihistamines, decongestants are not useful.
- WHO guidelines for diagnosing CSOM
  - Persistent otorrhoea lasting >2 weeks
  - Presence of the tympanic membrane perforation

- Spectral Gradient Acoustic Reflectometry: measures the condition of the middle ear by assessing the response of the TM to a sound stimulus. Equivalent to tympanometry for dx of middle ear effusions
- Diagnostic tympanocentesis & myringotomy: involves puncturing the tympanic membrane and aspirating middle ear fluid to relieve pressure. Only used if the primary and secondary line treatment fail.
- With the increasing incidence of drug resistant strains of S. pneumoniae, CDC recommends the capacity of clinicians to be efficient in using tympanocentesis.

Differential diagnosis

1. Bullous myringitis
2. Otitis media
3. Cholesteatoma
4. Tympanosclerosis
5. Tonsillitis — referred pain from the pharyngeal region to the middle ear
6. TMJ disorder
7. Ramsay hunt
8. OME trauma

Investigations

- Tympanometry — AOM, OME (type A curve normal)
- Tympanocentesis — recurrent AOM
- Full blood count
- Ear swab if otorrhoea present
- CT scan or MRI
  - Persistent otorrhoea despite topical Treatment for 12 weeks
  - Presence of complications
  - Prior to surgical interventions

GOALS OF TREATMENT:

1. Prevent hearing impairment
2. Prevent effect on language development

AOM	CSOM	OME
Observe	Aural toilet	Observe
Systemic antibiotics	Topical medications	Myringotomy with or without ↓
Analgesia	Fluoroquinolone drops	Adenoidectomy ↓
Myringotomy with or without ↓	Systemic antibiotics	
Adenoidectomy ↓	Tympanomastoidectomy	

Antibiotics:

TABLE 2 RECOMMENDED ANTIBIOTICS FOR TREATMENT OF ACUTE OTITIS MEDIA

INITIAL IMMEDIATE OR DELAYED TREATMENT		TREATMENT AFTER INITIAL TREATMENT FAILURE (48-72 H)	
Recommended first-line treatment	**Alternative treatment (if penicillin allergy)**	**Recommended first-line treatment**	**Alternative treatment**
Amoxicillin (80-90 mg/kg/d in 2 divided doses)	Cefdinir (14 mg/kg/d in 1 or 2 doses)	Amoxicillin-clavulanate (90 mg/kg/d amoxicillin, with 6.4 mg/kg/d clavulanate in 2 divided doses)[a]	Ceftriaxone, 3 d clindamycin (30-40 mg/kg/d in 3 divided doses), with or without third-generation cephalosporin
OR	Cefuroxime (30 mg/kg/d in 2 divided doses)	OR	Failure of second antibiotic
Amoxicillin-clavulanate (90 mg/kg/d amoxicillin, with 6.4 mg/kg/d clavulanate [amoxicillin to clavulanate ratio, 14:1] in 2 divided doses)[a]	Cefpodoxime (10 mg/kg/d in 2 divided doses)	Ceftriaxone (50 mg IM or IV for 3 d)	Clindamycin (30-40 mg/kg/d in 3 divided doses) plus third-generation cephalosporin
	Ceftriaxone (50 mg IM or IV daily for 1 or 3 d)		Tympanocentesis[b]
			Consult specialist[b]

Note: Cefdinir, cefuroxime, cefpodoxime, and ceftriaxone are highly unlikely to be associated with cross-reactivity with penicillin allergy based on their distinct chemical structures.
[a]May be considered in patients who have received amoxicillin in previous 30 d or who have otitis-conjunctivitis syndrome.
[b]Perform tympanocentesis/drainage if skilled in procedure, or seek consultation from otolaryngologist for tympanocentesis/drainage. If tympanocentesis reveals multidrug-resistant bacteria, seek infectious disease specialist consultation.
Abbreviations: IM, intramuscular; IV, intravenous.
From: Lieberthal AS, et al.[2]

Duration of Treatment

< 2 years → 20 days

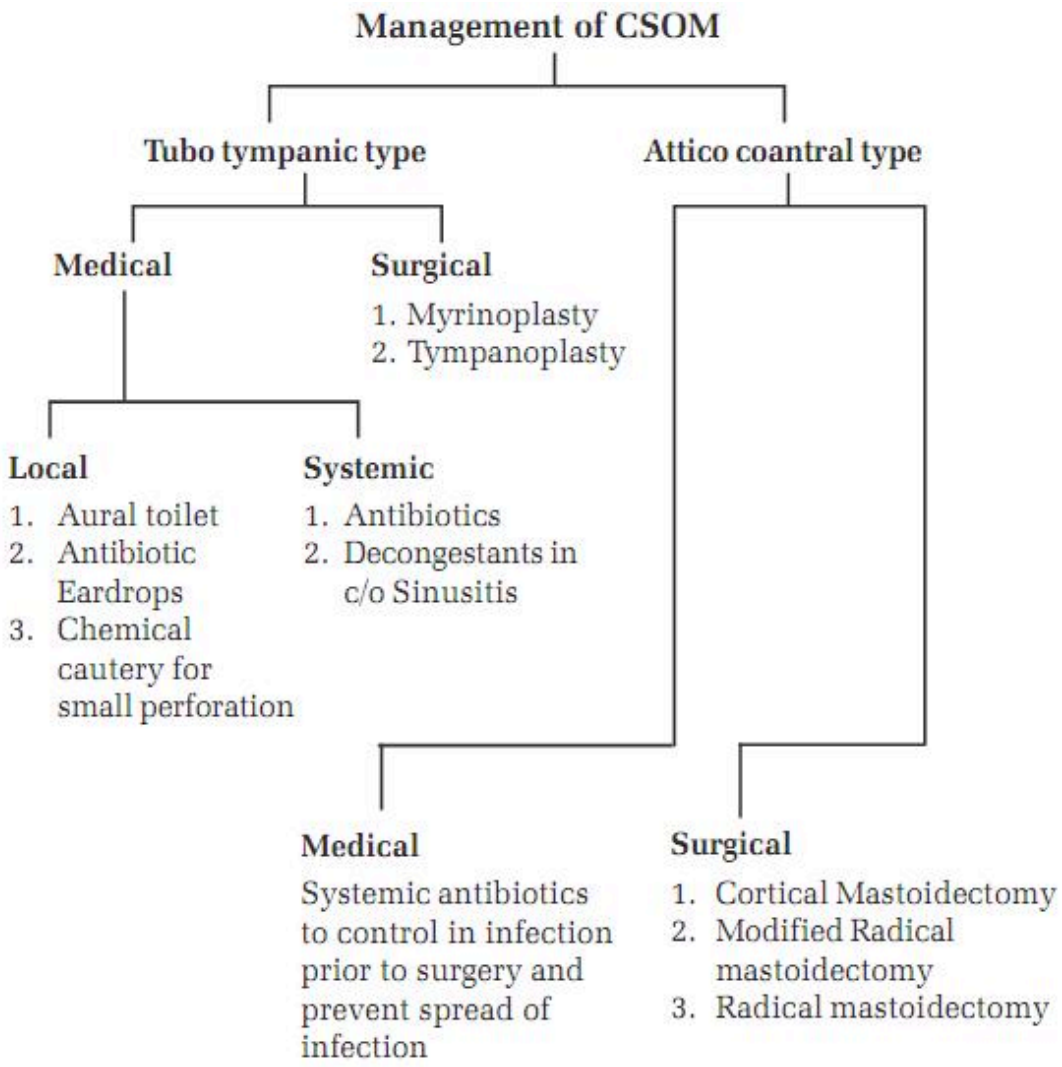
5 years → 5 – 7 days

Treatment:

1. *Myringotomy*
   - Children with persistent fluid and infection in their ears undergo this operation in which very small plastic tubes (1-2 mm) are inserted in the ears to remove fluid, restore normal hearing, and reduce infections. The tubes drain out the fluid, which equalises the pressure between the middle and outer ear. People with otitis media are advised not to fly if possible.
   - Relieve pressure and pain

2. *Antibiotics*
   - Amoxicillin: 20-40 mg/kg/day tid for 10-14 days or,
   - Augmentin: 45 mg/kg/day po bid for 10-14 days
   - Auralgan: analgesic/adjunct for ear pain 2-4 drops tid
   - Cefzil
   - Pediazole (erythromycin/sulfisoxazole)
   - Bactrim (trimethoprim/sulfamethoxazole
   - These medications are used as secondary agents if the primary antibiotic has failed after 10 days and the symptoms persists.

CSOM MANAGEMENT



- Antibiotic drops +/-steroids
- Regular aural toilet
- Quinolones: Ciprofloxacin, ofloxacin most effective
- Solutions of ear irrigation
  - 1.5% acetic acid
  - Dilute H2O2

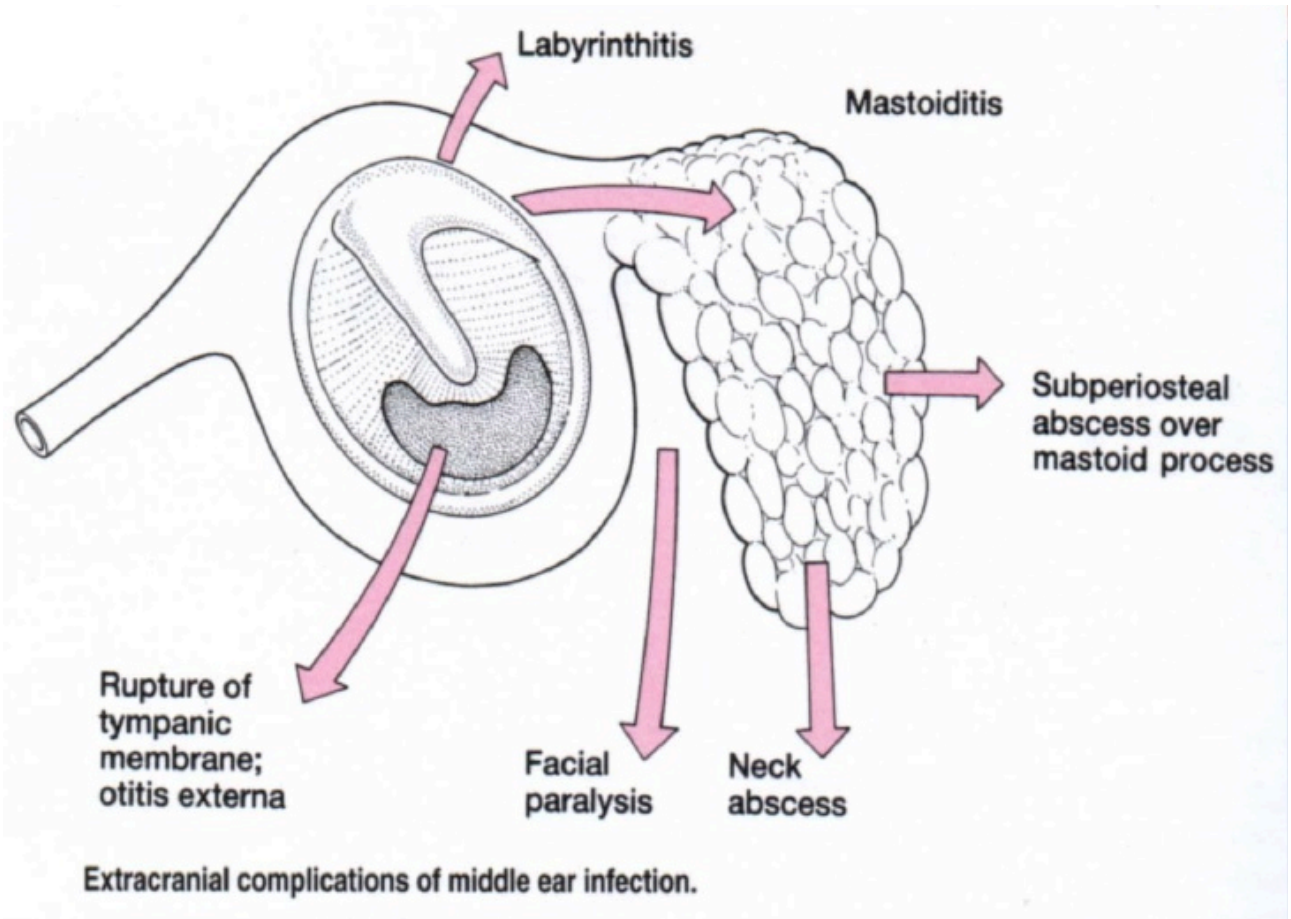
Differentials of OME

- CSF otorrhoea
  Peri - lymphatic fluid fistula

Complications:

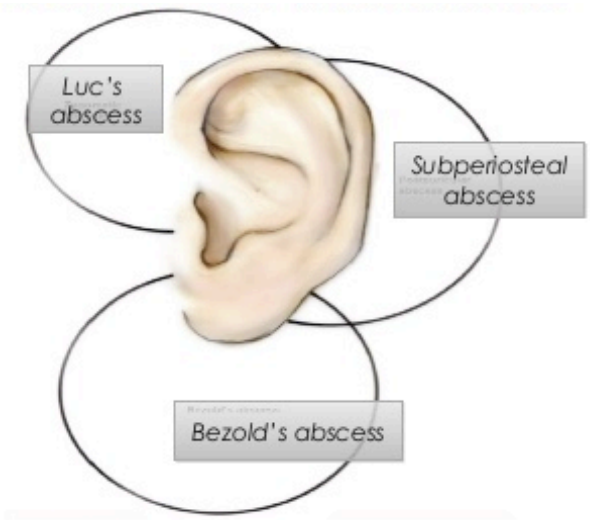
- Hearing loss: conductive, sensoneural, mixed)
- Acute mastoiditis: before the advent of antibiotics
- Chronic perforation of the TM
- Tympanosclerosis
- Cholesteatoma
- Chronic suppurative OM
- Cholesterol granuloma: 'Blue drum syndrome'
- Facial nerve paralysis
- Intracranial complications
- Bacterial meningitis
- Epidural abscess
- Subdural empyema
- Brain abscess
- Otitic hydrocephalus
- Lateral sinus thrombosis

Extra-temporal complications



Extracranial complications of middle ear infection.

- Mastoiditis: develops when infection tracks under the periosteum of the temporal bone to cause a sub-periosteal abscesses.



- Luc's abscess (infection spreads from the middle ear, resulting in a sub-periosteal collection beneath the temporal muscle)
- Bezold's abscess (infection breaks through the mastoid tip to cause a neck abscess deep to the sternocleidomastoid muscle)

Intratemporal

- Hearing loss
- CSOM
- Retraction pockets
- Cholesteatoma:
  - A mass of keratinising squamous epithelium and cholesterol in the middle ear usually resulting from chronic otitis media, with squamous metaplasia or extension of squamous epithelium inward to line an expanding cystic cavity that may involve the mastoid & erode surrounding bone. Usually present in the anterior superior aspect through the TM.
- Facial nerve palsy

Intracranial

- Otitis meningitis
- Epidural abscess
- Subdural abscess
- Focal encephalitis
- Otitis hydrocephalus
- Sigmoid and lateral sinus thrombosis
- Intra-parenchymal abscess & other intracranial suppurative complications
- Dural venous thrombophlebitis (usually sigmoid sinus)

MANAGEMENT OF NECK MASS

Differentials (KITTENS)

- (K) Congenital
- Infections or Iatrogenic
- Toxins or Trauma
- Endocrine
- Neoplasms
- Systemic

A. Congenital:

- Bronchial clefts cysts
- Cystic hygromas
- Teratoma and dermoid cysts
- External laryngocoele
  - Manifests later in life
  - Blow wind instruments — like a flute-isa
- Thyroglossal duct cyst:
  - Move up and down with tongue protrusion

B. Infections:

- Bacterial or viral lymphadenitis
- Tuberculosis — painless. matted LNs
- Persistent generalised lymphadenopathy
- Infectious mononucleosis
- Deep neck infections or abscess → fluctuation and inflammation
  - Parapharyngeal space
  - Submandibular

C. Toxins or Trauma and Endocrine:

Toxins OR Trauma

- Haematoma
- Oedema

Endocrine

- Parathyroid cyst
- Thymic cyst
- Thyroid hyperplasia
- Aberrant thyroid tissue

D. Neoplasms and Systemic diseases:

- Neoplasms:
  - Metastatic or regional malignancies
  - Thyroid neoplasms
  - Lymphoma
  - Salivary gland tumours
  - Vascular tumours
  - Neurogenic tumours
  - Lipomas
- Systemic diseases
  - Granulomatous disease
  - HIV
- Others
  - Acquired cysts — plunging ranula and sebaceous cyst

History

- Character:
  - Onset, duration, progression and pain
- Contributing factors:
  - RTIs, TB contact, risk for malignancy, trauma, recent travel, immunodeficiency
- Associated symptoms:
  - Fever nasal or aural or throat symptoms, night sweats, weight loss and malaise

Physical Examination

- Character of mass:
  - Size, distribution, mobility, tenderness, fluctuant, consistency, Transillumination
  - Solitary vs generalised cervical lymphadenopathy
  - Character of overlying skin
  - Thorough head and neck examination for primary malignancy
  - Palpate over lymphatic sites, liver, spleen and thyroid gland
  - Auscultation for vascular abnormalities

Ancillary tests

- **Lab test:**
  - TBS with differentials, ESR
  - Other blood tests
- **FNAs:**
  - For MCS and/or cytology
  - For non-resolving masses or if malignant is suspected

- **Panendoscopy** where mass is suspected to be metastatic
- **Open biopsy:**
  - Contraindicated except for persistent idiopathic adenopathy or high suspicion of malignancy where FNA is negative or indeterminate and complete work up does not reveal primary site
  - Prepare for completion of neck dissection if frozen section shows malignancy
- **Radiology:**
  - CT or MRI to define nature and extent of lesion
  - Angiography for vascular masses
  - U/S identifies cystic masses with Doppler defining vascular lesion

Treatment:

- Depends on nature and/or cause of lesion
- May include one or a combination of:
  - Medical treatment
  - Surgery
  - Radiotherapy
  - Chemotherapy

HEAD AND NECK CANCER

Definition:

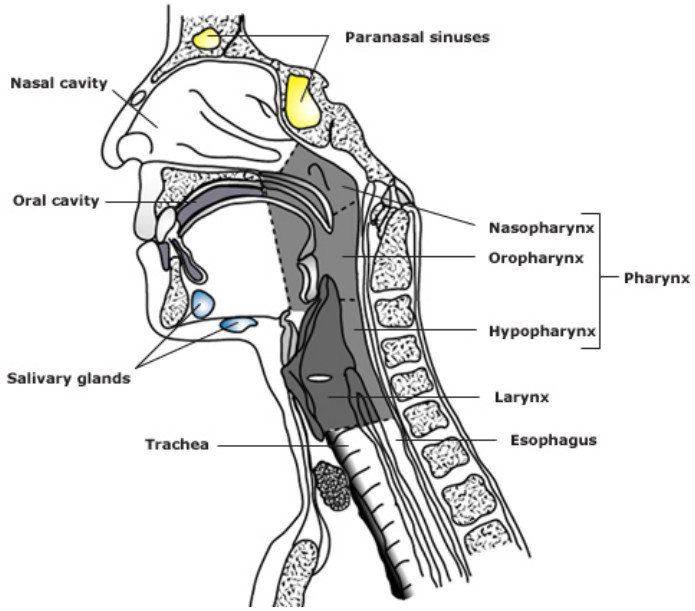
- Tumours arising from the epithelial lining of the upper aerodigestive tract
- **Squamous cell cancer** or a variant is the most common histologic type

Cites

Table 7-1. Head and Neck Cancer: Primary Sites

Oral Cavity
Lip
Floor of mouth
Oral tongue
Buccal mucosa
Alveolar ridges
Hard palate
Retromolar trigone
Pharynx
Nasopharynx (includes superior surface of soft palate)
Oropharynx (includes inferior surface of soft palate, uvula)
Hypopharynx (pyriform sinus, postcricoid, posterior wall)
Larynx
Supraglottic larynx (false cords, arytenoids, epiglottis)
Glottic larynx (includes commissures)
Subglottic larynx
Nasal Cavity and Paranasal Sinuses
Nasal cavity
Maxillary sinuses
Ethmoid sinuses
Frontal sinuses
Sphenoid sinuses

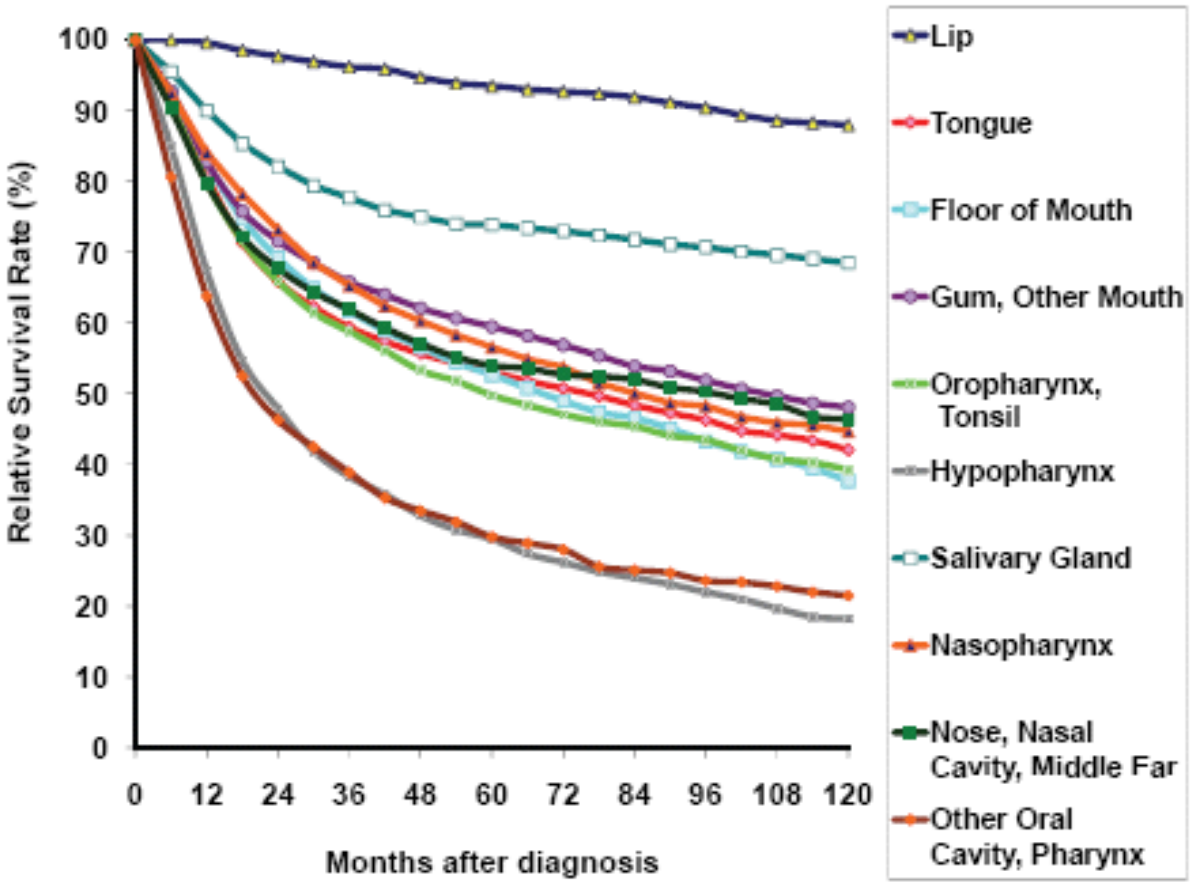
Review of anatomy



Epidemiology

- M:F is 2:1 but as high as 7:1 in CA-larynx
- **Retrospective hospital-based descriptive study(KNH)**
  - The larynx was the most common site for aerodigestive malignancies, followed in order of frequency, by the tongue, the mouth, and the nasopharynx.
  - The eye followed by the thyroid were the most commonly affected sites outside the aerodigestive tract.
  - Squamous cell carcinoma was the most common malignancy, with sarcomas being rare
  - Gender and age distribution showed an overall male preponderance and a wide age range.
  - Specific tumour sites and tumour types showed varying patterns of gender and age distribution.
  - The study therefore confirmed the relative prominence of laryngeal, oral and nasopharyngeal CA's among the African population.

Relative Survival Rate (percentage) By Primary HNC Site, 1988-2001



Risk Factors

1. **Tobacco Products** —
   - *Smoking, Tobacco, Cigarettes, Cigars, Pipes, Chewing Tobacco, Snuff*

- *Numerous studies have concluded that tobacco, which contains carcinogens such as polonium 210, nitrosamines, and aromatic hydrocarbons, is directly linked to development of carcinomas.*

2. **Ethanol Product**s —
   - Alcohol acts as a solvent to enhance mucosal exposure to carcinogens, increasing cellular uptake of these.
3. **Chemicals** —
   - *Asbestos, Chromium, Nickel, Arsenic, Formaldehyde*
4. **Other Factors**:
   - *Ionising Radiation, Plummer-Vinson Syndrome, Epstein-Barr Virus, Human Papilloma Virus*
5. Possible occupational risks
   - **Wood Working**
     - Prone to carcinomas of the paranasal sinuses
     - Though rare, an occupational exposure history is important in patients with recurrent epistaxis, nasal obstruction, or facial pain
   - Leather manufacturing
   - Nickel Refining
   - Textile industry
   - Radium dial painting

Carcinogens and Viruses:

- Smokeless tobacco and other oral chewed carcinogens - Betel quid are associated with the development of cancers of the oral cavity.
- The Plummer-Vinson syndrome, seen in women younger than 50, associated with iron-deficiency anaemia, hypo pharyngeal webs, dysphasia, and a higher risk of cancers of the post-cricoid and hypo pharynx.
- Maxillary sinus: are associated with certain occupational exposures (e.g., nickel, radium, mustard gas, chromium, and byproducts of leather tanning and woodworking).
- HPV is associated with oral cancers (oropharynx and tonsillar areas), most common types are 16 and 18.

HPV-related oral cancer risk factors

1. Younger age
2. Current oral HPV infection
3. High-risk sexual behaviours
   - First sexual experience at young age
   - Increasing number of vaginal- and oral-sex partners

- Specifically linked to squamous cell carcinomas of the base of the tongue, tonsil, and epiglottis
- Associated with a 9-fold increased risk of oropharyngeal cancer

Warning signs for HN Cancer

- **Hoarseness —** frequently occurs in the very earliest laryngeal glottic cancers

- **Erythroplasia** — is a clinical term to describe any erythematous (red) area on a mucous membrane, that cannot be attributed to any other pathology
- **Referred otalgia —** may accompany a cancer of the larynx, pharynx or oral cavity
- **Persistent sore throat —** One longer than two weeks in a patient with a smoking history should arouse suspicion for a possible cancer in the larynx and/or pharynx.
- **Epistaxis**
- **Nasal obstruction**
- **Serous otitis media**
- **Neck mass**
- **Non-healing ulcer**
- **Dysphagia**
- **Submucosal mass**

What do they mean?

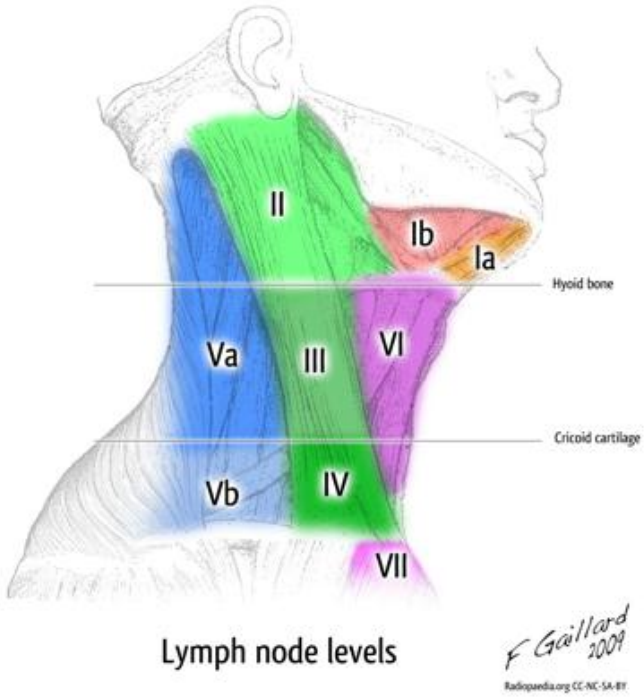
- Epistaxis, nasal obstruction, and serous otitis media can all herald a nasopharyngeal cancer
- Early cancer in many sites, e.g., the epiglottis, pyriform sinus, nasopharynx and paranasal sinuses, are silent with few signs
- Although not an early sign, a neck mass may be the first presenting symptom. Any high-risk patient with a neck mass should be thoroughly evaluated for a head and neck primary cancer.
- A non-healing ulcer, dysphagia or a submucosal mass may also serve as warning signs of potential carcinomas

Site of Lymphatic Drainage:

- Level I - all nodes above hyoid bone, below mylohyoid muscle, and anterior to posterior edge of submandibular gland Level IA - all nodes between medial margins of anterior digastric muscles, above hyoid bone, below mylohyoid muscle Level IB - all nodes below mylohyoid muscle, above hyoid bone, posterior and lateral to medial anterior digastric muscle and anterior to submandibular gland
- Level II - all nodes below skull base at jugular fossa to hyoid bone, anterior to posterior edge of sternocleidomastoid muscle and posterior to submandibular gland
  - Level IIA - all nodes that lie posterior to internal jugular vein and are inseparable from the vein or lie anterior, lateral or medial to the vein
  - Level IIB - all nodes that lie posterior to internal jugular vein and have a fat plane separating the nodes and the vein
- Level III - all nodes between hyoid bone and cricoid cartilage arch and anterior to posterior sternocleidomastoid muscle, and lateral to the internal carotid artery Level IV - all nodes between cricoid cartilage arch and clavicle, anterior to posterior sternocleidomastoid muscle and posterolateral to anterior scalene muscle and lateral to common carotid artery
- Level V - all nodes from skull base posterior down to posterior border of sternocleidomastoid muscle to level of clavicle, anterior to trapezius muscle

- Level VA - all nodes between skull base and cricoid cartilage arch, behind posterior edge of sternocleidomastoid muscle
- Level VB - all nodes between cricoid cartilage arch and clavicle, behind sternocleidomastoid muscle

- Level VI - all nodes inferior to hyoid bone and above top of manubrium, between medial margins of bilateral common carotid and internal carotid arteries Level VII - all nodes behind the manubrium between medial margins of common carotid arteries bilaterally, extending inferiorly to level of innominate vein
- Level VII
  - superior mediastinal nodes
  - between CCAs, below superior aspect of manubrium to level of the brachiocephalic vein



Match between the site and lymphatic drainage:

Cancer from the…	Site of drainage
Oral cavity	I
Larynx	II & III
Nasopharynx	II & V
Thyroid	IV & V

Staging:

- Clinical staging used, not pathologic = physical + radiographic.
- TNM staging system used.
- T – site-specific, but in general:
  - T1-3 = increasing size of tumour
  - T4= invasion of muscle, cartilage or bone
    - T4a = surgically resectable disease
    - T4b= locally unresectable disease
- N — nodal involvement is the same for all EXCEPT nasopharyngeal

Management:

- Find it, usually late
  - over 80% of tumours are late stage
- Surgery (Excision)
- Radiation Therapy
- Chemotherapy
- Combine the above

Key to curing the cancer:

- Stop all smoking (causes more cancer deaths than any other factor)
- Wait 30 years (time required for relative risk of lung cancer to return to 1 after smoking cessation)
- Ignore cancers due to:
  - Low level exposures
  - Multifactorial genetic predisposition
  - Stochastic phenomena
- **Prevention**
  - definition of more subtle genetic and environmental risk factors
- **Targeted Therapy**
  - Molecular and otherwise
- **Screening**
  - Molecular Screening for early disease
  - Genetic screening for inherited cancer susceptibility
  - Conventional screening for non-genetic risk factors
    - Pap smear, colonoscopy, etc
- **Clinical Presentation/Diagnosis**
  - pathologic LN in the neck may suggest primary site
  - oral cavity CA spread to level I
  - larynx CA — level II and III
  - disease in IV, V → suspect thyroid or primary below neck
- Previously Untreated stage I, II, Low-bulk stage III
  - Single-modality therapy with surgery or radiation

  - Cure rates are 52-100% depending on primary site
  - Which modality is chosen depends on local expertise, anticipated functional outcome, and patient preference
- Previously Untreated Higher bulk stage III, IV (T3,T4,N2,N3)
  - If resectable — surgery followed by RT +/- chemo based on pathology (favoured option for oral cavity) OR chemo and radiation, with surgery upon relapse
  - If unresectable — chemo and radiation together
  - Cure rates are 10-65% and often at the cost of cosmetic and functional disability

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Management — Recurrent/Relapsed Head and Neck Cancer

- Recurrent disease – If salvage surgery feasible, surgery
- OR if no prior radiation, then radiation indicated + chemo
- Median survival is 5-9 months.

Principles of surgery:

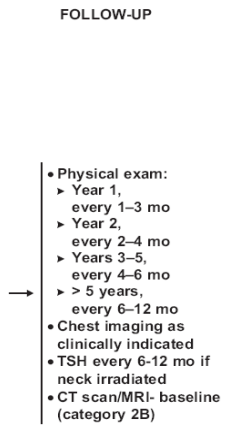
- Goal: Complete removal of the tumour with negative margins.
- A comprehensive neck dissection involves the en bloc removal of all five lymph node levels. The sternocleidomastoid muscle, the internal jugular vein, and the spinal accessory nerve are jeopardised. If not called radical neck dissection.
- Done when cancerous lymph nodes are suspected or known to be present.
- Selective neck dissections are used, whereby fewer than five lymph node levels are removed, done when there are no palpable lymph nodes.

Principles of radiotherapy:

- Can be used as a single modality to treat early-stage disease.
- Standard, once-daily fractionation consists of 2.0 Gy per day with a total dose of 70 Gy or greater to the primary site and gross adenopathy and 50 Gy or greater to uninvolved nodal stations at risk.
- When given postoperatively, the total dose to the primary site and involved nodal stations is 60 Gy or greater, and the dose to uninvolved nodal stations at risk is 50 Gy or greater.
- Postoperative radiation generally begins 4 to 6 weeks after surgery.
- Hyperfractionation being studied: but no significant differences in overall survival were demonstrated, a recent metaanalysis indicated a significant improvement in absolute survival at 5 years (3.4%; $p = 0.003$) with altered-fractionation approaches.
- Increased acute toxicity and hence not recommended routinely.
- IMRT is being used.

Principles of chemotherapy:

- Chemotherapy as a single modality is not curative for patients with H&N cancer
- In unresectable squamous cell CA of H&N, concurrent chemo RT has been shown to increase survival as compared to RT alone
- For patients with locally advanced CA hypo-pharynx/larynx — *ChemoRT with surgery reserved for salvage compared to upfront surgery offers a significant chance of preservation of the larynx without compromising survival*
- Drugs used:
  - Cisplatin and infusional 5-FU → response in 60-90% of previously untreated patients; clinical CR in 20-50%
  - Other agents: MTX, carboplatin, paclitaxel, docetaxel, ifosfamide, topotecan, irinotecan response rates are 13-31%
- When possible surgery is the first option;
- Unless we are trying to save the organ
- We then try chemotherapy and radiation together.
- **Adjuvant chemo RT** —
  - Cisplatin + RT adjuvant cat 1 if positive margins and extra capsular extension in involved LN's.
  - For everything else like positive LN, perineurial involvement only adjuvant RT, cat 1.
- **Targeted therapies** —
  - Cetuximab studied in combination with RT and compared to RT alone.
  - Showed improved loco regional and OS rates.



Summary:

- Multimodality therapy for all but very early stages: surgery, radiation with adjuvant chemotherapy
- Significant morbidity due to therapy is possible: cosmesis, decreased saliva, swallowing dysfunction, social dysfunction
- Novel molecular directed therapies incorporated into next generation trials