Anatomy of Nose
External Nose

- bony pyramid
- cartilaginous pyramid
- lobule
- soft tissue
Vestibule
Nasal cavity proper
Roof

- Frontal & nasal bones
- Cribiform plate of ethmoid
- Body of sphenoid
Floor

- Nasal bone
- Frontal sinus
- Perpendicular plate of ethmoid
- Septal cartilage
- Vomer
- Maxilla
- Vomer bone
- Palatine bone
- Cribiform plate of ethmoid bone
- Sphenoid sinus
- Pharyngeal tonsil
- Vomeronsal cartilage
Medial wall -

- Nasal spine of frontal bone
- Crest of nasal bone
- Membranous septum
- Columellar septum
- Ant. nasal spine of maxilla
- Septal cart.
- Vomer
- Perp. plate of ethmoid
- Rostrum of sphenoid
- Crest of maxilla
- Crest of palatine bone
Blood supply of septum

Fig. 33.1 Blood supply of nasal septum.
Kiesselbachs and Woodruffs plexus
Lateral wall of nose
Drainage of sinuses

- Semilunar hiatus - Openings of frontal, maxillary and anterior ethmoidal sinuses
- Ethmoid bulla - Opening of the middle ethmoid sinus
- Opening of sphenoid sinus
- Opening of eustachian tube
- Opening of nasolacrimal duct

(C) teachmeanatomy, The #1 Applied Human Anatomy Site on the Web
Inferior meatus
Middle meatus
Osteomeatal complex [Picadli’s circle]

The osteomeatal complex
Lining membrane of the nose

- Olfactory regions
- Respiratory regions
- Nasal vestibules
Nerve supply of nasal cavity
Olfactory pathway
Disorders of smell
Autonomic innervation
FACIAL INFECTION

- DANGEROUS AREA OF FACE
  1. A triangular area bounded with an apex opposite the medial angles of eyes & nose and a base formed by the upper lip
  2. It is drained by facial vein
  3. It has important communications with cavernous sinus:

  supraorbital & superior ophthalmic veins

  Facial vein

  deep facial vein → pterygoid plexus of veins → emissary veins

  Cavernous sinus
Danger Triangle of Face
Lymphatic drainage
Questions

- Frontonasal duct opens into:
  - A] Inferior meatus
  - B] Middle meatus
  - C] Superior Meatus
  - D] Inferior turbinate
• Paranasal Sinus opening in the middle meatus are
• -Maxillary
• -anterior ethmoid
• -posterior ethmoid
• -frontal
• -Sphenoid
• Maxillary sinus opens into the middle meatus at the level of
• -Hiatus Semilunaris
• -Bulla Ethmoidalis
• -Infundibulum
• -None of the above
• Quadrilateral cartilage is attached to all except
  • -Ethmoid
  • -Vomer
  • -Sphenoid
  • -Maxilla
• Nasal valve is formed by
• -Upper lateral cartilage
• -Lower Lateral cartilage
• -Lower end of the upper lateral cartilage
• -Upper end of the Lower lateral cartilage
Functions of the nasal cavity

• Nasal Respiration
• Protection of lower respiratory tract ‘
• Vocal resonance
• Olfaction
• Outlet to lacrimal secretions
• Inspiratory and expiratory current
Questions

• During inspiration the normal flow of the air through the nasal cavity is through:
• -Middle part of the cavity in the meatus in a parabolic curve
• -lower part of the nasal cavity in the inferior meatus in a parabolic curve
• -superior part of the cavity in the superior meatus
• -through olfactory area
• Ciliary Movement rate of the nasal mucosa is
• 1-2 mm/min
• 2-5 mm/min
• 5-10 mm/min
• 10-12 mm/min
Choanal atresia

• Etiology:
  Persistence of bucconasal membrane

• Types:
  • Bony (90%) and membranous (10%).
Choanal atresia

- **Clinical features:**
  - Bilateral atresia presents with respiratory obstruction as the new born being a natural breather does not breathe from mouth.
  - Bilateral atresia is an emergency
CHARGE Syndrome

- C
- H
- A
- R
- G
- E
• Diagnosis:
  1. Presence of mucoid discharge in nose
  2. Absence of air bubbles in the nasal discharge.
  3. Failure to pass a catheter from nose to pharynx.
  4. Installing radiopaque dye in to the nose and taking a lateral x-ray.
Treatment:

- Emergency airway:
  - 1. Mc’Govern’s technique: A feeding nipple with a large hole provides good oral airway.
  - 2. Tracheostomy

- Definitive treatment:
  - 1. Correction of atresia at 1.5 years of age.
Meningocoele /meningoencephalocoele

- Etiology
- Types
Clinical features:

Examination:

Treatment:
Glioma

• What is it?
• Clinical features
• Examination
• Treatment
Dermoid

- Etiology
- Clinical features
- Diagnosis
- treatment
<table>
<thead>
<tr>
<th></th>
<th>Meningocoele/meningoencephalocele</th>
<th>Glioma</th>
<th>Dermoid</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Content</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Pathology</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Compressibility test</strong></td>
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<td></td>
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<tr>
<td><strong>Transillumination test</strong></td>
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<tr>
<td><strong>Frustenberg test [cough impulse]</strong></td>
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</tbody>
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Diseases of external nose and septum

- Midline deformities

- Lateral nasal deformity
Saddle nose and Hump nose
Lateral nasal deformity

• Crooked nose

• Deviated nose
## Fractures of nasal septum

<table>
<thead>
<tr>
<th></th>
<th>Jarjaway fracture</th>
<th>Chevallet fracture</th>
</tr>
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<tbody>
<tr>
<td><strong>Direction of blow</strong></td>
<td>From front</td>
<td>From below</td>
</tr>
<tr>
<td><strong>Direction of fracture</strong></td>
<td>Horizontal</td>
<td>Vertical</td>
</tr>
</tbody>
</table>

![Diagram of nasal septum fractures](image)
Diseases of septum

- Deviated nasal septum
- Septal abscess
- Septal hematoma
- Septal perforation
Deviated nasal septum

• Aetiology:
  • 1. Trauma
  • 2. Developmental deformities of palate
Deviated nasal septum - Types

Fig. 26.4 Types of deviated nasal septum.
Clinical features

- Nasal obstruction:
- Headache
- Sinusitis
- Epistaxis
- Anosmia
- External deformity
- Middle ear infection
Cottle’s line

Drawn from frontal spine to anterior nasal spine. Deviations anterior to it can be treated by septoplasty only. Posterior to it by SMR or septoplasty.
Cottles test
### Differences between septoplasty and SMR

<table>
<thead>
<tr>
<th></th>
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<th>Septoplasty</th>
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</thead>
<tbody>
<tr>
<td></td>
<td>Radical</td>
<td>conservative</td>
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<td>Children ??</td>
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<tr>
<td>Elevation of flaps</td>
<td></td>
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<tr>
<td>Removal of cartilage</td>
<td></td>
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<tr>
<td>Caudal dislocation</td>
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<tr>
<td>Septal perforation</td>
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<td>Supratip deformity and columnellar retraction</td>
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<td>Revision surgery</td>
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</tbody>
</table>
• Septal surgery is usually done after the age of 17 years so as not to interfere with the growth of nasal septum.
• For deviated nasal septum surgery is required for all except?
• -septal spur with epistaxis
• -septal deviation
• -persistent rhinorrhea
• -recurrent ear pain and discharge
• Thudicums nasal speculum is used to visualize
  • -Anterior nasal cavity
  • -Posterior nares
  • -tonsils
  • -larynx
• Which is not visualized in posterior rhinoscopy.

• -eustachian tube
• -inferior meatus
• -middle meatus
• -superior concha
• Cottles test tests the patency of the nares in

• Atrophic rhinitis
• Rhinosporidiosis
• Deviated nasal septum
• Hypertrophic inferior turbinate
Septal haematoma

- What is it?
- Etiology:
- Clinical features:
  1. obstruction – b/l or u/l ???
  2. Pain over the bridge of nose
  3. NO FEVER
Treatment:
Small – aspiration with a wide bore needle
Large – incision and drainage

What is important??
Compressed nasal packing following drainage to prevent reaccumulation
Septal abscess

• Etiology:
  - Secondary to infection in a septal hematoma
  - Rarely following furunculosis

• Clinical features:
  Fever
  Headache
  Enlarged submental or submandibular lymphnode
• Treatment:
  1. Aspirate pus plus remove a piece of cartilage
  2. Systemic antibiotics

• Complications:
  Saddle nose deformity
  Meningitis and cavernous sinus thrombosis
• Septal hematoma is accumulation of blood between perichondrium and cartilage – True /false

• In which condition do you drain immediately septal abscess or hematoma ?? And why ??
Perforation of septum

-Most common cause: Traumatic perforation

-Pathological perforations:
1. Cartilagenous – Lupus, TB, Leprosy
2. Bony – Syphilis
3. Total septum – Wageners granuloma
• Small perforations – whistling sounds
• Large perforations – nasal obstruction and epistaxis
138 Septal abscess

A Is always secondary to a septal haematoma.
B Pain is localized to the tip of the nose.
C Systemic symptoms are unusual.
D A septal perforation may ensue.
E Initial treatment consists of incision and drainage plus systemic antibiotics.

139 Septal perforation

A Most cases are due to nose-picking, syphilis or cocaine abuse.
B Chromic acid is a recognized industrial cause.
C Large perforations characteristically cause a whistling noise.
D The cartilaginous septum is most commonly involved, except in syphilitic cases, when it is the bony septum.
E Surgical repair is usually advisable because of the danger of severe epistaxes.
• Bony septal perforation occurs in

1. Tuberculosis
2. Leprosy
3. Syphilis
4. Sarcoidosis
Epistaxis

• Classification of epistaxis:
  • Anterior epistaxis
  • Posterior epistaxis

• MC site: Littles area

• Above the level of middle turbinate
• Below the level of middle turbinate
• Posterior part of nasal cavity
• Causes of epistaxis

1) Local causes
   - Trauma
   - Infection
   - Foreign body
   - Neoplasm of nose and paranasal sinus
   - Deviated nasal septum

2) General cause

3) Idiopathic

4. Mediastinal tumors can cause elevation of venous pressure and bleeding from nose.
<table>
<thead>
<tr>
<th></th>
<th>Anterior epistaxis</th>
<th>Posterior epistaxis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Incidence</td>
<td>More common</td>
<td>Less common</td>
</tr>
<tr>
<td>Site</td>
<td>Littles area</td>
<td>Woodruffs plexus</td>
</tr>
<tr>
<td>Age</td>
<td>Children</td>
<td>After 40 years of age</td>
</tr>
<tr>
<td>Cause</td>
<td>Trauma</td>
<td>HTN</td>
</tr>
<tr>
<td>Bleeding</td>
<td>Mild can be controlled by local pressure or anterior pack</td>
<td>Bleeding is severe, requires hospitalization and postnasal pack</td>
</tr>
</tbody>
</table>
General causes of epistaxis

- Hypertension
- Disorders of blood and blood vessels
- Liver disease
- Kidney disease
- General infection
- Vicarious menstruation
Protocol for epistaxis

1. Assessment and first aid
   - RESUS
2. Initial medical review
3. Nasal preparation
   - Visualise bleeding point
     - Controlled
     - Yes
       - Cauterise
         - Controlled
       - No
         - Bleeding
           - Anterior pack
             - Controlled
             - Posterior pack
               - Controlled
               - Bleeding
                 - Surgery
                 - Follow up
             - Observe
             - Control
           - Bleeding
             - Anterior pack
               - Controlled
               - Posterior pack
                 - Controlled
                 - Bleeding
                   - Surgery
                   - Follow up
         - Observe
Digital Pressure (Trotter’s Method)

- Application of digital pressure over Kiesselbach’s plexus for at least 15-20 minutes
• Ligation of artery
  1] Sphenopalatine A
  2] Internal maxillary artery
  3] Ethmoidal artery
  4] External carotid artery
Herditary hemorrhagic telengectasia

• Autosomal dominant condition
• Affects the blood vessels
Management

- Recurrent bleeds
  - Blood transfusion required
    - Mild: Laser photocoagulation
    - moderate: Packing and antifibrinolytic agents
    - Severe: Nasal closure Youngs operation
  - Blood transfusion not required
    - Septodermoplasty
Recurrent epistaxis in a 15 year old female the most common cause is:

1. Juvenile nasopharyngeal angiofibroma
2. Rhinosporidiosis
3. Foreign body
4. Haematopoietic disorder
A 70 year old patient with epistaxis, patient is hypertensive with BP of 200/100 mm hg. On examination no active bleeding is noted next step in the management is
- Observation
- Internal maxillary artery ligation
- Anterior and posterior nasal packing
- Anterior nasal packing
Source of epistaxis after ligation of external carotid artery is

- Maxillary artery
- Greater Palatine artery
- Superior labial artery
- Ethmoidal Artery
- In case of uncontrolled epistaxis ligation of internal maxillary artery is done in the
  - Maxillary antrum
  - Pterygopalatine fossa
  - at the neck
  - Medial wall of orbit
## Granulomatous disease of nose

<table>
<thead>
<tr>
<th>Bacterial</th>
<th>Fungal</th>
<th>Unspecified cause</th>
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<tbody>
<tr>
<td>Rhinoscleroma</td>
<td>Rhinosporidiosis</td>
<td>Wageners granuloma</td>
</tr>
<tr>
<td>Syphilis</td>
<td>Aspergillosis</td>
<td>Non healing granuloma</td>
</tr>
<tr>
<td>Tuberculosis</td>
<td>Mucormycosis</td>
<td>Sarcoidosis</td>
</tr>
<tr>
<td>Lupus</td>
<td>Candidiasis</td>
<td></td>
</tr>
<tr>
<td>Leprosy</td>
<td>Histoplasmosis</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Blastomycosis</td>
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</tr>
</tbody>
</table>
Rhinoscleroma

- **Etiology**: Frisch bacillus or Klebsiella rhinoscleromatis
- **Gram negative bacillus**
- **Endemic in northern India**
Rhinoscleroma

- Clinical features:
  1) **Atrophic stage**: foul smelling purulent nasal discharge and crust.
  
  2) **Granulomatous stage**: nodules in the nose giving WOODY feel. Nodules are seen in lower part of external nose and lip. Nodules are painless and non-ulcerative.
  
  3) **Cicatrising stage**: stenosis of nares, nasopharynx and oropharynx.
Rhinoscleroma: Spread

• Starts in nose and spreads to pharynx, larynx, trachea and bronchus.

• Subglottic larynx is most commonly affected
• Diagnosis
- Miculicz cells
- Russel bodies
Rhinoscleroma-treatment

• Treatment:
  1. Streptomycin and tetracycline for a minimum period of 4-6 weeks.
  2. Steroids are given to decrease fibrosis.
  3. Treatment is stopped only when two cultures are negative.
Lupus Vulgaris

- Low grade tubercular infection
- Sites: Anterior part of nose
- APPLE JELLY NODULES
• Diagnosis:
  • Smear for AFB
  • Tissue TB PCR

• Treatment:
  • ATT
Leprosy

- Organism: Tb or LL
- Affected parts:
  - Anterior part of septum and IR
- Late sequelae:
  - Depression over bridge of nose, retrusion of columella
- Treatment:
  - Dapsone, Rifampin, Isoniazid
Rhinosporidiosis

• Causative organism: Rhinosporidium Seeberi
• Prevalance: India, Pakistan, Srilanka.
• More common in Southern India than in North India
• Characteristic appearance: Leafy, pink, purple, polypoidal mass.

• It is studded with white dots representing the sporangia of fungus.

• Mass is very vascular and bleeds to touch.
• Diagnosis is by biopsy
• This fungus can not be cultured

• Treatment: complete excision of mass with diathermy knife and cauterization of its base.
• Dapsone is partially effective
Mucormycosis

- Rapidly progressing and fatal
- Uncontrolled diabetics
- Fast spread
- c/f
- Treatment
• Atrophic dry nasal mucosa, extensive crustings, with woody hard feel of external nose is suggestive of
  • -Rhinosporidiosis
  • -Rhinoscleroma
  • -Atrophic rhinitis
  • -carcinoma of nose
Allergic rhinitis
• Allergy is mediated by which immunoglobulin??

• What are the symptoms of allergy??
• **Seasonal:**
  
  • Only during a particular season when the pollens of plant are released.

• **Perennial:** Symptoms are present throughout the year.
• Etiology:
  1] Inhalant
  2] Ingestant
  3] Genetic predisposition
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
Clinical features

• Usually seen at 12—16 years of age.
• The cardinal symptoms of seasonal nasal allergy include
  • Paroxysmal sneezing,
  • Nasal obstruction,
  • Watery nasal discharge
  • Itching in the nose.
• Itching may also involve eyes, palate or pharynx.
• Some may get bronchospasm.
Symptoms of perennial allergy are not so severe as that of the seasonal type.

They include frequent colds, persistently stuffy nose, loss of sense of smell due to mucosal oedema,

Postnasal drip, chronic cough and hearing impairment due to eustachian tube blockage or fluid in the middle ear.
• **Ocular signs include**

1] *Oedema of lids,*

2] *Congestion and cobble-stone appearance of the conjunctiva,*

3] *Dark circles under the eyes (allergic shiners).*
Signs

• *Nasal signs* –
• **Otologic signs:**

• *Retracted tympanic membrane* or serous otitis media as a result of eustachian tube blockage.
• *Pharyngeal signs include granular pharyngitis*

• A child with perennial allergic rhinitis may show all the features of prolonged mouth breathing as seen in adenoid hyperplasia.

• *Laryngeal signs include hoarseness of voice and oedema of the vocal cords*
Diagnosis

• Clinical
• Investigations:
  • Total and differential count. Peripheral eosinophilia may be seen but is an inconsistent finding.
  • Nasal smear shows large number of eosinophils in allergic rhinitis.
  • Skin tests help to identify specific allergen. They are prick, scratch and intradermal tests.
  • Radioallergosorbent test (RAST) measures specific IgE antibody concentration in the patient's serum.
  • Nasal provocation test.
Complications:

- Recurrent sinusitis
- Nasal polypi.
- Serous otitis media.
- Orthodontic problems and other ill-effects of prolonged mouth breathing especially in children.
- Bronchial asthma. Patients of nasal allergy have four times more risk of developing bronchial asthma.
• Avoidance of allergen
• Treatment with drugs
• Immunotherapy
• Antihistamines.
• Sympathomimetic drugs (oral or topical).
• Corticosteroids. [Oral and Topical]
• Sodium chromoglycate
• **Immunotherapy**: Immunotherapy or hyposensitisation is used when treatment fails to control symptoms or produces intolerable side effects.
• Allergen is given in gradually increasing doses till the maintenance dose is reached.
• Immunotherapy suppresses the formation of IgE.
• It also raises the titre of specific Ig antibody.
• Immunotherapy has to be given for a year or so before significant improvement of symptoms can be noticed. It is discontinued if uninterrupted treatment for 3 years shows no clinical improvement.
Vasomotor rhinitis: Non allergic rhinitis

• It is non – allergic rhinitis but clinically simulating nasal allergy with symptoms of nasal obstruction, rhinorrhea and sneezing.

• One or the other of these symptoms may predominate. The condition usually persists throughout the year and all the tests of nasal allergy are negative.
Pathogenesis

• Imbalance between sympathetic and parasympathetic system.
• So, emotions play a significant role.
• Paroxysmal sneezing - Bouts of sneezing start just after getting out of the bed in the morning.
• Excessive rhinorrhoea - This accompanies sneezing or this may be the only predominant symptom. It is profuse and watery and may even wet several handkerchiefs.
• The nose may drip when the patient leans forward, and this may need to be differentiated from CSF rhinorrhoea.
• Nasal obstruction: This alternates from side to side.
• Usually more marked at night. It is the dependent side of nose which is often blocked when lying on one side.
• Post nasal drip.
• **Treatment**

• **Medical:**
  • Antihistaminics and oral nasal decongestants
  • Topical steroids (e.g. beclomethasone dipropionate, budesonide or fluticasone), used as spray or aerosol, are useful to control symptoms.
  • Systemic steroids can be given for a short time in very severe cases.

• **Surgical**
  • Reduction of turbinates and
  • Vidian neurectomy
Rhinitis medicamentosa

- Due to excessive use of topical decongestants which cause rebound on withdrawal.
- Treatment
  1. Stop topical decongestants.
  2. Short course of systemic steroids.

Drug induced rhinitis

- Sympathetic blockers: Reserpine, Guanethidine, propanolol
- Cholinergic drugs like neostigmine (anti-cholinesterase)
Sludders neuralgia
Nasal polyp

• What is polyp?
• Is it benign or malignant?
• Types ?
Antrochoanal polyp

- Origin: maxillary antrum
- Single/Unilateral
- It grows into the nasal cavity reaching nasopharynx
- Young age
- Male predominant
- Etiology: infectious
Clinical features

• Nasal obstruction
• Hyponasal voice
• Mucoid discharge from the nose
• Eustachian tube block leading to CHL
• o/e polyp visible on anterior rhinoscopy and posterior rhino scopy as a greyish white mass
Treatment

• Role of medical therapy ???/
• Surgery :
  1] Avulsion of polyp
  2] Intranasal polypectomy
  3] Caldwell luc operation
  4] Functional endoscopic sinus surgery
Ethmoidal polyp

- Origin: Ethmoidal air cells
- Age: adults
- Etiology: allergic origin or diseases of ciliary motility
- Associated syndromes:
  1. chronic rhinosinusitis
  2. asthma
  3. cystic fibrosis
  4. kartagener syndrome: Bronchiectasis, sinusitis, situs inversus.
  5. young syndrome: Sinopulmonary syndrome and azoospermia.
  6. churg strauss syndrome: Asthma, fever, eosinophilia, vasculitis, granuloma
  7. aspirin intolerance: Sampters triad: Nasal polypi, asthma, aspirin intolerance.
• Sampters triad:
  • Asthma
  • Aspirin insensitivity
  • Nasal polyposis
Symptoms:
1] Bilateral nasal obstruction
2] Nasal discharge
3] Excessive sneezing
4] Anosmia
5] Snoring
6] Headache

Examination:

Multiple sessile pedunculated
Bunch of grapes appearance
Insensitive to touch and bleeding
Treatment

• Conservative :
  • 1] antihistamine
  • 2] steroids
  • 3] nasal decongestants

• Surgery :
  • FESS:
    • Intranasal ethmoidectomy
    • ExTRANASAL ethmoidectomy
<table>
<thead>
<tr>
<th></th>
<th>AC pollyp</th>
<th>Ethmoidal polyp</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td></td>
<td></td>
</tr>
<tr>
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<td></td>
<td></td>
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<td>Recurrence</td>
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Foreign body in nose

• Inanimate /animate
• Age – toddlers
• Clinical features :
  • Unilateral foul smelling discharge
  • Nasal obstruction
  • Vestibulitis
  • Epistaxis
• Treatment – removal under L/A or G/A
Rhinolith

- Mineralized foreign body in the nose
- Nucleus: FB or blood clot surrounding deposits and calcium and magnesium
- Age: adults
- Unilateral nasal obstruction and discharge
Nasal Myiasis

- Etiology: Ova of chrysosmia species in nose seen in atrophic rhinitis
- Most commonly seen from August to October.

- Clinical features:
  - Intense irritation
  - Itching
  - Sneezing headache
  - Bleeding from nose
  - Foul smelling nasal discharge
Complications:
1. Destruction of nose, paranasal sinuses, soft tissue of face, palate.
2. Fistula between nose and palate.
3. Death due to meningitis

Treatment:
1. Instillation of chloroform into nose followed by removal of maggots with forceps.
2. Isolate patient with mosquito nets.
Atrophic Rhinitis

- Chronic inflammation of nose characterised by atrophy of nasal mucosa and turbinate bones.
- The nasal cavities are roomy and full of foul smelling crusts.
Types of atrophic rhinitis

• Primary atrophic rhinitis:
  Exact cause is not known

• Secondary atrophic rhinitis:
  Secondary to infections like syphilis, lupus, leprosy, rhinoscleroma, radiotherapy to nose or excessive surgical removal of turbinates or severe deviated septum
SECONDARY ATROPHIC RHINITIS

3. Severe deviated nasal septum
Primary atrophic rhinitis: Cause

- Cause is not known. Various theories have been put forward:
  - 1. Hereditary: More than one member in the family are affected.
  - 2. Endocrinological: The disease starts at puberty, involves females more than males, disease tends to subside after menopause.
  - 4. Infective: Klebsiella Ozaenae (Perez bacillus), E.Coli, Diptheroids. But they are all secondary invaders responsible for foul smell rather than primary causative organism.
Clinical features

• **Merciful anosmia:** Patient himself is unaware of the smell due to marked anosmia.

• Patient may complain of **nasal obstruction** in spite of unduly wide nasal chambers.
Examination findings

• Nose: Filled with crusts.
  Roomy
  Atrophic pale mucosa.
  Small turbinates
• Paranasal sinuses: Small with thick walls
• Atrophic pharyngitis
• Atrophic laryngitis
Medical management

• Nasal irrigation with: Normal saline or an alkaline solution made by dissolving powder with sodium bicarbonate 1 part, sodium bborate 1 part, sodium chloride 2 parts in 280 ml of water.

• 25 % glucose in glycerine.

• Local antibiotics: Kemicetine antiozaena solution (chlormycetin, oestradiol, vitamin D2)
• Oestradiol spray: increases vascularity
• Placental extract submucosal injection
• Systemic streptomycin
• Potassium iodide.
Surgical

• Youngs operation: Mucosal flaps are elevated to close nasal cavity
• Narrowing of nasal cavity:
  • 1. Submucosal injection of teflon.
  • 2. Section and medial displacement of lateral wall of nose.
  • 3. Insertion of fat, cartilage, bone under mucoperiosteum of floor and lateral wall of nose.
ANATOMY OF PHARYNX
- Nasopharynx
- Oropharynx
- Hypopharynx

**Division of Pharynx**

- The nasal part – **NASOPHARYNX/EPIPHARYNX**
  (extends from base of skull to soft palate)

- The oral part – **OROPHARYNX**
  (extends from hard palate to hyoid bone)

- The laryngeal part – **LARYNGOPHARYNX/HYPOPHARYNX**
  (extends from upper border of epiglottis to lower border of cricoid cartilage)
Nasopharynx

- Base of skull
- Choanae
- Eustachian tube opening
- Pharyngeal recess (Fossa of Rosenmüller)
- Retropharyngeal space

Nasal septum
Section 1 The Oral Cavity

Consists of two parts
- Oral vestibule: between cheeks and lip and teeth
- Oral cavity proper: within arch of teeth

Oral vestibule leads, by the space behind the molar teeth, into the oral cavity proper

Boundaries
- Anterior and lateral: gum and teeth
- Posterior: oropharyngeal isthmus
- Roof: palate
- Floor: tongue, muscles and mucous membrane
Oropharynx
OROPHARYNX

It is the middle part of the pharynx situated behind the oral cavity.
Functions of oropharynx

- 1] Conduit for air and food
- 2] Helps in speech
- 3] Taste
- 4] Immunity and local defense
Hypopharynx

• It lies opposite 3rd, 4th, 5th, and 6th cervical vertebra
• It lies behind and on sides of larynx
Hypopharynx – subdivisions

- Pyriform fossa
- Post cricoid region
- Posterior pharyngeal wall
Applied anatomy

• Internal laryngeal nerve runs submucosally in the lateral wall of pyriform sinus hence through this nerve pain is referred to ear

• Post cricoid region is a common site for carcinoma in females suffering from Plummer vinson syndrome
Functions

1] Common pathway for air and food
2] Provides resonance for speech
3] Helps in deglutition
Structure of pharyngeal wall

- Mucous membrane
- Pharyngobasilar fascia
- Muscular coat
  - External layer – Superior, Middle and inferior constrictor
  - Internal layer – Stylopharyngeus, Salpingopharyngeus, Palatopharyngeus
- Buccopharyngeal fascia
Killians Dehiscence
WALDEYER'S RING

An interrupted circle of protective lymphoid tissue at the upper ends of the respiratory and alimentary tracts

- **Pharyngeal tonsil (adenoid)**
- **Tubal tonsil**
- **Palatine tonsil**
- **Lingual tonsil**

**Upper midline in nasopharynx**

**Around openings of auditory tube**

**Either side of oropharynx**

**Under mucosa of posterior third of tongue**
Adenoids
Location

• Location:
  
  • Also called nasopharyngeal tonsils, as they are situated at the junction of the roof and posterior wall of nasopharynx.
# Tonsils vs Adenoids

<table>
<thead>
<tr>
<th></th>
<th>Adenoids</th>
<th>Tonsils</th>
</tr>
</thead>
<tbody>
<tr>
<td>Location</td>
<td>Nasopharynx</td>
<td>Oropharynx</td>
</tr>
<tr>
<td>Number</td>
<td>Single</td>
<td>Paired (two)</td>
</tr>
<tr>
<td>Lining epithelium</td>
<td>Ciliated columnar</td>
<td>Stratified squamous</td>
</tr>
<tr>
<td>Capsule</td>
<td>Absent</td>
<td>Present on lat surface</td>
</tr>
<tr>
<td>Crypts</td>
<td>Absent</td>
<td>Present on medial surface</td>
</tr>
<tr>
<td>Growth</td>
<td>1. Present at Birth</td>
<td>Persist through out life</td>
</tr>
<tr>
<td></td>
<td>2. <strong>Enlarges until 6 years of age.</strong></td>
<td></td>
</tr>
<tr>
<td></td>
<td>3. Atrophys at puberty and <strong>completely disappears by 20 years of age.</strong></td>
<td></td>
</tr>
</tbody>
</table>
Symptoms: Ear, Nose and throat are involved

Nasal obstruction and Resultant sinusitis

Mouth breathing because of Nasal obstruction

Obstructs Eustachian Tube.
Causes Serous otitis Media, ASOM,CSOM
Adenoid facies

1. Elongated dull long expressionless face.
2. Pinched in nose due to disuse atrophy of alae nasi muscles.
3. Open mouth and drooping lower jaw giving face an elongated appearance.
4. Hitched up upper lip
5. Crowded upper teeth
6. High arched palate as the moulding effect of tongue on palate is lost.
Adenoid facies

- 1. Elongated dull long expressionless face.
- 2. Pinched in nose due to disuse atrophy of alae nasi muscles.
- 3. Open mouth and drooping lower jaw giving face an elongated appearance.
- 4. Retracted jaw.
- 4. Hitched up upper lip
- 5. Crowded upper teeth
- 6. High arched palate as the moulding effect of tongue on palate is lost.
Treatment

• Mild symptoms: Conservative with breathing exercises, decongestants, antihistaminics.
• Moderate to severe symptoms: Surgery.
Tonsills: Location

- Location: Situated in oropharynx between anterior and posterior pillars.
- Anterior pillar is formed by palatoglossus
- Posterior pillar is formed by palatopharyngeus
- Bed of tonsil: Superior constrictor and styloglossus muscle.
Bed of tonsil

- Bed of tonsil: Superior constrictor and styloglossus muscle.
Blood supply

1. Tonsillar branch of facial artery is the main artery.
2. Ascending pharyngeal
3. Ascending palatine.
4. Dorsal linguae branches of lingual artery
5. Descending palatine branch of maxillary artery.
Lymphatic supply of tonsils

• Upper deep cervical nodes particularly the jugulodigastric node situated below the angle of mandible.
Tonsillitis

• Aetiology: Hemolytic streptococcus is the most common infecting organism

• Age: Most commonly affects school going children but can affect adults also.

• Rare in infants and after the age of 50 years
Types

• 1. Acute superficial tonsillitis.
• 2. Acute follicular tonsillitis.
• 3. Acute parenchymal tonsillitis.
• 4. Acute membranous tonsillitis.
Which type?

- Here tonsils are congested along with oral mucosa
Which type?

- Prominent follicles
Which type?

- Whitish membrane over tonsil.
Which type?

- Tonsil is congested and enlarged
Symptoms

• Fever: High or low?
• Sore throat
• Difficulty in swallowing
• Ear ache ... Why?
Signs

- Throat redness with hyperemia of pillars, soft palate and uvula.
- Jugulodigastric lymphnodes are enlarged.
Treatment

• Bed rest.
• Plenty of fluids.
• Analgesics
• Antibiotics:
  • DOC ???
  • Alternative DOC ???
• Duration of antibiotics
Complications

- Chronic tonsillitis
- Paratonsillar abscess
- Parapharyngeal abscess
- Cervical abscess due to suppuration of jugulodigastric lymphnodes.
- Acute otitis media
- Rheumatic fever
- Acute glomerulonephritis
- Subacute bacterial endocarditis
Chronic tonsillitis

- Etiology –
  - Chronic infection in the teeth or sinus may be a predisposing factor
  - Subclinical infection of the tonsil

- c/f – recurrent attacks of sore throat,
  - Cough with throat irritation
  - Bad taste in mouth
  - Thick speech with swallowing difficulty
• Types
  • Chr follicular
  • Chr parenchymatous
  • Chr fibroid

• Clinical feature
  • Enlarged bt yellow pus+
  • Enlarged
  • Small but on applying pressure there is pus
Membrane over tonsil

- Membranous tonsillitis
- 2. Diphtheria.
- 3. Vincent's angina
- 4. Infectious mononucleosis.
- 5. Agranulocytosis.
- 7. Aphthous ulcers.
- 8. Malignancy tonsil
- 9. Traumatic ulcer
- 10. Candidial infection
Tonsillectomy indications

- Absolute
  - Huge tonsils causing oropharyngeal obstruction
  - Malignancy of tonsil

- Relative indications
  - Recurrent tonsillitis
  - 2nd attack of quinsy
  - Chronic tonsillitis
  - Tonsillitis causing febrile seizures, cardiac disease, IGA nephropathy

- Non tonsillar indication – excision of styloid process and UPPP
Contraindications for tonsillectomy

- Polio epidemic
- Submucous cleft palate
- Acute tonsillar infection
- Age <3 years
- Recent acute upper respiratory tract infection
Ludwig's Angina
• **Etiology**:
• Dental infections
• Submandibular sialadenitis
• Infections of the oral mucosa
• The symptoms include:
• pain or tenderness in the floor of your mouth, which is underneath your tongue.
• difficulty swallowing.
• drooling.
• problems with speech.
• neck pain.
• swelling of the neck.
• redness on the neck
• 1. Systemic antibiotics.
• 2. Incision and drainage of abscess.
  Intraoral: if the infection is localised to sublingual space.
  External— If infection involves submaxillary space.
• 3. Tracheostomy: If the airway is endangered
Juvenile Nasopharyngeal Angiofibroma
ANATOMY OF NASOPHARYNX

- It opens anteriorly into the nasal cavity.
Nasopharyngeal Angiofibroma

- **Is it benign or Malignant:**
  Benign but locally Invasive.

- **Is it common or uncommon:**
  Rare but most common benign tumor of nasopharynx.

- **Males or Females:**
  Males only. Never seen in Females.

Occurs in young males at the time of puberty – 2nd decade - probably testosterone dependent from a Hamartomatous Nidus.
Site of origin

Arises in posterior nasal cavity, near superior border of sphenopalatine foramen
Other sites of origin

1. Anterior wall of sphenoid sinus.
2. Roof of nasopharynx
3. Lateral wall of nasopharynx
Pathology

Endothelium lined blood vessels
Without muscularis layer

1. No Vasoconstriction
2. No effect of adrenaline
3. Recurrent Unprovoked profuse Epistaxis
4. No biopsy

Benign and Non-Encapsulated:
Locally invasive
Extension and Clinical Symptoms

1. Nasal Obstruction
2. Widening of Nasal Bridge
3. Swelling of Cheek
4. Proptosis

Frog Face Deformity

- 2. Bulge in Palate
- 3. Serous Otitis Media
- 4. Cranial signs and cavernous sinus
Clinical Features

- Age: Pubertal (Second Decade).
- Sex: Exclusively Males
- Why? Testosterone Dependant.
Clinical features

<table>
<thead>
<tr>
<th>Bleeding</th>
<th>Obstructive</th>
<th>Cranial spread</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Profuse recurrent epistaxis. [Anaemia]</td>
<td>• Nasal obstruction and hyponasal speech.</td>
<td>• Cranial involvement. (2,3,4,5,6 nerves palsy)</td>
</tr>
<tr>
<td></td>
<td>• ET tube block-serous otitis media and Conductive hearing loss.</td>
<td></td>
</tr>
<tr>
<td></td>
<td>• Frog face deformity</td>
<td></td>
</tr>
</tbody>
</table>
Frog Face Deformity

1. Nasal Obstruction
2. Widening of Nasal Bridge
3. Swelling of Cheek
4. Proptosis
Clinical Examination

• Anterior rhinoscopy - Globular pink mass post part of nasal cavity, septum being pushed to opposite side.

• Post nasal examination – Pink to purple globular mass with dilated blood vessel.

• Probe test – Contraindicated.

• Digital palapation of tumour is contraindicated.
Evaluation

• History : Main stay
• Examination :
• X-Ray neck lateral view.
• X-Ray PNS
• CT scan.
• MRI
• Angiography
• NO BIOPSY
Plain X-Ray (Limited Role)

1. Opacification of sphenoid sinus.

2. Holmann-Miller sign:
   Anterior Bowing of Posterior wall of
   maxillary sinus
Role of CT Scan: CECT is investigation of choice

- CT better delineates bony details of the skull base, including bony erosion, in particular, the depth of invasion into the bone of the sphenoid sinus, a main predictor of recurrence.

- CT diagnosis is based on two constant features which are:
  - 1. mass in the nose and pterygopalatine fossa;
  - 2. erosion of bone behind the sphenopalatine foramen at the root of the pterygoid plate and clinical findings.
Role of MRI

• MRI is crucial for highlighting soft tissue elements of the tumor and assesses the relation of the tumor to critical structures such as the internal carotid artery, cavernous sinus, and pituitary gland.

• Recurrence and residual tumors are best appreciated on MRI.
Role of angiography

• Confirmation of the JNA diagnosis is usually provided by angiography,
• It is also used as treatment with embolization.
• Angiography provides information on the specific blood supply of the tumor.
Blood Supply

- The **internal maxillary artery** is the most common vascular source from which JNAs arise.

Other known vessels include:
- Ascending pharyngeal artery
- External, internal, and common carotid arteries.

- JNAs have also been known to develop from blood supply contralateral to the side they form
When is biopsy done

• No Biopsy for JNA
• Exception – only when Radiotherapy is contemplated
Treatment

• Treatment of choice for JNA is Surgery with Pre-operative embolisation.

• To decrease vascularity:
  • 1. Embolisation 24-48 hours before surgery.
  • 2. A course of estrogen therapy [stilbestrol 2.5 mg thrice daily for 3 weeks]
  • 3. Cryotherapy
• The surgical approaches can be:

• **Inferior approaches** include transpalatal and transoral-transpharyngeal routes.

• **Anterior approaches** include transnasal, Le Fort I maxillotomy, medial maxillectomy and maxillary swing depending on the location.

• **Lateral approach** includes the infratemporal fossa approach
Role of radiotherapy

• Indications:
  • 1. Intracranial involvement.
  • 2. Direct blood supply from internal carotid artery
  • 3. Unresectable disease
  • 4. Multiple recurrences may be good candidates for radiation treatment

• Recurrence rate of 20–30% can be expected with radiation treatment alone.

• Newer techniques in radiotherapy treatment such as intense-modulated conformal radiotherapy (IMRT) and gamma knife have shown good results for management of JNA.
Hormonal therapy and chemotherapy

• Diethylstilbesterol with flutamide because it is testosterone dependant.

• Chemo for residual /recurrent disease – doxorubicin, vincristine and dacarbazine
Summary

• Most common benign tumour of nasopharynx.
• Locally aggressive.
• Extremely vascular - no muscle layer in the blood vessel.
• Origin – Sphenopalatine foramen.
• Pubertal male.
• Testosterone dependent.
• Recurrent unprovoked profuse epistaxis.
• Nasal obstruction and hyponasal speech.
• Conductive hearing loss with serous OME.
• Proptosis, broad nasal bridge, swelling of cheek, frog face deformity
• Involvement of 2,3,4,5,6 cranial nerves
• CT-Scan/X-ray: Holmann Miller sign, Antral sign
• Carotid angiography to know the vascularity of the tumour.
• Biopsy contraindicated.
• Pre-op embolization and hormone therapy.
• Treatment of choice – surgery.
A 14 year old male child has unilateral nasal obstruction, epistaxis, swelling over cheek. The diagnosis is ??

1. Nasal polyp
2. NPC
3. Angiofibroma
4. Foreign body
Nasopharyngeal Cancer
Aetiology

1. Chinese have a higher genetic susceptibility. In India it is common in North-east regions where people are predominantly of Mongoloid origin.

2. Ebstein Barr virus.

3. Environmental: Smoking of tobacco, opium Nitrosamines from dry salted fish Smoke from burning of incense sticks
• Most common Histopathological type: Squamous cell carcinoma
• Commonest presentation: Cervical Lymphadenopathy because of rich lymphatics.
• MC lymphnodes involved: Lymph nodes between angle of jaw and mastoid and lymph nodes along spinal accessory in post triangle of neck.
• Males are three times more commonly affected.
Extension and Clinical Symptoms

1. Nasal Obstruction
2. Epistaxis
3. Proptosis

4. Cranial signs and cavernous sinus
3. Serous Otitis Media
2. Bulge in Palate
Cranial nerves involvement

• Nearly all the cranial nerves can be involved.
• Horner's syndrome can also occur due to involvement of sympathetic chain.
• Jugular foramen syndrome: due to involvement of IX, X, XI nerves in jugular foramen.
Unilateral serous otitis media

• Presence of unilateral serous otitis media in an adult should raise suspicion of nasopharyngeal carcinoma.
• Presence of unilateral serous otitis media in a teenage boy should raise suspicion of nasopharyngeal angiofibroma.
What is Trotter's Triad?
• Unilateral conductive hearing loss
• Ipsilateral earache & facial pain
• Ipsilateral immobilization of the soft palate

Trotters triad:
• 1. Conductive deafness due to block of eustachian tube.
• 2. Ipsilateral temporoparietal neuralgia due to Vth nerve involvement.
• 3. Palatal paralysis (Xth nerve involvement.)
Lymphatic spread

- MC lymphnodes involved: Lymph nodes between angle of jaw and mastoid and lymph nodes along spinal accessory in post triangle of neck in HO’s Triangle
• Treatment of choice for NPC: Irradiation. Supervoltage therapy delivering 6000-7000 rads.
Common sites of malignancy in the oropharynx are

1. Base (posterior 1/3 rd of tongue)
2. Tonsil and tonsillar fossa.
3. Palatine arch i.e. soft palate and anterior pillar
4. Posterior and lateral paharyngeal wall.
• Oro-pharyngeal tumors (Mainly Base of tongue tumors) are asymptomatic in initial phase. So these areas should be thoroughly examined in patients with metastases with unknown primary.
• Most common histo-pathological type: Squamous cell carcinoma.
• Oro-pharyngeal carcinomas of adenocarcinoma variant arise from Minor salivary glands.
• Most common lymph node involved in oro-pharyngeal tumors are Jugulo-digastric LN.
• Palatine arch tumors are superficially spreading, rarely undergo metastases and have good prognosis.

• Base of tongue and tonsil tumors are ulcerative or infiltrative, undergo early metastases and have bad prognosis.

• Commando operation: Hemimandibulectomy and neck dissection
Styalgia (Eagles syndrome)

- Elongated styloid process.
- Pain in tonsillar fossa and neck which radiates to ipsilateral ear.
- Diagnosis: trans oral palaption and X-ray.
- Treatment: Excision if symptomatic.
Tumors of hypopharynx
<table>
<thead>
<tr>
<th></th>
<th>Carcinoma pyriform sinus</th>
<th>Carcinoma Post-Cricoid</th>
<th>Carcinoma Post-Pharyngeal wall</th>
</tr>
</thead>
<tbody>
<tr>
<td>Incidence</td>
<td>Most common</td>
<td>Intermediate</td>
<td>Least Common</td>
</tr>
<tr>
<td>Gross feature</td>
<td>Ulcerative or exophytic</td>
<td>ulcerative</td>
<td>Exophytic</td>
</tr>
<tr>
<td>Lymphatic spread</td>
<td>Early because of rich lymphatic supply</td>
<td>Intermediate</td>
<td>Intermediate</td>
</tr>
<tr>
<td>Lymph nodes involved</td>
<td>Upper and middle deep cervical LN</td>
<td>Para tracheal and mediastinal Lymph nodes</td>
<td>Retropharyngeal</td>
</tr>
<tr>
<td>Voice change</td>
<td></td>
<td>Most common as it involves recurrent laryngeal nerve at cricoarytenoid joint</td>
<td></td>
</tr>
<tr>
<td>Dysphagia</td>
<td>seen</td>
<td>seen</td>
<td>seen</td>
</tr>
<tr>
<td>Skip lesions</td>
<td></td>
<td>Common when oesophagus is involved and cells spread along submucous lymphatics</td>
<td></td>
</tr>
</tbody>
</table>
## Tumors of Hypopharynx

<table>
<thead>
<tr>
<th></th>
<th>Carcinoma pyriform sinus</th>
<th>Carcinoma Post-cricoid</th>
<th>Carcinoma posterior pharyngeal wall</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Prognosis</strong></td>
<td>Bad because of early lymphatic spread</td>
<td>Bad because of skip lesions</td>
<td>Intermediate</td>
</tr>
</tbody>
</table>

Post-Cricoid tumors are a part of Plummer Vincent syndrome.

Treatment for early lesions without lymph node involvement: radiotherapy.
Treatment for advanced lesions with lymph node involvement: Surgery.
Abscesses in relation to pharynx
Anatomy of peritonsillar space

- Bed of tonsil is formed by ???

- Peritonsillar space is between capsule of tonsil and superior constrictor muscle
Peritonsillar abscess or quinsy

Acute tonsillitis

Crypta magna gets infected and sealed off

Infection from crypta magna ruptures into tonsill causing intratonsillar abscess

Intratonsillar abscess bursts through capsule and spreads to peritonsillar space causing peritonsillar abscess or quinsy
Symptoms

• Age:  
• General: They are due to septicemia and patient has high grade fever
• Local:
  • 1. Severe unilateral throat pain
  • 2. Severe odynophagia leading to dehydration
  • 3. Muffled and thick voice often called “Hot potato voice”
  • 4. Foul breath
  • 5. Trismus due to spasm of pterygoid muscles which are in close proximity to superior constrictor
Signs

- Congested pillars engulfing tonsils.
- Uvula is swollen, oedematous and pushed to opposite side.
- Mucopuss over tonsil
- Jodulodgastric nodes are enlarged.
- Torticollis: Patient keeps the neck tilted to the side of abscess
Treatment

• IV fluids to combat dehydration
• Antibiotics
• Analgesics: like PCM, pethidine. Avoid aspirin as it can cause bleeding
Surgical treatment

• Option 1: Incision and drainage of abscess followed by interval tonsillectomy (4-6 weeks after an attack of quinsy)

• Option 2: Hot tonsillectomy. This is tonsillectomy during quinsy. This has the risk of rupture of abscess during anaesthesia and excessive bleeding at the time of operation.
Anatomy of retropharyngeal and prevertebral space

1. Location
2. Horizontal extent
3. Contents
4. Source of infection
Anatomy of retropharyngeal and prevertebral space
<table>
<thead>
<tr>
<th>Location</th>
<th>Retropharyngeal space</th>
<th>Prevertebral space</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Between buccopharyngeal fascia covering constrictors of pharynx and prevertebral fascia</td>
<td>Between vertebral fascia and vertebrae.</td>
</tr>
<tr>
<td>Vertical extension</td>
<td>From base of skull to bifurcation of trachea (T4) in mediastinum.</td>
<td>From base of skull to coccyx</td>
</tr>
<tr>
<td>Horizontal</td>
<td>Divided into two spaces (spaces of gillette) which do not communicate across midline</td>
<td>Single cavity across midline</td>
</tr>
<tr>
<td>Contents</td>
<td>Retropharyngeal nodes which disappear by 4-5 years.</td>
<td>Nil</td>
</tr>
<tr>
<td>Usual source of infection</td>
<td>Infection of lymph nodes. As lymph nodes disappear with age, this infection is common in children</td>
<td>Caries of spine which is usually tubercular</td>
</tr>
<tr>
<td>Aetiology and Lymph nodes involved</td>
<td>Retropharyngeal abscess</td>
<td>Retrovertebral abscess</td>
</tr>
<tr>
<td>-----------------------------------</td>
<td>-------------------------</td>
<td>-----------------------</td>
</tr>
<tr>
<td>Suppuration of retropharyngeal lymphnodes secondary to acute infections of adenoids, nose, paranasal sinuses.</td>
<td></td>
<td>Secondary to caries of spine</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Posterior pharyngeal wall bulge</th>
<th>Retropharyngeal abscess</th>
<th>Retrovertebral abscess</th>
</tr>
</thead>
<tbody>
<tr>
<td>Unilateral and present to one side</td>
<td></td>
<td>Central midline bulge</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Downward extension</th>
<th>Retropharyngeal abscess</th>
<th>Retrovertebral abscess</th>
</tr>
</thead>
<tbody>
<tr>
<td>Downwards it can extend down behind the oesophagus in to mediastinum</td>
<td></td>
<td>This space extends from the base of skull to coccyx and so can extend down up to coccyx</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Incision and drainage</th>
<th>Retropharyngeal abscess</th>
<th>Retrovertebral abscess</th>
</tr>
</thead>
<tbody>
<tr>
<td>A vertical incision is given in the most fluctuant area of the abscess, by transoral route.</td>
<td></td>
<td>It can be done through a vertical incision along the anterior border of sternocleidomastoid (for low abscess) and along its posterior border (for high abscess)</td>
</tr>
</tbody>
</table>
• In acute retropharyngeal infections, incision and drainage is usually done without anaesthesia as there is risk of rupture during intubation.
Pharyngeal pouch

- **Synonyms:** Zenkers diverticulum

- It is a **pulsion** diverticulum where pharyngeal mucosa herniates through **Killian’s dehiscence** - a weak area between two parts of the inferior constrictor.

- **Pathology:** Herniation of poch starts in the midline. It is at first behind the oesophagus and then comes to lie on its left. **Mouth of the sac is wider than the opening of oesophagus** and food preferentially enters the sac.
Clinical features

1. Initial few swallows, food enters the “wide mouthed” diverticulum preferentially. So patient won’t be having dysphagia but food does not enter stomach.

2. After few swallows - pouch gets filled. Further swallows produces gurgling sound.

3. After few swallows when pouch gets fully filled, it presses on oesophagus causing dysphagia.

4. Food may regurgitate at night during sleep - cough
• Diagnosis: Barium swallow.
• Treatment:
• 1. Excision of pouch and cricopharyngeal myotomy through cervical approach.
• 2. Dolhman’s procedure: The partition wall between the oesophagus and the pouch is divided by diathermy through an endoscope. This is done in poor risk debilitated patients.
Anatomy of Larynx
• Lies opposite 3\textsuperscript{rd} to 6\textsuperscript{th} cervical vertebra

• Laryngeal cartilages
• Paired – Thyroid, Cricoid, Epiglottis
• Unpaired – Arytenoid, Corniculate, Cunneiform
THYROID CARTILAGE

ANTERIOR ASPECT
Cricoid cartilage
Epiglottis
Arytenoid cartilage
Corniculate and cuneiform cartilages
Membranes

• Extrinsic:
  1] thyrohyoid
  2] cricothyroid
  3] cricotracheal

Intrinsic
  1] cricovocal
  2] quadrangular membrane
Quadrangular membrane

- Greater cornu of hyoid
- Thyrohyoid membrane
- Opening for neurovascular bundle
- Quadrangular membrane
- Corniculate cartilage
- Muscular process of arytenoid cartilage
- Vocal process of arytenoid cartilage

Cricovocal membrane

- Cut surface of hyoid bone
- Epiglottis
- Thyroepiglottic ligament
- Cut surface of thyroid cartilage
- Vestibular ligament
- Vocal ligament
- Cricovocal membrane
- Cricoid cartilage
Muscles of larynx

Intrinsic muscles

- Acting on the vocal cord
  - ABDUCTOR – Posterior cricoarytenoid
  - ADDUCTOR
    1] Lateral cricoarytenoid
    2] Interarytenoid
    3] Thyroarytenoid
  - Tensor – Cricothyroid /Vocalis

- Acting on the laryngeal inlet
  - Opener
    - Thyroepiglottic
  - Closer
    - interarytenoid/aryepiglottic
Lymphatics

- Supraglottis – drains into upper deep cervical lymphnodes

Subglottis – prelaryngeal and pretracheal lymphnodes

Glottis-No lymphatics
Spaces of larynx

- Preepiglottic space of Boyer
- Paraglottic space
- Reinkes space
Pre-epiglottic Space of Boyer

- **Boundary** -
  - Antr.ly – Thyrohyoid Membrane
  - Hyoid bone
  - Postr.ly - Epiglottis
  - Supr.ly – Hyoepiglottic Ligament

- **Contents** – Fat, Areolar tissue, Lymphatics

- It is continuous laterally with Paraglottic space.
Paraglottic space

Paraglottic Space

- Bounded
  - Laterally – Thyroid Cartilage
  - Medially – Conus Elasticus
    - Quadrangular Membrane
  - Posteriorly – Mucosa of Pyriform Fossa

It encompasses the laryngeal ventricle & saccule
Functions of larynx

• Protection of lower airways
• Phonation
• Respiration
• Fixation of the chest
Acute and chronic inflammations of larynx
Three common inflammatory conditions of larynx

• 1. Acute laryngitis: Inflammation of larynx
• 2. Acute epiglottitis: Inflammation of epiglottis
• 3. Acute Laryngo-tracheo-bronchitis: Inflammation of larynx-trachea and bronchus.
Few important points to remember

• **Acute laryngitis** is caused due to **low grade** infections or vocal abuse and epiglottitis and laryngotracheobronchitis are due to rapidly progressive infections.

• **Epiglottitis** is due to bacterial infection (H.Influenza) and laryngotracheo-bronchitis is due to viral infections (parainfluenza).

• **Epiglottitis** and laryngotracheobronchitis are commonly seen in children.

• **Epiglottitis** and laryngotracheobronchitis are paediatric emergencies
• Chances of resp obstruction are higher in children because of smaller airways and also in rapidly progressive infections.
• Involvement of epiglottis alone does not cause cough.
• Voice is affected only if vocal cords are involved
• Racemic adrenaline and steroids are used to decrease edema.
<table>
<thead>
<tr>
<th>Structures involved</th>
<th>Acute laryngitis</th>
<th>Acute Epiglottitis</th>
<th>Laryngo-tracheo-bronchitis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Entire larynx</td>
<td>Only supraglottic structures: epiglottis, aryepiglottic folds and arytenoids. Vocal cords are spared</td>
<td>Larynx-trachea and bronchus</td>
<td></td>
</tr>
<tr>
<td>Cause</td>
<td>Infectious or non-infectious (vocal abuse)</td>
<td>Infectious</td>
<td>Infectious</td>
</tr>
<tr>
<td>Age</td>
<td>Adults and sometimes children</td>
<td>Children very common (2-7) years</td>
<td>Children (3 mon to 3 years)</td>
</tr>
<tr>
<td>Organisms</td>
<td>Strept.pneumoniae, H.influenza, streptococcus, S.aureus</td>
<td>H.Influenza B</td>
<td>Para influenza virus type I and II. Can be secondarily invaded by bacteria</td>
</tr>
<tr>
<td></td>
<td>Acute Laryngitis</td>
<td>Acute epiglottitis</td>
<td>Acute Laryngotracheo-Bронchitis</td>
</tr>
<tr>
<td>----------------------</td>
<td>------------------</td>
<td>--------------------</td>
<td>---------------------------------</td>
</tr>
<tr>
<td>Onset</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Progression</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Malaise, Fever</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sore throat</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cough</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hoarseness of voice</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Dyspnoea and Stridor</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Acute laryngitis</td>
<td>Acute Epiglottitis</td>
<td>Acute laryngo-tracheo-bronchitis</td>
</tr>
<tr>
<td>------------------</td>
<td>------------------</td>
<td>--------------------</td>
<td>---------------------------------</td>
</tr>
<tr>
<td>Examination</td>
<td></td>
<td>DO NOT EXAMINE IN</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>OPD. CHILD MAY HAVE</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>LARYNGEAL</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>OBSTRUCTION.</td>
<td></td>
</tr>
<tr>
<td>Hospitalisation</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Radiological features</td>
<td></td>
<td><strong>Thumb sign</strong> due</td>
<td><strong>Steeple sign</strong> on AP view</td>
</tr>
<tr>
<td></td>
<td></td>
<td>to swollen epiglottis in</td>
<td>of neck.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>lateral x-ray</td>
<td></td>
</tr>
<tr>
<td>Voice rest</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cough suppressants</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Analgesics</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Antibiotics</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Steroids</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Racemic adrenaline nebulisation or injection</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Intubation or tracheostomy</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hydration</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
• **Thumb sign** due to swollen epiglottis in lateral x-ray

• **Steeple sign**: Smooth constriction seen on AP view due to subglottic stenosis.
Polypoid degeneration of vocal cords (Reinke’s oedema)

- Bilateral symmetrical swelling of the whole of membranous part of vocal cords.
- Oedema of subepithelial space (Reinke’s space) of the vocal cords.
- Chronic irritation due to various causes.
- Also seen in myxoedema.
• Symptoms: Hoarseness of voice: common and the patient uses false cords for voice production which gives him a low pitched and rough voice.
• Examination: On indirect laryngoscopy, vocal cords appear as fusiform swellings with pale translucent look.
• Treatment:
  • 1. Voice rest and speech therapy
  • 2. Decortication of vocal cords: One cord first. Second cord after 3-4 weeks.
  • 3. Stop smoking
Tuberculosis of larynx

- Secondary to pulmonary tuberculosis
- Bronchogenic spread or hematogenous: Bacilli are carried by sputum and settle and penetrate the intact laryngeal mucosa.
Impairement of adduction of vocal cord and weakness of Voice: First sign

Pseudooedema: laryngeal mucosa appears pale red and swollen due to cellular infiltration

Posterior part of larynx affected: 1. Interarytenoid folds 2. ventricular bands 3. Vocal cords and 4. epiglottis are affected in order.

1. Swelling in the inter-arytenoid region giving a mamillated appearance

1. Ulceration of vocal cord giving mouse nibbled appearance.

2. Pseudoedema of epiglottis: turban epiglottis

Lupus of larynx

• Indolent tubercular infection associated with lupus of nose and pharynx.
• No pulmonary tuberculosis.
• Asymptomatic
• Affects anterior part of larynx, epiglottis is first involved
<table>
<thead>
<tr>
<th></th>
<th>TB larynx</th>
<th>Lupus Larynx</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Pulmonary TB</td>
<td>Yes</td>
<td>No</td>
</tr>
<tr>
<td>2. Activity of TB</td>
<td>Active</td>
<td>No</td>
</tr>
<tr>
<td>3. Parts affected</td>
<td>Posterior</td>
<td>Anterior</td>
</tr>
<tr>
<td>4. Symptomatic</td>
<td>Yes</td>
<td>No</td>
</tr>
</tbody>
</table>
Congenital lesions of larynx and stridor
Laryngomalacia

**Diagram:** Sagittal section through the larynx.
Laryngomalacia - inspiration and expiration
Laryngomalacia-supine and prone
Laryngomalacia- Omega shaped epiglottis
Subglottic stenosis
Laryngeal web
Subglottic hemangioma
<table>
<thead>
<tr>
<th></th>
<th>Laryngomalacia</th>
<th>Congenital subglottic stenosis</th>
<th>Laryngeal web</th>
<th>Subglottic hemangioma</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pathology</td>
<td>Excessive flaccidity of supraglottic larynx which is sucked in during inspiration</td>
<td>Abnormal thickening of cricoid cartilage or fibrous tissue below vocal cords</td>
<td>Due to incomplete recanalisation of larynx: web at the level of vocal cords</td>
<td>Hemangioma in subglottic region</td>
</tr>
<tr>
<td>Age</td>
<td>At birth</td>
<td>Asymptomatic</td>
<td>At birth</td>
<td>Asymptomatic at birth. By 3-6 months hemangioma grows in size and becomes symptomatic</td>
</tr>
<tr>
<td>Course</td>
<td>Usually appears by 2 years of age</td>
<td>Spontaneously resolves as larynx grows with age</td>
<td>Depends of thickness and extent of web</td>
<td>Depends on size but usually resolves with age</td>
</tr>
<tr>
<td></td>
<td>Laryngomalacia</td>
<td>Congenital subglottic stenosis</td>
<td>Laryngeal web</td>
<td>Subglottic hemangioma</td>
</tr>
<tr>
<td>--------------------------</td>
<td>----------------</td>
<td>-------------------------------</td>
<td>---------------</td>
<td>---------------------</td>
</tr>
<tr>
<td><strong>Stridor</strong></td>
<td>In inspiration</td>
<td>No stridor. Stridor is seen only after infection</td>
<td>Present</td>
<td>Present</td>
</tr>
<tr>
<td><strong>Affect of crying on stridor</strong></td>
<td>Increases</td>
<td>Normal</td>
<td>Increases</td>
<td>Increases as hemangioma increases in size during crying</td>
</tr>
<tr>
<td><strong>Prone</strong></td>
<td>Decreases</td>
<td>No effect</td>
<td>No effect</td>
<td>No effect</td>
</tr>
<tr>
<td><strong>Cry</strong></td>
<td>Normal</td>
<td>Normal</td>
<td>Weak cry or aphonia</td>
<td>Normal</td>
</tr>
<tr>
<td><strong>Direct laryngoscopy</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1. Omega shaped epiglottis</td>
<td></td>
<td>Subglottic diameter less than 4mm in full term neonate (normal 4.5-5.5mm)</td>
<td>Web is seen between the vocal cords and has a concave posterior margin</td>
<td>Reddish blue mass in subglottic region</td>
</tr>
<tr>
<td>2. Floppy aryepiglottic folds</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3. Prominent arytenoids</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Treatment</strong></td>
<td>Conservative</td>
<td>Conservative</td>
<td>Thin web: Cut with CO2 laser or knife. Thick web: Laryngofissure and placement of a silicon keel</td>
<td>Steroids may decrease the size of lesion. Majority of lesions involute with time. So do tracheostomy and observe.</td>
</tr>
</tbody>
</table>
- Laryngo-oesophageal cleft:
- Due to failure of fusion of cricoid lamina.
- Coughing, choking and cyanosis during feeding.
Stirdor

- Inspirator stridor: Supraglottis or pharynx.
- Biphasic stridor: lesions of glottis, subglottis and cervical trachea
- Expiratory stridor: Thoracic trachea, primary and secondary bronchus
Character of stridor

• Nasopharynx: Snoring or snorting sound.
• Pharyngeal cause: Gurgling sound and muffled voice
• Laryngeal cause: Hoarse cry or voice
• Stridor decreasing in prone: Laryngomalacia, micrognathia, macroglossia, innominate artery compression.

• If 3.5 mm bronchoscope can be passed intubation of child is possible.
Laryngeal paralysis
Causes

• Many
• Most common cause of paralysis is Bronchial carcinoma
• Left recurrent laryngeal nerve is most affected because of its long course.
• Thyroidectomy is also a common cause.
• Diabetes, syphilis, lead poisoning, viral infections.
Pearls
### Nerve supply of larynx

<table>
<thead>
<tr>
<th></th>
<th>Recurrent laryngeal nerve</th>
<th>Superior laryngeal nerve</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sensory supply</td>
<td>Inferior part of larynx</td>
<td>Superior part of larynx</td>
</tr>
<tr>
<td>Motor supply</td>
<td>All muscles of larynx except cricothyroid</td>
<td>Crico thyroid</td>
</tr>
<tr>
<td>Paralysis</td>
<td>Impaired abduction mainly.</td>
<td>Impaired tensor function</td>
</tr>
</tbody>
</table>

---

Superior Laryngeal Nerve

Recurrent laryngeal Nerve
All laryngeal muscles receive unilateral innervation except interarytenoid which receive bilateral innervation.
Phonation

During phonation vocal Cords adduct and become Tense.

In paralysis of cricothyroid, Vocal cords remain flaccid, Floppy and fail to adduct, Completely.

So voice is affected in Cricothyroid paralysis.
Inhalation

Deep inhalation requires, Vocal cords to abduct to let Air inside.

In RLN paralysis abduction is impaired
Position of vocal cords

Cricothyroid: Tensor and sec adductor: Supplied by SLN

Abductors: supplied by RLN

Cadaveric position: When both abductores and adductors are Paralysed. Vocal cords are 3.5mm from midline which is neutral position
Position of vocal cords

- Median
- Paramedian
- Intermediate (cadaveric)
- Slight abduction
- Full abduction
# Position of vocal cord in health & disease

<table>
<thead>
<tr>
<th>Position of cord</th>
<th>Location of cord from midline</th>
<th>Situations in health</th>
<th>Situations in disease</th>
</tr>
</thead>
<tbody>
<tr>
<td>Median</td>
<td>Midline</td>
<td>Phonation</td>
<td>RLN paralysis</td>
</tr>
<tr>
<td>Intermediate (cadaveric)</td>
<td>3.5 mm.  (This is neutral position of cricoarytenoid joint. Abduction and adduction takes place from this position.)</td>
<td>-</td>
<td>Combined paralysis (both RLN &amp; SLN)</td>
</tr>
<tr>
<td>Gentle abduction</td>
<td>7 mm</td>
<td>Quiet respiration</td>
<td>Paralysis of adductors</td>
</tr>
<tr>
<td>Full abduction</td>
<td>9.5 mm</td>
<td>Deep inspiration</td>
<td>-</td>
</tr>
<tr>
<td></td>
<td>RLN- Unilateral palsy</td>
<td>RLN- Bilateral palsy</td>
<td>SLN- Unilateral palsy</td>
</tr>
<tr>
<td>----------------</td>
<td>-----------------------</td>
<td>----------------------</td>
<td>-----------------------</td>
</tr>
<tr>
<td>Sensory loss</td>
<td>Ipsilateral anaesthesia of larynx below vocal cords.</td>
<td>Bilateral anaesthesia of larynx below vocal cords.</td>
<td>In SLN palsy, ipsilateral anaesthesia above vocal cords.</td>
</tr>
<tr>
<td>Motor loss</td>
<td>Ipsilateral paralysis of all intrinsic muscles except cricothyroid</td>
<td>Bilateral paralysis of all intrinsic muscles except cricothyroid</td>
<td>Ipsilateral paralysis of cricothyroid</td>
</tr>
<tr>
<td></td>
<td>Normal</td>
<td>RLN-Unilateral palsy</td>
<td>RLN-Bilateral palsy</td>
</tr>
<tr>
<td>--------------------------</td>
<td>--------------------------------------------</td>
<td>----------------------</td>
<td>---------------------</td>
</tr>
<tr>
<td>Vocal cord position</td>
<td>Normal position is gentle abduction as</td>
<td>Median or paramedian</td>
<td>Median or paramedian</td>
</tr>
<tr>
<td>during respiration</td>
<td>cricothyroids are inactive</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Phonation</td>
<td>Median</td>
<td>Paralysed cord is median. Normal cord becomes median. Voice is not much affected. Asymptomatic</td>
<td>Both cords are already in median position. So voice is not affected</td>
</tr>
<tr>
<td></td>
<td>Normal</td>
<td>RLN-Unilateral palsy</td>
<td>RLN-Bilateral palsy</td>
</tr>
<tr>
<td>------------------</td>
<td>-----------------</td>
<td>----------------------</td>
<td>---------------------</td>
</tr>
<tr>
<td>Deep inspiration</td>
<td>Full abduction</td>
<td>Paralysed cord in median position but normal cord fully abducts. So sufficient airway and no stridor</td>
<td>Both cords fail to abduct and remain in median position—insufficient airway—<strong>stridor</strong></td>
</tr>
<tr>
<td>Cough</td>
<td>Normal</td>
<td>Normal</td>
<td>Ineffective as cords fail to meet</td>
</tr>
<tr>
<td>Aspiration</td>
<td>Nil as cords approximate</td>
<td>nil</td>
<td>Common as cords fail to meet and ineffective cough</td>
</tr>
<tr>
<td></td>
<td>RLN-Unilateral palsy</td>
<td>RLN-Bilateral palsy</td>
<td>SLN-Unilateral palsy</td>
</tr>
<tr>
<td>----------------</td>
<td>----------------------</td>
<td>---------------------</td>
<td>----------------------</td>
</tr>
<tr>
<td>Treatment</td>
<td>Nil</td>
<td>Lateralisation</td>
<td>Epiglottopexy and tracheostomy</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Medialisation of vocal cord

- Injection of Teflon paste
- Muscle-cartilage implant
- Arthrodesis of cricoarytenoid joint
Lateralisation of vocal cord

- 1. Arytenoidectomy
- 2. Cordectomy
Benign tumors of larynx
Vocal nodule and polyp

Nodules  Polyp
<table>
<thead>
<tr>
<th></th>
<th>Vocal Nodules</th>
<th>Vocal polyp</th>
</tr>
</thead>
<tbody>
<tr>
<td>Alternate names</td>
<td>Singer or screamer’s nodule</td>
<td></td>
</tr>
<tr>
<td>Cause</td>
<td>Chronic Vocal abuse</td>
<td>Sudden Vocal abuse like shouting</td>
</tr>
<tr>
<td>Laterality</td>
<td>Bilateral and symmetrical</td>
<td>Unilateral</td>
</tr>
<tr>
<td>Location</td>
<td>Free edge of vocal cord at the junction of anterior 1/3 and posterior 2/3</td>
<td>Same</td>
</tr>
<tr>
<td></td>
<td>rd and posterior 2/3rds. This is the area of maximum vibration of vocal cord</td>
<td></td>
</tr>
<tr>
<td>Appearance</td>
<td>Reddish in early stages and white in chronic stage</td>
<td>Pedunculated polyp</td>
</tr>
<tr>
<td>Symptoms</td>
<td>Hoarseness and vocal fatigue</td>
<td>HOV and diplophonia (double voice due to different vibratory frequencies of two vocal cords.</td>
</tr>
<tr>
<td>Treatment</td>
<td>Initially voice rest. If it fails—Microscopic excision without injuring vocal ligament</td>
<td>Microscopic excision</td>
</tr>
</tbody>
</table>
Laryngocele

- It is an air filled cystic swelling due to dilatation of the saccule.
- Types:
  - Internal: confined within the larynx and presents as distension of false cord and aryepiglottic fold.
  - External: distended saccule herniates through the thyroid membrane and presents in neck.
  - Combined: Both internal and external components are seen.
• Cause: it is due to raised transglottic air pressure as in trumpet players, glass blowers or weight lifters.

• Symptoms: Hoarseness, cough and airway obstruction if large.

• Examination: Reducible swelling in neck which increases in size on coughing or performing valsala
• Treatment:
• 1. Surgical excision through a neck incision.
• 2. Marsupialisation by laryngoscopy, but chances of recurrence are there.
# Squamous papillomas

<table>
<thead>
<tr>
<th></th>
<th>Juvenile Papillomas</th>
<th>Adult-onset papillomas</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Age</strong></td>
<td>Infants and young children</td>
<td>30-50 years</td>
</tr>
<tr>
<td><strong>Sex</strong></td>
<td>Male twice more common</td>
<td>Male twice more common</td>
</tr>
<tr>
<td><strong>Number</strong></td>
<td>Multiple</td>
<td>Single</td>
</tr>
<tr>
<td><strong>Location</strong></td>
<td>True and false cords and epiglottis</td>
<td>Anterior half of vocal cord or anterior commisure.</td>
</tr>
<tr>
<td><strong>Aggressive</strong></td>
<td>Aggressive, recurrence is common after surgical excision</td>
<td>Less aggressive, does not recur after excision</td>
</tr>
<tr>
<td><strong>Affect of age</strong></td>
<td>Tend to disappear spontaneously after puberty</td>
<td></td>
</tr>
</tbody>
</table>
Laryngeal papillomas
• Symptoms: Hoarseness, stridor.
• Clinical examination: White, glistening, friable, bleeding growths.
• **Treatment:**
  2. Others: Cryotherapy, microelectrocautery.
• **Prevention of recurrence:** Interferon therapy.
Laryngeal Carcinoma
• Mostly affects males

• Aetiology:
  • 1. Benzopyrene in cigarette smoke.
  • 2. Alcohol
  • 3. Previous radiation
  • 4. Occupational exposure to asbestos, mustard gas, petroleum.

• Histopathology: Squamous cell carcinoma.
Types of Laryngeal carcinoma

- Supraglottic
- Glottic
- Subglottic
Pearls

1. Morphology

2. Lymphatic spread

3. Symptoms

4. HPE
<table>
<thead>
<tr>
<th>Incidence</th>
<th>Supraglottis</th>
<th>Glottis</th>
<th>Subglottis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Site</td>
<td>Most common in epiglottis</td>
<td>Most commonly at free edge and superior surface of anterior and middle 1/3 rd of vocal cord</td>
<td>Most common in midline in anterior half.</td>
</tr>
<tr>
<td>Appearance</td>
<td>Suprahyoid - Exophytic</td>
<td>Nodule or ulcer or thickening of vocal cord.</td>
<td>Submucosal Nodule</td>
</tr>
<tr>
<td>Vocal cord</td>
<td>Vocal cord involvement is very late. So hoarseness of voice is late.</td>
<td>Vocal cord involvement is early. So hoarseness of voice is early. But vocal cord fixation is very late. Vocal cord fixation indicates infiltration of thyroarytenoid muscle and is a very bad prognostic sign</td>
<td>Late</td>
</tr>
<tr>
<td></td>
<td>Supraglottis</td>
<td>Glottis</td>
<td>Subglottis</td>
</tr>
<tr>
<td>----------------</td>
<td>------------------------------------------------------------------------------</td>
<td>-------------------------------------------------------------------------</td>
<td>----------------------------------------------------------------------------</td>
</tr>
<tr>
<td>Metastases</td>
<td>Very early due to rich lymphatic supply</td>
<td>Never because of scant lymphatics in vocal cords</td>
<td>Early</td>
</tr>
<tr>
<td></td>
<td>Bilateral lymphnodes are involved in case of epiglottis carcinoma as it is midline.</td>
<td></td>
<td>Bilateral</td>
</tr>
<tr>
<td></td>
<td>Upper and middle deep cervical lymphnodes are involved</td>
<td></td>
<td>Lower deep cervical, pretracheal and para Tracheal</td>
</tr>
<tr>
<td>Symptoms</td>
<td>Silent until late.</td>
<td>Hoarseness of voice</td>
<td>Silent</td>
</tr>
<tr>
<td></td>
<td>Dysphagia, throat pain, pain in ear, respiratory distress are late features</td>
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</tr>
<tr>
<td>Nature</td>
<td>Anaplastic and highly malignant</td>
<td>Well differentiated and less malignant</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Supraglottis</td>
<td>Glottis</td>
<td>Subglottis</td>
</tr>
<tr>
<td>---------------------</td>
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<td>--------------------------------</td>
<td>--------------------------------</td>
</tr>
<tr>
<td><strong>prognosis</strong></td>
<td>Bad because</td>
<td>Good because</td>
<td></td>
</tr>
<tr>
<td></td>
<td>1. Symptoms are late</td>
<td>1. Presents early</td>
<td></td>
</tr>
<tr>
<td></td>
<td>as it is silent most</td>
<td>with HOV.</td>
<td></td>
</tr>
<tr>
<td></td>
<td>of the time</td>
<td>2. No lymphatic</td>
<td></td>
</tr>
<tr>
<td></td>
<td>2. Early lymphatic</td>
<td>metastases.</td>
<td></td>
</tr>
<tr>
<td></td>
<td>metastases</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>because of rich</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>metastases</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>3. Anaplastic and</td>
<td>3. Well differentiated</td>
<td></td>
</tr>
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<td></td>
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<td></td>
<td></td>
</tr>
</tbody>
</table>
## Treatment of laryngeal carcinoma

<table>
<thead>
<tr>
<th>T1 carcinoma</th>
<th>Radiotherapy</th>
</tr>
</thead>
<tbody>
<tr>
<td>T2 carcinoma</td>
<td>Radiotherapy</td>
</tr>
<tr>
<td>T3 Carcinoma</td>
<td>Total laryngectomy</td>
</tr>
<tr>
<td>T4 Carcinoma</td>
<td>Total laryngectomy</td>
</tr>
<tr>
<td>More advanced T4</td>
<td>Combined</td>
</tr>
</tbody>
</table>

### Exceptions:
- T1 or T2 carcinoma with involvement of anterior commissure or arytenoids: Fronto-lateral partial laryngectomy

### Indications:
1. T3,T4
2. Cord fixation or bilateral cordal lesions
3. Invasion of thyroid or cricoid cartilage with perichondritis
4. Bilateral arytenoid involvement
5. Involvement of posterior commissure
6. Transglottic cancer
7. Cervical metastases

### Contraindication:
- Distant Metastases
Few important points

• Any patient in cancer age group having persistent or gradually increasing HOV for 3 weeks must have laryngeal examination to exclude cancer.

• Impairement of vocal cord mobility indicates deeper infiltration in to thyroarytenoid muscle, crico-arytenoid joint or invasion of recurrent laryngeal nerve.

• Toludine blue helps in differentiating carcinoma in situ and carcinoma from leukoplakia.
• Radiotherapy is curative in early vocal cord lesion with normal mobility in 90%.
• Disadvantages of total laryngectomy: Loss of voice and permanent tracheostome.
Vocal rehabilitation

• 1. Oesophageal speech.
• 2. Tracheo-oesophageal speech
• 3. Artificial Larynx:
  1. Electrolarynx
  2. Transoral pneumatic device
Oesophageal speech

- Air swallowed and slowly ejected from oesophagus into the pharynx
- Patient can speak 6-10 words before re-swallowing air
- Voice rough but loud and understandable
Tracheo-oesophageal speech

- Prosthesis: Bloom singer valve
- Looks like natural phonation
Electrolarynx

An external mechanical sound source is substituted for the larynx. Anatomic structures for articulation and resonance are usually unaltered.
MCQ

• Stridor occurring immediately following thyroid surgery may be due to:

• A. unilateral recurrent laryngeal nerve paralysis
• B. combined superior and recurrent laryngeal nerve paralysis in one side
• C. bilateral recurrent laryngeal nerve paralysis
• D. laryngomalacia
• E. none of the above
MCQ

• Stridor in an infant is most commonly due to-

  a. Diphtheria
  b. Acute epiglottitis
  c. Foreign body aspiration
  d. Laryngomalacia
Foreign bodies of air passages
Size of FB and Location

- A large foreign body will lodge in Supraglottis or glottis or subglottis?
Size of FB and Location

• A small foreign body will lodge in Supraglottis or glottis or subglottis?
• Metallic vs Vegetable FB? Which is more dangerous

• Vegetable bronchitis: Vegetables swell up and also cause congestion and oedema of tracheobronchial mucosa causing vegetable bronchitis.
Tracheal Foreign Body

- A smooth FB will move up and down the trachea with inspiration and expiration producing “Audible slap” and “palpatory thud.”
- Asthmatoid wheeze may also be present.
Bronchial FB is most likely to lodge in right or left?
Clinical features

1. Initial period of choking, gagging and coughing.
2. Symptomless period where the mucosa adapts to foreign body
3. Late symptoms: can cause cough, stridor, hoarseness of voice.
4. Stage of complications: Depends on extent of obstruction
Bronchial FB
Causing partial obstruction

- Allows air in and out.
- Only wheeze.
Causing complete obstruction

- Air doesn’t enter into lungs.
- Lung collapses causing atelectasis.
Causing one way obstruction like a valve

- Air goes out but doesn’t enter into lungs.
- Lung collapses causing atelectasis.
Causing one way obstruction like a valve

• In this case the FB is acting like a valve allowing air in to the lung but not out.
• Causes swelling of lung with air or Emphysema.
• Emphysematous bullae may rupture causing spontaneous pneumothorax
In summary the possible complications are:

- Wheeze
- Atelectasis
- Emphysema
- Spontaneous pneumothorax
- Vegetable bronchitis.
- Foreign body can get infected causing pneumonitis, bronchiectasis or lung abscess.
Investigations

- X-ray neck, thorax, abdomen
- Is X-ray indicated for radiolucent FB?
X-ray is indicated for radiolucent FB

• 1. radiolucent FB sometimes can also be seen as radiolucent shadow.
• 2. Indirect signs like atelectasis, emphysema.

• If X-ray is negative, it does not rule out a foreign body.
• Bronchogram, video laryngoscopy, clinical examination may show a radiolucent FB.
Management of Laryngeal FB

Partial or no Obstruction:

- What do you call this procedure?

Complete obstruction:

- The above manoeuvre is contraindicated. It can further increase obstruction.

- Emergency tracheostomy followed by removal of FB by laryngoscopy
Management of Bronchial FB

• Not an emergency.
• Emergency removal is indicated only in case of complete obstruction or in presence of vegetable FB.

• Methods:
• 1. Rigid or flexible bronchoscopy
• 2. Dormia baskets.
• 3. Thoracotomy or bronchotomy.
MCQ

• When an emergency opening into the airway is required, the cricothyroid membrane should be pierced immediately
  • a) superior to the thyroid cartilage.
  • b) inferior to the thyroid cartilage.
  • c) inferior to the cricoid cartilage.
  • d) superior to the thyroid isthmus.
MCQ

- **Laryngocele arises as a herniation of laryngeal mucosa through the following membrane:**
  - A. Cricotracheal
  - B. Crisosternal
  - C. Thyrohyoiod
  - D. Cricothyroid
MCQ

• All of the following statements about Laryngomalacia are true, Except:

• A. Stridor is increased on crying and relieved on lying prone
• B. Surgical Tracheostomy is the treatment of choice
• C. It is the most common congenital anomaly of the larynx
• D. It is associated with an omega shaped epiglottitis