ENT MODEL QUESTIONS WITH SOLUTIONS

QUESTIONS DRAWN FROM THE TAMILNADU DR MGR UNIVERSITY PREVIOUS YEARS QUESTIONS

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About this Resource

This resource includes all the questions from Otolaryngology MBBS question papers of The Tamilnadu Dr. MGR Medical University along with answer key. There may be repetitions also. Some of the keys provided could be very exhaustive. This has been done to provide complete resource to the student.

The questions are arranged under the following headings:

Essay

Short notes

Ultrashort notes

This resource contains key to nearly 200 essay questions, 250 short notes, and 250 ultra-short notes. This is the first initiative of this kind.

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ENT QUESTION BANK

ESSAY

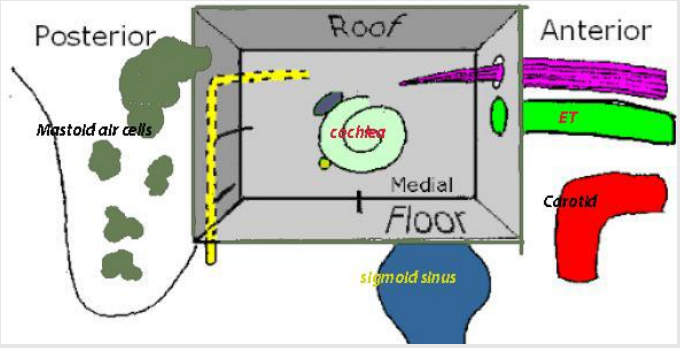
EAR

1. Describe in detail with suitable diagrams the anatomy of the middle ear.

   Synonyms: Middle ear cleft, Tympanum

   The middle ear cleft includes the tympanum (middle ear cavity proper), the eustachean tube, and the mastoid air cell system. The tympanic cavity is an air-filled irregular space contained within the temporal bone. It also contains the three auditory ossicles (malleus, incus and stapes) along with their attached muscles. For the purpose of description, the tympanic cavity may be considered as a box with four walls, a roof and a floor. The corners of this hypothetical box are not sharp.

   Lateral wall: The lateral wall of the tympanum / middle ear is partly bony and partly membranous. The central portion of the lateral wall is formed by the tympanic membrane, while above and below the tympanic membrane there is bone, forming the outer lateral walls of the epitympanum (attic) and hypotympanum respectively. The lateral wall of the epitympanum (attic) also includes that part of the tympanic membrane lying above the anterior and posterior malleolar folds - this portion of the ear drum is also known as pars flaccida. This portion of the tympanic membrane lacks the middle fibrous layer, hence the name. The lateral attic wall (bony portion) is wedge shaped, its lower portion is also called the outer attic wall (scutum). Scutum actually means shield in Latin. This bony portion is thin and its lateral surface forms the superior portion of the deep portion of the external meatus.



Three openings are present in the bone of the medial surface of the lateral wall of the tympanic cavity. The first opening is the posterior canaliculus for the chorda tympani nerve. This opening is situated at the junction between the lateral and posterior walls of the tympanic cavity. This opening is usually present at the level of the upper end of the handle of the malleus. This opening leads to the bony canal which descends through the posterior wall of the tympanic cavity. Since chorda tympanic nerve traverses this canal it is also known as the canal for chorda tympani nerve. This canal also contains a branch from the stylomastoid artery which usually accompanies the chorda tympani nerve.

The second opening is the petrotympanic (Glasserian) fissure. This fissure opens anteriorly just above the attachment of the tympanic membrane. This opening is in fact a small slit about 2 mm long. It receives the anterior malleolar ligament. It also transmits the anterior tympanic branch of the maxillary artery to the tympani cavity.

The third is the canal of Hugier. It lies medial to the Glasserian fissure. The chorda tympani nerve enters through this.

Roof: The roof of the middle ear cavity is formed by the tegmen tympani. It is this tegmen tympani which separates the middle ear cavity from the dura of the middle cranial fossa. This tegmen tympani is formed in part by the petrous portion of the temporal bone, and the squamous portion of the temporal bone. The suture line between these two components is known as the Petro squamous suture line. This suture line is unossified in the young, and does not close until adult life is reached. Through this suture veins from the middle ear may pass to the superior petrosal sinus.

Floor: The floor is much narrow. In fact, it is narrower than the roof of the middle ear cavity. This portion of the middle ear cavity lies in close relationship with the jugular bulb. The middle ear cavity is separated from the jugular bulb by a thin piece of bone. Rarely, the floor may be deficient and the jugular bulb in these patients is separated from the middle ear cavity only by fibrous tissue and mucous membrane. At the junction of the floor and the medial wall of the middle ear there is a small opening which allows the entry of tympanic branch of glossopharyngeal nerve to pass into the middle ear. This nerve takes an important part in the formation of tympanic plexus.

Anterior wall: The anterior wall of the tympanic cavity is very narrow. This is because the medial and lateral walls converge anteriorly. The anterior wall can be divided into two portions; the upper and lower portions. The lower portion of the anterior wall is larger than the upper portion. It has a thin plate of bone which separates this portion from the internal carotid artery as it enters the skull. This plate has two openings for the carotico tympanic nerves. The upper opening transmits the superior carotico tympanic nerve and the inferior opening transmits the inferior carotico tympanic nerve. It is through these nerves that sympathetic nerves reach the tympanic plexus. The upper smaller part of the anterior wall

has two tunnels placed one below the other. The upper tunnel transmits the tensor tympani muscle, and the lower tunnel transmits the bony portion of the eustachean tube.

Medial wall: The medial wall separates the middle ear from the inner ear. The most prominent portion of the medial wall of the middle ear cavity is the promontory. It is a rounded projection occupying most of the central portion of the medial wall of the middle ear. This projection is raised by the underlying basal turn of the cochlea. The promontory has numerous small grooves on its surface. These grooves contain the tympanic plexus of nerves. Behind and above the promontory is the oval window (fenestra vestibuli). This is a oval shaped opening connecting the tympanic cavity with the vestibule. In life this is closed by the foot plate of stapes and its surrounding annular ligament. The long axis of the fenestra vestibuli is horizontal. Its inferior border is concave. The size of the oval window varies, but on an average, it is 3.25mm long and 1.75 mm wide. Above this fenestra vestibuli is the canal for facial nerve (horizontal portion) and below lies the promontory. Hence the fenestra vestibuli lies at the bottom of a depression also known as fossula that can be of varying depths depending on the position of the facial nerve and the prominence of the promontory.
The fenestra cochlea (round window) lies just below and behind the oval window. It is closed in life by a membrane known as the round window membrane (secondary tympanic membrane). The secondary tympanic membrane appears to be divided into an anterior and posterior portion by the presence of a transverse thickening. The diameter of the round window membrane is between 1.8 to 2.3 mm. It is made up of three layers; the outer mucosal, middle fibrous and an inner endothelial layer. The membrane of the fenestra cochleae does not lie at the end of the scala tympani but forms part of its floor. The ampulla of the posterior semicircular canal is the closest vestibular structure to this membrane. The nerve supplying the ampulla of the posterior semicircular canal (singular nerve) lies close to this secondary tympanic membrane. The secondary tympanic membrane forms a landmark for the position of the singular nerve. This is useful during surgical procedures like singular neurectomy for treatment of intractable vertigo. These two windows (oval & round) are separated by the posterior extension of the promontory. This is known as the subiculum. Rarely a spicule of bone arises from the promontory above the subiculum and runs to the pyramid on the posterior wall of the middle ear cavity. This spicule of bone is known as the ponticulus. The round window faces inferiorly and a little posteriorly, lying completely under the cover of the promontory and hence usually is difficult to visualise. The round window niche is usually triangular in shape, having anterior, posterosuperior and posteroinferior walls. The posterosuperior and posteroinferior walls meet posteriorly leading on to the sinus tympani. This sinus tympani is a difficult area to visualise. Cholesteatoma may lurk in this area making it difficult to remove. This is one of the commonest causes of cholesteatoma recurrence after mastoidectomy. Small mirrors known as the zinne mirror can be used to visualise this area indirectly. Since sinus tympani lies under the pyramid, removal of the pyramid during surgery will bring the sinus tympani area into view. The facial nerve canal is another important anatomical structure present in this wall. This nerve runs above the promontory and fenestra vestibuli in an

anteroposterior direction. The canal may occasionally be deficient leaving an exposed facial nerve. This is a dangerous anatomical variant because this nerve can easily be traumatized during any surgical procedures in the middle ear cavity. Even infections of the middle ear mucosa can cause facial nerve palsy in patients with an exposed facial nerve. The anterior end of the facial nerve canal is marked by the presence of a bony process known as processus cochleariformis. This curved projection of bone is concave anteriorly and it houses the tendon of the tensor tympani muscle as it turns laterally to the handle of the malleus. Behind the fenestra vestibuli, the facial nerve turns inferiorly to begin its descent in the posterior wall of the tympani cavity.

The region above the level of the facial nerve canal forms the medial wall of the epitympanum or attic. The dome of the lateral semicircular canal extends a little lateral to the facial canal and is the major feature of the posterior portion of the epitympanum. In well pneumatised bones this dome of the lateral canal can be very prominent.

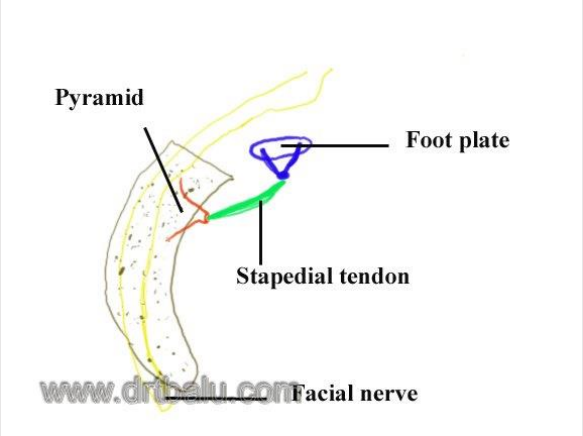


Figure showing Pyramid of middle ear

Posterior wall: The posterior wall of the middle ear is wider above than below. In its upper part it has an important opening known as the aditus. This aditus helps the middle ear communicate with the mastoid air cell system. Aditus is a large irregular opening connecting the mastoid antrum to the middle ear cavity. Below the aditus is a small depression known as the fossa incudis. Fossa incudis houses the short process of the incus. Below the fossa incudis lies the pyramid.

Pyramid is a small conical projection which is hollow and its apex pointing anteriorly. It contains the stapedius muscle, the tendon of which passes forwards to insert into the neck of the stapes. The canal within the promontory curves downwards and backwards to join the descending portion of the facial nerve canal. Between the promontory and the tympanic annulus is the facial recess. The facial recess is bounded medially by the facial nerve and laterally by the tympanic annulus. Running through the wall between the two with varying degrees of obliquity is the chorda tympani nerve. This nerve always run medial to the tympanic membrane. Drilling over the facial recess area between the facial nerve and the

annulus in the angle formed by the chorda tympani nerve can lead into the middle ear cavity. This surgical approach to the middle ear cavity through this area is known as the facial recess approach. This approach is suitable for surgeries involving the round window niche like placement of electrodes during cochlear implant procedures. Hypotympanum can also be approached through this approach.

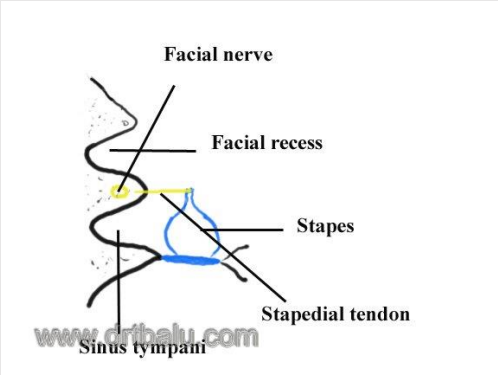


Diagram showing sinus tympani of middle ear cavity

Contents of the middle ear:

The most important content of the middle ear is air. The air flows into the middle ear through a patent eustachean tube. The other contents are:

Chain of three ossicles which help in sound transmission; the malleus, incus and stapes. Two muscles, chorda tympani nerve and the tympanic plexus of nerves.

Malleus: This bone is shaped like a hammer hence the name. This is the largest of the three ossicles of the middle ear cavity. It has a head, neck and three processes arising from below the neck. The overall length of the malleus ranges between 7.5 - 9 mm. Its head lies in the attic region of the middle ear effectively dividing the attic into an anterior portion and a posterior one. The anterior portion lie anterior to the handle of the malleus, while the posterior portion lie behind the handle of the malleus. During surgical procedures for attic cholesteatoma clipping of this head will improve the exposure in the attic region. The head of the malleus on its posteriomedial surface has an elongated saddle shaped cartilage covered facet for articulation with the incus. This articular surface is constricted near its middle dividing the articular facet into a larger superior and a smaller inferior portion. The inferior portion of the articular facet lies at right angles to that of the superior portion. This projecting lower portion is also known as the cog or spur of the malleus. Below the neck the bone broadens and gives rise to the following: the anterior process from which a slender anterior ligament arises to insert into the petrotympanic fissure; the lateral process which receives the anterior and posterior malleolar folds from the annulus tympanicum, and the handle which runs downwards, medially and slightly backwards between the mucous and fibrous layers of the tympanic membrane. On the deep medial surface of the handle there is

a small projection into which the tendon of the tensor tympani muscle inserts. Additionally, the malleus is supported by the superior ligament which runs from the head to the tegmen tympani.

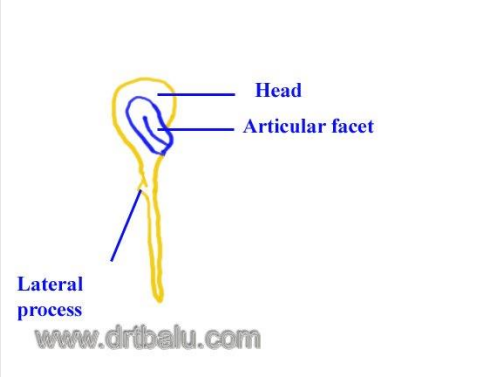


Image showing Malleus along with its articular facet

Incus: This bone is shaped like an anvil. It articulates with the malleus and has a body and two processes. The body lies in the attic and has a cartilage covered articular facet corresponding to that of the malleus. The short process projects backwards from the body to lie in the fossa incudis. It is in fact attached to the fossa incudis by a short ligament. The long process of the incus descends into the mesotympanum behind and medial to the handle of the malleus. At its tip there is a small medially directed lenticular process which articulates with the stapes. The long process of the incus has precarious blood supply. This portion of the incus is prone for undergoing necrosis in disease conditions.

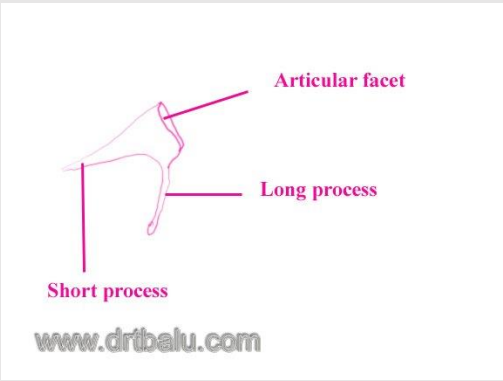


Image showing incus along with its articular facets

The stapes: The stapes consists of a head, neck, two crura and a base (footplate). The head of the stapes points laterally and has a small cartilage covered depression for articulation with the lenticular process of the incus. The tendon of the stapedius muscle attaches to the posterior part of the neck and the upper part of the posterior crura. The neck of the stapes gives rise to two crura, the anterior crura is thinner and less curved than the posterior crura. The two crura join the foot plate which closes the oval window during life. The average dimensions of the foot plate are 3mm x

1.4mm. The long axis of the foot plate is almost horizontal, with the posterior end being slightly lower than the anterior.

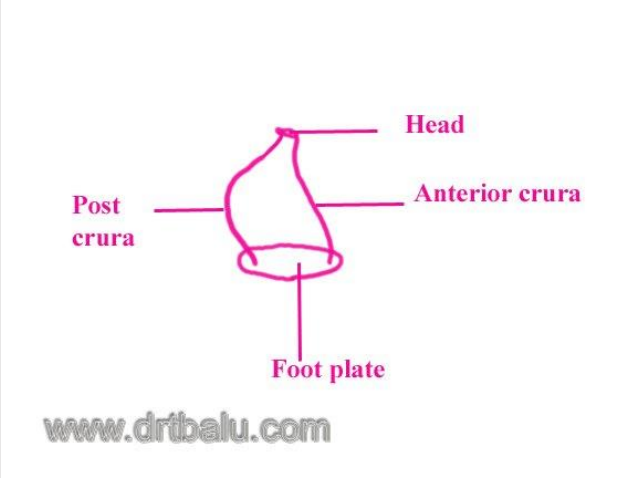


Image showing stapes

Muscles of the middle ear:

Stapedius muscle: arises from the walls of the conical cavity within the pyramid. A slender tendon emerges from the apex of the pyramid and inserts into the stapes. This muscle is supplied by a small branch from the facial nerve. The stapedial tendon is inserted into the neck of the stapes. On contraction this muscle rocks the stapes backwards holding it firm against the annular ligament preventing excessive transmission of sound into the inner ear. This muscle has a protective role to play. It protects the inner ear from insults caused by loud noise. Patients with facial nerve palsy have hyperacusis because of lack of action of this muscle.

Tensor tympani muscle: This long slender muscle arises from the walls of the bony canal which lie above the canal for the eustachean tube. Parts of the muscle also arise from the cartilaginous portion of the eustachean tube and the greater wing of sphenoid. From these origins the muscle passes backwards into the tympanic cavity lying on the medial wall of the middle ear just below the level of the facial nerve. The bony covering of the canal is often deficient in its tympanic segment where the muscle is replaced by its tendon. This tendon enters the processus cochleariformis, turns at right angles inserting into the medial aspect of the upper end of the handle of the malleus. This muscle is supplied by the mandibular nerve by way of a branch from the medial pterygoid nerve, which passes through the otic ganglion without synapsing. This muscle tenses the tympanic membrane by holding the handle of the malleus thus helping the middle ear in better sound perception.

Chorda tympani nerve:

This is a branch of the facial nerve. It enters the middle ear cavity through the posterior canaliculus which is present at the junction of the lateral and posterior walls. It runs across the medial surface of the tympanic membrane between the mucosal and fibrous layers passes medial to the upper portion of the handle of the malleus. Here it lies above the tendon of the tensor tympani muscle, continues forwards and leaves by way of the anterior canaliculus placed within the petrotympanic fissure. It joins the lingual branch of the V nerve with which it is distributed to the anterior 1/3 of the tongue.

Tympanic plexus:

Is found over the promontory. It is formed by the tympanic branch of the glossopharyngeal nerve, carotico tympanic nerves which supplies the sympathetic component. The tympanic plexus provides the following branches:

1. Branches to the mucous membrane lining the tympanic cavity, eustachean tube, mastoid antrum and its air cells

2. A branch joining the greater superficial petrosal nerve.

3. The lesser superficial petrosal nerve, which contain all the parasympathetic fibers of the IX nerve. This nerve leaves the middle ear through a small canal below the tensor tympani muscle where it receives parasympathetic fibers from the VII nerve by way of a branch from the geniculate ganglion. The full nerve passes through the temporal bone to emerge lateral to the greater superficial petrosal nerve on the floor of the middle cranial fossa, outside the dura. It then passes through the foramen ovale with the mandibular nerve and accessory meningeal artery to the otic ganglion. Post ganglionic fibers from the otic ganglion supply secretomotor fibers to the parotid gland by way of the auriculotemporal nerve.

The mucosal lining of the middle ear cavity varies according to the location. The attic or the epitympanum is lined by pavement epithelium, while the middle ear proper is lined by cuboidal epithelium and the hypotympanum is lined by ciliated columnar epithelium.

2. Describe causes and clinical features of various conditions presenting with pain in one ear.

Unilateral pain in the ear can be classified into:

i. External ear causes

ii. Middle ear causes

iii. Pain referred from adjacent areas due to segmental innervation.

External ear causes: Otitis externa is the most common cause of unilateral pain in the ear. This is due to infection of the external auditory canal. This condition is really a very painful one.

Otitis external can be classified into:

1. Acute diffuse otitis externa (commonly caused by bacteria)

2. Acute localised otitis externa (commonly furuncle)

3. Chronic otitis externa

4. Eczematous otitis externa

5. Fungal otitis externa

6. Malignant otitis externa

Predisposing factors for otitis externa:

Under normal conditions the skin lining the external auditory canal is well protected by its self-cleansing mechanism. In diseased conditions several factors may come into play in the pathogenesis of otitis externa.

1. Absence of cerumen: The cerumen plays an important role in the protection of the external canal. It protects the external canal from moisture. It also has anti-bacterial properties which helps in the protection of the external canal. The cerumen also lowers the pH of the external canal making it difficult for the bacterial pathogens to colonize.

2. Removal of cerumen by ear buds: is one of the common causes of otitis externa. The act of removal traumatizes the skin lining of the external canal making it vulnerable to infections.

3. Frequent exposure to water: external canal when constantly bathed in water loses its ability to protect itself. The presence of water macerates the skin lining of the external canal and also increase the pH of the external canal making it more favorable for bacterial colonization. This condition is common in swimmers.

Acute diffuse otitis externa:

This is also known as the swimmers ear. This is an inflammatory condition involving the external canal in a diffuse manner. This condition is common in swimmers because of the propensity for the external canal to be exposed to water for long durations. This exposure leads to maceration of the external canal skin, and also lowers the pH of the external canal providing an environment favorable to infections.

Main symptoms:

1. Itching in the external canal

2. Tenderness on palpation

3. Aural fullness rarely occur due to the reduction in size of the external canal lumen due to oedema

4. Rarely stenosis of the external canal may occur causing accumulation of debris and secretions

Common signs:

1. Erythema of the external canal

2. Oedema of external canal

3. Secretions from the external canal (weeping canal)

4. Pain on mastication

5. Pulling of helix in a postero superior direction cause pain

6. In advanced cases fever and lymphadenopathy may occur (pre and post auricular nodes may be involved)

Stages of acute diffuse otitis externa: (Senturia)

Preinflammatory stage: is characterized by intense itching, edema and sensation of fullness in the ear.

Inflammatory stage: may be divided into mild, moderate and severe.

Mild acute inflammatory stage: here the cardinal features are increased itching, pain, mild erythema and oedema of the external canal skin. At later stages exfoliation of skin with minimal amount of cloudy secretions may be seen in the external canal.

Moderate acute inflammatory stage: in this stage the itching and tenderness of the external canal intensifies. The external canal is narrowed due to oedema and accumulation of epithelial debris.

Severe acute inflammatory type: In this stage pain becomes intolerable to such an extent the patient may refuse to eat, the lumen of the external canal becomes totally obliterated due to oedema and accumulated epithelial debris. Otorrhoea may become purulent. In addition, regional nodes may also be involved. Infections from the external canal may involve the parotid gland via the fissure's of santorini.

Common organisms involved: Psuedomonas aeruginosa and staphylococcus aureus are commonly cultured from the external canal of these patients. The normal commensals like staphylococcus epidermidis and corynebacteria are conspicuously absent.

Management:

The aim is twofold:

1. Resolving the infection

2. Promoting the external canal skin's recovery to its original state.

Firstly, the canal is cleaned atraumatically by gentle suctioning and debridement under microscope. Topical hydrogen peroxide solution instilled will help the process of debridement.

A cotton wick dipped in I.G. paint can be inserted in to the external canal and allowed to stay for a day. This will reduce the external canal skin oedema and will increase the size of the meatus. Ear drops containing a mixture of neomycin and 1% hydrocortisone may be instilled as ear drops at least three times a day. In addition to the antibiotic and anti-inflammatory effects this drug reduces the pH of the external canal making it more resistant to the organisms.

In severe cases oral antibiotics and anti-inflammatory drugs can be resorted to. Quinolones are commonly used oral antibiotic.

Acute localized otitis externa:

This condition is otherwise known as furunculosis or circumscribed otitis externa. This is a localised infection usually found to involve the lateral 1/3 of the external canal. It also has a propensity to involve the posterior superior aspect of the external canal. This is caused due to obstruction of the apopilosebaceous units found extensively in this area.

Trauma to skin in this area followed by infection is commonly attributed cause. The organism responsible is commonly staph aureus.

Symptoms:

1. Localised pain

2. Localised itching

3. Purulent discharge if the abscess ruptures

4. If oedema or abscess occludes the external canal hearing loss can occur.

Signs:

1. Erythema of the skin

2. Localised abscess formation

Management:

If the abscess is pointing it can be treated by incision and drainage. Oral antibiotics should be used. The preferred drug of choice is penicillin of first generation cephalosporins. Anti-inflammatory drugs can be used to reduce inflammation and pain.

These patients must be advised to cut their nails short and to keep their hands clean, since this is the commonest route of infection.

Chronic otitis externa:

This is a chronic infection / inflammation involving the skin lining of the external canal. There is thickening of the skin lining of the external canal due to persistent low-grade infection / inflammation.

Symptoms:

1. Unrelenting pruritus

2. Mild pain

3. Presence of dry skin in the external canal

Signs:

1. Asteatosis (lack of cerumen)

2. Hypertrophic external canal skin

3. Presence of dry flaky skin in the external canal

4. Mild tenderness on ear manipulation

5. Rarely muco purulent Otorrhoea

Cultures from the external canal of these patients are highly unreliable because they would have been using various antibiotic drops to surmount the problem.

Management:

Involves extensive use of acetic acid ear drops. This helps to reduce the pH of the skin lining the external canal making it more resistant to bacterial infections. In intractable cases steriod drops can be tried. Antibiotic drops may not be useful in these patients.

Surgery is indicated in extreme cases. A canalplasty is performed to widen the external canal. The involved skin may be removed to be replaced by a split thickness graft.

Eczematous otitis externa:

This condition includes various dermatologic conditions involving the skin of the external canal. It may range from atopic dermatitis, contact dermatitis, seborrheic dermatitis, neuro dermatitis, infantile eczema etc.

This condition is characterized by intense itching, in fact this could be the only complaint of the patient. On examination, erythema of the external canal skin may be seen. There may also be associated scaling and oozing from the canal skin.

Success lies in the management of the underlying dermatologic condition

Fungal otitis externa:

This is the commonest type of otitis externa in tropical countries. This condition is associated with increased ear canal moisture, or following treatment of otitis external by prolonged use of topical antibiotics. The protective cerumen layer is absent in these patients. This condition is more common in diabetics.

Symptoms:

1. Intense itching

2. Pain when otitis externa is coexistent

3. Blocking sensation due to the presence of fungal balls

Signs:

1. Inflamed external canal skin

2. External canal tenderness

3. Fungal debris (black in case of aspergillus and white in the case of candida). Invariably the infection is mixed type.

Management:

The condition is managed by careful aural toileting to remove the fungal balls. The best way to remove fungus from the ear canal is by aural syringing. Antifungal ear drops of clotrimazole can be administered. If secondary infections are present oral antibiotics and anti-inflammatory drugs may be resorted to.

Malignant otitis externa:

This rare but sinister form of otitis externa is known to affect elderly diabetics. This condition is caused by pseudomonas infection of the external ear. These patients have a unique nocturnal deep boring type of pain.

The patient gives history of trivial trauma to the external canal. Granulations can be seen at the junction of bony cartilaginous portion of the external canal.

This condition can cause complications like facial nerve involvement, and spread to the intracranial structures.

Middle ear causes of otalgia:

Acute otitis media is one of the common middle ear causes of otalgia. This condition is common in children. It is caused by eustachean tube block causing pent up secretions to accumulate in the middle ear cavity. Pain gets relieved when the ear drum perforates and starts to drain the middle ear cavity. Children are commonly affected because of their short, wide and straight eustachean tube.

Otitic barotrauma: This is caused due to sudden changes in altitudes as in deep sea diving / flying unpressurized airplanes.

Referred otalgia:

Pain to the ear can be referred from disorders affecting other portions of head and neck. These include:

1. Temporomandibular joint dysfunction

2. Dental pain

3. Quinsy

4. Tonsillitis

5. Post tonsillectomy pain always radiates to the ipsilateral ear.

Causes for referred otalgia should be diligently searched for in a patient with ear pain, with clinically normal ear.

3. Give an account of aetiology, clinical features and treatment of Glue Ear.

Synonyms:

Secretory otitis media, glue ear, serous otitis media, non-purulent otitis media.

Definition:

Otitis media with effusion is defined as chronic accumulation of mucus within the middle ear, and rarely this could involve the mastoid air cell system. This accumulation causes conductive hearing loss.

Histology and histopathology of eustachean tube:

The pseudostratified ciliated columnar epithelium of respiratory tract extends up the eustachean tube as far as the anterior part of the middle ear cavity. These cells are capable of producing mucous. There are also goblet cells seen in their midst. These cells are also capable of secreting mucous material. Otitis media with effusion is caused by inflammation of this epithelium in the eustachean tube and hypotympanum. In established cases of glue ear, the cuboidal epithelium of middle ear and mastoid air cells gets replaced by thickened pseudostratified columnar epithelium. The cilia of these cells have also been found to be ineffective in propelling the secretions into the nasopharynx. The submucosa is found to be oedematous, inflamed with dilated blood vessels with increased number of macrophages and plasma cells.

Etiology:

1. In many children otitis media with effusion is preceded by an episode of acute otitis media. This is common in children who is more prone for upper respiratory infections. Common being viral infections which damages the eustachean tube epithelium.

2. Craniofacial abnormalities: Children with cleft palate have deficient palatal muscles causing a poor eustachean tube function leading on to Otitis media with effusion. This occurs despite a successful surgical repair of the cleft palate. Children with Down's syndrome are also more prone for OME.

Note: Children with bifid uvula do not appear to have higher incidence of OME

3. Allergy: Previously nasal allergy has been postulated as an important factor in the development of Otitis media with effusion. Studies have been unequivocal.

4. Gastroesophageal reflux: GERDS has been commonly demonstrated radiologically in children with OME. Furthermore, biochemical analysis of middle ear fluid have demonstrated significant amounts of pepsin (in 80% of cases).

5. Parental smoking has been attributed as an important predisposing factor for the development of OME.

Age of occurrence: OME shows classically a bimodal distribution. The first peak occurs around 2 years of age, and the second peak occurs at about 5 years of age. This distribution occurs roughly around the ages when the child goes to preschool and primary school.

Seasonal association: OME commonly occurs during winter season, when there is more likelihood of upper respiratory infections, and also because of the possibility of closer contact with affected children. This is seen in temperate zones. In non-temperate zones it is commonly seen during rainy season.

Clinical features:

A high index of suspicion is necessary to identify this condition. Every child with upper respiratory infection must be otoscopically examined.
Otoscopic findings: The tympanic membrane may be bulging, or retracted with a distorted cone of light. The ear drum may appear yellow, blue or simply clear white. Pneumatic otoscopy will reveal a ear drum which has a restricted mobility.

Microbiology of OME: Commonly middle ear effusions due to glue ear is sterile. Rarely bacteria could be cultured. The incidence of these pathogens are higher in children under the age of 2, and in children with recurrent upper respiratory infections.

Investigations:

Puretone audiometry: Demonstrates mild to moderate conductive deafness.

Tympanograms (Type B) is commonly associated with OME. Type A is infrequently associated while Type C falls somewhere in between. Tympanometry can be used as a screening test to identify patients with OME.

Free field audiometry: Demonstrates deafness.

Management:

1. Antibiotics: Amoxycillin is the drug of choice followed by cephalosporins.

2. Nasal decongestants like oxymetazoline / xylometazoline may help in some cases.

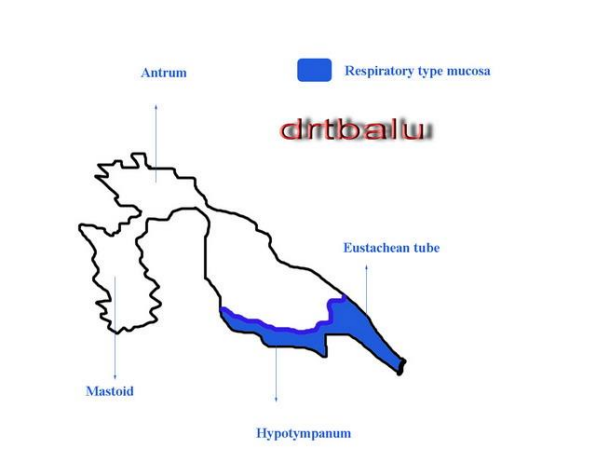
3. Topical nasal steroids can be used in resistant cases.

4. Auto inflation of eustachean tube by performing valsalva maneuver. Balloon blowing may also help.

Surgical management:

1. Adenotonsillectomy

2. Myringotomy and insertion of ventilation tubes



Eustachean tube along with its lining epithelium

4. Enumerate causes of conductive deafness with intact tympanic membrane. Discuss clinical features and management of otitis media with effusion in a 4-year-old child.

Some of the causes of conductive deafness with intact ear drum include:

External ear causes:

Congenital causes:

Atresia of external auditory canal
Anotia
Hypoplastic tympanic membrane

Acquired causes:

Impacted cerumen
Acquired stenosis of external auditory canal
Impacted FB. Fully occluding the external auditory canal
Exostosis of external auditory canal
Tympanosclerosis of ear drum

Middle ear causes:

Congenital:

Congenital fixation of head of the malleus
Congenital fixation of foot plate of stapes
Hypoplastic middle ear cavity

Acquired:

Glue ear
Hemotympanum
Ossicular chain discontinuity due to trauma
Otosclerosis
Adhesive otitis media

Salient points of otitis media with effusion:

Synonyms: Glue ear, Secretory otitis media, Serous otitis media. Among these synonyms the term serous otitis media is a misnomer since the fluid accumulated within the middle ear cavity always contain some amounts of mucoid material.

Prevalence: It has a bimodal distribution. The first peak (largest) at about two years of age, and a second peak at about 5 years of age.

Clinical features:

Natural resolution of the disorder is the order of the day. In the majority of children OME causes only a mild hearing impairment thereby there is no significant disability. These children have a minimal (10dB) residual hearing loss even after resolution of the disease. A small percentage of children may have attic retraction. This potential of formation of attic retraction and cholesteatoma can be minimized by inserting ventilation tubes in these patients.
Some of these children may manifest with vestibular symptoms i.e. Giddiness and unsteadiness. The child could become clumsy in his / her activities because of lack of co-ordination.

Tympanosclerosis and atrophy of the ear drum can be caused by OME. The risk is further increased when ventilation tubes are inserted.

Management:

Role of antibiotics in these patients are highly controversial and is not advised routinely.
Use of anti-inflammatory drugs is not advised
Use of antihistamines and mucolytics have been suggested by some authors.

Grommet insertion:

Ventilation tubes will benefit only those children who have 25dB hearing loss over a 12-week period.

Adenoidectomy when performed reduces the recurrence rate of the disease. Adenoidectomy can be combined with grommet insertion.

5. Enumerate conditions producing conductive deafness. Discuss etiology, clinical features and treatment of acute suppurative otitis media

Causes for conductive deafness include:

External ear causes:

Congenital:

Atresia of external auditory canal

Microtia

Anotia

Acquired:

Impacted cerumen (commonest)
Acquired stenosis of external auditory canal
Exostosis of external auditory canal

Ear drum causes:

Tympanosclerosis
Perforation involving the ear drum
Retracted ear drum

Middle ear causes:

Congenital:

Congenital fixation of head of malleus (Fixed malleus syndrome)
Congenital fixation of foot plate of stapes

Acquired:

Ossicular chain necrosis due to CSOM (long process of incus is commonly eroded as it receives precarious blood supply)

Middle ear effusion

Adhesive otitis media

Glue ear

Hemotympanum

Glomus jugulare

Ossicular chain discontinuity due to trauma

Otosclerosis

ASOM:

Definition:

Acute suppurative otitis media is defined as suppurative infection involving the mucosa of the middle ear cleft. By convention it is termed acute if the infection is less than 3 weeks in duration.

Pathophysiology:

Obstruction to the eustachean tube seem to be the most important antecedent event in the pathophysiology of acute suppurative otitis media. Majority of acute suppurative otitis media is

triggered by upper respiratory infections which might find its way into the middle ear cavity through the eustachean tube orifice. Infections involving the nasopharynx may find its way into the middle ear through the pharyngeal end of eustachean tube. The infection is initially commonly viral in origin, allergy could also play an important role in the pathogenesis. Later the middle ear mucosa becomes secondarily infected by pathogenic bacteria. The bacteria commonly implicated in this disorder is S Pneumoniae, H. Influenza, and M Catarrhalis.

The majority of otitis media prone children have a patulous eustachean tube or an hypotonic eustachean tube. Children with neuromuscular disorders or with abnormalities of the first or second arch have a patulous eustachean tube leading on to this problem. To become pathogenic the bacteria must become adherent to the mucosa lining the middle ear cavity, this is made possible by prior infection of the middle ear mucosa by viruses.

Flask model explaining the role of eustachean tube in middle ear infections:

The eustachean tube, middle ear, and mastoid air cell system can be likened to a flask with a long narrow neck. The mouth of the flask represents the nasopharyngeal end, the narrow neck, the isthmus of the eustachean tube, and the bulbous portion, the middle ear and mastoid air chamber. The fluid flow through the neck of the flask would be dependent on the pressure at either end, the radius and length of the neck, and the viscosity of the liquid. When a small amount of liquid is instilled into the mouth of the flask, liquid flow stops somewhere in the narrow neck owing to capillarity within the neck and the relative positive air pressure that develops in the chamber of the flask.

The basic geometry is considered to be critical for the protective function of the eustachean tube - middle ear system. Reflux of liquid into the body of the flask occurs if the neck of the flask is excessively wide, or the length of the neck of the flask is too short as seen in children. Because infants have a shorter eustachean tube than adults, reflux is more likely to occur in the baby. The position of the flask in relation to the liquid is another important factor. In humans, the supine position enhances flow of liquid into the middle ear; thus, infants might be at risk for developing reflux otitis media because they are commonly supine. Reflux of liquid into the vessel can also occur if a hole is made in the bulbous portion of the flask, because this prevents the creation of positive pressure in the bulbous portion. This positive pressure is useful in the prevention of reflux of material from the neck of the flask.

If negative pressure is applied to the bulbous portion of the flask then this pressure is sufficient to cause aspiration of contents from the neck of the flask. This scenario is represented by high negative pressure in middle ear as it occurs in nose blowing, crying, closed nose swallowing, diving or airplane descent. The neck of the eustachean tube is supposed to be compliant hence compliance plays a vital role in prevention of reflux of secretions.

Clinical features:

Acute suppurative otitis media passes through 4 stages: 1. Stage of hyperemia

2. Stage of exudation

3. Stage of suppuration

4. Stage of resolution.

The progression of these stages depends on the virulence of the infecting organisms, resistance of the host, adequacy of antibiotic therapy. If the infecting organism is virulent or if the antibiotic treatment is not sufficient then the disease may progress to a stage of coalescent mastoiditis with its attendant complications.

Stage of hyperemia:

Initial infection by infection results in hyperemia of the mucous membrane causing otalgia, fever and fullness in the affected ear. This stage is characterized by oedema of the mucoperiosteum due to vascular engorgement. Otoscopy show dilated vessels along the handle of malleus and along the rim of the tympanic membrane. Antibiotic therapy during this stage will help in resolution of the disease. Amoxycillin is the drug of choice.

Stage of exudation:

Absence of treatment during the stage of hyperemia leads to the stage of exudation. In this stage there is outpouring of fluid from the dilated vessels of the mucoperiosteum. This fluid is serous in nature containing fibrin, red cells, and polymorphs. This exudate fills the tympanomastoid compartment really fast, and the whole middle ear cavity is under intense pressure due to this retained secretion. Pain is the most prominent feature of this stage. The patients may have fever and fullness in the ear. Otoscopy shows a bulging ear drum with loss of all landmarks. The drum is reddish and bulging in nature. These patients have also coexistent mastoid tenderness due to mastoiditis.

Stage of suppuration:

Failure of treatment during the stage of exudation leads on to stage of suppuration. The exudate present in the middle ear cavity is a very good culture medium and hence there is secondary bacterial infection leading on to suppuration.

Stage of resolution:

is preceded by either rupture of the ear drum leading on to a serous / serosanguinous / purulent discharge from the ear. When the middle ear is free from the exudate / pus the stage of resolution sets in. The patient has reduction in otalgia, fever subsides. The patient has considerable clinical improvement.

Stage of complication:

If the infection persists beyond a period of 2 weeks then there is associated thickening of the mucoperiosteum especially in the air cells around the peri antral area leading to a block in the drainage from the antral cells. The pent-up secretions in the mastoid air cell system causes intense pressure, venous stasis and local acidosis. This acidosis cause dissolution of calcium from the bone causing decalcification and coalescence of the mastoid air cell system. This condition is known as coalescent mastoiditis. This stage is characterized by emergence of otalgia and low-grade fever. Erosion of the outer cortex in the mastoid lead to the formation of abscess under the periosteum of the mastoid cortex. This condition is known as subperiosteal abscess.

Management:

Acute suppurative otitis media is a self-limiting condition. If appropriate antibiotics is started early then it resolves. Amoxycillin is the drug of choice. Cephalosporins may also be started in refractive cases. Anti-inflammatory drugs like ibuprofen is also prescribed in order to alleviate pain. Patients who are refractory to medical management may undergo myringotomy in order to decompress the middle ear cavity. This procedure is done using a myringotome.

Coalescent otitis media and subperiosteal abscess are surgical complications. These patients must be taken up for surgery under adequate antibiotic cover.

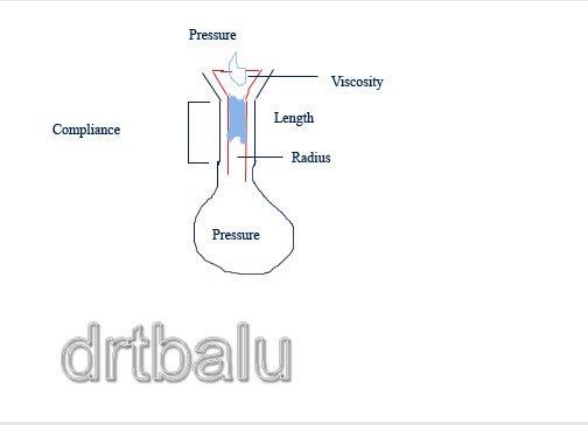


Figure explaining the flask model of normal eustachean tube function

Figure showing the differences between eustachean tubes of adult and a child

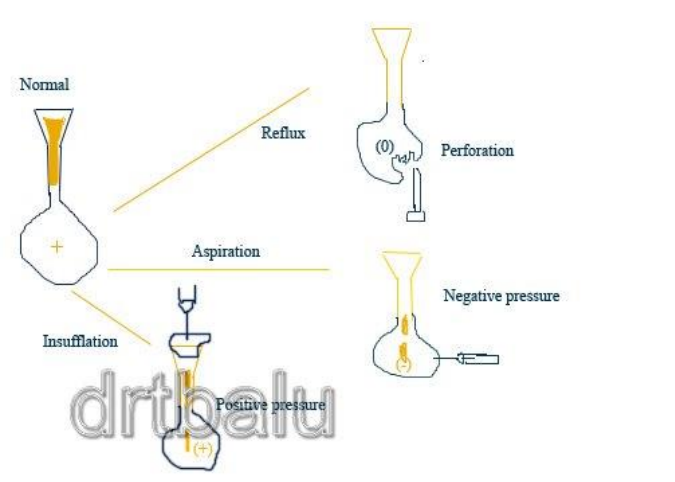


Figure showing various pathophysiological factors involved in middle ear diseases

6. Discuss the aetiopathogenesis, Clinical features and Management of tubotympanic type of chronic suppurative otitis media.

Definition:

Chronic suppurative otitis media is defined as a chronic infection of the mucosa lining the middle ear cleft. Middle ear cleft includes the eustachean tube, hypotympanum, mesotympanum, epitympanum, aditus and mastoid air cell system.

Tubotympanic disease: This is also known as safe disease because it is bereft of any serious complications. The infection is limited to the mucosa and the antero inferior part of the middle ear cleft, hence the name. This disease does not have any risk of bone erosion. The discharge any will flow through a perforation present in the pars tensa portion of the ear drum. This perforation is usually surrounded by a rim of remnant ear drum or at least the annulus is intact. (Central perforation). The perforation is usually reniform (kidney shaped) because of poor blood supply to the affected portion of the tympanic membrane.

The infective activity of safe disease is related to the frequency of upper respiratory tract infections, the discharge tending to increase with increasing frequency of upper respiratory infections.

Aetiology:

1. Could be a sequela to inadequately treated acute otitis media.

2. Acute suppurative otitis media causing persistent perforation which could be infected from bacteria in the external auditory canal. This condition is known as persistent perforation syndrome.

Microbiology of CSOM: In all varieties of CSOM the major organism found in the discharge are gram negative bacilli i.e. Ps. aeruginosa, E. coli, and B. proteus. These organisms are not commonly found in the upper respiratory tract, but they are found in the skin of external auditory canal.

Clinical features of tubotympanic disease:

1. The discharge in this condition is profuse and mucopurulent in nature.

2. The discharge is not foul smelling.

3. Since the infected area is open at both ends i.e. the eustachean tube end and the perforation in the ear drum, the discharge does not accumulate in the middle ear.

4. The ossicular chain is not at risk in this type of disorder, the conductive deafness caused is due to the presence of perforation in the tympanic membrane and thickening of the tympanic membrane.

5. Conductive deafness may also be accentuated by thickening of round window membrane due to the presence of secretions. Hearing loss is usually about 30 - 40 dB.

6. These patients have poorly pneumatised / sclerosed mastoid air cell system. This feature has been attributed to repeated attacks of middle ear infections during childhood causing inadequate pneumatization of mastoid air cell system. In patients with pneumatised mastoid air cell system repeated middle ear infections can cause sclerosis with evidence of new bone formation. Mastoids in these patients may be sclerotic.

7. Pain in the ear when present is always associated with otitis externa. This commonly occurs when the patient attempts to clean the ear off the purulent secretions with a ear bud or cotton tipped applicator.

Pathology of tubotympanic disease:

Pathological changes depend on the stage of the disease. The stages are as follows:

Acute stage: This is where the ear is actively discharging. The mucosa of the middle ear cavity is hypertrophied, and congested.

Inactive stage: This condition is characterized by dry perforation of ear drum, commonly in its antero inferior part, close to the eustachean tube orifice. The middle ear mucosa is normal.

Quiescent stage: Perforation of ear drum is present; the middle ear is dry and mucosa may be normal or hypertrophied.

Healed stage: Here the perforation of ear drum has healed by formation of thin scar. There may even be tympanosclerotic patches / chalky deposits on the ear drum. The ossicular chain is invariably intact.

Tuning fork tests show:

Rinne - Negative on the affected side

Weber - Lateralized to the good ear

Absolute bone conduction test - Not reduced

Pure Tone audiometry show conductive hearing loss. The hearing loss is invariably under 40 dB.

Management of tubotympanic disease:

Conservative management:

If the disease is active - with active ear discharge

Aural toileting - must be done using dry cotton swabs.

Suction method can be used to suck out secretions from the external canal and the middle ear cavity. The only disadvantage of this procedure is the risk of noise induced deafness.

Syringing the affected ear with warm saline mixed with acetic acid (1.5%) can be used to syringe the affected ear. This solution not only clears the ear of its purulent secretions, it also helps to remove crusts if present. The presence of weak acetic acid has bacteriostatic effect.

Role of antibiotics in the management of tubotympanic disease:

Antibiotics can be administered depending on the culture report. The best route of administration is topical because the presence of a large central perforation enables adequate concentration of antibiotics to reach the middle ear mucosa. Ototoxic drugs are to be avoided because the increased vascularity present in the middle ear mucosa will cause easy absorption of the drug into the inner ear fluids causing sensori neural hearing loss. Ciprofloxacillin can be administered topically.

Oral amoxycillin in adequate doses or penicillins in adequate doses may be beneficial.

Role of antihistamines and nasal decongestants: Is questionable. Their role is to decongest the nasal and naso pharyngeal mucosa, pharyngeal end of eustachean tube. Since there is associated perforation of tympanic membrane, secretions don't tend to accumulate inside the middle ear cavity. Topical nasal decongestants should not be used for more than a week, because of their propensity to cause rhinitis medicamentosa.

Precautions:

1. The ear must be kept dry. This can be achieved by keeping the ears plugged when taking head bath. Swimming must be avoided till the perforation heals.

2. Preexisting sinus infections if any must be treated aggressively.

3. Presence of focal sepsis in the throat (tonsils commonly) must be ruled out.

Surgical management:

1. Surgical management aims at correcting the causative problems if any.

The presence of deviated nasal septum must be corrected as this could predispose to chronic sinus infections.

If focal sepsis is identified in the tonsils and adenoid then adenotonsillectomy needs to be performed.

After eradicating the possible focal sepsis only attempt must be made to definitively treat the perforation. If the ear drum has managed to stay dry for more than 6 months myringoplasty can be performed. Temporalis fascia is used as grafting material because of its availability in close proximity, its thickness is more or less similar to that of normal ear drum. One other added advantage is its low basal metabolic rate.

If middle ear mucosa is wet and oedematous then cortical mastoidectomy should be resorted to if conservative management fails. Mastoidectomy can always be combined with myringoplasty in the same sitting.

7. What is cholesteatoma? Describe its pathogenesis, etiology, clinical features and management.

Definition of cholesteatoma:

Cholesteatoma is defined as a cystic bag like structure lined by stratified squamous epithelium on a fibrous matrix. This sac contains desquamated squamous epithelium. This sac is present in the attic region. Cholesteatoma is also defined as 'skin in wrong place'. Cholesteatoma is known to contain all the layers of skin epithelium. The basal layer (germinating layer) is present on the outer surface of cholesteatoma sac in contact with the walls of the middle ear cleft.

Theories of bone invasion by cholesteatoma:

1. Pressure theory - states that increase in the pressure caused by enlarging cholesteatoma cause bone erosion. Ischemia has been attributed as the cause in this theory.

2. Enzymatic theory: Inside the cholesteatoma are present multinucleated osteoclasts and histiocytes. These cells release acid phosphatase, collagenase and other proteolytic enzymes. These enzymes are known to cause bone erosion.

3. Pyogenic osteitis: Pyogenic bacteria may release enzymes which could cause bone resorption.

Types of cholesteatoma:

1. Congenital cholesteatoma

2. Primary acquired cholesteatoma

3. Secondary acquired cholesteatoma

Congenital cholesteatoma: is known to arise from embryonic cell rests present in the middle ear cavity and temporal bone. These cell rests are known to commonly occur in cerebello pontine angle and petrous apex. In fact, congenital cholesteatoma is seen as a whitish mass behind an intact tympanic membrane.

Derlacki and Clemis laid down the following as criteria to diagnose congenital cholesteatoma:

1. The patient should not have previous episodes of middle ear disease

2. Ear drum must be intact and normal

3. It is purely an incidental finding

4. If discharge and ear drum perforation is present then it should be construed that congenital cholesteatoma has managed to erode the tympanic membrane.

Clinical features: The disorder is an incidental finding. The common location of congenital cholesteatoma is the antero superior quadrant of tympanic membrane, postero superior quadrant being the next common site of involvement. Anteriorly situated congenital cholesteatomas are known to affect the eustachean tube function causing conductive deafness due to middle ear effusion, whereas posterior congenital cholesteatoma is known to cause conductive deafness due to impairment of ossicular chain mobility.

Staging of congenital cholesteatoma:

Staging as suggested by Derlacki and Clemis: They were the first to stage congenital cholesteatoma. They classified congenital cholesteatoma into

1. Petrous pyramid cholesteatoma

2. Cholesteatoma involving the mastoid cavity

3. Cholesteatoma involving the middle ear cavity.

Potsic suggested the following staging mechanism:

Stage I : Single quadrant involvement with no ossicular / mastoid involvement.

Stage II : Multiple quadrant involvement with no ossicular / mastoid involvement

Stage III : Ossicular involvement without mastoid involvement

Stage IV : Mastoid extension

Nelson's staging:

Type I : Involvement of mesotympanum without involvement of incus / stapes

Type II : Involvement of mesotympanum / attic along with erosion of ossicles without extension into the mastoid cavity

Type III : Involvement of mesotympanum with mastoid extension

Staging this disease will help in deciding the modality of treatment and in predicting the long term prognosis.

Acquired Cholesteatoma: can be divided into two types, primary acquired and secondary acquired cholesteatomas.

Primary acquired cholesteatoma: In this condition there is no history of preexisting or previous episodes of otitis media or perforation. Lesions just arise from the attic region of the middle ear.

Secondary acquired cholesteatoma: always follows active middle ear infection which manages to destroy the ear drum along with the annulus. This type of destruction is common in acute necrotizing otitis media following exanthematous fevers like measles etc.

Theories to explain pathogenesis of cholesteatoma:

Various theories have been postulated to explain the pathogenesis of cholesteatoma. They are:

1. Cawthrone theory: This theory suggested by cawthrone in 1963 suggested that cholesteatoma always originated from congenital embryonic cell rests present in various areas of the temporal bone.

2. Theory of immigration: This theory was suggested by Tumarkin. He was of the view that cholesteatoma was derived by immigration of squamous epithelium from the deep portion of the external auditory canal into the middle ear cleft through a marginal perforation or a total perforation of the ear drum as seen in acute necrotizing otitis media.

3. Theory of invagination: This theory was suggested by Toss. He theorized that persistent negative pressure in the attic region causes invagination of pars flaccida causing a retraction pocket. This retraction pocket becomes later filled with desquamated epithelial debris which forms a nidus for the infection to occur later. Common organisms known to infect this keratin debris are Pseudomonas, E. coli, B. Proteus etc.

Toss also classified attic retraction pockets into 4 grades:

1. Grade I: The retracted pars flaccida is not in contact with the neck of the malleus.

2. Grade II: The retracted pars flaccida is in contact with the neck of the malleus to such an extent that it seems to clothe the neck of the malleus.

3. Grade III: Here in addition to the retracted pars flaccida being in contact with the neck of the malleus there is also a limited erosion of the outer attic wall or scutum.

4. Grade IV: In this grade in addition to all the above said changes there is severe erosion of the outer attic wall or scutum.

4. Metaplastic theory: This theory was first suggested by Wendt in 1873. He took into consideration the histological changes seen in various portions of the middle ear cavity. The attic area of the middle ear cavity is lined by pavement type of epithelium. This epithelium undergoes metaplastic changes in response to subclinical infection. This metaplastic mucosa is squamous in nature there by forming a nidus for cholesteatoma formation in the attic region.

Of all the above-mentioned theories, the theory of invagination appears to be the most plausible one currently explaining the various pathologic features of cholesteatoma.

Clinical features of acquired cholesteatoma:

Ear discharge: is scanty and foul smelling. In fact the odor is best described as musty in nature. This is due to the presence of saprophytic infection and osteitis.

Hearing loss: is commonly conductive in nature. Some patients may even surprisingly have a normal hearing despite the presence of a huge cholesteatoma. This normal hearing could be attributed to the bridging effects of cholesteatomatous mass.

Sensorineural hearing loss if present could be attributed to the absorption of toxins through the round window membrane, or may be due to use of ototoxic antibiotics topically on a long-term basis.

Ear ache: if present could be attributed to the presence of co-existing otitis externa, or presence of extradural abscess.

Tinnitus if present may indicate imminent sensorineural hearing loss.

Vertigo may be present if there is erosion of lateral semicircular canal by the cholesteatomatous matrix. Fistula test if performed is positive in these patients.

Fistula test: This test is positive if there is a third window is present in the labyrinth due to the erosion of the labyrinthine bone. This commonly occurs in the lateral semicircular canal area. This test is performed using a snugly fitting siegles pneumatic speculum and slowly applying pressure by compressing the pneumatic bulb. If labyrinthine fistula is present the patient will feel giddy and will have nystagmus.

Facial palsy may indicate erosion of facial nerve canal with involvement of facial nerve.

On examination:

There is destruction of the outer attic wall, with presence of attic perforation. Cholesteatomatous flakes may be seen through the perforation like cotton wool.

There is associated sagging of the posterior superior meatal wall.

Hearing tests indicate conductive deafness commonly if labyrinth is uninvolved. It may turn out to be sensorineural hearing loss if there is associated erosion of the labyrinth.

X ray mastoids may show sclerosis with presence of cavity.

Management:

Since this is a surgical problem modified radical mastoidectomy is advocated in almost all of these patients.

The aims of the surgical procedure is as follows:

1. To exteriorize the disease

2. To create adequate ventilation to the middle ear cavity

3. To create a permanent skin lined cavity exposed to the exterior.

The various modifications of mastoidectomy procedures are discussed elsewhere.

8. Enumerate complications of CSOM. Discuss briefly the management of attico antral type of CSOM

Otitis media is common in developing countries among population of low socioeconomic strata. Advent of potent antibiotics has reduced the incidence of complications of otitis media. Even though the incidence of dangerous complications due to chronic otitis media is on the decrease they are still present.

Types of complications following CSOM:

Complication	Types
Extracranial	Subperiosteal abscess Labyrinthitis Facial nerve palsy
Intracranial	Brain abscess Temporal lobe abscess Cerebellar abscess Meningitis Lateral sinus thrombosis Perisinus abscess Extradural abscess Subdural emphysema

Management:

Since this is a surgical problem modified radical mastoidectomy is advocated in almost all of these patients.

Investigations:

Otomicroscopy:

Examination under microscope will help in accurate assessment of the extent of the disease. Suction of cholesteatomatous flakes can be performed under direct vision as both hands are free.

Pus for culture & sensitivity:

Pus if present in the attic can be sent for culture and sensitivity as this will ensure optimal antibiotic therapy following surgery.

Imaging of temporal bone:

CT scan of temporal bone will help in accurate identification of the extent of the disease. This investigation is a must if intracranial complications are suspected. This will also serve as a road map to the surgeon if surgery is contemplated.

The aims of the surgical procedure are as follows:

1. To exteriorize the disease

2. To create adequate ventilation to the middle ear cavity

3. To create a permeant skin lined cavity exposed to the exterior.

In patients with minimal attic retraction with cholesteatoma and if the neck of the sac is wide enough then periodical suction clearance can be resorted to. This will not only remove cholesteatomatous flakes but will also manage to keep the ear dry.
If then neck of the cholesteatoma sac is narrow then atticotomy can be performed under general anesthesia. In this surgical procedure the outer attic wall can be removed thereby exposing the cholesteatomatous sac in its entirety. If cholesteatoma is found to extend to the anterior epitympanic recess then the head of the malleus can be nipped in order to provide access to this area.
Extension of cholesteatomatous sac into the mesotympanum, hypotympanum and mastoid cavity will have to be managed by modified radical mastoidectomy with reconstruction of the hearing mechanism.

Modified radical mastoidectomy:

This surgery is ideally performed under general anesthesia.
Mastoid bone is exposed via a post aural incision of William Wilde. Henle's spine is identified as this is a vital landmark for the position of antrum which is supposed to lie 1.5 cm below and in front of this bony spiky projection.
Using motor and burr bits mastoid cortex is opened up. Aditus is identified. The same is widened. Posterior canal wall is thinned down. Outer attic wall is removed totally exposing the attic area. Cholesteatoma sac from the attic region is removed in toto. Posterior canal wall is removed. Anterior and posterior buttress of bone is also removed. Facial ridge is lowered up to the level of lateral semicircular canal.
Meatoplasty wide enough to accommodate the thumb of the surgeon is fashioned. The wound is closed in layers after packing the operated cavity with ointment impregnated gauze. This

procedure makes the entire middle ear cavity, aditus, antrum and external auditory canal into a single cavity there by exteriorizing the entire disease process.

9. Complications of Chronic Otitis Media-Factors in the spread of infection, Classification. Discuss in detail about Lateral Sinus Thrombosis.

Complications of otitis media occur as a result of infection spreading from the mucosa of the middle ear cleft to the adjacent structures. Usually the middle ear space is separated from adjacent structures by bone. During preantibiotic era complications commonly followed acute otitis media. With the advent of antibiotics, it the chronic otitis media which is causing complications.

Even though the incidence of these complications have drastically reduced after the advent of potent antibiotics, the morbidity and mortality caused by the complications are still very high.

The complications of otitis media fall under two categories:

1. Complications within the cranium

2. Complications within the temporal bone

Intracranial complications:

These can be further subclassified into extradural and intradural complications.

Extradural complications:

. Extradural abscess

. Meningitis

. Sigmoid sinus thrombosis

Intradural complications:

. Subdural abscess

. Brain abscess

. Otitic hydrocephalus

Intratemporal complications:

. Facial palsy

. Labyrinthitis

. Petrositis

. Subperiosteal abscess

. Internal carotid artery aneurysm

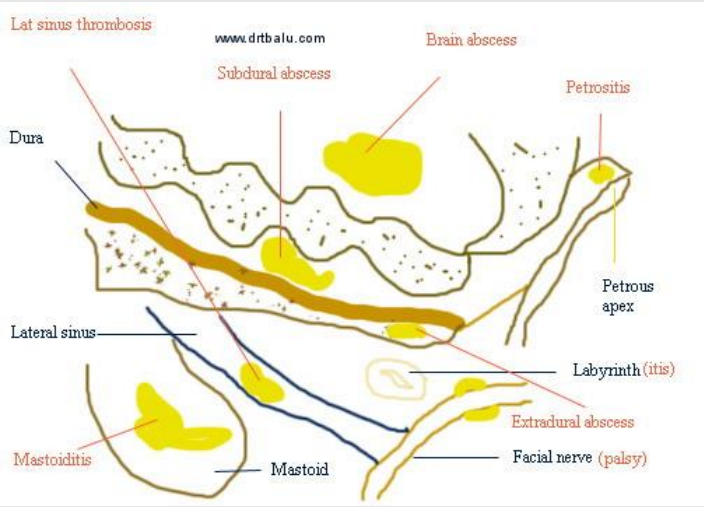
Extratemporal complications:

. Subclavian vein thrombosis

. Luc's abscess

. Citelli's abscess

. Bezold's abscess



Diagrammatic representation of intracranial complications of otitis media

Route of spread of infection from the ear:

Whether acute or chronic, the infection from the middle ear spreads via:

1. Extension through bone that has been demineralized during acute infections, or resorbed by cholesteatoma, or osteitis in chronic disease of the ear. Demineralization is brought about by various enzymes that are released during the acute infections. Cholesteatoma causes bone erosion either due to pressure necrosis, or halisterisis. Halisterisis is also known as hyperemic decalcification. As the term itself suggests decalcification is caused by hyperemia.

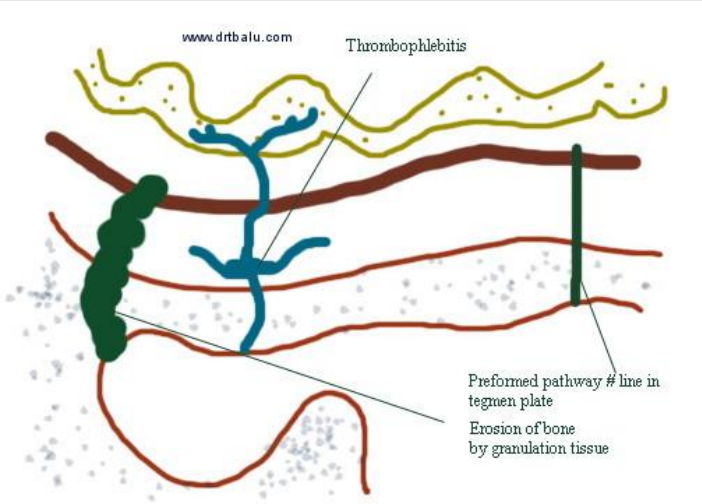
2. Spread through venous channels: Spreading of infected clot within small veins through the bone and dura into the dural venous sinuses. If spread via this route occurs then the infection may find its way into the brain without involving the bone or dura. Thrombophlebitis from the lateral sinus may spread to the cerebellum, and from the superior petrosal sinus may spread to the temporal lobe of the brain.

3. Spread through normal anatomical pathways: Spread may occur through oval / round windows into the internal auditory meatus. Spread may also occur through the cochlear and vestibular aqueducts. Certain areas may have dehiscent bone as a normal variant i.e. bony covering of the jugular bulb, dehiscent areas in the tegmen tympani, and dehiscent suture lines of the temporal bone.

4. Spread may occur through non anatomical bony defects like those caused due to trauma, (accident, surgical) or by erosion due to neoplasia.

5. Spread may occur through surgical defects as caused by fenestration of the oval window during stapedectomy procedures.

6. Spread may occur directly into the brain tissue through the peri arteriolar spaces of Virchow Robin. This spread does not affect the cortical arterioles parse, hence abscess occur in the white matter without the involvement of gray matter of brain.



Diagrammatic representation showing the various routes of spread of infection from the middle ear cavity.

Chronic middle ear disease cause complications by progressive and relentless erosion of the bone barriers, exposing the structures at risk to damage - the facial nerve, labyrinth and the dura. Acute infections cause early complications via the thrombophlebitis mechanism or extension through already available anatomical pathways.

Factors that determine the spread of infection:

I. Patient attributes: Patient's general condition and immunologic status play an important role in the spread of infections.

II. Bacterial attributes like the virulence of the infecting organism is also important. For example, acute infections caused by Strep. pneumoniae type III, and H. Influenza type B have immense potential to spread.

III. Adequacy / Inadequacy of treatment of the middle ear condition may also play an important role.

Extradural abscess:

Is always associated with involvement of dura mater by the spreading disease, constituting pachymeningitis. This is commonly preceded by loss of bone, either through demineralization in acute infection or erosion by cholesteatoma in chronic disease. If the cholesteatoma is non infected it may simply expose the dura without any inflammatory reaction. If cholesteatoma is infected it is associated with formation of granulation tissue over the dura. Dura is tough and resists infection. It attempts to wall off the infection, and collection of pus occur between the dura and the bone. This is known as extradural abscess and is the commonest of all intracranial complications.

A middle cranial fossa extradural mass may strip the dura from bone on the inner surface of squamous temporal bone.

Such an enlarging mass may cause increasing intracranial tension, causing focal neurological signs and papilledema. Sometimes it could erode the skull from inside to the exterior causing a subperiosteal abscess i.e. the classic Pott's puffy tumor. Rarely an extradural abscess may develop medial to the arcuate eminence over the petrous apex. This irritates the Gasserian ganglion of the trigeminal nerve, and the 6th cranial nerve. This produces the classic Gradenigo syndrome (includes facial pain, diplopia and aural discharge). Posterior fossa extradural abscess is limited by the attachments of the dura laterally to the sigmoid sinus. Posterior extension of this abscess around the sigmoid sinus produces the Perisinus abscess. This could also extend to the neck through the jugular vein.

Clinical features:

Depends on the site of the abscess, its size, duration and rate of development. In most patients the symptoms are vague, and nonspecific. Sometimes it could be a incidental finding during mastoid surgery. The common complaint of the patient being headache accompanied by malaise. If the abscess communicates with the middle ear the patient may have interim relief following an episode of aural discharge.

Lateral sinus thrombosis:

Thrombophlebitis can develop in any of the veins adjacent to the middle ear cavity. Of these the lateral sinus, which comprise of the sigmoid and transverse sinuses is the largest and most commonly affected. Initially it is usually preceded by the development of an extradural Perisinus abscess. The mural thrombus partly fills the sinus. The clot progressively expands and eventually occlude the lumen. The clot may later become organized, and partly broken down and may even be softened by suppuration. During this stage there is a release of infecting organism and infected material into the circulation causing bacteremia, septicemia and septic embolization.

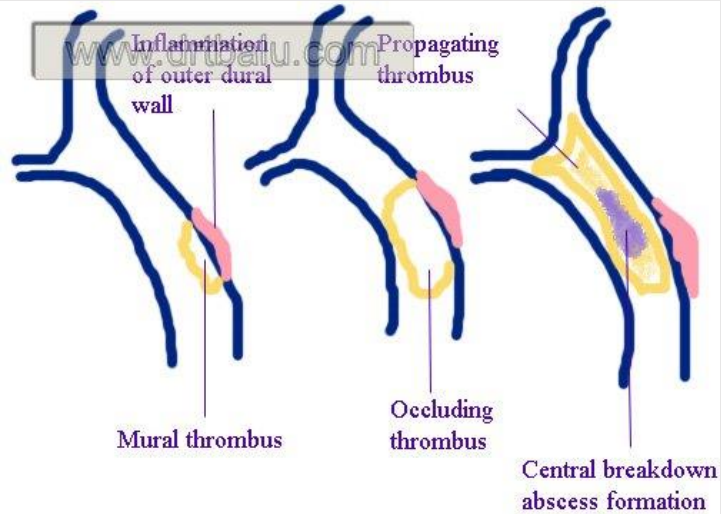


Image showing various stages of lateral sinus thrombosis

Extension / propagation of the thrombus upwards may extend to the confluence of the sinuses, and beyond that to the superior sagittal sinus. Invasion of the superior and inferior petrosal sinuses may cause the infection to spread to the cavernous sinus. This spread of venous thrombophlebitis into the brain substance accounts for the very high association of this complication with brain abscess. Downward progression of thrombus into and through the internal jugular vein can reach the subclavian vein.

The harmful effects are caused by the release of infective emboli into the circulation, and also from the hemodynamic disturbances caused to venous drainage from inside the cranial cavity. The use of antibiotics has greatly reduced the incidence of lateral sinus thrombosis these days.

Formerly it was commonly associated with acute otitis media in childhood; now it is commonly seen in patients with chronic ear disease. In the preantibiotic era the commonest infecting organism was beta hemolytic streptococci. This organism was known to cause extensive destruction of red blood cells causing anemia. Now a days the infection is by a mixed flora.

Clinical features:

The patients manifest with severe fever, wasting illness in association with middle ear infection. The fever is high and swinging in nature, when charted it gives an appearance of 'Picket fence'. It is always associated with rigors. The temperature rose rapidly from 39 - 40 degree Centigrade. Headache is a common phenomenon, associated with neck pain. The patient appears emaciated and anemic. When the clot extended down the internal jugular vein, it will be accompanied by perivenous inflammation, with tenderness along the course of the vein. This tenderness descended down the neck along with the clot, and would be accompanied by perivenous oedema or even suppuration of the jugular lymph nodes. Perivenous inflammation around jugular foramen can cause paralysis of the lower three cranial nerves. Raised intracranial pressure produce papilledema and visual loss. Hydrocephalus could be an added complication if the larger or the only lateral sinus is occluded by the thrombus, or if the clot reaches the superior sagittal sinus. Extension to the cavernous sinus can occur via the superior petrosal sinus, and may cause chemosis and proptosis of one eye. If circular sinus is involved it could spread to the other eye. The propagation of the infected emboli may cause infiltrates in the lung fields, and may also spread to joints and other subcutaneous tissues. These distant effects usually developed very late in the disease; these could be the presenting features if the disease is insidious in onset. Masking by antibiotics could be one of the causes. Patients always feel ill, and persisting fever is usual. The patients may have ear ache, in association with mastoid tenderness, and stiffness along the sternomastoid muscle. The presence of anemia is rare now a days. Papilledema is still a common finding. Other coexisting intracranial complications must be expected in more than 50 percent of patients.

Extension of infected clot along the internal jugular vein is always accompanied by tenderness and oedema along the course of the vein in the neck, and localised oedema over the thrombosed internal jugular vein may still be seen. One rare finding is the presence of pitting oedema over the occipital region, well behind the mastoid process, caused by clotting within a large mastoid emissary vein, this sign is known as the Griesinger's sign. In fact, there is no single pathognomonic sign for lateral sinus thrombosis and a high index of suspicion is a must in diagnosing this condition.

Investigations:

A lumbar puncture must be performed, if papilledema does not suggest that raised intracranial pressure may precipitate coning. Examination of CSF is the most efficient way of identifying meningitis. In uncomplicated lateral sinus thrombosis, the white blood count in the CSF will be low when the cause is chronic middle ear disease, and somewhat raised in acute otitis media. The CSF pressure is usually normal. The variations in the level of CSF proteins and sugar are not useful.

Queckenstedt test: This is also known as Tobey - Ayer test. This is recommended whenever lumbar puncture for a possible intracranial infection is performed. The test involves measurement of the CSF pressure and observing its changes on compression of one or both internal jugular veins by fingers on the neck. In normal humans' compression of each internal jugular vein in turn is followed by an increase in CSF pressure, of about 50 - 100mm above the normal level. When the pressure over the internal jugular vein is released then there is a fall in the CSF pressure of the same magnitude. In patients with lateral sinus thrombosis pressure over the vein draining the occluded sinus cause either no increase, or a low slow rise in CSF pressure of 10 - 20 mm. Compression of the normal internal jugular vein produces a rapid pressure rise ranging from 2 - 3 times the normal level. This test is also prone for false negative results due to the presence of collateral channels draining the venous sinuses. False positives can occur if a normal lateral sinus is small or absent that creating an erroneous impression of lateral sinus thrombosis.

CT scanning: is an essential investigation in these patients. It may show filling defects within the sinus, and increased density of fresh clots. When contrast materials like Iothalamate (conray) is used failure of opacification of the affected lateral sinus may become evident. The presence of septic thrombosis shows intense inflammatory enhancement of the sinus walls and of the adjacent dura. This enhancement of the walls, but not of the contents of the sinus constitutes the empty triangle or 'delta' sign. It can also exclude accompanying complications like brain abscess and subdural empyema.

Angiography: is a definitive investigation of lateral sinus thrombosis. It helps to demonstrate the obstruction, its site and the anatomical arrangement of the veins. There is an impending risk of displacing the infected thrombus.

Arteriography: performed with radio opaque dye injected into the carotid artery can show the venous outflow during the venous phase. This can be clearly visualized in digital subtraction angiography. This technique involves precise superimposition of a negative arteriogram on a positive film of bone structures. This effectively cancels out the skeletal image thus clearly revealing the vascular pattern.

MRI: Is sufficiently diagnostic hence angiography can be avoided if MRI could be taken. Established thrombus shows increased signal intensity in both T1 and T2 weighted images. MRI can also be used to show venous flow. Gadolinium enhancement may show a delta sign comparable with that seen-on CT scans

Management: Treatment involves administration of antibiotics, together with exposure of lateral sinus and incision of the sinus and removal of its contents. Anticoagulants are not advocated at present. Before exposing the lateral sinus and clearing its contents it is imperative to clear the ear of any infections by doing a cortical mastoidectomy. The involved sinus may feel firm, appear white and opaque thus suggesting occlusion of the lumen with clot. Dissemination of clot can be prevented by ligation of the affected internal jugular vein. Now a days the only indication of internal jugular vein ligation is the presence of septicemia which is resistant to antibiotics.

10. Enumerate the complications of Chronic Suppurative Otitis Media. Write in detail about lateral sinus thrombosis.

Already discussed …..

11. 50 year old male presents with history of left ear discharge for 3 years. What are the routes of spread of infection to the intracranial structures in this patient? Enumerate the intracranial complications expected. Describe in detail about otogenic brain abscess.

Whether acute or chronic, the infection from the middle ear spreads via:

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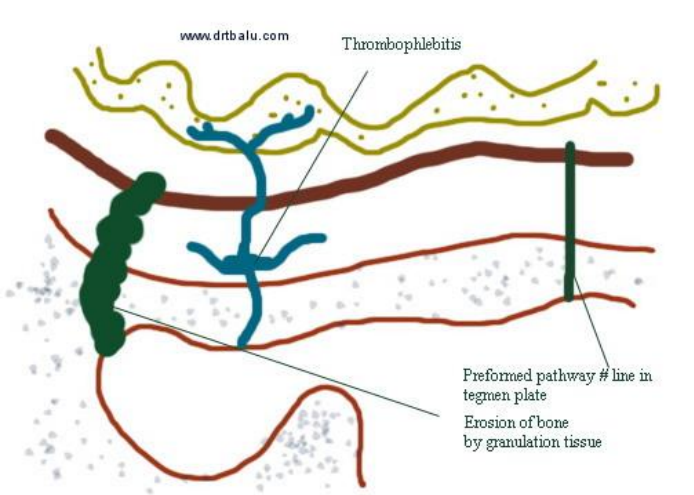


Diagrammatic representation showing the various routes of spread of infection from the middle ear cavity.

Intracranial complications:

These can be further subclassified into extradural and intradural complications.

Extradural complications:

. Extradural abscess

. Meningitis

. Sigmoid sinus thrombosis

Intradural complications:

. Subdural abscess

. Brain abscess

. Otitic hydrocephalus

Brain abscess:

Otogenic brain abscess always develop in the temporal lobe or the cerebellum of the same side of the infected ear. Temporal lobe abscess is twice as common as cerebellar abscess. In children nearly 25% of brain abscesses are otogenic in nature, whereas in adults who are more prone to chronic ear infections the percentage rises to 50%. The routes of spread of infection has already been discussed above, the commonest being the direct extension through the eroded tegmen plate. Although dura is highly resistant to infection, local pachymeningitis may be followed by thrombophlebitis penetrating the cerebral cortex, sometimes the infection could extent via the Virchow - Robin spaces in to the cerebral white matter. Cerebellar abscess is usually preceded by thrombosis of lateral sinus. Abscess in the cerebellum may involve the lateral lobe of the cerebellum, and it may be adherent to the lateral sinus or to a patch of dura underneath the Trautmann's triangle.

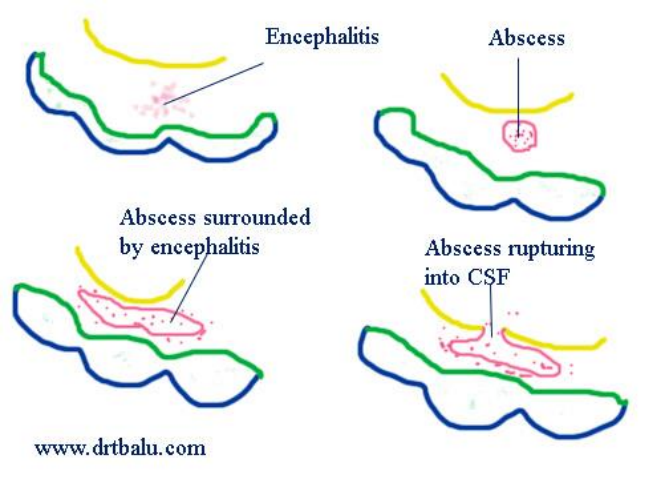


Diagram illustrating stages of brain abscess

Stages of formation of brain abscess:

Stage of cerebral oedema: This is infact the first stage of brain abscess formation. It starts with an area of cerebral oedema and encephalitis. This oedema increases in size with spreading encephalitis.

Walling off of infection by formation of capsule: Brain attempts to wall off the infected area with the formation of fibrous capsule. This formation of fibrous tissue is dependent on microglial and blood vessel mesodermal response to the inflammatory process. This stage is highly variable. Normally it takes 2 to 3 weeks for this process to be completed.

Liquefaction necrosis: Infected brain within the capsule undergoes liquefactive necrosis with eventual formation of pus. Accumulation of pus cause enlargement of the abscess.

Stage of rupture: Enlargement of the abscess eventually leads to rupture of the capsule containing the abscess and this material finds its way into the cerebrospinal fluid as shown in the above diagram.

Cerebellar abscess which occupy the posterior fossa cause raised intra cranial tension earlier than those above the tentorium. This rapidly raising intra cranial pressure cause coning or impaction of the flocculus or brain stem into the foramen magnum. Coning produces impending death. If the walling off process (development of capsule) is slow, softening of brain around the developing abscess may allow spread of infection into relatively avascular white matter, leading to the formation of secondary abscesses separate from the original or connected to the original by a common stalk. This is how multilocular abscesses are formed. Eventually the abscess may rupture into the ventricular system or subarachnoid space, causing meningitis and death.

The mortality rate of brain abscess is around 40%, early diagnosis after the advent of CT scan has improved the prognosis of this disease considerably.

The bacteriological flora is usually a mixture of aerobes and obligate anaerobes. Anaerobic streptococci are the commonest organisms involved. Pyogenic staphylococci is common in children. Gram negative organism like proteus, E coli and Pseudomonas have also been isolated.

Clinical features:

The earliest stage where the brain tissue is invaded (stage of encephalitis) is marked by the presence of headache, fever, malaise and vomiting. Drowsiness eventually follows. These early features may be masked by the complications such as meningitis or lateral sinus thrombosis. If this stage progresses rapidly to generalized encephalitis before it could be contained by the formation of the capsule, drowsiness may progress to stupor and coma followed by death. Usually the period of local encephalitis is followed by a latent period during which the pus becomes contained within the developing fibrous capsule. During this latent phase the patient may be asymptomatic.

During the next state (stage of expansion) the enlarging abscess first cause clinical features due to the alteration of CSF dynamics, and site-specific features may also be seen due to focal neurological impairment. The pulse rate slows with rising intracranial pressure, the temperature may fall to subnormal levels. Drowsiness may alternate with periods of irritability. Papilledema is also found due to elevated CSF pressure.

Clinical features also vary according to the site of involvement. Hence the differences that are seen between the cerebral and cerebellar abscess.

Cerebral (Temporo sphenoidal abscess):

A cerebral abscess in the dominant hemisphere often cause nominal aphasia, where in the patient has difficulty in naming the objects which are in day to day use. He clearly knows the function of

these objects. Visual field defects arise from the involvement of optic radiations. Commonly there is quadrantic homonymous hemianopia, affecting the upper part of the temporal visual fields, more rarely it may also involve the lower quadrants. The visual field loss are on the side opposite to that of the lesion. This can be assessed by confrontation method. Upward development affects facial movements on the opposite side, and then progressively paralysis of the upper and lower limbs. If the expansion occurs in inward direction then paralysis first affects the leg, then arm and finally the face.

Cerebellar abscess:

The focal features associated with cerebellar abscess is weakness and muscle incoordination on the same side of the lesion. Ataxia causes the patient to fall towards the side of the lesion. Patient may also manifest intention tremors which may become manifest by the finger nose test. This test is performed by asking the patient to touch the tip of the nose with the index finger first with the eyes open and then with the eyes closed. The patient may often overshoot the mark when attempted with the eyes closed in case of cerebellar abscess. The patient may also have spontaneous nystagmus. Dysdiadokinesis is also positive in these patients.

Investigations:

CT scan and MRI scans are the present modes of investigation. Scan is ideally performed using contrast media. These scans not only reveal the position and size of the abscess, the presence of localised encephalitis can be distinguished from that of an encapsulated abscess. Associated conditions such as subdural abscess, and lateral sinus thrombosis can also be seen.

Lumbar puncture:

Is fraught with danger because of the risk of coning. Lumbar puncture must be performed in these patients only in a neurosurgical unit where immediate intervention is possible if coning occurs.

Treatment: involves use of large doses of antibiotics. Ideally the abscess should be controlled neurosurgically and with antibiotics. After the patient recovers mastoidectomy is performed to remove the focus of infection. Abscess can be drained by placement of burr holes, and excision of the necrotic tissue along with the capsule.

12. Discuss the aetiopathology of conductive deafness. Describe the types of tympanoplasty

Etiopathology of conductive deafness:

Any condition that impedes conduction of sound from the environment into the middle ear cavity cause conductive hearing loss. Conditions that causes obstruction to transmission of sound in the external auditory canal (impacted cerumen) can cause conductive deafness. Conditions that reduce

the size of the vibrating ear drum (perforations) can cause conductive deafness. Conditions that prevent coupling of sound from the ear drum to the middle ear ossicles can cause conductive deafness. Conditions that affect the mobility of the middle ear ossicles can cause conductive deafness.

Causes of conductive deafness:

Outer ear causes:

Congenita microtia / Anotia

Otitis externa

Trauma

Squamous cell carcinoma

Exostosis

Osteoma

Cerumen / FB

External canal stenosis

Psoriasis

Middle ear causes

Congenital atresia / ossicular chain malformation

Otitis media

Cholesteatoma

Otosclerosis

Perforation of ear drum

Temporal bone trauma

Otitis barotrauma

Glomus tumors

Tympanoplasty is defined as the surgical procedure which enables reconstruction of middle ear cavity and the conducting ossicular system (tympano- ossicular system). Wullstein and Zollner classified Tympanoplasty according to the type of ossicular
reconstruction needed. Five types of Tympanoplasty have been classified.

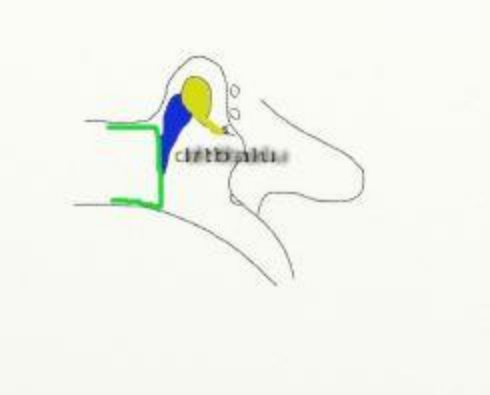
Type I Tympanoplasty: This is indicated in patients with presence of all the middle ear ossicles with normal mobility. Ossicular chain reconstruction is not needed in these patients. Efforts are made to close the perforated ear drum using temporalis fascia graft (Hong Kong flap). This procedure is also known as myringoplasty.

Type II Tympanoplasty: In this procedure the tympanic membrane is grafted to the intact incus and stapes. This procedure is very rarely used, since it is very rare for erosion of the handle of malleus to be present alone without the involvement of other ossicles. The neotympanum created is draped over the existing incus and stapes. There is a certain amount of obliteration of middle ear space. Since the ossicular chain lever ratio is not normally maintained in these patients, they tend to have at least 30 dB hearing loss even after a successful surgery.

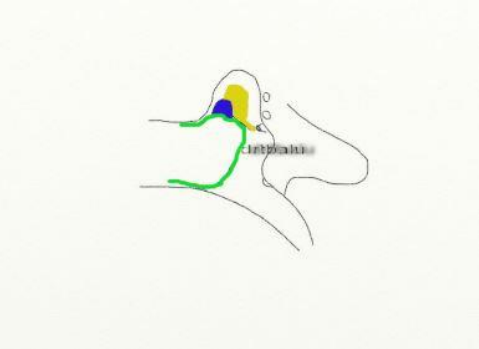
Type III Tympanoplasty: This technique is used only when a mobile suprastructure of stapes alone is present. In this surgical procedure the tympanic membrane graft is draped over the mobile suprastructure of stapes. This is also known as Columella effect. This type of middle ear is commonly seen in birds. The middle ear space is really nonexistent. Even after successful surgery these patients still manifest with 30 – 40 dB hearing loss.
This surgical procedure is useful in patients without malleus and incus. Incus has the most precarious blood supply among the three ossicles.

Type IV Tympanoplasty: This surgical procedure is performed in patients only with mobile foot plate of stapes. The grafted ear drum is draped over the mobile foot plate. In these patients there is virtually no middle ear space at all. The grafted ear drum virtually drapes the promontory.
Even after successful surgery these patients still have about 40 – 50 dB hearing loss.

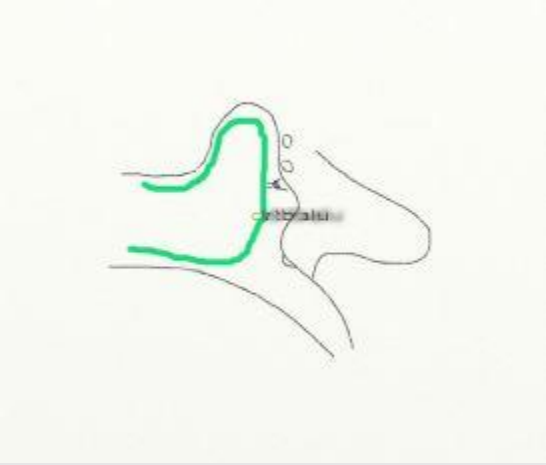
Type V Tympanoplasty: In this surgical procedure a third window is created over the lateral semicircular canal. (Fenestra over lateral canal). This surgical procedure is outdated these days.



Type I tympanoplasty



Type II tympanoplasty



Type III tympanoplasty

Type IV tympanoplasty

13. Describe the etiology, pathology, clinical features and management of otosclerosis

Synonyms: Otospongiosis, Ankylosis of foot plate of stapes.

Definition: Otosclerosis is a hereditary localised disease of the bone derived from the otic capsule characterised by alternating phases of bone resorption and new bone formation. The mature lamellar bone is removed by osteoclasis and replaced by woven bone of greater thickness, cellularity and vascularity.

History: In 1741 Antonio Valsalva described ankylosis of stapes while doing a postmortem on the body of a deaf patient.
In 1894, Adam Politzer introduced the term "otosclerosis" and described the histopathology of the disease for the first time.
In 1912 Siebenmann introduced the term otospongiosis to denote active otosclerotic foci.

Pathophysiology:

The primary pathological change occurs in the bony labyrinth with secondary effects upon middle ear and inner ear function. The otosclerotic focus may be asymptomatic, or if present in the area of foot plate of stapes it may give rise to ankylosis of foot plate with resultant conductive deafness. Otosclerotic foci may involve other portions of labyrinth causing sensori neural hearing loss and vestibular abnormalities.
A combination of effects are possible in otosclerosis. They are:
Histological otosclerosis: Otosclerotic foci doesnot cause any symptoms and hence known as histological otosclerosis.
Stapedial otosclerosis: is the classical otosclerosis with fixation of stapedial foot plate causing conductive deafness.
Cochlear otosclerosis: The foci involves the cochlea causing sensorineural deafness.
Combined otosclerosis: Here in addition to fixation of foot plate of stapes there is also associated sensorineural hearing loss due to involvement of cochlea.

Otospongiosis: European otologists prefer to use this term to indicate the active phase of otosclerosis.

Incidence: Otosclerosis is common in caucasian races. It is rarely found in Mongoloid and Negro population.

Sex incidence: In clinical practice otosclerosis is seen more often in women than in men. The ratio was found to be 2:1. Nowadays the authors believe the apparent female preponderance may be due to the fact that unilateral otosclerotic deafness is less common in women than in men. Noticeable deterioration in hearing also occur during pregnancy due to hormonal changes. Deafness due to otosclerosis may be initiated or made worse by pregnancy.Causative factors / etiology: Many theories have been proposed to explain the etiological factors of otosclerosis. They are:

1. Metabolic

2. Immune disorders

3. Vascular disease

4. Infection (Measles) currently accepted

5. Trauma : The petrous bone doesnot have regenerative capacity. This is because of the fact that the enzymes released during reparative phase are very toxic to the inner ear hair cells.
Pockets of tissue capable of regeneration may be sequestered in various portions of labyrinthine bone. These tissue could be activated by the presence of regenerative enzymes in the blood following bone fracture elsewhere in the body.

6. Temporal bone abnormalities (congenital)
Genetic factors predisposing to otosclerosis: The tendency for otosclerosis to run in families has been documented. Authors have postulated an autosomal dominant mode of inheritance with varying degrees of penetration.
Otosclerosis is associated with osteogenesis imperfecta in 0.15 % of cases. This is known as Van der Hoeve syndrome or Adair - Dighton syndrome.

Sites affected by otosclerosis: The commonest site for apperance of otosclerotic bone is fissula ante fenestrum. This fissula is constantly seen in the labyrinthine capsule lying in front of the oval window. This area may contain unossified cartilage persisting even in adults. This area was referred to as Cozzolino's zone by Perozzi in memory of his teacher. Otosclerosis may occur in this area due to bony ossification of the cartilage.
Residual cartilage may be present in the following areas of labyrinth:
1. Fissula ante fenestram
2. Fissula post fenestram
3. Intracochlear

4. Round window
5. Semicirular canals
6. Petrosquamous suture
7. Base of styloid process

In normal development the fissula appears as fibrous connective tissue bundle joining the vestibule with the tympanic cavity. This fibrous tissue is encased in primary cartilage which later gets replaced by bone. From the fissula the bone acquires a connective tissue lining which later becomes converted into perichondrium. The fissula is reduced in size by the production of new secondary cartilage from the perichondrium. These changes are completed by birth. The secondary cartilage remains throughout life as a stable, dormant cartilage and hence may even be considered as normal structure. It is only when this secondary cartilage gets ossified otosclerosis occur (Bast & Anson).

Otosclerotic changes may appear as a result of interaction between bone growth promoting substances circulating in the blood stream, and the unstable cartilagenous elements in the capsule of the labyrinth. Otosclerosis is often seen at times when the bone growth promoting substances are circulating in the blood as in pregnancy and following fractures of other bones.

Histopathology of otosclerosis:

The normal endochondral bone of labyrinthine capsule in which otosclerotic focus begins is compact in type. Ultra-structurally, lamellae composed of fine fibrils lying in a ground substance are concentrically disposed around haversian canals containing blood vessels and connective tissue. In otosclerosis there is sharply defined new bone formations that could be differentiated from normal bone by their deep carmine stain and by marked enlargement of bone spaces and haversian canals. The following are the changes which occur in a otosclerotic foci:

1. Focal / diffuse replacement of normal compact bone by irregular, loose cancellous bone with more deeply staining lamellae.
2. There is an associated increase in size of Haversian canals, cell spaces and marrow spaces with corresponding increase in vascularity. The blood vessels are frequently surrounded by a narrow margin of blue staining material that Manassee described first as Blue Mantle zone.
3. Increase in osteocytes, and appearance of osteoblasts and osteoclast cells.

Histologically otosclerosis may be classified into:

1. Early focal otosclerosis
2. Diffuse active otosclerosis
3. Quiescent otosclerosis
4. Cochlear otosclerosis

Early focal otosclerosis: In this type the abnormalities are localised to one or two small areas of an otherwise normal foot plate section. The abnormal areas show an enlarged marrow space surrounded by a blue staining area on H&E staining.

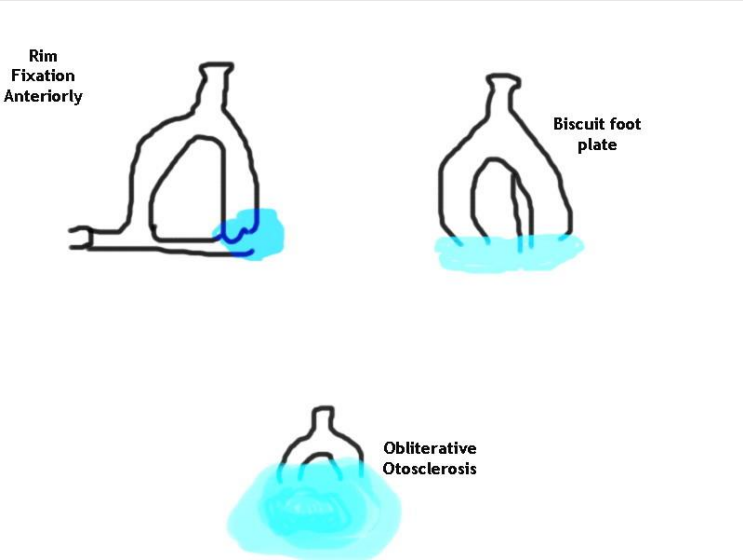
Diffuse active otosclerosis: In this type there is abnormal vascularity with a great increase in size and number of marrow spaces. Most of these spaces are lined by osteoblasts. In places around the circumference of the marrow spaces there is a scalloped appearance where bone has been recently absorbed. The number of osteocytes is greatly increased.

Quiescent otosclerosis: Here even though there may be some increase in the size and number of marrow spaces there is no evidence of bone formation or bone destruction. Osteoblasts and osteoclasts are only occasionally seen. This could be considered as a burnt-out phase of the disease spectrum.

Cochlear otosclerosis: This condition causes pure sensorineural deafness without stapes fixation. Otosclerotic foci may occur in the otic capsule without the involvement of stapedial foot plate. The process of bone erosion and new bone formation which occur in otosclerosis releases enzymes like amylase, SGOT, SGPT etc. which can enter into the endolymph via the round window membrane. These enzymes are toxic to the sensitive hair cells of the cochlea causing sensorineural hearing loss.

Clinical types of otosclerosis: Classification of various clinical types of otosclerosis is based on microscopic appearances of the diseased foot plate.

Rim fixation: Here the otosclerotic foci starts from the anterior portion of the oval window niche. It gradually expands to involve the anterior portion of the foot plate causing fixation of the anterior portion of the foot plate only leaving the centre of the plate free.



Diagrammatic representation of various clinical types of otosclerosis

Biscuit foot plate: This type occurs less frequently. The focus originates in the foot plate itself and as it expands it gives rise to the biscuit or rice grain foot plate with delineated margins.

Obliterative otosclerosis: Rarely a large mass of otosclerotic new bone fills up the oval window niche obscuring the entire foot plate. This condition is known as obliterative otosclerosis. It is a difficult condition to manage surgically.
Clinical features:

Deafness: Typically, deafness in otosclerosis is bilateral and gradually increasing in nature. Unilateral otosclerosis occur in 15% of patients. Frequently it occurs between third and fifth decades of life. In majority of cases the deafness is conductive in nature. The deafness will not be noticed by the patient till the loss reaches 30 dB level. This is the deafness level in which understanding speech becomes difficult. These patients may hear better in noisy environment because the speaker has a tendency to raise his voice because of excessive ambient noise. This phenomenon a feature of otosclerosis is known as Paracusis Willisii.
In cochlear otosclerosis the deafness is purely sensorineural in nature. Some patients may have both conductive and sensorineural hearing loss (mixed deafness) because of the tendency of bone reparative enzymes to damage the inner hair cells.

Patients with otosclerosis have characteristically quiet voice with good tone and the change in speech pattern may be detected only by close relatives.
Tinnitus: is a common symptom and occasionally could be the only presenting feature. The presence of tinnitus should alert the physician about the presence of cochlear otosclerosis. It could also be seen in some patients without cochlear degeneration due to abnormally increased vascularity of the otosclerotic bone. Mostly tinnitus indicates sensorineural degeneration. Tinnitus may be unilateral or bilateral. It is usually roaring in nature.

Vertigo: Transient attacks of vertigo is not uncommon in patients with otosclerosis. This could be due to the action of toxic enzymes released by the lesion into the vestibular labyrinth. These patients may even have coexisting Meniere's disease.

Clinical examination: The ear drum in these patients is normal (mint condition). Rarely during active phase of the disease, the increased vascularity of the promontory may be seen through the ear drum. This sign is known as Flamingo’s flush sign or Schwartz's sign. This indicates otospongiosis (active otosclerosis).

Hearing assessment can be done using tuning forks. For detailed description of tuning fork tests read the chapter titled clinical examination of the ear.

Pure tone audiometry will show precisely the amount and type of hearing loss. The presence of Carhart's notch is a classic audiometric feature in these patients. This Carhart's notch is present in bone conduction. There is a dip centered around 2000 Hz. This is actually an artifact. In cochlear otosclerosis audiometry reveals sensorineural hearing loss.

Stapes fixation causes an elevation in the bone conduction thresholds of 5dB at 500Hz, 10dB at 1000 Hz, 15 dB at 2000 Hz, and 5 dB at 4000 Hz. In the audiogram this creates a peculiar pattern known as Cookie bite audiogram. The bone conduction audiogram appears like a cookie having been bitten.

Impedance audiometry is an useful investigation to diagnose otosclerosis. Middle ear compliance is often reduced. When stapes is fixed stapedial reflex is absent. The typical impedance curve is As curve.

All these patients with pure conductive deafness have excellent speech discrimination thresholds.

Management:

Medical: The aim of medical management is to convert an active otosclerotic foci into an inactive or quiescent focus. Fluoride is the drug of choice.

Indications of fluoride therapy:

1. Patients with surgically confirmed otosclerosis who show progressive sensorineural deafness disproportionate to age.

2. Patients with pure sensorineural loss with family history, age of onset, audiometric pattern and good auditory discrimination indicate the possibility of cochlear otosclerosis.

3. Patients with radiological demonstration by CT scan of spongiotic changes in the cochlear capsule

4. Patients with positive Schwartz sign.

5. Post op treatment: If patients are found to have an active focus during surgery, fluoride therapy is prescribed for 2 years.

Contraindications of fluoride therapy:

1. Patients with chronic nephritis and nitrogen retention

2. Patients with chronic rheumatoid arthritis

3. Patient who are pregnant / lactating

4. In children before skeletal growth has been completed

5. Patients who show allergy for the drug

6. Patients with skeletal fluorosis

Fluorides act on otosclerotic foci by reducing osteoclastic bone resorption with a corresponding increase in osteoblastic bone formation. Fluoride also has antienzymatic action thereby it can neutralize the toxic enzymes released from the otospongiotic foci.

Dose: A daily dose of 50 mg of sodium fluoride is given for a period of 2 years. In patients with positive Schwartz's sign the dose can be increased up to 75 mg per day.

Adverse effects of sodium fluoride therapy:

1. Gastric disturbance

2. Arthritis

3. Skeletal fluorosis

Surgical treatment: Stapedectomy

Hearing aids: These patients will benefit from the use of hearing aids if surgery is not acceptable to the patient or if it is risky. There is always a 1% risk of producing a dead ear during surgery even in the best of hands.

14. Define Meniere's disease. Discuss the etiopathology, clinical features and management of the same.

In late 19th century Prosper Meniere described a condition characterized by ear block, tinnitus, and vertigo. He even correctly identified the site of lesion to be labyrinth. It won't be an understatement to say that precious little has been added to the knowledge and understanding of the disorder since then. Prosper Meniere in fact lived far ahead of his time. He was born in 1799 in France. In 1848 he began to translate the text book on hearing loss authored by Kramer. The book was written in German. This kindled his interest in otology. In his classical seminal reports, he goes on to describe a series of patients who presented with neural deafness, with hearing loss greater for low frequencies. Deafness was commonly unilateral in these patients. These patients usually present with tinnitus, vertigo, nausea and vomiting. He reported that these patients had a normal ear drum. He also reported that these symptoms were completely reversible.

The exact etiology of Meniere's disease is unknown, however various etiologies have been suspected.

Etiological factors of Meniere's disease:
1. Genetic
2. Anatomical causes
3. Traumatic
4. Viral infection
5. Allergy
6. Autoimmunity
7. Psychosomatic and personality disorders.

Etiological features of secondary endolymphatic hydrops:

1. Developmental insult
2. Abnormal metabolic / endocrine states
3. Syphilis
4. CSOM
5. Viral infection
6. Autoimmunity
7. Otosclerosis
8. Abnormal fluid balance
9. Leukemia

Genetic causes:
Familial tendency has been observed in nearly 20% of patients with Meniere's disease. Studies have demonstrated that Meniere's disease is attributable to a mutation on chromosome 6. Transmission is supposedly autosomal dominant in nature.
Anatomical causes:
1. Small vestibular aqueduct: Radiological studies of patients with Meniere's disease demonstrated a smaller vestibular aqueduct in nearly 10% of patients. Considerable difficulty was experienced in visualizing the endolymphatic duct / sac in ears affected by Meniere's disease.

2. Reduction in the rugose portion of the endolymphatic sac has been detected in a significant number of Meniere's disease patients.
Traumatic causes:
Association between Meniere’s disease and trauma (physical / acoustic) has been implicated in Meniere's related symptoms. Trauma may cause biochemical dysfunction in the cells of the membranous labyrinth, or may simply cause release of debris into the endolymph causing obstruction of the endolymphatic duct / sac.
Viral infection: Damage to the endolymphatic sac and duct by viral infection has been proposed as an etiological mechanism in Meniere's disease. Neurotrophic viruses have been implicated in this process.

Researches in Sweden have identified a higher reactivity to herpes simplex virus type I in patients with Meniere's disease. DNA of herpes virus has been isolated from the endolymphatic sac of affected individuals. Circulating levels of group specific proteins of enterovirus VP1 have been found to be elevated in patients with active disease. Absence of the protein can be correlated with remission.

Allergy:

Nearly 80% of patients with Meniere's disease have history of childhood allergy. Both food and inhalant allergens have been implicated. Treatment of allergy with immunotherapy caused a remission of the disease in majority of these patients. IgE changes have not been demonstrated in these patients causing a doubt regarding this etiological factor.

Autoimmunity:

Autoimmunity as an etiological factor has been considered in Meniere's disease. The endolymphatic sac has been shown to contain immunoglobulin and lymphocytes and is capable of generating immune response. Immunoglobulins have been found to be deposited in the walls and the luminal fluid of endolymphatic sacs of Meniere's disease. Elevated levels of immune complexes have been demonstrated in nearly 20% of patients with bilateral Meniere's disease. ESR has been found to be elevated. Circulating immune complexes have been found to be elevated in Meniere's disease. Antibodies directed against type II collagen have been found in the serum of these patients.

Psychosomatic features:

Patients with Meniere's disease have an increased incidence of personality disorders.

Secondary hydrops possible etiological factors involved:
Developmental insult:
Developmental insults can cause symptomatic endolymphatic hydrops. Mondini's deformity is commonly associated with secondary Meniere's disease. The true Mondini deformity occurs secondary to an arrest at the seventh week. Only the basilar turn of the cochlea has undergone complete development.
Typically, the interscalar septum or osseous spiral lamina is incomplete; resulting in a confluency of the apical and middle cochlea turns (incomplete partition). The vestibule and semicircular canal may or may not be normal.

Abnormal metabolic / endocrine states:

Certain abnormal metabolic and endocrine states predispose to the development of secondary hydrops. Both high and low blood glucose levels have been associated with dysfunction of inner ear. The hearing may fluctuate with blood glucose levels. It has been demonstrated that induced hypoglycemia resulted in a decrease in potassium concentration of endolymph associated with a

rise in endolymphatic sodium levels. These changes resemble the changes seen in Meniere's disease. Hyperlipoproteinemia has been associated with Meniere's like symptoms.
Endocrine disorders causing secondary hydrops:

1. Hypothyroidism

2. Nephrogenic diabetes insipidus

3. Adrenal insufficiency Syphilis:

Syphilis is a known cause of endolymphatic hydrops. This could be caused due to the inner ear's reaction to syphilitic organism.

Meniere's disease by definition is idiopathic endolymphatic hydrops characterized by roaring tinnitus, vertigo, fluctuating hearing loss. Even though sometimes erroneously used interchangeably, Meniere's disease is different from endolymphatic hydrops. It should be borne in mind that the term endolymphatic hydrops indicates the underlying pathophysiological mechanism of Meniere's disease. Endolymphatic hydrops can in fact be classified as primary and secondary according to the causative factors involved. Primary endolymphatic hydrops is in fact the classic Meniere's disease where in the underlying etiology is unknown. In secondary hydrops the etiopathogenesis of the underlying disorder is clearly elicitable.

Stages of Meniere's disease and their possible pathogenesis:
Any theory postulating the pathogenesis of Meniere's disease should be able to explain the clinical stages of Meniere's disease, Lermoyez's syndrome and Tumarkin attacks. It should also be able to explain the vertigo that occurs in congenital syphilis and secondary endolymphatic hydrops. In early stages of Meniere's disease, attacks of vertigo commonly predominate. Hearing is affected only transiently. During this stage the sac is said to be functioning well and has the ability to completely clear the duct. Hydrops occurs only briefly before each attack of vertigo and is completely cleared after each episode. In later stages of Meniere's disease, glycoprotein secretion causes some amount of functional damage within the sac reducing its ability to reabsorb excess fluid. During this stage there is persistent endolymphatic hydrops within the cochlea. The attacks of vertigo persists in these patients and hearing also does not immediately improve since it is very difficult for all the excess fluid to drain through the narrowed duct. In some ears (Lermoyez's syndrome) cochlear function improves even during the initial clearance of endolymph. These ears theoretically should have larger vestibular aqueducts. Burnt out stage (Late stage) the endolymphatic sac is no longer capable of clearing the fluid. Once the duct is blocked completely, there can no longer be any acute vertigo. In these patients continuing secretion of saccin will increase the hydrops within the ear adversely affecting the patient's hearing.

Predisposing factors for development of Meniere's disease:
1. Fibrosis of endolymphatic sac and vestibular epithelia
2. Altered glycoprotein metabolism

3. Inner ear viral infection
4. Tightly adherent dura in the region of endolymphatic sac
5. Lack of periaqueductal pneumatization
6. Anterior / medial displacement of lateral sinus causing a reduction in the size of Trautmann's triangle. This displacement also causes impediment to the venous drainage locally resulting in a disruption of hydrodynamics of the region.
Currently accepted theory explaining the pathogenesis of Meniere's disease is the drainage theory.

Drainage theory: This theory makes a sincere attempt to encompass all the previously mentioned aspects of anatomy, physiology and pathophysiology.

According to drainage theory the excess endolymph volume which is present in endolymphatic hydrops accumulate in the apical end of cochlea, where the membranes are laxer than elsewhere. When the situation is normal mild increase in the volume of endolymph can be removed by radial fold whereas larger increase in its volume needs an intact longitudinal flow for efficient removal. When the excess volume of endolymph reaches the endolymphatic duct, the sinus can temporarily accommodate the excess volume which the sac is not prepared to receive. This excess of fluid can usually be removed without causing any vestibular disturbance as the endolymphatic valve of Bast isolates the pars superior and prevents endolymphatic fluid draining out of the utricle. If the bony endolymphatic duct is narrow / occluded by accumulation of debris, endolymph may build up excessively in the endolymphatic duct during the longitudinal flow. Overflow begins to occur opening the valve of Bast so that endolymph enters the pars superior. This excessive volume of endolymph entering the saccule distorts the crista in one direction causing vertigo to occur. As the excess endolymph is cleared, the amount of endolymph decreases and the stretched cristae reduces in size, thereby causing a reversal in the direction of nystagmus. Progression of the disease decreases the functionality of the sac due to damage to the cells lining the sac. During the late stages of the disease, the valve of Bast remains patent and during the longitudinal flow a sudden drainage of endolymph from the utricle causing drop attacks to occur (Tumarkin's crisis).

Clinical manifestations of Meniere's disease:
1. Episodic attacks of rotatory vertigo
2. Ipsilateral hearing loss
3. Aural fullness
4. Roaring tinnitus
Episodic vertigo: Is always associated with vegetative signs such as nausea and vomiting. This is supposed to be the most debilitating symptom manifested by the patient. The vertigo begins all of a sudden in a otherwise normal individual. It is accompanied by pallor, sweating, nausea, diarrhea and vomiting. During the attack the patient is fully conscious, oriented in time and space. The patient suffers no residual neurological symptoms after the attack is over. If there is diplopia then it could be due to acute vertigo causing it. The general rule of the thumb is that attacks of vertigo in Meniere's disease last somewhere between 24 minutes and 24 hours. The frequency of these attacks is also highly variable. Patients with severe hydrops suffer attacks on a daily basis while others have long quiescent periods in between attacks.

After the acute phase is over, the symptoms gradually subside, and the patient invariably falls asleep. Some patients may complain of disequilibrium and motion intolerance within the first 24 hours after the initial attack.

Shea's symptomatic classification of Meniere's disease:
Stage I: The patient has solely cochlear symptoms
Stages II - IV: These patients have progressively more cochlear and vestibular symptoms
Stage V: End stage Meniere's disease.

Hearing loss: This is sensorineural in nature and is a cardinal feature of Meniere's disease. The hearing loss is typically fluctuating and progressive. Hearing may in fact fluctuate significantly during the early phases of the disease. The deafness is classically known to involve lower frequencies as compared with s/n loss caused by noise exposure which involves higher frequencies. End stage Meniere's disease is characterized by profound sensorineural hearing loss. Diplacusis is the common complaint in a majority of Meniere's disease patients. Here the same frequency sound is perceived to be different by both the ears.

Tinnitus: Tinnitus in a Meniere's patient is highly variable. It is commonly roaring in nature. It could in fact be the first symptom of the attack. It could be continuous / intermittent. It is invariably non pulsatile in nature. The pitch of the tinnitus usually corresponds to the region of cochlea having the most severe hearing loss. Aural fullness: This is one of the most important symptoms of Meniere's disease. This is mostly caused by enlarging membranous labyrinth. This pressure symptom is limited usually to one ear.

Meniere's disease can be diagnosed by:

1. Vertigo: Vertigo is spontaneous, lasting minutes to hours. It could be recurrent, and if recurrent the patient must have atleast
2 episodes within 20 minutes. These episodes should be accompanied by nystagmus.
2. Hearing loss: In frequencies (200, 500, 1000 Hz) 15 dB. Hearing loss is sensorineural in nature covering the lower frequencies. When compared with the other ear, it should be less by 25 dB in all the frequency ranges studied audiometrically.
3. Tinnitus: Roaring in nature
4. Aural fullness.

Criteria for diagnosis of Meniere's disease:Possible Meniere's disease:

1. Episodic vertigo of Meniere's type without documented hearing loss
2. Fluctuating hearing loss with dysequilibrium but without definite episodes

Probable Meniere's disease:

1. One definitive episode of vertigo
2. Audiometrically documented hearing loss at least on one occasion
3. Tinnitus / aural fullness in the treated ear

Definite Meniere's disease:

1. Two or more definitive episodes of spontaneous vertigo one at least lasting for 20 minutes
2. Audiometrically documented hearing loss at least on one occasion
3. Tinnitus and aural fullness in the treated ear.

Investigations:

Audiological assessment: It is very important to assess cochlear function in a patient with Meniere's disease. Cochlear function can easily be assessed by pure tone audiometry. Patients with Meniere's diseased usually manifest with a flat audiometry curve. Most of these patients have low frequency sensori neural hearing loss.

The pure tone audiometric features seen in Meniere's disease include:
1. Peaked pattern
2. Downward sloping pattern Serial audiometry performed over a period of time may demonstrate fluctuating hearing loss. Fluctuations are often seen in the frequency range between 250 - 1000 Hz. Special audiometric tests need to be performed to ascertain whether the hearing loss is a cochlear or hair cell related disease.
Presence of recruitment can be demonstrated by alternate binaural loudness balance test in unilateral disease, or SISI test in unilateral / bilateral disorders.

Stapedius reflex thresholds: Are within normal limits in these patients. Speech discrimination thresholds: Closely resemble pure tone thresholds in most patients. Poor speech discrimination out of proportion to the pure tone thresholds should arouse suspicion of retro cochlear lesion. The phenomenon known as' roll over' a marked decrease in discrimination is seen in retrocochlear lesions.

Evoked response audiometry: Evoked response audiometry has been found to be instrumental in the diagnosis of Meniere's disease. This test determines the electrical activity occurring in the cochlea and central auditory pathways in response to sound stimuli.

Electrocochleography: Belongs to the battery of tests under evoked response audiometry. It evaluates the evoked potential activity of the cochlea and 8th cranial nerve. Electrocochleography is the best existing objective test for Meniere' s disease. This test measures the electrical events generated either within the cochlea or by primary afferent neurons. The recorded potentials include: Cochlear microphonic potential and summating potential from cochlea, and the whole nerve action potential from the cochlear division of 8th nerve. Cochlear microphonics is an alternating current, the polarity of which is identical to that of the auditory stimulus. It is infact

thought to be the sum of the individual hair cell intracellular potentials. Most of the cochlear microphonic potential is produced by outer hair cells within the first few millimeters of the basal turn of cochlea. In patients with Meniere's disease these cochlear microphonic potentials are small and distorted. In some patients a marked 'after ringing' (a sinusoidal wave) of the cochlear microphonic is seen.

Summating potential: This potential is of short latency (0.3 msec) and is usually present at high stinulus intensities. It is actually a DC shift from the base line of response, generally in a negative direction. This potential occurs for the entire duration of the stimulus. Major component of summating potential is derived from the asymmetry in the vibration induced deflection of the basilar membrane. In normal ears, at high stimulus intensities, the basilar membrane vibrates more upwards towards the scala media than down wards generating a negative summating potential. Endolymphatic hydrops accentuates this asymmetry by stretching and stiffening the basilar membrane, limiting its downward vibration. This mechanical deformity of the basilar membrane is greatest at its basal end and this is the region where the majority of summating potentials are generated from. The normal upgoing asymmetry is enhanced, leading to a negative summating potential of increased amplitude and width.

In Meniere's disease, the common findings on electrocochleography include:
1. Increased summating potential and action potential ratio: A summating potential /action potential ratio of up to 1:3 is within normal range; a higher ratio is suggestive of hydrops.
2. Widened summating potential and action potential complex: The normal width of the summating potential / action potential complex is 1.2 - 1.8 ms and a widening of greater than 2ms is usually significant.
3. Small distorted cochlear microphonic.

Glycerol dehydration test:

This test was originally introduced by Klockhoff and Lindblom in 1966. The drug initially used to cause dehydration was chlorthalidone which promoted sodium excretion without appreciable potassium loss. Pure tone audiometry was performed before and after the administration of the diuretic. A rise in threshold of at least 10dB in three consecutive octave bands were considered diagnostic of Meniere's disease. This test became sensitive when it was combined with trans tympanic electrocochleography. Glycerol was later substituted for chlorthalidone. During glycerol dehydration the marked negative summating potential is seen to decrease. Positive result to glycerol testing can occur if the patient has a fluctuating hearing loss due to endolymphatic hydrops. Glycerol is administered orally in doses of 1.5 mg /kg body weight in the fasting state, and the test can only be considered positive only if there is an increase in serum osmolality of at least 10 mOs/kg to verify the effectiveness of the dehydration process. After one hour the amplitude of the action potential appeared to diminish by 12%.

Side effects of glycerol administration:

1. Headache
2. Nausea / vomiting
3. Drowsiness

Glycerol can also be administered parenterally to shorten the duration of the test. Intravenous administration is performed using 200 ml of 10% glycerol solution.

Acetazolamide test:

This is also another one of the dehydration tests used in the diagnosis of Meniere's disease. This drug is carbonic anhdrase inhibitor. It has been used to increase the cochlear hydrops. This test is hence also known as "reverse glycerol test". Azetazolamide 500 mg in aqueous is injected intravenously over one minute, and electro cochleogram is recorded continuously for 45 minutes. Pure tone audiometry and speech audiometry are also performed. Ecocg showed an enhanced negative summating potential within 10 - 15 minutes of drug infusion, reversing towards the pre-infusion base line level and 45 - 60 minutes. No change was seen in normal individuals or in those with other cochleo vestibular pathologies. This test is useful in patients who have intense vomiting when glycerol is adminsitered.

Medical management of Meniere's disease includes:
1. Dietary management
2. Physiotherapy
3. Psychological support
4. Pharmacologic intervention

Dietary management: This includes reduction of sodium in the diet. In fact it was Frustenberg in 1934 who introduced a low salt diet for patients with Meniere's disease. Pathophysiology of Meniere's disease is enlargement of membranous labyrinth due to excess accumulation of endolymphatic fluid. Any attempt to reduce this fluid level will help in alleviate the symptoms of the patient.

Medical management is mainly used to treat patients during the acute phase of the attack. Vestibular suppressants are commonly used. Drugs used to control attacks of vertigo have varying levels of anticholinergic, antiemetic and sedative properties. Drugs used to alleviate symptoms include phenothiazines (prochlorpherazine and perphenazine), antihistamines like (cinnarizine, cyclizine, dimenhydrinate, and meclizine hydrochloride), benzodiazepines like (lorazepam and diazepam).
Vestibular suppressants:
Diazepam: when used acts as vestibular depressant. It also alleviates the anxiety associated with this disorder. The beneficial effects of diazepam ib vestibular system is presumed to be due to an increase in the cerebellar GABA-ergic system. Stimulation of cerebellar GABA-ergic system

mediates inhibition on the vestibular response. This drug is very useful in alleviating vertigo especially when associated with anxiety. Usual dose is 5 mg administered orally every 3 hours. The initial dose may also be administered intravenously.

Antiemetic drugs: Drugs belonging to this group helps to alleviate vomiting in Meniere's disease.
Anticholinergic drugs: Glycopyrrolate an anticholinergic drug when combined with diazepam is helpful in controlling inner ear symptoms of nausea and vomiting. In adults it is administered in doses of 1-2 mg. It may also be administered as intramuscular injection (0.1 - 0.2 mg) every 4 hours. Side effects (reversible) of this drug includes dry mouth, distortion of visual acuity, exacerbation of symptoms in patients with prostatic hypertrophy. This drug is contraindicated in patients with glaucoma and prostatic hypertrophy.

Antidopaminergic drugs:
Droperidol: This is an antidopaminergic drug used to alleviate the symptoms of Meniere's disease. This drug is administered in doses of 2.5 - 10 mg orally in adults. If administered intravenously it is given as 5 mg bolus. This drug has fewer incidence of side effects like extrapyramidal symptoms / sedation / hypotension. Prochlorperazine: This drug belongs to phenothiazine group. It is used as an antiemetic and a potentiator of analgesic and hypnotic drugs. Usual recommended dose is 10 mg given orally or intramuscularly every 4 - 6 hours in adults. This drug has excellent antiemitic effect.

Antihistamines:
Dimenhydrinate: is useful in preventing and treating vertigo associated with Meniere's disease. It is also very effective in controlling nausea and vomiting. Only side effect of this drug is its propensity to cause drowsiness. It is administered as 50 -100 mg doses thrice a day. This drug can also be administered intramuscularly / intravenously. Diphenhydramine: This drug is not useful in treating acute vertigo. It may be useful in prevention of vertigo. The usual duration of action is 4-6 hours. Usually this drug is administered as an initial loading dose of 50 mg orally.

Meclizine: This drug is one of the most useful antiemetics to prevent / treat nausea and vomiting associated with vertigo of vestibular origin. It has a slower onset and a longer duration of action (24 hours). For vertigo the usual dose administered in adults is 25 - 100 mg daily in divided doses. Side effects of this drug include: drowsiness, blurred vision, drowsiness.
Promethazine: This drug has pronounced antihistaminic activity in addition to its strong central cholinergic blocking activity. It is effective in the treatment of vertigo and motion sickness. It is administered usually in doses of 25 mg every 4 to 6 hours. One major advantage of this drug is that it can be administered rectally, when severe vomiting prevents its effective oral administration. Most common side effect of this drug is sedation.
Maintenance therapy: The goal of maintenance therapy is
1. To prevent acute attacks of vertigo
2. To maintain hearing in Meniere's disease. This therapy usually includes dietary modifications combined with pharmacological intervention. Dietary modifications: The mainstay of diet modifications is to reduce sodium intake. A very low sodium intake or low sodium diet is usually recommended. A strict low sodium diet means a daily allowance of 1500 mg. This is a very stringent

diet and patients find it very difficult to comply with this diet. A more practical approach would be to advise the patient to avoid excessively salty food. Restrictions are also imposed on the intake of caffeine, nicotine and alcohol.

Diuretics: The use of diuretics in the maintenance therapy is based on the supposition that these drugs can alter the fluid balance of inner ear, leading to a depletion of endolymph and a correction of hydrops. In 1934 Furstenburg demonstrated that the symptoms of Meniere's disease were due to retention of sodium. He went on to recommend a low sodium diet / use of diuretics to control Meniere's disease. Boles in 1975 demonstrated that most patients had their vertigo controlled with an 800 -1000 mg of sodium diet / day.
Hydrochlorthiazide: This diuretic causes natriuresis and kaliuresis by blocking sodium reabsorption in the loop of Henle. Potassium supplementation is required in patients using this drug. Side effects of this drug include: hypokalemia, hyperglycemia, hypotension, and hyperuricemia. It is usually administered as 50 mg tabs orally / day in adults.
Potassium supplements is usually required in these patients.
Dyazide: Is a potassium sparing diuretic. It can be conveniently administered as a single daily dose.
Frusemide: This is a loop diuretic. It is a very potent diuretic. It can cause electrolyte and volume depletion more rapidly than other diuretics. It usually causes hypokalemia. Usual adult dose is 10 - 80 mg/day. The duration of action lasts for about 4 hours.
Amiloride: This is a potassium sparing diuretic acting on the distal tube of Henle. Its diuretic potency is highly limited. It is usually used in combination with other diuretics in order to minimize potassium loss.
Carbonic anhydrase inhibitors:
Acetazolamide: Is a carbonic anhydrase inhibitor. It causes a decrease in the sodium - hydrogen exchange in the renal tubule inducing diuresis.
Methazolamied: Is another carbonic anhydrase inhibitor shown to be effective in controlling symptoms of Meniere's disease. This drug is usually administered in doses of 50 mg / day, 5 days a week for 3 months.

Medical ablative therapy:
Aminoglycosides: Ototoxic effects of aminoglycosides are well documented. Streptomycin and gentamycin are predominantly vestibulo toxic. Intramuscular injections of streptomycin administered twice daily for periods of days to weeks have been used in patients with debilitating bilateral disease / unilateral disease in the only hearing ear. Complete ablation causes disabling oscillopsia. Many authors have suggested lower doses and fewer injections to achieve partial ablation, thereby reducing the incidence of severe ataxia. Currently the recommended daily dose is 1 g of streptomycin intramuscularly 5 days a week until vestibular ablation occurs as manifested by absence of ice water caloric test. Intratympanic injections of these drugs have also been used with success.
Vasodilators: The use of vasodilators is based on the idea that Meniere's disease results from ischemia of the stria vascularis. Betahistine has been used with varying degrees of success. This drug can be used for short term control of vertigo and for maintenance therapy. Nicotinic acid is

another vasodilator which when administered 30 minutes before meals in doses of 50 - 400 mg helps in resolving the acute crisis associated with Meniere's disease.
Calcium channel blockers: Nimodepine a highly lipophilic drug is very useful in the medical management of Menierie's disease. It readily crosses the blood brain barrier. This drug is useful in patients who have failed diuretic medical therapy.
ACE inhibitors: These are very effective vasodilators. These drugs block the renin angiotensin aldosterone system. They produce vasodilatation by blocking angiotensin II induced vasoconstriction.
Lipoflavins and vitamins: Combination of lipoflavins and vitamins have been tried as a management modality with varying degrees of success. Restricting tea and coffee intake to once daily will help these patients in reducing endolymphatic fluid volume. Ingestion of excessive amounts of caffeine and alcohol cause enormous fluid shifts in the physiological fluid compartments.

Surgical management:

Should be contemplated only when conservative management fails. Commonly performed surgery for this disorder is Endolymphatic sac decompression. This surgery can be performed under general anesthesia via mastoid approach.

15. Enumerate the peripheral vestibular causes of vertigo. Discuss the Pathophysiology, Clinical features and Management of BPPV (Benign Positional Paroxysmal Vertigo).

Benign paroxysmal positional vertigo is the most commonly diagnosed vestibular disorder. This is commonly caused by dysfunction of the posterior semicircular canal. Lateral and superior semicircular canals can also be involved on rare occasions. It is characterized by brief spells of severe vertigo (often lasting for just a few seconds) that are experienced only with specific movements of the head.

History:

This disorder was first described by Barany in 1921. He documented the various components of this disorder as 1. Nystagmus, 2. Fatiguability of the nystagmus and 3. Vertigo. He failed to correlate the onset of nystagmus with specific positions of the head.

Dix & Hallpike 1952 described the Dix Hallpike maneuver for eliciting the nystagmus. They also described the unique features of nystagmus accompanying this disorder. These features were 1. Very short latency, 2. Directional features, 3. Brief duration, and 4. Reversibility on returning the patient to a seated position.

Schuknecht postulated that BPPV was caused by loose otoconia from the utricle which in certain positions, displaced the cupula of the posterior canal. (Schuknecht theory). He later modified his theory and proposed that it was due to the deposition of otoconia on the cupula of the posterior

semicircular canal. He termed this theory as cupulolithiasis. The cupulolithiasis theory proposes that calcium deposits become embedded on the cupula making the posterior semicircular canal sensitive to gravity.

Hall & Ruby suggested that BPPV could result from deflection of the posterior canal cupula caused by debris within the posterior canal. This theory became known as the canal lithiasis theory. In this theory the calcium debris does not become adherent to the cupula but float freely within the canal. Head movements like looking up, down, or rolling over to the affected ear may result in the displacement of the sludge causing the classic symptoms.

Hall & Ruby described 2 types of BPPV:
1. BPPV with a fatigable nystagmus, where the deposits are freely mobile within the cupula of the posterior canal,

2. BPPV with a non-fatiguing nystagmus where the calcium deposits are fixed on the cupula of the posterior canal.

Typical features of BPPV as described by Hall & Ruby:

1. Canalithiasis mechanism - This explains the latency of the nystagmus as a result of the time needed for motion of the material within the posterior canal to be initiated by the gravity.

2. Duration of the nystagmus - is correlated with the length of time required for the dense material to reach the lowest part of the posterior canal.

3. The vertical (upbeating) and torsional (superior poles of the eye beating towards the lowermost ear). The nystagmus is more vertical when the patient looks away from the lowermost ear, and more torsional when looking towards the lowermost ear.

4. The reversal of nystagmus when the patient returns to the sitting position is due to retrograde movement of material in the lumen of the posterior canal back towards the ampula, resulting in ampulo petal deflection of the cupula.

5. The fatiguability of the nystagmus evoked by repeated Dix Hallpike positional testing is explained by dispersion of material within the canal.

Incidence:

BPPV is the most common cause of vertigo constituting 20 - 40% of all patients with peripheral vestibular disease. Mean age of onset ranging between 4th and 5th decades. women outnumbering men by 2:1.

History: Patient c/o severe vertigo associated with change in head position. Symptoms are always sudden in nature, never lasting more than a minute. The patient may even volunteer provocating postures.

On examination: the classic eye movements associated with Dix Hallpike maneuver is seen.

Dix-Hallpike maneuver: The patient is positioned on the examination table in such a way that when he/she is placed supine, the head extends over the edge. The patient is lowered with the head supported and turned 45 degrees to one or the other side. The eyes are carefully observed; if no abnormal eye movements are seen, the patient is returned to the upright position.

This same maneuver is repeated with the head in the opposite direction and the patient's symptoms are noted.

The pattern of response consists of the following:

1. Nystagmus is a combination of vertical upbeating & rotatory (torsional) beating towards the downward eye. Pure vertical nystagmus is not seen in BPPV.

2. There is often a latency of onset of nystagmus

3. Duration is less than a minute

4. Vertiginous symptoms are invariably seen

5. Nystagmus disappears with repeated testing (fatiguability)

6. Symptoms often recur with the nystagmus in opposite direction on return of the head to upright position.

Canalithiasis involving the posterior canal is the commonest cause of BPPV. Posterior canal BPPV may rarely be bilateral, but while testing the head must be positioned in the plane of the posterior canal during testing of unaffected ear otherwise the debris in the affected side can rest against the cupula and stimulate an excitatory nystagmus from the unaffected ear.

Lateral canal BPPV:

Lateral canal has also been identified as the offender in 17 % of cases with BPPV. Lateral canal BPPV can be detected by a variation of Dix Hallpike maneuver. The patient's head is first brought to the supine position resting on the examination table (not hyperextended). The head is then turned rapidly to the right so that the patient's right ear rests on the table. The eye movements of the patient are monitored with Frenzel's glasses for 30 seconds. The patient's head is then turned to the supine position (eyes looking upward) and is then rapidly turned to the left so that the left ear rests

on the table. Eye movements are monitored. The nystagmus with lateral canal BPPV is horizontal and may beat toward (geotropic) or away (ageotropic) from the downward ear. It begins with a short latency, increases in magnitude progressively, and is less susceptible to fatigue with repetitive testing than the vertical torsional nystagmus of posterior canal BPPV.

Cupulolithiasis, either alone or in combination with Canalithiasis is more likely to be involved in the etiology of lateral canal BPPV than in the case of posterior canal BPPV. If the nystagmus is geotropic, the particles are likely to be in the long arm of the lateral canal relatively far from the ampulla, if the nystagmus is ageotropic, the particles could be in the long arm relatively close to the ampulla or on the opposite side of the cupula either floating within the endolymph or embedded in the cupula.

Superior canal BPPV: Incidence of superior canal BPPV is very rare.

Standard electrooculography or 2-dimensional video nystagmography devices do not record the typical eye movements associated with BPPV. Thus, clinical examination of the patient is of paramount importance.

Management:

Medical:

Repositioning maneuver: Currently BPPV is managed by repositioning maneuvers that, in cases of Canalithiasis use gravity to move canalith debris out of the affected semicircular canal and into the vestibule. For posterior canal BPPV the maneuver developed by Epley is effective.

Epley maneuver - This is performed by placing the head of the patient in the Dix Hallpike position that evokes the vertigo. The posterior canal on the affected side is in the earth vertical plane when the head is in this position. After the cessation of initial nystagmus, the head is rolled through 180 degrees, (this is done in two 90-degree increments, stopping in each position until the nystagmus resolves) to the position in which the offending ear is up. The patient is then brought to the upright sitting position. This procedure is likely to be successful when nystagmus of the same direction continues to be elicited in each of the new position (as the debris continues to move away from the cupula). This maneuver is repeated until no nystagmus is elicited. This is successful in 90 % of cases. Posterior canal BPPV can be converted to lateral canal BPPV during Epley maneuver. The lateral canal BPPV resolves in several days. Drugs are usually not prescribed, but low dose meclizine or calmpose can be given 1 hour before the procedure if the patient is anxious or prone to vomiting.

Sermont maneuver - is also effective in posterior canal BPPV, but is most difficult to perform and it has no significant advantages over the Epley maneuver. This is being described here for the sake of completion. In this maneuver the patient is moved quickly in to the position that provokes the

vertigo and remains in that position for 4 minutes. The patient is then turned rapidly to the opposite side ear down, and remain in the second position for 4 minutes before slowly getting up.

In both these maneuvers gravity is the stimulus that move the particles within the canal, so there is no need to turn the head on the body, enbloc movement of the head and body as much as possible is the plan.

Vibrator therapy:

Some physicians use a small hand held vibrator over the mastoid to agitate the particles and make it move. This mastoid vibrator is to be avoided in patients with retinal detachment or in patients who may be susceptible to retinal detachment due to high myopia.

After these repositioning maneuvers patients are instructed to avoid bending over and are told to sleep with the head elevated at least 45 degrees for the next couple of days.

Brandt Doroff exercises - can be performed by the patient in the home environment. These exercises are performed in 3 sets / day for 2 weeks.

It is started like this:

Position 1 - The patient must be seated upright on the bed. Then he moves to side lying position (position 2) the head is kept angled upwards about half way. The patient should stay in this position at least for 30 seconds or till the giddiness subsides. If the giddiness does not subside thee patient must revert back to position 1. After 30 seconds the procedure is repeated on the opposite side. Most of the patients get relief within a period of 10 minutes.

Treatment maneuvers for lateral canal BPPV:

In these patients with geotropic nystagmus lying on one side with the affected ear up for 12 hours has been found to be effective.

Surgical management:

Singular neurectomy - is a very demanding procedure. The posterior canal is supplied by singular branch of vestibular nerve. This nerve when preferentially sectioned alleviates the patient's symptom due to posterior canal BPPV.

Posterior canal plugging procedure - is a easier procedure. Through a mastoidectomy incision the labyrinth is exposed. The posterior canal is drilled exposing the membranous portion of the canal. The canal is sealed and packed off thereby preventing the debris from floating. After the procedure the patient may feel slightly giddy. The patient needs to be kept in the hospital till giddiness subsides.

16. Enumerate causes of deafness in children. What are the investigations to assess hearing in children? Write about auditory training & hearing aids in such children.

Causes of deafness in children can be classified into:

1. Congenital causes
2. Acquired causes

Congenital causes can be subdivided into:

Disorders affecting the external auditory canal:
These include atresia of external auditory canal, microtia, anotia.

Disorders affecting middle ear:
These include fixity of ossicles (head of malleus, and foot plate of stapes)

Disorders affecting inner ear:

Various developmental abnormalities (congenital) can affect development of inner ear causing deafness. They include defects affecting semicircular canal (Mondini / Schibe deformities)

Acquired causes of deafness in children:

External ear causes:

Impacted cerumen (commonest)
Exostosis of external canal
Stenosis of external auditory canal

Ear drum causes:

Ear drum perforation
Tympanosclerosis

Middle ear causes:

CSOM
ASOM
Adhesive otitis media
Secretory otitis media
Otosclerosis
Meniere's disease

Inner ear causes:

Labyrinthitis
Noise induced hearing loss
Drug induced hearing loss

The following are the ways to identify hearing loss in children:

Startle reflex:

Neonates with normal hearing manifest startle reflex on sudden exposure to sound.

Speech and language milestones observation:

9 months - Demonstrates an understanding of simple words like "mummy", "daddy" etc.
10 months - Child develops babbling sounds like da
1 year - The child speaks one / more words
18 months - Understands simple phrases, retrieves familiar objects and points to body parts. Has a spoken vocabulary of 20-50 words.
24 months - Has a vocabulary of 150 words
3-5 years - Uses spoken language to express wants and emotions

Auditory investigations that needs to be performed in children include:

1. Free field audiometry
2. Evoked response audiometry
3. Evoked acoustic emissions
4. BERA
5. Play audiometry
6. Pure tone audiometry in grown up child

Auditory training:

Children with moderate to severe deafness should be fitted with hearing aids:

Types of hearing aids:

Body worn hearing aids

Behind the ear hearing aids

BERA can be used in a child with severe deafness

Children need to be fitted with hearing aids to both ears if deafness is bilateral. Hearing aids should be fitted to a child as soon as hearing loss is identified.

Digital hearing aids should be provided to children with sensorineural hearing loss.

Aggressive speech and language therapy should be provided to the child.

1. Describe the anatomy of lateral wall of nose. Describe the types and management of nasal polyps.

Introduction:

With the common use of nasal endoscopes as a diagnostic and surgical tool, the anatomy of the lateral nasal wall has been completely rewritten. In fact, the present description of the lateral nasal wall anatomy confirms with the endoscopic anatomy of the lateral nasal wall. The anatomy of the lateral nasal wall is highly variable, and a thorough understanding of the anatomy is a must before proceeding with any nasal endoscopic procedure.

Nasal turbinates: The turbinates are the most prominent feature of the lateral nasal wall. They are usually three or sometimes four in number. These turbinates appear as scrolls of bone, delicate, covered by ciliated columnar epithelium. These turbinates sometimes may contain an air cell, in which case it is termed as a concha.

These turbinates project from the lateral wall of the nose. Out of these turbinates the following are present in all individuals:

The superior, middle and inferior turbinates. A small supreme turbinate may be present in some individuals. Among these turbinates the superior and the middle turbinates are components of the ethmoidal complex where as the inferior turbinate is a separate bone. Commonly a prominence may be seen at the anterior attachment of the middle turbinate. This prominence is known as the agger nasi cell. This prominence varies in size in different individuals. These agger nasi cells overlie the lacrimal sac, separated from it just by a thin layer of bone. In fact this agger nasi cell is considered to be a remnant of naso turbinal bones seen in animals.
When the anterior attachment of the inferior and middle turbinates is removed, the lacrimal drainage system and sinus drainage system can be clearly seen.

The inferior meatus is present between the inferior turbinate and the lateral nasal wall. The nasal opening of the naso lacrimal duct opens in the anterior third of the inferior meatus. This opening is covered by a mucosal valve known as the Hasner's valve. The course of the naso lacrimal duct from the lacrimal sac lie under the agger nasi cell.

The middle meatus lie between the middle turbinate and the lateral nasal wall. The middle turbinate is part of the ethmoidal complex. The sinuses have been divided into the anterior and posterior groups. The anterior group of sinuses are frontal, maxillary and anterior ethmoidal sinuses. These sinuses drain into the middle meatus, i.e. under the middle turbinate.

Uncinate process: actually, forms the first layer or lamella of the middle meatus. The uncinate process is a wing or boomerang shaped piece of bone. It attaches anteriorly to the posterior edge of

the lacrimal bone, and inferiorly to the superior edge of the inferior turbinate. Superior attachment of the uncinate process is highly variable, may be attached to the lamina papyracea, or the roof of the ethmoidal sinus, or sometimes to the middle turbinate. The configuration of the ethmoidal infundibulum and its relationship to the frontal recess depends largely on the behavior of the uncinate process. The uncinate process can be classified into 3 types depending on its superior attachment. The anterior insertion of the uncinate process cannot be identified clearly because it is covered with mucosa which is continuous with that of the lateral nasal wall. Sometimes a small groove is visible over the area where the uncinate attaches itself to the lateral nasal wall.

Type I uncinate: Here the uncinate process bends laterally in its upper most portion and inserts into the lamina papyracea. Here the ethmoidal infundibulum is closed superiorly by a blind pouch called the recesses terminalis (terminal recess). In this case the ethmoidal infundibulum and the frontal recess are separated from each other so that the frontal recess opens in to the middle meatus medial to the ethmoidal infundibulum, between the uncinate process and the middle turbinate. The route of drainage and ventilation of the frontal sinus run medial to the ethmoidal infundibulum.

Type II uncinate: Here the uncinate process extends superiorly to the roof of the ethmoid. The frontal sinus opens directly into the ethmoidal infundibulum. In these cases, a disease in the frontal recess may spread to involve the ethmoidal infundibulum and the maxillary sinus secondarily. Sometimes the superior end of the uncinate process may get divided into three branches one getting attached to the roof of the ethmoid, one getting attached to the lamina papyracea, and the last getting attached to the middle turbinate.

Type III uncinate process: Here the superior end of the uncinate process turns medially to get attached to the middle turbinate. Here also the frontal sinus drains directly into the ethmoidal infundibulum.

Rarely the uncinate process itself may be heavily pneumatised causing obstruction to the infundibulum.

Ethmoidal infundibulum: is a cleft like space, which is three dimensional in the lateral wall of the nose. This structure belongs to the anterior ethmoid. This space is bounded medially by the uncinate process and the mucosa covering it. Major portion of its lateral wall is bounded by the lamina papyracea, and the frontal process of maxilla to a lesser extent. Defects in the medial wall of the infundibulum is covered with dense connective tissue and periosteum. These defects are known as anterior and posterior fontanelles. Anteriorly the ethmoidal infundibulum ends blindly in an acute angle.

Hiatus semilunaris: Lies between the anterior wall of the Bulla and the free posterior margin of the uncinate process. This is in fact a two-dimensional space. Through this hiatus a cleft like space can be entered. This is known as the ethmoidal infundibulum. This ethmoidal infundibulum is bounded medially along its entire length by the uncinate process and its lining mucosa. The lateral wall is formed by the lamina papyracea of the orbit, with participation from the frontal process of the

maxilla and the lacrimal bone. The anterior group of sinuses drain into this area. In fact this area acts as a cess pool for all the secretions from the anterior group of sinuses.

Osteomeatal complex: This term is used by the surgeon to indicate the area bounded by the middle turbinate medially, the lamina papyracea laterally, and the basal lamella superiorly and posteriorly. The inferior and anterior borders of the Osteomeatal complex are open. The contents of this space are the agger nasi, nasofrontal recess (frontal recess), infundibulum, bulla ethmoidalis and the anterior group of ethmoidal air cells.
Some authors divide this Osteomeatal complex into anterior and posterior. The classic Osteomeatal complex described already has been described as the anterior Osteomeatal complex, while the space behind the basal lamella containing the posterior ethmoidal cells is referred to as the posterior ethmoidal complex, thus recognizing the importance of basal lamella as an anatomical landmark to the posterior ethmoidal system. Hence the anterior and the posterior Osteomeatal complex has separate drainage systems. So when the disease is limited to the anterior compartment of the Osteomeatal complex, the ethmoid cells can be opened and diseased tissue removed as far as the basal lamella, leaving the basal lamella undisturbed minimizing the risk during surgery.

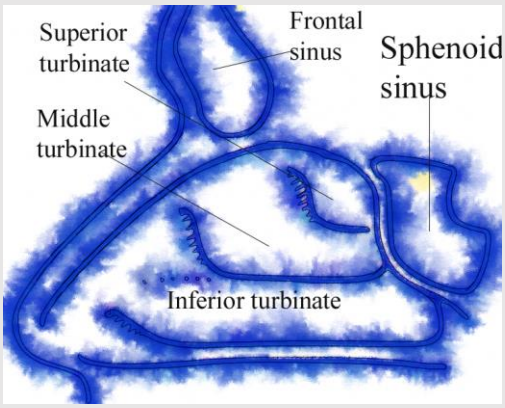


Image showing turbinates in the lateral wall

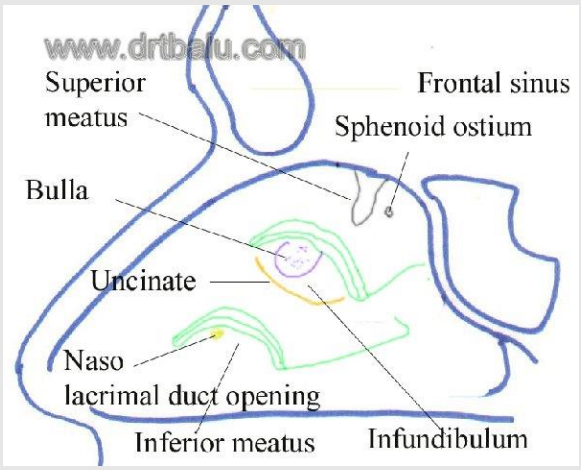
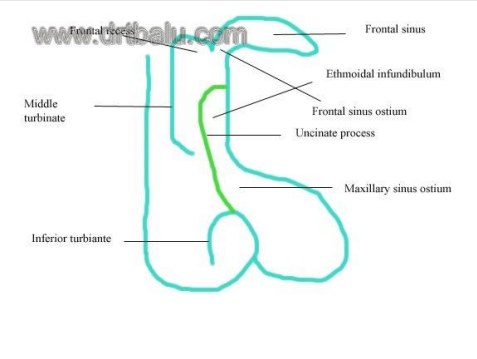
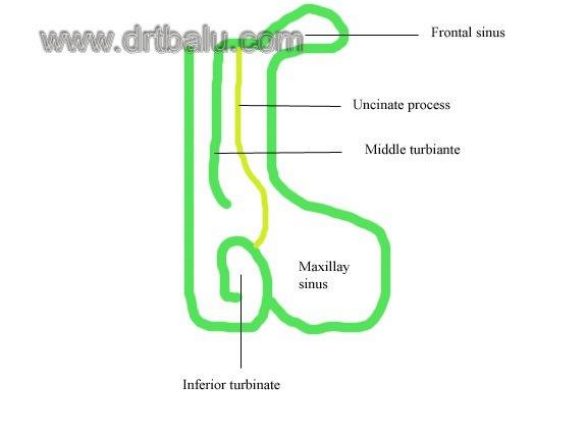


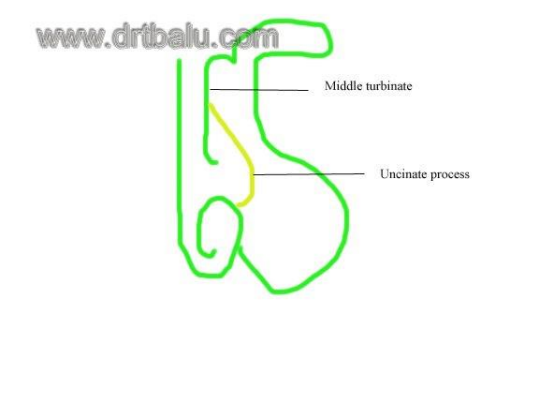
Diagram of lateral wall of nose after removal of middle turbinate



Type I uncinate



Type II uncinate



Type III uncinate

Types of nasal polyp:

Polyp can be solitary / multiple / Unilateral / Bilateral

Antrochoanal polyp:

This is solitary and unilateral. It arises from the maxillary sinus, exits out via the enlarged accessory ostium and enters the nasal cavity. Once inside the nasal cavity it grows posteriorly and exits out of the choana. When it reaches the nasal cavity, it causes unilateral nasal obstruction. When it exits out of the choana obstruction becomes bilateral.
It commonly occurs in young and adolescents. Infection is probably the etiological cause.

Management:

Endoscopic polypectomy. If there is recurrence and when the patient has reached adulthood then Caldwell Luc surgery is preferred.

Multiple bilateral ethmoidal polypi:

Polyp is defined as benign oedematous hypertrophic mucosa. Ethmoidal polypi are multiple and bilateral. Allergy is the probable etiology. Ethmoidal polypi is a medical disorder. Steroids cause the polypoidal tissue to shrink in size. If the patient does not respond to administration of antihistamines and steroid then bilateral endoscopic polypectomy can be performed. Use of debrider cause less trauma to normal nasal mucosa.

Patients should be advised to continue topical nasal steroid spray for a period of 9 months following surgery.

All types of polypoidal tissue appears like a bunch of grapes. They are pale and glistening. They should be differentiated from oedematous turbinate tissue.

Differentiation between nasal polyp and polypoidal turbinates:

Turbinates are sensitive on probing while polypoidal tissue are insensitive to touch. On probing the probe can be passed all around the polypoidal tissue, but not all around the turbinate tissue as turbinates are attached to lateral nasal wall.
If the nasal cavity is packed with xylocaine impregnated gauze polypoidal turbinate shrinks in size while polyp does not shrink that much.

Difference between ethmoidal and Antrochoanal polyp:

Ethmoidal polyp	Antrochoanal polyp
Seen in adults	Seen in children and adolescents
Allergy is the common cause	Infection is the common cause
Multiple like a bunch of grapes	Unilateral and single
Arises from ethmoidal labyrinth	Arises from maxillary sinus
Seen easily on anterior rhinoscopy	Seen in posterior rhinoscopy
Recurrence is common	Recurrence is uncommon
Polypectomy needs to be performed	Caldwell Luc surgery is indicated in recurrent cases

2. List the causes of the epistaxis. Describe the management of juvenile nasopharyngeal angiofibroma.

Epistaxis is defined as bleeding from the nasal cavity. This is a Greek word meaning nose bleed. Since it is a very common problem its true incidence is very difficult to predict.

Etiology: The etiology of epistaxis is not just simple or straight forward. It is commonly multifactorial, needing careful history taking and physical examination skill to identify the cause. For purposes of clear understanding the etiology of epistaxis can be classified under two broad heads, i.e. local and systemic causes.

Local factors causing epistaxis: include vascular anomalies, infections and inflammatory states of the nasal cavity, trauma, iatrogenic injuries, neoplasms and foreign bodies. Among these causes the commonest local factors involved in epistaxis is infection and inflammation. Infections and inflammation of the nasal mucous membrane may damage the mucosa leading on to bleeding from the underlying exposed plexus of blood vessels. Chronic granulomatous lesions like rhinosporidiosis can cause extensive epistaxis.

Aneurysms involving the internal carotid artery may occur following head injury, injury sustained during surgical procedures. These extradural aneurysms and aneurysms involving the cavernous sinus may extend into the sphenoid sinus wait for the opportune moment to rupture. It can cause sudden fatal epistaxis, or blindness. Urgent embolization is the preferred mode of management of this condition.

Trauma is one of the common local causes of epistaxis. It is commonly caused by the act of nose picking in the Little's area of the nose. This is commonly seen in young children. Acute facial trauma may also lead to epistaxis. Patients undergoing nasal surgeries may have temporary episodes of epistaxis.

Irritation of the nasal mucous membrane: any disruption of normal nasal physiology can cause intense drying and irritation to the nasal mucosa causing epistaxis. These episodes are common during extremes of temperature when the nasal mucosa is stressed to perform its air-conditioning role of the inspired air. In these conditions there is extensive drying of nasal mucosa causes oedema of the nasal mucous membrane. This oedema is caused due to venous stasis. Ultimately the mucosa breaches exposing the underlying plexus of blood vessels causing epistaxis.

Anatomical abnormalities: Common anatomical abnormality causing epistaxis is gross septal deviation. Gross deviations of nasal septum cause disruption to the normal nasal airflow. This disruption leads to desiccation / drying of the local mucosa. The dry mucosa cracks and bleeds.

Septal perforations: Chronic non healing septal perforations can cause bleeding from the granulation tissue around the perforation.

Neoplasms: involving the nose and paranasal sinuses can cause epistaxis. Neoplasms include benign vascular tumors like hemangioma, juvenile nasopharyngeal angiofibroma, and malignant neoplasms like squamous cell carcinoma. If epistaxis occurs along with secretory otitis media then nasopharyngeal carcinoma should be the prime suspect.

Systemic causes for epistaxis:

Hypertension is one of the common systemic causes of epistaxis. Accumulation of atherosclerotic plaques in the blood vessels of these patients replaces the muscular wall. This replacement of muscular wall reduces the ability of the blood vessels to constrict facilitating epistaxis. This is one of the common causes of posterior nasal bleeds. It commonly arises from the Woodruff's plexus found close the posterior end of the middle turbinate.

Hereditary hemorrhagic telangiectasia is another systemic disorder known to affect the blood vessels of the nose. This disease causes loss of contractile elements within the blood vessels causing dilated venules, capillaries and small arteriovenous malformations known as telangiectasia. These changes can occur in the skin, mucosal lining the whole of the respiratory passage and urogenital passage. Bleeding from these telangiectasia is difficult to control. Bleeding invariably starts when the patient reaches puberty. Common cause of mortality in these patients is gastrointestinal bleed.

Systemic diseases like syphilis, tuberculosis & wegner's granulomatosis cause epistaxis because of their propensity to cause ulceration of the nasal mucous membrane.

Blood dyscrasias can also cause epistaxis. A low platelet count is one common cause of nasal bleed in this category. In thrombocytopenia the platelet count is less than 1 lakh. Epistaxis can start when the platelet count reduces to 50,000. Platelet deficiency can be caused by ingestion of drugs like aspirin, indomethacin etc. Hypersplenism can cause thrombocytopenia in idiopathic thrombocytopenic purpura. These patients need to be transfused fresh blood in adequate quantities. Only when thee platelet count increases will the nasal bleed stop.

Management of JNA:

Juvenile nasopharyngeal angiofibroma is a locally eroding vascular mass involving the nose and nasopharynx. This is a highly vascular tumor. Many of these patients seek medical attention only after spontaneous unprovoked epistaxis.

CT scan:

This should be performed in order to accurately assess the extent of the tumor mass.

Contrast CT can be performed in order to ascertain blood supply of the tumor mass

Surgery is the ideal treatment of choice for these patients.
Large quantities of blood should be available on the table during surgery.
Surgical approach depends on the extent of the tumor mass.

3. Enumerate in detail the various causes of epistaxis. How will you manage epistaxis in a 6-year-old child?

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Irritation of the nasal mucous membrane: any disruption of normal nasal physiology can cause intense drying and irritation to the nasal mucosa causing epistaxis. These episodes are common during extremes of temperature when the nasal mucosa is stressed to perform its air-conditioning role of the inspired air. In these conditions there is extensive drying of nasal mucosa causes oedema of the nasal mucous membrane. This oedema is caused due to venous stasis. Ultimately the mucosa breaches exposing the underlying plexus of blood vessels causing epistaxis.

Anatomical abnormalities: Common anatomical abnormality causing epistaxis is gross septal deviation. Gross deviations of nasal septum cause disruption to the normal nasal airflow. This disruption leads to desiccation / drying of the local mucosa. The dry mucosa cracks and bleeds.

Septal perforations: Chronic non healing septal perforations can cause bleeding from the granulation tissue around the perforation.

Neoplasms: involving the nose and paranasal sinuses can cause epistaxis. Neoplasms include benign vascular tumors like hemangioma, juvenile nasopharyngeal angiofibroma, and malignant neoplasms like squamous cell carcinoma. If epistaxis occurs along with secretory otitis media then nasopharyngeal carcinoma should be the prime suspect.

Systemic causes for epistaxis:

Hypertension is one of the common systemic causes of epistaxis. Accumulation of atherosclerotic plaques in the blood vessels of these patients replaces the muscular wall. This replacement of muscular wall reduces the ability of the blood vessels to constrict facilitating epistaxis. This is one of the common causes of posterior nasal bleeds. It commonly arises from the Woodruff's plexus found close the posterior end of the middle turbinate.

Hereditary hemorrhagic telangiectasia is another systemic disorder known to affect the blood vessels of the nose. This disease causes loss of contractile elements within the blood vessels causing dilated venules, capillaries and small arteriovenous malformations known as telangiectasia. These changes can occur in the skin, mucosal lining the whole of the respiratory passage and urogenital passage. Bleeding from this telangiectasia is difficult to control. Bleeding invariably starts when the patient reaches puberty. Common cause of mortality in these patients is gastrointestinal bleed.

Systemic diseases like syphilis, tuberculosis & wegner's granulomatosis cause epistaxis because of their propensity to cause ulceration of the nasal mucous membrane.

Blood dyscrasias can also cause epistaxis. A low platelet count is one common cause of nasal bleed in this category. In thrombocytopenia the platelet count is less than 1 lakh. Epistaxis can start when the platelet count reduces to 50,000. Platelet deficiency can be caused by ingestion of drugs like aspirin, indomethacin etc. Hypersplenism can cause thrombocytopenia in idiopathic

thrombocytopenic purpura. These patients need to be transfused fresh blood in adequate quantities. Only when thee platelet count increases will the nasal bleed stop.

Management of Epistaxis in a 6-year-old child:

Cause of nasal bleed should be assessed.
Anterior rhinoscopic examination should be performed.

Anterior nasal packing should be performed. In fact, almost all cases of nasal bleed can be managed successfully by anterior nasal packing. If bleeding is from enlarged and dilated retro columellar vein then the same should be cauterized. Refractory Little's area bleed can be managed by cauterizing the area.

4. Enumerate causes of epistaxis. Describe management of epistaxis.

The etiology of epistaxis is not just simple or straight forward. It is commonly multifactorial, needing careful history taking and physical examination skill to identify the cause. For purposes of clear understanding the etiology of epistaxis can be classified under two broad heads, i.e. local and systemic causes.

Local factors causing epistaxis: include vascular anomalies, infections and inflammatory states of the nasal cavity, trauma, iatrogenic injuries, neoplasms and foreign bodies. Among these causes the commonest local factors involved in epistaxis is infection and inflammation. Infections and inflammation of the nasal mucous membrane may damage the mucosa leading on to bleeding from

the underlying exposed plexus of blood vessels. Chronic granulomatous lesions like rhinosporidiosis can cause extensive epistaxis.

Aneurysms involving the internal carotid artery may occur following head injury, injury sustained during surgical procedures. These extradural aneurysms and aneurysms involving the cavernous sinus may extend into the sphenoid sinus wait for the opportune moment to rupture. It can cause sudden fatal epistaxis, or blindness. Urgent embolization is the preferred mode of management of this condition.

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Incidence: The incidence of epistaxis is known to be slightly higher in males. It also has a bimodal distribution affecting young children and old people.

Evaluation: While evaluating a patient with epistaxis it is absolutely necessary to assess the quantum of blood loss. The blood pressure and pulse rate of these patients must be constantly monitored. These patients will have tachycardia. Infusion of fluid must be started immediately. Initially ringer lactate solution will suffice. If the patient has suffered blood loss of more than 30% of their blood volume (about 1.5 liters) then blood transfusion becomes a must. Further examination should be started only after the patient's general condition stabilizes.

History: Careful history taking is a must. History taking should cover the following points:

1. History regarding the frequency, severity and side of the nasal bleed.

2. Aggravating and relieving factors must be carefully sought.

3. History of drug intake must be sought.

4. History of systemic disorders like hypertension and diabetes mellitus must be sought.

Physical examination:

The nasal pack if any must be removed. Anterior nasal examination should be done, first attempted without the use of nasal decongestants. If visualization is difficult due to oedema of the nasal mucosa then nasal decongestants can be used to shrink the nasal mucosa. The solution used for anesthetizing the decongesting the nose is a mixture of 4% xylocaine and xylometazoline.

Nasal endoscopy can be performed under local anesthesia to localize posterior bleeds.

Investigations:

If bleeding is minimal no investigation is necessary.

If bleeding is more than a complete blood work up to rule out blood dyscrasias is a must. It includes bleeding time, clotting time, platelet count and partial thromboplastin time.

Imaging studies like CT scan of the para nasal sinuses must be done to rule out local nasal conditions of epistaxis. Imaging must be done only after 24 hours of removing the nasal packing. Scans done with the nasal pack or immediately after removing the nasal pack may not be informative.

In difficult and intractable cases angiography can be done and the internal maxillary artery can be embolized in the same sitting. This procedure should be reserved only for cases of intractable nasal bleeding.

Management:

Conservative:

Nasal packing: Anterior nasal packing using roller gauze impregnated with liquid paraffin is sufficient to manage a majority of anterior nasal bleeds. The liquid paraffin acts as a lubricant, and as a moistening agent. The tamponing effect of a nasal pack is sufficient to stop nasal bleeding. This type of roller gauzes can be kept inside the nasal cavity only up to 48 hours after which it has to be removed and changed. The newer packs like the BIPP (Bismuth Iodine paraffin paste) packs can be left safely in place for more than a week.

Merocel pack

To manage post nasal bleed a post nasal pack is a must. Post nasal packing can be done in 2 ways:

Post nasal packing (conventional): A gauze roll about the size of the patient's naso pharynx is used here. Three silk threads must be tied to the gauze roll. One at each end and the other one at the middle. The patient should be in a recumbent position. After anesthetizing the nasal cavity with 4% xylocaine the mouth is held open. Two nasal catheters are passed through the nasal cavities till they reach just below the soft palate. These lower ends of the catheters are grasped with forceps and pulled out through the mouth. The silk tied to the ends of the gauze is tied to the nasal catheters. The post nasal pack is introduced through the mouth and gradually pushed into the nasopharynx, at the same time the nasal catheters on both sides of the nose must be pulled out. When the pack

snugly sits inside the nasopharynx, the two silk threads tied to its end would have reached the anterior nares along with the free end of the nasal suction catheter.

The two silk threads tied to the suction catheters are untied. The catheters are removed from the nose. The silk thread is used to secure the pack in place by tying both the ends to the columella of the nose. The silk tied to the middle portion of the gauze pack is delivered out through the oral cavity and taped to the angle of the cheek. This middle portion silk will help in removal of the nasal pack. In addition to the postnasal pack anterior nasal packing must also be done in these patients.

Postnasal pack using balloon catheters: Specially designed balloon catheters are available. This can be used to perform the post nasal pack. Foleys catheter can be used to pack the post nasal space. Foley's catheter is introduced through the nose and slid up to the nasopharynx. The bulb of the catheter is inflated using air through the side portal of the catheter. Air is used to inflate the bulb because even if the bulb ruptures accidentally there is absolutely no danger of aspiration into the lungs. After the foleys catheter is inflated the free end is knotted and anchored at the level of the anterior nares.

Problems of nasal packing:

1. Epiphora (watering of eyes) occur due to blocking of the nasal end of the nasolacrimal duct.

2. Heaviness /headache due to blocking of the normal sinus ostium.

3. Prolonged post nasal pack can cause eustachean tube block and secretory otitis media.

4. Prolonged nasal packing can cause secondary sinusitis due to blockage of sinus ostium.

Newer packing materials: Newer packing materials made of silicone are available. The advantages of these material are that they are not irritating, patient can breathe through the nose with the pack on through the vent provided, these packs can be retained inside the nasal cavity for more than 2 weeks. They can be removed and repositioned if necessary. The only disadvantage is that they are expensive.

Surgical management:

Endoscopic cauterization can be tried if the bleeders are localised and accessible. If not accessible, ligation of the internal maxillary artery can be done through Caldwell approach. Sphenopalatine artery clipping can be done endoscopically. It is accessible close to the posterior end of the middle turbinate. In rare cases external carotid artery ligation at the neck can be resorted to. External carotid artery is differentiated from the internal carotid in the neck by the fact that internal carotid artery does not give rise to branches in the neck, while the external carotid artery does so.

Ethmoidal artery ligation: If epistaxis occurs high in the nasal vault, anterior and posterior ethmoidal arteries may be ligated using Liga clips. These arteries can be accessed using an external ethmoidectomy incision. The anterior ethmoidal artery is usually found 22mm from the anterior lacrimal crest. If ligation of the anterior ethmoidal artery does not stop bleeding then posterior ethmoidal artery should also be ligated. The posterior ethmoidal artery can be found 12mm posterior to the anterior ethmoidal vessel.

Epistaxis caused by the presence of tumors both benign and malignant calls for definitive treatment of the tumor parse.

5. Describe the Aetiology, clinical features and management of Rhinosporidiosis

Rhinosporidiosis has been defined as a chronic granulomatous disease characterised by production of polyps and other manifestations of hyperplasia of nasal mucosa. The etiological agent is Rhinosporidium seeberi.

Clinical classification of Rhinosporidiosis:

Nasal
Nasopharyngeal
Mixed
Bizarre (ocular and genital)
Malignant rhinosporidiosis (cutaneous rhinosporidiosis)

Common sites affected:

Nose - 78%
Nasopharynx - 68%
Tonsil - 3%
Eye - 1%
Skin - very rare
Gross features of rhinosporidiosis:

Lesions in the nose can be polypoidal, reddish and granular masses. They could be multiple pedunculated and friable. They are highly vascular and bleed easily. Their surface is studded with whitish dots (sporangia). They can be clearly seen with a hand lens. The whole mass is covered by mucoid secretion. The Rhinosporidium in the nose is restricted to the nasal mucous membrane and does not cross the muco cutaneous barrier.

Histopathology of nasal rhinosporidiosis:

There is papillomatous hyperplasia of nasal mucous membrane with rugae formation. The epithelium over the sporangia is thinned out, foreign body giant cells can be seen. Accumulation of mucous in the crypts seen with increased vascularity. The increased vascularity is responsible for excessive bleeding during surgery. Increased vascularity is due to the release of angiogenesis factor from the Rhinosporidial mass. Rhinosporidial spores stain with Sudan black, Bromophenol blue etc.

Features of rhinosporidiosis:

The cardinal features of rhinosporidiosis are
1. chronicity,
2. recurrence
and
3. dissemination.

The reasons for chronicity are

1. Antigen sequestration - The chitinous wall and thick cellulose inner wall surrounding the endospores is impervious to the exit of endosporal antigens from inside, and is also impermeable to immune destruction. However, this sequestered antigen may be released after phagocytosis.

2. Antigenic variation - Rhinosporidial spores express varying antigens thereby confusing the whole immune system of the body.

3. Immune suppression - ? possible release of immuno suppressor agents

4. Immune distraction - Studies of immune cell infiltration pattern has shown that immune cell infiltration has occurred in areas where there are no spores, suggesting that these infiltrates reached the area in response to free antigen released by the spores. This serves as a distraction.

5. Immune deviation

6. Binding of host immunoglobins

Treatment:

Surgery is the treatment of choice. Rhinosporidial mass can be removed intranasally, the only problem being bleeding. Post operatively the patient is started on T. Dapsone in dose of 100 mg / day for a period of 6 months.

Unsolved problems:

Habitat - Breeds in ponds (highly theoretical, spores have not been isolated from ponds even on intense effort)

Lifecycle - In the absence of viable ways to culture the organism the life cycle remains highly speculative
Pathogenicity - does not fulfill any of the 4 criterial laid down by Koch regarding the infectivity.

6. Discuss the clinical features and management of deviated nasal septum.

Septal deviations are pretty common occurrence. In fact a dot central nasal septum is a clinical curiosity. Even though septal deviations are common they are usually not severe enough to cause symptoms.

Aetiology:

Direct trauma - Many septal deviations are a result of direct trauma and this is frequently associated with damage to other parts of the nose such as fractures of nasal bone.

Birth moulding theory - Many patients with septal deviation do not give history of trauma. Birth moulding theory was propounded by Gray. According to him abnormal intrauterine posture may result in compression forces acting on the nose and upper jaws. Displacement of septum can occur in these patients due to torsion forces that occur during parturition. Dislocations are more common in primipara and when the second stage of labor lasted for more than 15 minutes. Dislocations are generally to the right in the case of left occipitoanterior presentations and to the left with right occipitoanterior presentations. Subsequent growth of nose accentuates these asymmetries.

Differential growth between nasal septum and palate - This is the most acceptable theory today. When the nasal septum grows faster in certain individuals than the palate then the nasal septum starts to buckle under pressure.

Pathophysiology:

Deformity of nasal septum may be classified into:

1. Spurs

2. Deviations

3. Dislocations

Spurs - These are sharp angulations seen in the nasal septum occurring at the junction of the vomer below, with the septal cartilage and / or ethmoid bone above. This type of deformity is the result of vertical compression forces. Fractures that occur through nasal septum during injury to the nose

may also produce sharp angulations. These fractures heal by fibrosis that extend to the adjacent mucoperichondrium. This increases the difficulty of flap elevation in this area.

Deviations - May be C shaped or S shaped. These can occur in either vertical or horizontal plane. It may also involve both cartilage and bone.

Dislocations - In this the lower border of the septal cartilage is displaced from its medial position and projects into one of the nostrils.

In patients with septal deviation a compensatory hypertrophy of the turbinates and bulla may occur on the side opposite to the deviation. If compression forces are involved the septal deviations are often asymmetrical and may also involve the maxilla, producing flattening of the cheek, elevation of the floor of the affected nasal cavity, distortion of the palate and associated orthodontic abnormalities. The maxillary sinus is usually slightly smaller on the affected side.

Anterior septal deviations are often associated with deviations in the external nasal pyramid. Deviations may affect any of the three vertical components of the nose causing:

1. Cartilaginous deviations

2. The C deviation

3. The S deviation.

Cartilaginous deviations:

In these patients the upper bony septum and the bony pyramid are central, but there is a dislocation / deviation of the cartiagenous septum and vault.

The C deviation:

Here there is displacement of the upper bony septum and the pyramid to one side and the whole of the cartiagenous septum and vault to the opposite side.

The S deviation:

Here the deviation of the middle third (the upper cartiagenous vault and associated septum) is opposite to that of the upper and lower thirds. With deviations of the nose, the dominant factor is the position of the nasal septum, hence the adage 'as the septum goes, so goes the nose'. The first step, therefore in treating the twisted nose is to straighten the septum, and if this objective is not achieved, there is no hope of successfully straightening the external pyramid.

Effects of septal deviation:

Nasal obstruction - This is always found on the side of the deviation, and can also be present on the opposite side as a result of hypertrophic changes of the turbinates.

Mucosal changes - The inspiratory air currents are abnormally displaced and frequently gets concentrated on small areas of nasal mucosa, producing excessive drying effect. Crusting will occur and the separation of the crusts often produces ulceration and bleeding. Since the protective mucous layer is lost the resistance to infection is reduced. The mucosa around a septal deviation may become oedematous as a result of Bernoulli's phenomenon. This oedema further increases nasal obstruction.

Neurological changes - Pressure may be exerted by septal deviations on adjacent sensory nerves can produce pain. This was first explained by Sluder and the resultant condition became known as 'the anterior ethmoidal nerve syndrome'. In addition to these direct neurological effects, reflex changes perhaps may result from septal deformities which affect the nasopulmonary and nasal reflexes.

Symptoms:

The symptoms caused by septal deviations are entirely the result of their effects on nasal function. The dominant symptom being nasal obstruction, but this is rarely severe enough to cause anosmia.

Signs:

Septal deviations are evident on anterior rhinoscopy. This should be done without the use of nasal speculum because the insertion of speculum is sufficient to straighten the nasal septum. When the tip of the nose is lifted septal deviation become evident. Nasal obstruction may also be present on the opposite side (paradoxical nasal obstruction). This is due to the presence of hypertrophied turbinates. If the hypertrophy is limited to turbinate mucosa alone then it will shrink when decongestant drugs are used in the nasal cavity. If the hypertrophy is bony then decongestant drops is useless.

Septal deviations in the region of the nasal valve area cause the greatest obstruction, since this is the narrowest part of the nasal cavity. This can be identified by the Cottle test. A positive Cottle test will confirm the fact that narrowing is present in the nasal valve area. This is done by asking the patient to pull the cheek outwards and this maneuver is supposed to open up the area thus reducing the block. The septum should not be considered in isolation and it is necessary to do a careful examination of the lateral wall of the nasal cavity. Whenever sinus complications like sinusitis is suspected due to obstruction to the drainage channel of the sinuses by the deviation x-ray sinus must be taken.

Septal deviation in new born is associated with asymmetry of the nostrils, an oblique columella and tip which points in the direction which is opposite to the deviation. Most of these patients are

diagnosed by the use of Gray's struts. These struts are 4mm wide and 2mm thick and after lubrication, are inserted into the nostrils and then gently pushed backwards along the floor of the nasal cavity, hugging the nasal septum. Normally these struts can be introduced for a distance of 4 - 5 cms, but in cases of septal deviation a frank obstruction is encountered, usually 1 - 2 cms from the nostril.

Cottle has classified septal deviations into three types:

Simple deviations: Here there is mild deviation of nasal septum, there is no nasal obstruction. This is the commonest condition encountered. It needs no treatment.

Obstruction: There is more severe deviation of the nasal septum, which may touch the lateral wall of the nose, but on vasoconstriction the turbinates shrink away from the septum. Hence surgery is not indicated even in these cases.

Impaction: There is marked angulation of the septum with a spur which lies in contact with lateral nasal wall. The space is not increased even on vasoconstriction. Surgery is indicated in these patients.

Indications for submucous resection of nasal septum:

1. Marked septal deviation occurring behind the vertical line passing between the nasal processes of the frontal and maxillary bones. This deviation must be the cause for the patient's symptoms.

2. Closure of septal perforations

3. Source of grafting material

4. To obtain surgical access in hypophysectomy, and Vidian neurectomy

Surgically the septum is divided into anterior and posterior segments by a vertical line passing between the nasal processes of frontal and maxillary bones

Procedure:

Submucosal resection of nasal septum is ideally performed under local anesthesia. 4% xylocaine is used as topical anesthetic agent by nasal packing. 2% xylocaine is used as infiltrative anesthetic agent. It is mixed with 1 in 1 lakh adrenaline. Infiltration is done at the mucocutaneous junction on both sides just behind the columella. The floor of the nasal cavity is also infiltrated on the concave side. Killian's incision is preferred for SMR operations. Killian's incision is the commonly used incision. It is an oblique incision given about 5mm above the caudal border of the septal cartilage.

The cartiagenous and bony nasal septum is exposed by elevation of mucoperichondrial and mucoperiosteal flaps on both sides. This is done by slicing the septal cartilage just above the columella to access the opposite side. Flaps are elevated on both sides of the nasal septum. the cartilage is fully exposed from both sides and is remove using a Luc's forceps or a Ballenger's swivel knife. The flaps are allowed to fall back in place and wound is closed with catgut. Bony deviations along the floor of the nose if any are also chiseled out before wound closure.
SMR should not be performed in children because it may affect growth.

Complications of SMR:

1. Septal hematoma

2. Septal abscess

3. Septal perforation

4. Nasal deformities due to excessive removal of dorsal strut of the septum

5. Removal of the columella cartilage will cause pig snout deformity

Septoplasty:

This is a more conservative procedure. The anesthesia is the same as described for SMR operation. The incision is always sited on the concave side of the septum. Freer's hemitransfixation incision is preferred. This is made at the lower border of the septal cartilage. A unilateral Freer's incision is sufficient for septoplasty. Three tunnels are created as shown in the figure.

Exposure: The cartiagenous and bony septum are exposed by a complete elevation of a mucosal flap on one side only. Since flap is retained on the opposite side the vascularity of the septum is not compromised.

Mobilization and straightening: The septal cartilage is freed from all its attachments apart from the mucosal flap on the convex side. Most of the deviations are maintained by extrinsic factors such as caudal dislocation of cartilage from the vomerine groove. Mobilization alone will correct this problem. When deviations are due to intrinsic causes like the presence of healed fracture line then it must be excised along with a strip of cartilage. Bony deviations are treated either by fracture and repositioning or by resection of the fragment itself.

Fixation:

The septum is maintained in its new position by sutures and splints.

Advantages of Freer's incision:

1. The incision is cited over thick skin making elevation of flap easy.

2. There is minimal risk of tearing the flap

3. The whole of the nasal septum is exposed.

4. If need arises Rhinoplasty can be done by extending the same incision to a full transfixation one.

Advantages of Septoplasty:

1. More conservative procedure

2. Performed even in children

3. Less risk of septal perforation

4. Less risk of septal hematoma

7. Discuss clinical features, diagnosis and management of chronic sinusitis.

Chronic sinusitis is defined as chronic inflammation of mucosal lining of paranasal sinuses lasting for more than 3 weeks.

Pathology: The mucosal lining of para nasal sinuses shows evidence of chronic inflammatory changes. The cilia of the lining epithelium are damaged, causing inadequate drainage of sinus cavity. This is especially more common in maxillary sinuses. Pent up secretions start to accumulate within the sinuses. This retained secretions again predispose to secondary infections and reinfections

causing a vicious cycle. There is also associated hypertrophy of the lining mucosa leading to polypoidal changes.

Clinical features:

Symptoms:

1. Nasal obstruction: This could be due to the result of underlying pathology like a deviated nasal septum / septal spur, polypoidal changes of nasal mucosa, hypertrophied turbinates. The patient in fact complains of stuffy nose.

2. Nasal discharge: The patient complains of excessive nasal discharge, which could be mucoid to begin with and may later get purulent due to super added infections. This also leads to post nasal drip causing irritation of throat and formation of granular pharyngitis.

3. Abnormalities of smell: Patients may complain of diminished acuity of smell. Patient may also present with cacosmia or parosmia

4. Headache: is another important feature of chronic sinusitis. Pent up secretions within the sinus cavity leads to head ache.

5. Epistaxis: Hyperemia of nasal mucosa due to repeated infections may lead to epistaxis.

6. Sinus tenderness could also be present

Investigations:

Xray para nasal sinuses water's view - shows hazy sinuses

CT scan paranasal sinuses plain both axial and coronal cuts are diagnostic

Management:

Medical:

1. Antibiotics: Amoxycillin is the drug of choice. Erythromycin can be considered in patients allergic to amoxycillin

2. Pain killers like acetaminophen can be used

3. Nasal decongestant drops like xylometazoline can be used

4. Antihistamines can be considered if allergy is suspected to be the cause

Surgical:

1. Antral lavage

2. Middle meatal antrostomy

3. FESS

8. Enumerate causes, clinical features and management of atrophic rhinitis.

Definition:

Atrophic rhinitis is defined as a chronic nasal disease characterized by progressive atrophy of the nasal mucosa along with the underlying bones of turbinates. There is also associated presence of viscid secretion which rapidly dries up forming foul smelling crusts. This fetid odor is also known as ozaena. The nasal cavity is also abnormally patent. The patient is fortunately unaware of the stench emitting from the nose as this disorder is associated with merciful anosmia.

Aetiology:

The etiology of this problem still remains obscure. Numerous pathogens have been associated with this condition, the most important of them are

1. Coccobacillus,

2. Bacillus mucosus

3. Coccobacillus foetidus ozaenae

4. Diptheroid bacilli

5. Klebsiella ozaenae.

These organisms despite being isolated from the nose of diseased patients have not categorically been proved as the cause for the same.

Other possible factors which could predispose to this disease are:
1. Chronic sinusitis
2. Excessive surgical destruction of the nasal mucosa and turbinates

3. Nutritional deficiencies
4. Syphilis.
5. Endocrine imbalances (Disease is known to worsen with pregnancy / menstruation)
6. Heredity (Autosomal dominant pattern of inheritance identified)
7. Autoimmune disease

The triad of atrophic rhinitis as described by Dr. Bernhard Fraenkel are 1. Fetor, 2. crusting and 3. atrophy.

Age of onset: Usually commences at puberty.

Females are commonly affected than males. Heredity is known to be an important factor as there appears to be increased susceptibility among yellow races, Latin races and American negro races. Poor nutrition could also be a factor. Bernat (1965) postulated iron deficiency could be a cause of this disorder.

Recently immunologists have considered atrophic rhinitis to be an autoimmune disorder. Fouad confirmed that there was altered cellular reactivity, loss of tolerance to nasal tissues. This according to him could be caused / precipitated by virus infection, malnutrition, immunodeficiency.

Pathology:

1. Metaplasia of ciliated columnar nasal epithelium into squamous epithelium.
2. There is a decrease in the number and size of compound alveolar glands
3. Dilated capillaries are also seen

Pathologically atrophic rhinitis has been divided into two types:
Type I: is characterized by the presence of endarteritis and periarteritis of the terminal arterioles. This could be caused by chronic infections. These patients benefit from the vasodilator effects of estrogen therapy.
Type II: is characterized by vasodilatation of the capillaries, these patients may worsen with estrogen therapy. The endothelial cells lining the dilated capillaries have been demonstrated to contain more cytoplasm than those of normal capillaries and they also showed a positive reaction for alkaline phosphatase suggesting the presence of active bone resorption. It has also been demonstrated that a majority of patients with atrophic rhinitis belong to type I category.

Once the diagnosis of atrophic rhinitis is made then the etiology should be sought. Atrophic rhinitis can be divided in to two types clinically:
1. Primary atrophic rhinitis - the classic form which is supposed to arise denovo. This diagnosis is made by a process of exclusion. This type of disease is still common in middle east and India. All the known causes of atrophic rhinitis must be excluded before coming to this diagnosis. Causative organisms in these patients have always be Klebsiella ozenae.

2. Secondary atrophic rhinitis: Is the most common form seen in developed countries. The most common causes for this problem could be:
1. Extensive destruction of nasal mucosa and turbinates during nasal surgery
2. Following irradiation
3. Granulomatous infections like leprosy, syphilis, tuberculosis etc.

Clinical features:
The presenting symptoms are commonly nasal obstruction and epistaxis. Anosmia i.e. merciful may be present making the patient unaware of the smell emanating from the nose. These patients may also have pharyngitis sicca. Choking attacks may also be seen due to slippage of detached crusts from the nasopharynx into the oropharynx. These patients also appear to be dejected and depressed psychologically.
Clinical examination of these patients show that their nasal cavities filled with foul smelling greenish, yellow or black crusts, the nasal cavity appear to be enormously roomy. When these crusts are removed bleeding starts to occur.

Why nasal obstruction even in the presence of roomy nasal cavity?
This interesting question must be answered. The nasal cavity is filled with sensory nerve endings close to the nasal valve area. These receptors sense the flow of air through this area thus giving a sense of freeness in the nasal cavity. These nerve endings are destroyed in patients with atrophic rhinitis thus depriving the patient of this sensation. In the absence of these sensation the nose feels blocked.

Radiographic findings:
Are more or less the same in both primary and secondary atrophic rhinitis. Plain xrays show lateral bowing of nasal walls, thin or absent turbinates and hypoplastic maxillary sinuses.

CT scan findings:
1. Mucoperiosteal thickening of paranasal sinuses
2. Loss of definition of Osteomeatal complex due to resorption of ethmoidal bulla and uncinate process
3. Hypoplastic maxillary sinuses
4. Enlargement of nasal cavity with erosion of the lateral nasal wall
5. Atrophy of inferior and middle turbinates

Management:
Conservative:
Nasal douching - The patient must be asked to douche the nose at least twice a day with a solution prepared with:

Sodium bicarbonate - 28.4 g
Sodium diborate - 28.4 g
Sodium chloride - 56.7 g

mixed in 280 ml of luke warm water.

The crusts may be removed by forceps or suction. 25% glucose in glycerin drops can be applied to the nose thus inhibiting the growth of proteolytic organism.
In patients with histological type I atrophic rhinitis oestradiol in arachis oil 10,000 units/ml can be used as nasal drops.

Kemecetine antiozaena solution - is prepared with chloramphenicol 90mg, oestradiol dipropionate 0.64mg, vitamin D2 900 IU and propylene glycol in 1 ml of saline.

Potassium iodide can be prescribed orally to the patient in an attempt to increase the nasal secretion.
Systemic use of placental extracts have been attempted with varying degrees of success.

Surgical management:

1. Submucous injections of paraffin, and operations aimed at displacing the lateral nasal wall medially. This surgical procedure is known as Lautenslauger's operation.
2. Recently teflon strips, and autogenous cartilages have been inserted along the floor and lateral nasal wall after elevation of flaps.
3. Wilson's operation - Submucosal injection of 50% Teflon in glycerin paste.
4. Repeated stellate ganglion blocks have also been employed with some success
5. Young's operation - This surgery aims at closure of one or both nasal cavities by plastic surgery. Young's method is to raise folds of skin inside the nostril and suturing these folds together thus closing the nasal cavities. After a period of 6 to 9 months when these flaps are opened up the mucosa of the nasal cavities have found to be healed. This can be verified by postnasal examination before revision surgery is performed. Modifications of this procedure has been suggested (modified Young's operation) where a 3mm hole is left while closing the flaps in the nasal vestibule. This enables the patient to breathe through the nasal cavities. It is better if these surgical procedures are done in a staged manner, while waiting for one nose to heal before attempting on the other side.

9. Discuss etiology, clinical features and management of chronic maxillary sinusitis.

Chronic maxillary sinusitis: Is defined as infections involving the maxillary sinus lasting for more than 3 months

Chronic infections are common in maxillary sinuses because of its:

1. Nondependent drainage pathway

2. The close proximity of its drainage channel to anterior ethmoidal air cells.

Aetiology:

1. Commonly follows acute sinusitis

2. In Kartagener's syndrome it is common

3. Dental sepsis (Due to the close proximity of upper premolars and molars to its floor)

4. Allergy

5. Deviated nasal septum blocking its drainage

6. Fungal infections

7. Iatrogenic causes: Nasal packing, Naso gastric tube insertion

Types of chronic maxillary sinusitis:

1. Hypertrophic / Polypoidal sinusitis: This is also known as catarrhal sinusitis. The inflammation mainly affects the efferent vessels and lymphatics. To start with there is periphlebitis and perilymphangitis. Repeated such attacks may cause oedema and polypoidal changes in the mucous membrane lining the sinus cavity.

2. Atrophic sinusitis: Also known as suppurative sinusitis. The main pathology lies in the afferent vessels causing a thickening of the vessel walls. Oedema is not a feature of this type of sinusitis. Both types of sinusitis can coexist in the same sinus.

Clinical features:

1. Nasal obstruction

2. Post nasal drip

3. Epistaxis

4. Abnormalities of smell (cacosmia, parosmia, or hyposmia)

5. Vestibulitis

6. Headache

7. Eustachean tube block

8. Secondary tonsillitis

9. Laryngitis

Clinical findings:

Anterior rhinoscopy shows swollen mucosa with pus in the middle meatus. Pus can be made to appear by making the patient to put the head between the knees for a few seconds before anterior rhinoscopic examination.

Patients may have post nasal drip and granular pharyngitis.

Investigations:

X-ray paranasal sinuses water's view shows haziness of the affected maxillary sinus.

CT scan is diagnostic. It will reveal the exact cause for this infection.

Management:

1. Antibiotics (Penicillin or amoxycillin are the drugs of choice)

2. Antihistamines

3. Decongestant nasal drops to decongest the nasal mucosa

Minor surgical procedure like antral wash.

Intranasal antrostomy

Caldwell luc surgery in resistant cases.

FESS is the preferred modality of treatment these days

10. Discuss etiology, clinical features, management and complications of acute frontal sinusitis

Introduction:

Acute frontal sinusitis is defined as inflammation of mucosal lining of frontal sinus and it's out flow tract of less than 3 weeks duration. The incidence of acute frontal sinusitis is considerably lower when compared with that of maxillary sinusitis in adults and ethmoidal sinusitis in children. Early diagnosis and management of acute frontal sinusitis will go a long way in preventing development of complications.

Incidence:

Acute sinusitis commonly affects 20% of population. Acute frontal sinusitis affects about 4% of these individuals. Acute frontal sinusitis commonly affects adolescent males and young men. The age predilection is due to the fact that frontal sinuses become vascular and enlarge rapidly during 7 – 15 years of life. Male predilection largely remains unexplained.

Etiopathogenesis:

Acute frontal sinusitis is commonly preceded by viral infections of upper respiratory tract. Rhino virus has been commonly implicated. Other viruses like corona virus, respiratory syncytial virus and Para influenza viruses have been implicated. Viral infections up regulate inflammatory cytokines like IL6, IL8, Tumor necrosis factor ά, histamine and bradykinin. These viruses are also known to suppress neutrophils, macrophages and lymphocytic functions inhibiting immune response. The induction of inflammatory cascade causes mucosal oedema, occlusion of sinus Ostia, impairing mucociliary clearance mechanism. This causes stasis of secretions within the frontal sinus. Mucus stasis forms a nidus within the sinus for super added bacterial infections.

Host risk factors involved in the pathogenesis of acute frontal sinusitis:

These factors which predispose to acute frontal sinusitis include

Deviated nasal septum
Nasal polyposis

Immune deficiency states
Since frontal sinus is derived from the anterior ethmoidal cells these cells can cause obstruction to the outflow tract causing sinusitis. These structures include: agger nasi cells anteriorly, bulla posteriorly, supraorbital cells laterally and type I – type IV frontal cells.

A series of accessory ethmoidal cells line the frontal sinus outflow tract. These cells receive various names according to their position in relation to the sinus outflow tract. These cells include:

1. The agger nasi cell

2. Frontal intersinus septal cells

3. Suprabullar cells

4. Frontal / infundibular cells

Bent and Khun classified frontal infundibular cells based on their proximity to agger nasi cell.

A – Agger nasi cell

I – Type I frontal cell (a single air cell above agger nasi)

II – Type II frontal cell (a series of air cells above agger nasi but below the orbital roof)

III – Type III frontal cell (this cell extends into the frontal sinus but is contiguous with agger nasi cell)

IV – Type IV frontal cell lies completely within the frontal sinus

Diagnosis:

Acute frontal sinusitis is a clinical diagnosis depending on the duration of symptoms, i.e. lasting for less than 4 weeks. CT scans may show false positive results.

Major diagnostic criteria include:

Pain / tenderness over frontal sinus (tenderness can be elicited by applying pressure in the floor of the frontal sinus.
Head ache showing classic periodicity (More during early morning hours and gets better as the day progresses). This is due to the gravitational effects of frontal sinus drainage.
Nasal obstruction
Purulent rhinorrhoea
Fever
Hyposmia / anosmia

Unless complications are suspected imaging is not a must in the diagnosis of acute frontal sinusitis.

Microbiology: Organisms causing acute infections of frontal sinus include S. Pneumoniae, H. Influenza, and Moraxella Catarrhalis.

Aims of treatment:

To control infections using antibiotics
To reduce oedema and remove obstruction to sinus ostium facilitating drainage
Medical treatment will suffice. Surgery is not needed.

Antibiotics chosen should be able to manage the infecting spectrum of organism.

Complications of acute frontal sinusitis:

Should be suspected in patients with:

Protracted symptoms with increasing severity
Periorbital oedema due to preseptal cellulitis

Painful and restricted eye movements (orbital cellulitis)
Neurological signs and symptoms indicate intracranial complications

Meningitis is one of the important intracranial complications of acute frontal sinusitis. Signs and symptoms of meningitis include:

High fever
Photophobia
Neck pain
Neck stiffness
Severe headache
Altered mental status

Osteomyelitis is one of the complications of acute frontal sinusitis. This is caused by direct extension of infection or by thrombophlebitis involving the diploic veins. In patients with osteomyelitis of anterior table of frontal bone may lead to formation of subperiosteal abscess which present as swelling over the forehead. This is also known as the "Pott's puffy tumor".

Cavernous sinus thrombosis is a complication of acute frontal sinusitis. Thrombophlebitis involves the diploic veins which are valve less. The infection spreads to cavernous sinus causing thrombophlebitis. Patients with cavernous sinus thrombosis present with ophthalmoplegia, proptosis, visual loss, trigeminal nerve deficits.

Surgery is indicated in recalcitrant cases. It includes frontal sinus trephining and endoscopic decompression.

11. Discuss the aetiopathology of malignant tumors of the maxilla. Add a note on investigations and its surgical management.

Cancers involving maxillary sinus are rather uncommon. Incidence ranges between 0.5-1% of all malignancies. It constitutes about 3% of all head and neck malignancies.

Etiological factors include:
Viral
infections – EB virus, and Human papilloma virus infections

Exposure to wood dust – Especially African Mahogany wood dust causes adenocarcinoma of maxillary sinus. People working in nickel and chrome industries are more prone to develop cancer of maxillary sinus. People working in leather industries are also known to develop cancer of maxillary sinus.
Iatrogenic causes – Post irradiation
Use of snuff have also been documented to be the causative factor
Commonest type of malignancy involving the maxillary sinus is squamous cell carcinoma about 80%. The second commonest tumor involving the maxillary sinus is adenocarcinoma.

The following are the various types of malignant tumors of maxillary sinus:

Squamous
cell carcinoma
Adenocarcinoma
Transitional
cell carcinoma
Anaplastic
carcinoma
Malignant
melanoma
Adenoid
cystic carcinoma
Olfactory
neuroblastoma
Lymphomas
Clinical features:

Face – Swelling of the cheek. Pain and paresthesia over the cheek.

Orbital – Proptosis, diplopia, loss of vision

Nasal – Nasal deformity, unilateral nasal obstruction, blood tinged nasal discharge, epistaxis, hyposmia (rare)

Neurological – Multiple cranial nerve paralysis

Oral – Loosening of teeth, ill-fitting dentures, swelling involving palate, trismus (due to involvement of pterygoid muscles)

Otological symptoms – Ear block due to eustachean tube involvement, referred otalgia

Cervical symptoms – Cervical nodal metastasis

Involvement of anterolateral wall of maxilla present as:

Infraorbital
nerve paresthesia / anesthesia
Swelling
over cheek
Involvement of inferior wall of maxilla present as:

Palatal
swelling
Swelling over buccogingival sulcus
Loosening of upper dentition
Oroantral fistula
Trismus is seen in patients with involvement of pterygoid muscles

Involvement of floor of orbit present as:

Restriction of ocular movement
Proptosis
Periosteal thickening over orbital rim
Involvement of medial wall presents as:

Mass inside nasal cavity

Investigations:

Nasal endoscopy – If there is involvement of medial wall of maxilla the mass could be seen to present itself inside the nasal cavity. If the mass could be seen within the nasal cavity biopsy can be taken from the lesion. Under endoscopic vision inferior meatal antrostomy can be performed and the interior of the maxillary sinus can be examined and biopsy can be taken from the lesion.

X-ray paranasal sinuses water's view – shows opacity with expansion of the involved maxillary sinus. Erosion of the floor / anterolateral wall of the orbit can also be seen if present.

CT scan paranasal sinuses – Shows the extent of lesion, involvement of adjacent areas, evidence of bone erosion if present.

MRI imaging shows better soft tissue delineation. Extension into pterygopalatine fossa can be clearly seen.

Biopsy from the lesion is virtually diagnostic.

Management:

The optimal management modality depends on the extent of tumor and the histological type.

Treatment modalities available:

Surgery
Radiotherapy
Chemotherapy
Combined management modality

If the tumor is confined to the inferior portion of the maxilla the condition is best managed by partial maxillectomy followed by irradiation.

Tumor involving the whole of the maxilla can be managed by total maxillectomy followed by irradiation.

Involvement of orbit can be managed by combining orbital exenteration along with total maxillectomy.

Tumors of maxilla extending to infratemporal fossa can be managed by extended maxillectomy using Barbosa technique. Maxillectomy is combined with condylectomy and resection of pterygoid plate and muscles attached to it.

Neck dissection can be resorted to if neck nodes are involved.

Irradiation:

Is given by using Telecobalt or linear accelerator. Dosage include 6500 rads in divided fractions over 5 weeks. It is usually administered 5 days a week.

Chemotherapy:

Cisplatin and 5flurouracil can be administered along with radiotherapy. This is preferred in advanced cases of malignancy involving the maxillary sinus.

12. 60 year old patient with bleeding fleshy mass from nose. Write differential diagnosis, investigations required to diagnose the underlying pathology. Add a note on chemotherapy and surgical treatment of Carcinoma Maxilla.

Differential diagnosis of bleeding fleshy mass from nose in elderly:

Benign masses:

1. Nasal polyp with squamous metaplasia due to exposure

2. Angioma nose

3. Fibroma

4. Rhinoscleroma

5. Inverted Papilloma

Malignant lesions:

1. Adenocarcinoma nose / sinuses

2. Squamous cell carcinoma nose / sinuses

Investigations:

Imaging:

CT scan of nose and sinuses will help in exactly identifying the extent of the lesion. Presence of bony erosion can be clearly seen in CT images.

Contrast CT images would provide details about the vascularity of the mass in some cases exact blood supply can also be identified.

MRI:

This would help in identification of soft tissue masses and extent of the tumor. It also clearly brings out intracranial extension of the mass if it has managed to breach the skull base.

Biopsy from the lesion would help in the final diagnosis.

Surgical treatment of malignant growth maxilla:

Total maxillectomy can be done to remove the mass in its entirety.

Chemotherapy:

Cisplatin and 5flurouracil can be administered along with radiotherapy. This is preferred in advanced cases of malignancy involving the maxillary sinus.

THROAT

1. Describe the aetiopathology, clinical features, diagnosis and management of chronic tonsillitis. Mention complications of tonsillitis.

The duration of illness is more than 3 weeks. These patients have milder symptoms when compared to those with acute tonsillitis. Tonsils are enlarged. Tonsillar enlargement can be graded under 4 groups:

Grade 0: The tonsils are fully inside the pillars.

Grade 1: Tonsils found to be enlarged and out of its pillars

Grade 2: Tonsillar enlargement extends just up to half the distance of the uvula

Grade 3: Tonsillar enlargement up to the level of the uvula.

Grade 4: Tonsillar enlargement is so huge that they are virtually in contact with each other i.e. Kissing tonsil.

The anterior pillars are congested. The jugulodigastric nodes are enlarged and tender.

Types of chronic tonsillitis:

Chronic follicular tonsillitis:

In these patients the tonsillar enlargement is associated with the presence of prominent inflamed follicles. Whitish material can be seen extruding from the follicles when the anterior pillars are

pressed with a tongue depressor. This is known as the squeeze test. A positive squeeze test always indicates the diagnosis of chronic follicular tonsillitis. Inflammation and blockage of crypta magna in these patients lead on to the formation of Quincy or peritonsillar abscess.

Chronic parenchymatous tonsillitis:

In these patients' tonsils are enlarged but the follicles are not prominent. Infection is found within the substance of the tonsil.

Infection in patients with chronic tonsillitis is always poly microbial with a predominance of gram negative and anaerobic organisms. Surgery is commonly indicated in these patients.

Faucial diphtheria:

Causes membranous tonsillitis. Membranous exudate is seen over tonsils and soft palate, followed by its distant toxic effects.

It is caused by Corynebacterium diphtheria. Three different strains of diphtheria have been identified, they are Gravis, Intermedius and Mitis. These organisms grow in Loeffler's media or Tellurite agar. These organisms ferment glucose. This infection is rare these days because of the success of universal immunization programme.

Pathogenesis:

Multiplication of organism leads to production of toxins which cause epithelial necrosis with collection of polymorphs and fibrin leading formation of false membranous formation (because it consists of necrotic layer of mucosa, where as true membrane is superimposed over the intact mucosa).

Clinical features:

1. The child is very quiet

2. Lassitude

3. Malaise

4. Head ache

5. Fever

6. Foetor

On examination a greyish / yellowish thick membrane on one or both tonsils extending up to the soft palate and uvula. The membrane can be removed leaving a raw under surface. Massive cervical adenitis is also seen i.e. Bull neck.

Diagnosis is by:

Signs and symptoms

Throat swab for culture and sensitivity

Lymphocyte count is raised

Albuminuria is seen

Schick test is positive

Differential diagnosis:

Acute streptococcal tonsillitis

Oral thrush

Infectious mono nucleosis

Quincy

Treatment:

The patient is kept in isolation for 2 weeks

If myocarditis has set in patient must be kept in bed rest till the ECG become normal

The patient must have 3 negative swabs before discharge

Anti diphthertic serum must be administered in acute cases:

- for mild cases 20,000 units

- moderate cases 40,000 units - 80,000 units

Half dose is given as intra muscular injection and the other half as intra venous injections.

Injection penicillin is administered in doses of 5 - 10 lakhs

Tracheostomy is done in patient's with stridor.

Complications:

1. Myocarditis & circulatory failure

2. Peripheral neuritis with palatal palsy

3. Ocular muscle palsy

4. Peripheral neuritis

2. Mention the differential diagnosis of white patch in Tonsil.

Differential diagnosis of membrane over the tonsil:

The following are different conditions that present with membrane in the tonsillar region.

1. Membranous tonsillitis:

It occurs due to pyogenic organisms. An exudative membrane forms over the medial surface of tonsils along with features of acute tonsillitis.

2. Diphtheria:

Unlike acute tonsillitis which is abrupt in onset, diphtheria is slower in onset with less local discomfort. The membrane in diphtheria extends beyond the tonsils on to the soft palate. The membrane is dirty white in color. This membrane is adherent and its removal would eventually leave a bleeding surface. Urine from these patients would show albumin. Smear and culture from throat swab will reveal Corynebacterium diphtheriae.

3. Vincent angina:

This condition is caused by the presence of fusiform bacilli and spirochetes. This condition is insidious in onset with less fever and less discomfort in throat. Membrane forms over one tonsil and can be easily removed revealing an irregular ulcer on the tonsil. Throat swab will reveal the two organisms.

4. Infectious mononucleosis:

This condition affects young adults. Both tonsils are enlarged, congested and covered with membrane. There is also marked local discomfort. Lymph nodes in the posterior triangle of neck is enlarged and there is also splenomegaly. This disease is suspected because of failure of antibiotic treatment. Blood smear may demonstrate more than 50% lymphocytes of which about 10% are atypical. Paul Bunnel test is positive.

5. Agranulocytosis:

This condition presents with ulcerative necrotic lesions on the tonsils and oropharynx. These patients are severely ill. In its acute fulminating form the total leukocyte count is decreased to less than 2000/cubic mm. This could even to down to as low as 50/cubic mm.

6. Leukemia:

This condition also causes membranes to appear over tonsil. 75% of leukemias in children are acute lymphoblastic and 25% acute myelogenous. In adults 20% of acute leukemias are lymphocytic and 80% nonlymphocytic.

7. Aphthous ulcers:

This condition may involve any portion of the oral cavity or oropharynx. Sometimes it is solitary and may involve tonsils and their pillars. It may be small or large and could cause lot of pain.

8. Malignancy tonsil

10. Candida infection of tonsil

3. Describe the diagnosis, complications and treatment of acute follicular tonsillitis

Acute follicular tonsillitis:

The duration of illness is less than three weeks old. The patient in fact may not give any history of recent similar attacks.

Clinically these patients have:

1. Fever

2. sore throat

3. Foul smelling breath

4. Odynophagia (painful swallowing)

5. Tender enlarged upper deep cervical node belonging to the jugulodigastric group. These nodes are palpable just below the angle of the mandible.

6. In young children this condition is almost always associated with enlarged adenoids which may cause nasal airway obstruction and obstructive sleep apnoea syndrome.

7. Generally these patients are lethargic and toxic.

These patients have enlarged tonsils with prominent follicles.

Causative organisms of acute follicular tonsillitis:

1. Streptococci pneumoniae (commonest)

2. Staphylococcus

3. Pneumococcus

4. H. Influenzae

5. Diphtheroid

6. Viral

Investigations:

Throat culture is a must to identify the presence of beta hemolytic streptococci. The major disadvantage is that it takes a minimum of 48 hours for the culture to be reported. There is also the false negative results to contend with. Cultures must be performed when the body temperature is more than 38.3 .C or when the illness is characterized by sore throat. But a culture cannot differentiate between acute and chronic infections.

Carrier states are common among false negative patients. These patients can be identified by ASO titer assessment. A carrier will have a positive culture for hemolytic streptococci with a negative ASO titer.

Rapid tests for streptococci have been introduced among which rapid strep test has proved accurate and cost effective.

Medical management:

Therapy is directed at aerobic pathogens i.e. beta hemolytic streptococci. Penicillin's are the drug of choice. Ampicillin / Amoxycillin in doses of 40 - 50 mg /kg body weight can be used. Anaerobes have been shown to be involved in recurrent tonsillitis hence clindamycin in considered in recurrent and resistant cases.

Complications of tonsillitis:

1. Peri tonsillitis

2. Quincy

3. Pharyngeal abscess

4. Otitis media

5. Septic foci leading on to subacute bacterial endocarditis, nephritis or rheumatic fever

6. Septicemia (rare)

4. Discuss differential diagnosis of acute tonsillitis.

Infectious mononucleosis should be considered in adolescent / young child with acute tonsillitis when it is accompanied by tender cervical, axillary, inguinal nodes, splenomegaly, low grade fever, severe lethargy. This condition is caused by Epstein Barr virus.

Herpes simplex virus infections of tonsils can present as swollen tonsils. There may be aphthous ulcer on their surfaces. Ulcers are commonly seen over the anterior pillars of the tonsils. Ulcers can also be seen over the posterior pharyngeal wall. There may be associated Herpes labialis, gingival stomatitis.

Lymphoma of tonsil:

This condition presents as uniform fleshy enlargement of tonsil. There is also associated cervical adenopathy. Majority of these cases belong to non-Hodgkin's category.

Diphtheria:

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Candida infection of tonsil

5. What are the diseases in ENT which are likely to produce trismus. Describe clinical features and management of quinsy.

Trismus is inability to open the mouth. Normal mouth opening ranges between 25 - 50mm. Any value less than this is known as trismus. (Roughly the opening should permit a minimum of three fingers when inserted sideways).

ENT causes of trismus:

Inflammatory causes involving oral cavity and throat:

1. Acute tonsillar infections like quinsy can cause inability to open the mouth fully due to spasm of masticatory muscles. This spasm is due to excessive pain due to the inflammation of tonsillar and peritonsillar tissues.

2. Inflammation involving parotid salivary gland. Parotid gland infections cause reflex spasm of muscles of mastication leading on to trismus.

3. Dental infections involving unerupted molar tooth, or dental abscess

4. Submucosal fibrosis (painless trismus)

5. Retro pharyngeal abscess

6. Ludwig's angina

Inflammatory conditions involving external ear:

Inflammations of external auditory canal i.e. furuncle can cause pain on mouth opening there by reducing it.

Otolaryngological tumors causing trismus:

1. Carcinoma cheek

2. Carcinoma mandible

3. Carcinoma maxilla

4. Malignant tumors involving parotid gland

5. Tumors of parapharyngeal spaces

6. Juvenile nasopharyngeal angiofibroma

Quinsy otherwise also known as peritonsillar abscess is a collection of pus in the peritonsillar space between the superior constrictor and capsule of the tonsil. It is usually unilateral, and commonly affects adolescent males.

Pathophysiology:
Infection usually starts in the crypta magna from where it spreads beyond the confines of the capsule causing peri tonsillitis initially, and peritonsillar abscess later.

Another proposed mechanism is necrosis and pus formation in the capsular area, which then obstructs the weber glands, which then swell, and the abscess forms.

Weber's glands:

These are mucous (minor) salivary glands present in the space superior to the tonsil, in the soft palate. There are 20 - 25 such glands in this area. These glands are connected to the surface of the tonsil by ducts. The glands clear the tonsillar area of debris and assist with the digestion of food particles trapped in the tonsillar crypts. If Weber's glands become inflamed, local cellulitis can develop. Inflammation causes these glands to swell up causing tissue necrosis and pus formation i.e. the classic features of quinsy. These abscesses generally form in the area of the soft palate, just above the superior pole of the tonsil, in the location of Weber's glands.

The occurrence of peritonsillar abscesses in patients who have undergone tonsillectomy further supports the theory that Weber's glands have a role in the pathogenesis.

Aetiology:
Recurrent attacks of tonsillitis cause obstruction and obliteration of intra tonsillar clefts and the infection spreads to peritonsillar area causing suppuration.

Smoking and chronic periodontal disease could also cause quinsy.

Clinical features:

1. Patient looks very ill and febrile
2. Odynophagia (painful swallowing)
3. Dribbling of saliva
4. Inability to open mouth
5. Muffled / Hot potato voice otherwise known as rhinolalia clausa

On examination:

The tonsil is found pushed downwards and medially; it blanches on slight pressure. The uvula is edematous and is pushed to the opposite side. Tonsillar pillars are congested. Patient also has halitosis (bad breath), trismus and tender enlarged jugulodigastric nodes.

Medical management:

1. Broad spectrum antibiotics. The anti-bacterial spectrum should ideally include gram positive, gram negative and anaerobes. Commonly used drugs are broad spectrum penicillin's like ampicillin / amoxycillin, in addition to which metronidazole or clindamycin can be combined to take care of anaerobes.
2. Anti-inflammatory drugs like Ibuprofen and antipyretics like paracetamol.

Surgical management:

Incision and drainage: This is performed with patient in sitting position to prevent aspiration of pus into the larynx. First the oral cavity and throat of the patient is sprayed with 4 % topical xylocaine spray to anaesthetize the mucosa.
A Saint claire Thompson quinsy forceps, or a guarded 11 blade can be used. The 11 blade is guarded to prevent the blade from penetrating the tonsillar substance deeply and damaging underlying vital structures like internal carotid artery.

Site of incision:
Is commonly over the point of maximum bulge. It can also be made at the junction between a horizontal imaginary line drawn from the base of the uvula to the anterior pillar and a vertical imaginary line drawn along the anterior pillar. After incision is made a sinus forceps is introduced to complete the drainage procedure.

Six weeks after I&D tonsillectomy is performed in this patient to prevent further recurrence. This is known as interval tonsillectomy.
Some authors prefer to do tonsillectomy immediately on a quinsy patient. This is known as Hot tonsillectomy. But this method is fraught with danger because of excessive bleeding and impending risk of thromboembolism.

6. List the causes of stridor. Describe the indications, operative technique and complications of tracheostomy

Stridor is an abnormal, high pitched sound produced by turbulent airflow through a partially obstructed airway at the level of supraglottis, glottis, subglottis.

Stridor should be differentiated from stertor, which is low pitched, snoring type sound generated at the level of nasopharynx, oropharynx and occasionally supraglottis.

Stridor is a symptom and not a diagnosis and the underlying cause must be determined. It may be inspiratory (common), expiratory or biphasic depending on its timing in the respiratory cycle.

Inspiratory stridor suggests a laryngeal obstruction

Expiratory stridor implies tracheobronchial obstruction

Biphasic stridor suggests a subglottic / glottic anomaly

Causes of stridor:

Acute stridor is caused by:

FB aspiration (common in children between 1-2 years of age)

Bacterial tracheitis

Retropharyngeal abscess

Peritonsillar abscess

Spasmodic croup

Epiglottitis

Chronic stridor:

Laryngomalacia

Subglottic stenosis - present with inspiratory / biphasic stridor

Bilateral abductor palsy

Malignant growth larynx especially in the vocal cords

Congenital malformations of larynx

Laryngeal papilloma

Laryngeal / Tracheal stenosis

Extrinsic compression of trachea due to vascular rings, double aortic arch.

Indications of tracheostomy:

"The main indication of tracheostomy is that when the surgeon thinks about it" (Mosher).

1. In upper air way obstruction (obstruction above the level of larynx). Tracheostomy is indicated in all cases of upper airway obstruction irrespective of the cause as an emergency lifesaving procedure. It is also indicated in impending upper airway obstruction as in the case of angioneurotic oedema of larynx.

2. For assisted ventilation: In comatose patients who do not have the required respiratory drive airway can be secured by performing a tracheostomy and the patient can be connected to a ventilator for assisted ventilation.

3. For bronchial toileting: Chronically ill patients who do not have sufficient energy to cough out the bronchial secretions may have to undergo tracheostomy with the primary aim of sucking out the bronchial secretions through the tracheostome.

4. In cases of prolonged intubation: tracheostomy will have to be performed to prevent subglottic stenosis.

Advantages of tracheostomy:

1. The procedure permanently secures the airway.

2. The anatomical dead space is reduced.

3. Tracheostomy bypasses the upper airway and hence it is useful in upper airway obstructions.

4. Suction can be applied through the tracheostome and bronchial secretions can hence be cleared.

5. If portex tube is used as tracheostomy tube it can be connected to a ventilator thus assisting the process of ventilation. The silastic tracheostomy tubes should not be sterilized using ethylene oxide because it has the ability to retain some to the gas for a period up to one week. The slow liberation

of toxic bye products such as ethylene glycol and ethylene chlorohydrin can cause severe mucosal damage. These tubes have reduced the frequency of tracheostomy tube changes.

Disadvantages of tracheostomy:

1. It is a surgical procedure and hence has morbidity and mortality rates associated with surgical procedures.

2. The tracheostomy tube will have to be cleaned periodically.

3. During early phases periodical suction must be applied hence hospital support is a must.

4. The patient may not be able to use the voice. Some tracheostomy tube like the Fuller's metal tube may have a speaking valve which could help the patient to speak, the patient must get used to plugging the hole while speaking.

5. Decannulation is a complicated procedure.

Surgical procedure:

Anesthesia: Under emergency situations it is performed under local infiltration anesthesia. Under elective conditions it is performed under general anesthesia.

Position: Supine with neck hyperextended.

Incision: Emergency tracheostomy is performed with a vertical incision extending from the lower border of cricoid cartilage up to 2cm above supra sternal notch. This area is also known as the Burn's space and is devoid of deep cervical fascia.

Elective tracheostomy is performed through a horizontal incision at 2cm above the sternal notch.

If performed under emergency settings local anesthesia is preferred. The drug used is 2 % xylocaine with 1 in 100000 adrenalines. 2 ml of this solution is infiltrated in to the Burns space.

Through a vertical incision extending from the lower border of cricoid cartilage up to 2cm above the sternal notch the skin, platysma, and cervical fascia are incised. Branches of anterior jugular vein if any are ligated and divided. Sternohyoid and Sternothyroid muscles are retracted using langenbachs retractors. The anterior wall of trachea is exposed after splitting the pretracheal fascia. The tracheal rings are clearly identified. Few drops of 2% xylocaine is instilled into the trachea through a syringe. This process serves to desensitize the tracheal mucosa while it is being incised. Incision over the trachea is sited between the second and the third tracheal rings. If the tracheostome is planned for a long duration then it is better to excise a portion of the tracheal ring completely. If tracheostomy is planned for a short duration of less than a month then the cartilage is

not completely removed but partially excised creating a flap based either superiorly or inferiorly. This is known as the Bjork flap. This flap can be sutured to the skin to keep the tracheostome open. Tracheostomy tube is inserted into the opening and the wound is closed with silk.

A wet gauze is placed over the tracheostome in order to moisturize the inspired air.

If the patient is to be connected to a ventilator, then a portex tube is used. If the tracheostomy is performed to relieve acute airway obstruction then a metal tracheostomy tube like the Fuller or Jackson is preferred.

Complications of tracheostomy:

1. Injury to thyroid isthmus causing troublesome bleeding

2. Too lateral dissection may cause extensive bleeding and possible injury to recurrent laryngeal nerve.

3. Injury to the apex of the lung (right)

4. Sudden apnoea when the trachea is opened, due to loss of hypoxic respiratory drive. This can be prevented by slow opening of the trachea, or by subjecting the patient to inhale carbogen a mixture of carbon dioxide and oxygen.

5. Subcutaneous emphysema if pretracheal fascia is not dissected properly, or too small a tube is introduced into the tracheostome.

6. Injury to great vessels. This can occur in children.

7. What are the causes of stridor in children? Discuss in detail the management of foreign body in the air way.

Stridor is an auditory manifestation of a disordered respiratory function. In simple terms it could be termed as noisy breathing. It should be borne in mind that not all noisy breathing is stridor. Voluntary or involuntary vocalizations, moist sounds such as bubbling of secretions in the larynx or pharynx are to be excluded.

There is one more noisy breathing known as stertor, derived from the Latin word stertor meaning to snore. This is caused by obstruction of airway above the level of the larynx. It is low pitched snoring or snuffly sound produced by vibrations of tissue of the naso pharynx, pharynx or soft palate. Stridor on the other hand is caused due to air flow changes within the larynx, trachea or bronchi. Stridor is in fact more often musical in character. The frequency of sound produced also ranges from low pitched to high pitched frequencies.

Aerodynamic explanation of stridor and stertor:

Stridor and stertor are both due to turbulence of the air flow within a partially obstructed respiratory tract. In order to understand its mechanism, we will have to go back to the physics of airflow. According to Bernoulli's theorem if air passes through a tube which has constrictions, the velocity of gas increases at the level of the narrowing, and in order to preserve the law of conservation of energy, the local gas pressure falls. The same principle can be applied to air flowing through the respiratory tract, which is flaccid and compressible in a child. There is a natural increase in the pressure gradient at the sites of constriction, resulting in a collapse of the airway and temporary cessation of airflow. Now Pascal's principle come into play, which states that in fluid / gas at rest the pressure changes in one part is transmitted without loss to every portion of the fluid or gas and to the walls of the container. In addition to this the resilience of the cartiagenous support of the airway in children cause the airway to spring open thus resuming the air flow. Thus, the fluttering vibrations caused are amplified by the various resonators giving rise to audible sounds which are known as either a stridor or a stertor.

Stertor is always inspiratory in its timing and has a low frequency, where as a stridor can occur both during inspiration or expiration with varying frequencies. Caution should be exercised while applying these criteria to differentiate stridor from stertor because there can always be a overlap between these conditions.

1. During inspiration, the relatively supple, poorly supported structures of the infantile supraglottis tend to be drawn into the glottic aperture due to the pressure differential that exist between the pharynx and trachea during inspiration, on the contrary expiration forces the prolapsed tissues out of the laryngeal inlet. The noise caused by this process is limited to the inspiratory phase and is often low pitched in character. Stridor of this type occur in laryngomalacia.

2. In smaller bronchi and bronchioles, during expiration there is accentuation of bronchial muscle contraction combined with high velocity gas flow cause collapse of the airway causing stridor during expiratory phase, it could be heard as a wheeze. This is commonly caused by retained secretions or foreign bodies.

3. The relatively rigid walls of rima glottis and trachea prevent collapse and hence stridor arising from these areas is due to turbulence of airflow alone. If the obstruction is severe to and fro stridor can occur (biphasic).

The associated signs / symptoms associated with stridor are

1. Dyspnea: Occurs in all patients with airway obstruction. It is also associated with other signs like flaring of the nostrils, use of accessory muscles of respiration, and cyanosis may also be present in extreme cases. During obstruction an extraordinarily high negative pressure causing indrawing of soft tissues in the subcostal, intercostal, suprasternal and substernal areas. Severe indrawing of the suprasternal area may cause a phenomenon known as the tracheal tug, where the trachea appears to be pulled down into the chest with each inspiration. In infants & neonates who have a soft compliant chest wall the indrawing of the subcostal space may be a normal phenomenon. Similarly, these signs of distress may not be seen in infants even during chronic severe airway obstruction.

The respiratory obstruction which produces stridor & stertor can lead to hypoxia, hypercapnia, pulmonary oedema, cor pulmonale, vomiting, aspiration & pneumonia.

2. Cough: Is usually harsh and barking in nature. This symptom is commonly associated with subglottic inflammation or tracheal compression.

3. Hoarseness: Suggests laryngeal inflammation, trauma, tumors or vocal cord mobility problems. In a child with stertor there will not be any hoarseness of voice but the cry is usually muffled.

4. Deglutition: The process of deglutition & respiration share the common pathway i.e. the oropharynx. Hence disorders of swallowing may affect breathing and the vice versa is also possible.

Causes of stridor are anatomically classified:

1. Supralaryngeal causes:

a. Nose - choanal atresia

Obstruction due to infection / trauma / tubes

b. Cranio facial anomalies:

These patients have narrowing of oropharynx, nasopharynx and nasal cavities. The may also additionally manifest with macroglossia. The various anomalies associated with respiratory difficulties are:

Pierre Robin syndrome

Treacher collin syndrome

Apert's syndrome

Cruzon's syndrome

Mobieus syndrome

c. Macroglossia:

Beckwith Wiedemann syndrome

Down's syndrome

d. Tumors:

Hemangioma

Neuroblastoma

e. Laryngomalacia: Is caused by an excessively elastic cartiagenous support to the airway seen in infants. This commonly affects the glottic and supra glottic airway of infants. This excessively soft and elastic cartilage causes inspiratory collapse of the arytenoid, aryepiglottic folds and epiglottis during inspiration. The omega shaped epiglottis seen often in the infants adds to the problem. This causes occlusion of the laryngeal inlet. These patients have inspiratory stridor which becomes better on prone position or when the child is calm. Stridor is worsened if the child is restless or excited.

The cry of the child is usually normal. The child may also have aspiration and feeding difficulties. It is commonly seen during the first few months of life.

Direct laryngoscopy shows indrawing and falling forwards of the arytenoid and the aryepiglottic folds. The epiglottis may be infolded.

This condition may be managed conservatively, as the cartilage in infants tend to become stiffer as the child grows. In difficult cases the patient may be subjected to tracheostomy to secure the airway and to prevent aspiration, and feeding gastrostomy to maintain the nutritional status of the child. Epiglottoplasty may be considered in resistant cases.

2. Glottic causes:

a. Vocal cord palsy: Is one of the commonest causes of airway obstruction. In 80% of patients it is unilateral.

Etiology: Could be caused due to injury to vagus nerve at the level of Nucleus ambiguus - it is often bilateral.

Injury to the left recurrent laryngeal nerve due to cardio vascular causes and thoracic causes.

It could be caused due to increased intracranial pressure - i.e. Meningomyelocele with Arnold Chiari malformation.

Clinical features: Inspiratory stridor at birth Weak, hoarse cry or aphonia.

If unilateral the patient feels better when placed on the side of the lesion.

Symptoms of FB is divided into three stages:

Phase I of choking, gagging and wheezing. This phase lasts for a short time. FB may be coughed out or may lodge in the larynx or further down in the tracheobronchial tree.

Phase II of symptomless interval. In this phase the respiratory mucosa adapts to the presence of FB and the initial symptoms disappear. This interval could vary with the size and nature of the FB. This phase could last a few hours or a few weeks.

Phase III or later symptoms. This phase is caused by airway obstruction, inflammation or trauma induced by FB and would always depend on the site of lodgment of the FB.

Laryngeal FB:

A large FB may totally obstruct the airway causing sudden death unless airway is secured on an urgent basis. A partially obstructing FB can cause discomfort / pain in the throat, hoarseness of voice, croupy cough, aphonia, dyspnea, wheezing and hemoptysis.

Tracheal FB:

A sharp FB will produce cough and hemoptysis. Loose FB could move up and down the trachea between the carina and under surface of vocal cords. This could cause auditory slap or a palpable thud. Wheeze could also be present. The wheeze could be best heard when the patient has the mouth open.

Bronchial FB:

Majority of FB enter the right main bronchus as it is more wider and is a direct continuation of trachea. The FB could cause total obstruction of a lobar/ segmental bronchus causing atelectasis. Sometimes it may cause ball valve like obstruction allowing only air entry while air exit is obstructed. This leads to a condition known as obstructive emphysema.

The emphysematous bulla may rupture causing spontaneous pneumothorax. The FB may also shift from one side to the other causing changes in the physical signs. A retained FB in the lung can cause pneumonitis, bronchiectasis or lung abscess.

Diagnosis:

Is usually made from history. History of sudden onset of coughing, wheezing is sufficient. Examination will reveal diminished air entry. A high index of suspicion is necessary to arrive at the diagnosis.

Radiology:

X-ray soft tissue neck lateral view with neck in extension will reveal radio-opaque FB. A coin in trachea will be seen as a face on view in the lateral films and edge on view in PA view. This is because laryngeal inlet opens in an anteroposterior direction.

X-ray chest:

May reveal radio-opaque FB. It size, shape and location can also be clearly seen.

It will also reveal lobar / segmental atelectasis (due to obstruction by FB).

Unilateral hyperinflation of a lobe / segment or entire lung can be clearly seen. Mediastinal shift to the opposite side is seen in hyperinflation.

Pneumomediastinum / pneumothorax is also clearly visible.

Prolonged sojourn of FB would cause pneumonitis / bronchiectasis which could be clearly seen in chest radiographs.

CT chest is diagnostic and can also be said to be the gold standard.

Management:

First aid measures include pounding on the back, turning the child upside down and using Heimlich maneuver.

Heimlich Maneuver:

This is performed by standing behind the patient with arms placed around the lower chest. Four to five abdominal thrusts are given pushing up the residual air in the lungs upwards. This could dislodge the FB providing some airway.

Airway can be secured by performing tracheostomy.

Tracheal / bronchial FB can be removed by using bronchoscopy under GA. Surgeon and anesthetist should share the airway.

8. What is Stridor? Enumerate the causes of Stridor. Describe briefly the management modalities of Stridor

   Repetition of previous questions

9. Write an essay on the operative procedure of Tracheostomy, its complications and types of Tracheostomy tubes. Briefly discuss the functions and indications of Tracheostomy.

Repetition of previous questions

10. Enumerate the causes of dysphagia. Describe about etiology, clinical features, investigations and treatment of Plummer Vinson syndrome.

Dysphagia: is a Greek word for disordered swallowing.

Common causes of dysphagia in an adult:

Dysphagia due to conditions affecting the oral phase of swallow:

Cannot hold food in the mouth anteriorly due to reduced lip closure
Cannot form a bolus or residue on the floor of the mouth due to reduced range of tongue motion or coordination
Cannot hold a bolus due to reduced tongue shaping and coordination
Unable to align teeth due to reduced mandibular movement
Food material falls into anterior sulcus or residue in the anterior sulcus due to reduced labial tension or tone.
Food material falls into lateral sulcus or residue in the lateral sulcus due to reduced buccal tension or tone.
Abnormal hold position or material falls to the floor of the mouth due to tongue thrust or reduced tongue control
Delayed oral onset of swallow due to apraxia of swallow or reduced oral sensation
Searching motion or inability to organize tongue movements due to apraxia of swallow
Tongue moves forward to start the swallow due to tongue thrust.
Residue of food on the tongue due to reduced tongue range of movement or strength
Disturbed lingual contraction (peristalsis) due to lingual dyscoordination
Incomplete tongue-to-palate contact due to reduced tongue elevation
Unable to mash material due to reduced tongue elevation

Adherence of food to hard palate due to reduced tongue elevation or reduced lingual strength
Reduced anterior-posterior lingual action due to reduced lingual coordination
Repetitive lingual rolling in Parkinson disease
Uncontrolled bolus or premature loss of liquid or pudding consistency in to the pharynx due to reduced tongue control or linguoaveolar seal
Piecemeal deglutition
Delayed oral transit time
Dysphagia caused by disorders affecting Pharyngeal phase of swallow:

Delayed pharyngeal swallow
Nasal penetration during swallow due to reduced velopharyngeal closure
Pseudo epiglottis (after total laryngectomy) - Fold of mucosa at the base of the tongue
Cervical osteophytes
Coating of pharyngeal walls after the swallow due to reduced pharyngeal contraction bilaterally
Vallecular residue due to reduced posterior movement of the tongue base
Coating in a depression on the pharyngeal wall due to scar tissue or pharyngeal pouch
Residue at top of airway due to reduced laryngeal elevation
Laryngeal penetration and aspiration due to reduced closure of the airway entrance (arytenoid to base of epiglottis)
Aspiration during swallow due to reduced laryngeal closure
Stasis of residue in pyriform sinuses due to reduced anterior laryngeal pressure
Delayed pharyngeal transit time
Dysphagia caused by oesophageal disorders:

Esophageal-to-pharyngeal backflow due to esophageal abnormality
Tracheoesophageal fistula
Zenker diverticulum
Reflux

Investigations:

1. Chest radiograph

2. Ultrasound abdomen

3. Barium swallow

4. CT / MRI scan neck

5. Videofluroscopy study of swallowing

6. Fibreoptic endoscopy

7. Scintigraphy for oesphageal disorders

8. Oesophageal pH monitoring - reflux oesophagitis

Management:

Dietary modification: This plays an important role in oropharyngeal dysphagia. Diet can be mashed and made into a puree to enable easy swallowing. If the patient's swallowing improves the consistency of the food can be improved.

Increased viscosity of liquid: If liquid diet gets aspirated while attempting to swallow then viscosity can be added to the liquid by addition of starch. This increases the consistency of the liquid preventing aspiration.

Ryles tube feeding: If the patient is suffering from dysphagia due to obstructing lesion in the pharynx, laryngopharynx, oesophagus due to tumors then Ryles tube can be inserted and feeding initiated.

Feeding gastrostomy / jejunostomy: Are advised in patients with absolute dysphagia due to tumors involving postcricoid area and oesophagus.

Exercises and facilitation techniques:

Methods belonging to this group are useful in patients with dysphagia due to paralysis of oropharyngeal muscles / lip muscles. Lip, tongue and jaw exercises play a vital role.

Facilitating maneuvers: These are helpful in preventing aspiration during swallow. They also help in swallowing process in a patient who has undergone total laryngectomy. These include:

Supraglottic swallow

Extended supraglottic swallow

Super supraglottic swallow

Mendelson maneuver - helps in cricopharyngeal opening and laryngeal elevation during swallow. This procedure depends on manually prolonging the duration of elevation of larynx there by keeping the cricopharyngeal sphincter open. This can be performed by placing the index finger over the midportion of laryngeal cartilage and pushing it upwards during the swallow.

Plummer Vinson syndrome is defined by the classic triad of dysphagia, iron deficiency anemia and esophageal webs. This syndrome has an increased risk of squamous cell carcinoma of cricopharynx and upper esophagus.

It is a disease of women (middle aged).

Etiology:

Iron deficiency anemia is the most important causative factor of this syndrome

Diagnosis:

This is based on the evidence of iron deficiency anemia and presence of esophageal webs. Webs can be detected by upper GI endoscopy and the web appears as smooth thin and gray thickening with eccentric or central lumen. Webs invariably occur in the proximal part of the esophagus.

Management:

1. Treatment of iron deficiency anemia

2. If dysphagia is severe then mechanical dilatation can be resorted to using bougies

3. Since these patients are having high risk of malignancy biopsy needs to be taken from this area

11. 65-year-old male presented with dysphagia. Write differential diagnosis and investigations required to diagnose. Discuss treatment options for Carcinoma lower third of esophagus.

The elderly is at an increased risk for development of dysphagia due to illness that affect the swallowing mechanism.

Swallowing represents a function that involves more than 30 nerves and muscles. Bolus preparation and passage of food from oral cavity into the esophagus are voluntary, while further

passage across the aerodigestive tract to the stomach is reflex in nature. After initiation of swallow, it takes less than a second for a bolus to reach the esophagus and an additional 10-15 seconds to complete the swallow. Swallowing centers are bilaterally represented within the central nervous system, and the degree of each hemispheric representation seems to be critical in determining recovery of swallowing function after dysphagic stroke.

Dysphagia due to aging:

Normal aging is associated with cerebral atrophy, deterioration of nerve function and a region dependent decline in muscle mass that could adversely affect swallowing mechanism. The effects of age on the temporal evolution of isometric and swallowing pressure seems to progress with time. Even in many asymptomatic elderly persons nearly 80% demonstrate videofluoroscopic changes in pharyngeal swallow compared with what is considered normal in healthy young adults. Despite physiologic changes in the swallowing mechanism due ot aging, dysphagia cannot be attributed to normal aging alone, and its presence suggests the need for investigation to identify potentially treatable causes.

Causes of dysphagia:

Structural (Mechanical) Disorders
- — Zenker diverticulum (Killian dehiscence)
- — Lateral pharyngeal pouch or diverticula
- — Cricopharyngeal achalasia, bar, rings, and stenosis
- — Proximal esophageal web (Plummer-Vinson)
- — Oropharyngeal and laryngeal tumors
- Oesphageal growth benign / Malignant
- — Head and neck surgery
- — Radiotherapy
- — Extrinsic compression (cervical osteophytes, skeletal abnormalities, and thyromegaly)

Motor Disorders

Central Nervous System
- — Stroke
- — Head trauma
- — Brain tumors
- — Extrapyramidal syndromes (Parkinson disease, Huntington disease, and Wilson disease)
- — Multiple sclerosis
- — Cerebral palsy
- — Dementia
- — Metabolic encephalopathies
- — Tardive dyskinesia (phenothiazine use)

Peripheral Nervous System
- — Amyotrophic lateral sclerosis
- — Bulbar poliomyelitis

— Tabes dorsalis
— Guillain-Barré syndrome
— Drugs (botulinum toxin, procainamide, and cytotoxins)

Myogenic

— Myasthenia gravis
— Dermatomyositis, polymyositis
— Mixed connective tissue disease
— Hyperthyroidism, hypothyroidism
— Cushing syndrome
— Amyloidosis
— Paraneoplastic syndromes
— Drugs (amiodarone, alcohol, and statins)

Oesphageal causes of dysphagia:

Structural (Mechanical) Disorders

Intrinsic Encroachment

— Mucosal rings and webs: Schatzki, Plummer-Vinson, or multiringed esophagus (eosinophilic esophagitis)
— Strictures (inflammatory or fibrotic): peptic, caustic, pill, or radiation-induced
— Esophageal tumors: adenocarcinoma, squamous cell carcinoma, metastatic (breast or melanoma), leiomyoma, lymphoma, or granular cell tumor
— Systemic diseases: scleroderma (multifactorial), pemphigus/pemphigoid, lichen planus, or Crohn's disease
— Miscellaneous: post surgery (laryngeal, esophageal, or gastric cancers), acute esophageal infections, esophageal diverticula, or foreign bodies

Extrinsic Compression

— Mediastinal masses: lung cancer, lymphoma, lymph node, or thyromegaly
— Vascular compression: dysphagia lusoria (aberrant right subclavian artery), dysphagia aortica (right-sided aorta), or cardio-megaly (enlarged left atrium)
— Miscellaneous: cervical spine osteophytes/spondylosis or fundoplication

Motor Disorders

— Primary: achalasia, diffuse esophageal spasm, hypertensive lower esophageal sphincter, ineffective esophageal motility disorder, or nutcracker esophagus
— Secondary: connective tissue diseases, scleroderma, CREST syndrome, diabetes, Chagas disease, or paraneoplastic syndrome.

Examination of oral cavity, head and neck may reveal poor dentition. Lymphadenopathy, goiter, prior surgery. Laryngoscopic examination would reveal the presence of growth in the larynx, pyriform fossa and post cricoid regions.

Neurologic examination is mandatory for evaluation of every dysphagia. All cranial nerves should be tested particularly the nerves involved in swallowing.

Presence of tremors, cogwheeling rigidity and gait disturbance may indicate Parkinson's disease, which can cause dysphagia. A combination of motor and sensory abnormalities, particularly in the setting of longer disease duration and significant motor abnormalities may suggest multiple sclerosis. Myasthenia gravis is frequently associated with facial and pharyngeal weakness, and dysphagia is seen in nearly quarter of these patients.

Five major tests are currently used for estimating and quantifying swallowing dysfunction.

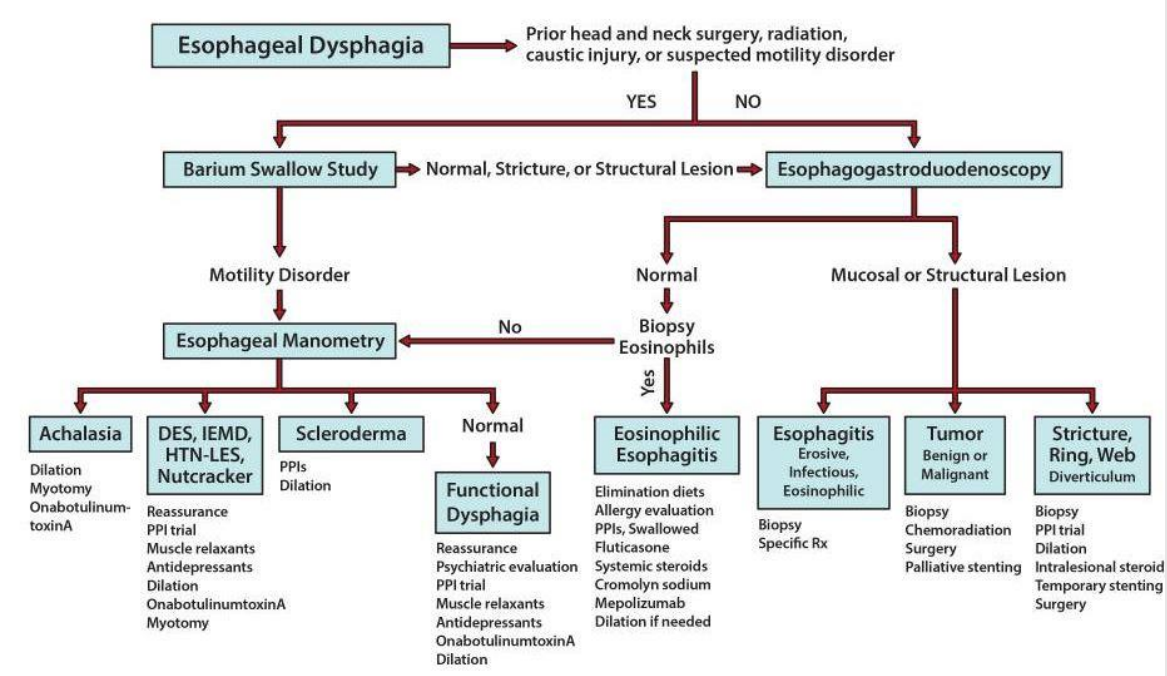
Barium radiography

Video fluoroscopic swallowing study

Fibreoptic endoscopic evaluation of swallowing

Upper GI endoscopy

Esophageal manometry



Management of carcinoma lower third of esophagus:

Esophagectomy remains the primary treatment modality for early stage esophageal cancers. Surgical resection must consist of a radical complete enbloc esophagectomy associated with

extended two field lymphadenectomy. Stomach can pulled up and anastomosed with the pharynx to compete the swallowing pathway anatomically.

Advanced malignant lesions of lower third esophagus can be managed by chemoradiation. Patient can be nourished by performing a jejunostomy for feeding purposes.

12. Discuss in etiology, clinical features, and management of Stricture Oesophagus

Disease processes that produces strictures of oesophagus can be grouped under three categories:

1. Intrinsic diseases that narrow the esophageal lumen through inflammation, fibrosis or neoplasia

2. Extrinsic diseases that compromise the lumen by direct invasion or lymph node involvement

3. Diseases that disrupt esophageal peristalsis and lower esophageal sphincter function by their effects on esophageal smooth muscle innervation

Diseases that cause esophageal stricture include:

Acid peptic disease

Autoimmune disease

Infections

Caustic

Congenital

Iatrogenic

Medication induced

Radiation induced

Malignant / idiopathic disease process

Etiology can be identified using radiologic and endoscopic modalities and can be confirmed by endoscopic visualization and tissue biopsy.

Use of manometry can be diagnostic when dysmotility is suspected as the primary process. CT scanning and endoscopic ultrasonography are valuable aids in staging malignant stricture.

Proximal / or mid esophageal stricture may be caused by:

Caustic ingestion (acid / alkali)

Malignancy

Radiation therapy

Infectious esophagitis - candida, herpes simplex virus, CMV, HIV

Medication induced stricture (pill esophagitis) - Ferrous sulphate, NSAID's potassium chloride, Quinidine, Tetracycline, ascorbic acid. Drug induced esophagitis occurs at the anatomic narrowing with the middle third of oesophagus behind the left atrium predominating.

Graft versus host disease

Idiopathic eosinophilic esophagitis

Extrinsic compression

Squamous cell carcinoma

Miscellaneous - Trauma to oesophagus from external forces, foreign body, congenital esophageal stenosis.

Distal esophageal strictures may be caused by the following:

Peptic stricture - Gastroesophageal reflux disease, Zollinger-Ellison syndrome

Adenocarcinoma

Collagen vascular disease - Scleroderma, systemic lupus erythematosus (SLE), rheumatoid arthritis

Extrinsic compression

Alkaline reflux following gastric resection

Sclerotherapy and prolonged nasogastric intubation

Crohn disease

Clinical features:

Difficulty in swallowing
Malnutrition
Severe cachexia in patients with carcinoma oesophagus

Management:

Benign strictures of esophagus:

Consideration should be paid to improve the nutritional status of the patient. Ryles tube may be inserted to facilitate regular feeding. If the patient is anemic it should be treated by regular administration of hematinic.

Benign strictures can be dilated using bougies.

Malignant strictures of oesophagus can be managed by chemoradiation if it is in an advanced stage. If it is in the early stage then complete resection of esophagus along with gastric pull up and anastomosis with the pharyngeal mucosa.

13. Discuss the aetiology, pathogenesis, Clinical features and Management of Retropharyngeal abscess.

Retropharyngeal abscess is a collection of pus between the posterior pharyngeal wall and the fascia and muscles covering the cervical vertebrae. It occurs in two forms –

1. The acute primary retropharyngeal abscess which is common in infants, and 2. Chronic retropharyngeal abscess which is common in adults. These two types of abscesses differ in their etiology and management.

Acute primary retropharyngeal abscess: Is the more dangerous type occurring in infants. It is common between the age group of 3 months to 3 years. The predisposing factors are malnutrition, gastroenteritis, poor hygiene etc.

Etiology: Abscesses may follow general debilitating illnesses like scarlet fever, measles etc. Infections from tonsils, adenoid and naso pharynx may even lead to the formation of retropharyngeal abscess. Rarely foreign bodies like bone pieces and pins may also cause retropharyngeal abscess.

Pathology: The disease consists of suppurative lymphadenitis of the retropharyngeal nodes of Henle, situated on either side of midline between the posterior pharyngeal wall and the aponeurosis over the bodies of the second and third cervical vertebrae. These glands receive the lymphatics of the post nasal space, pharynx, nose, eustachean tube and middle ear. These nodes

atrophy between the 3rd and 5th year of life hence acute retropharyngeal abscess is uncommon in children above the age of 4.

The Henle's node when infected from the lymphatics, there is first adenitis, then peri adenitis and abscess formation occur. The suppuration is usually one sided, and most prominent in the oro pharynx. If not evacuated in time or when it does not rupture, pus may spread along the esophagus or burst in different directions - towards the larynx, the angle to the jaw or even in to the external auditory canal. The pus is generally foul smelling yellow or whitish in color. It usually contains streptococci, and more rarely staphylococci and pneumococci.

Chronic retropharyngeal abscess: Is commonly known to occur in adults. This is usually caused by tuberculosis. The tuberculous foci occur in the bodies of the cervical vertebrae (Pott's disease) which later spread into the retropharyngeal space. Primary syphilis of the mouth and pharynx may also cause retropharyngeal abscess. This abscess usually is present in midline and is free to spread to either side also.

Symptoms: These patients have excruciating pain while swallowing (odynophagia). Young infants with retropharyngeal abscess will refuse feed, may have extensive drooling. In adults the head may be held straight. Torticollis is also common in these patients. These patients may have difficulty in breathing (stridor), in which case tracheostomy must be considered to secure the airway in the first place. Constitutional symptoms like fever / toxicity is very common in acute retropharyngeal abscess.

Investigations:

Complete blood count show leucocytosis. Blood cultures can also be performed to ascertain the appropriate antibiotics to be used.

C reactive proteins are also found to increased in these patients

Xray soft tissue neck - A.P. and lateral views.

These pictures show prevertebral soft tissue widening. This can be ascertained by estimating the size of the prevertebral soft tissue which is normally half the size of the body of the corresponding vertebra. If the widening is more than half the body size of the corresponding vertebra then retropharyngeal abscess must be considered. The cervical spine are straightened with loss of the normal lordosis (Ram Rod spine). Above the prevertebral shadow air shadow is seen in almost all cases of retropharyngeal abscesses. This gas shadow is caused by entrapped air which occur during breathing. Some bacteria esp. Clostridium are known to form gases which may be entrapped in the prevertebral space.

C.T. scan neck or MRI study of neck will also help in clinching the diagnosis. This must ideally be performed using intravenous contrast agents. It appears as a hypodense lesion in the

retropharyngeal space with ring enhancement. Other effects that could be seen are soft tissue swelling, and obliteration of normal fat planes.

C.T. scan is really helpful in differentiating cellulitis from abscess.

Management:

In majority of cases incision and drainage is done and the pus is immediately aspirated out using suction. The incision is made with 11 blade knife over the most prominent portion of the swelling. The I&D is done under local anesthesia. In the case of infants, it is preferable that the patient is held upside down while the surgery is being performed to prevent aspiration of pus into the lungs. When general anesthesia is preferred a cuffed endotracheal tube must be used to minimize the hazard of aspiration of pus into the lungs. The patient must be put in Rose position (tonsillectomy position) while the I&D is being done to reduce the threat of aspiration.

When the abscess points towards the neck then it should be opened through an incision over the neck, preferably along the posterior border of sternomastoid muscle. The dissection is carried out behind the great vessels of the neck and in front of the prevertebral muscles. The surgery is followed by a course of antibiotics mostly cephalosporin group. Clindamycin in dose of 600-900mg intravenously 8th hourly can be administered in adults. Injection penicillin G in doses of 24 million units per day as continuous infusion along with metronidazole injection in doses of 500mg three times a day can also be considered. Metronidazole is highly effective against anaerobes.

If tuberculosis is suspected to be the cause then surgery is deferred. Anti-tuberculous treatment is initiated.

Complications:

1. Mediastinitis

2. Airway obstruction

3. Atlanto occipital dislocation

4. Jugular vein thrombosis

5. Cranial nerve deficits especially the lower three ones

6. Hemorrhage secondary to involvement of the carotid artery

14. A male patient aged 60 yrs chronic smoker, comes with hoarseness of voice of 1-year duration. How do you investigate and treat him?

Commonest cause of hoarseness in an elderly smoker include:

Chronic laryngitis

Leukoplakia of vocal folds

Erythroplakia of vocal folds

Malignant lesions involving vocal folds

Paralysis of vocal folds

Malignant lesions of vocal folds:

Etiology:

It has been established that smoking and consumption of alcohol are potent risk factors responsible for laryngeal malignancy. Tobacco itself is a more potent risk factor than alcohol. Both these insults when combined increase the risk of malignancy by nearly 4 times.
Tobacco and Alcohol: These two substances have been proved to play a role in the etiology of Head and Neck malignancies. Of these two substances tobacco undoubtedly plays a vital role in the etiology of squamous cell cancers of head and neck region. Positive association also exists between tobacco usage and malignancy of upper aerodigestive tracts like oesophagus and lungs.

Studies have shown that concomitant exposure to tobacco and alcohol increased the risk of Head and neck malignancy by 4 times. The risk also increases proportionally as the number of cigarettes smoked per day increases. Cessation of smoking led to a reduction in the incidence of malignancy in the head and neck region. The type of
tobacco used also plays a vital role in the pathogenesis of malignancy. Air processed dark tobacco increases the risk of malignancy by 2 - 5 times when compared to light colored tobacco.

Management:

Laryngoscopic examination would reveal the lesion affecting the free margin of the vocal folds. Mobility of vocal folds also can be visualized. Common malignancy that causes vocal fold paralysis is malignant growth involving the post cricoid region.

Imaging:

CT imaging will reveal the exact extent of the lesion. It would also reveal the presence of secondary deposits in the neck region.

Growth vocal folds carries the best prognosis as the patients present to the hospital very early in the disease. Biopsy from the suspected lesion should be taken to confirm the diagnosis. In patients with carcinoma in situ, vocal fold stripping under general anesthesia would be helpful.

Early malignant lesions of the vocal fold which has not caused fixity of the cord can be managed by irradiation.

In patients with fixed vocal fold then partial / total laryngectomy is preferred. Some centres practice a combination of surgery with chemoradiation to manage these patients.

15. What are the causes of hoarseness of voice in male aged 55 years? How do you proceed with investigations? Write role of microlaryngeal surgery in papilloma larynx.

Introduction:
Human voice is so complex that it not only conveys meaning, it also is capable of conveying subtle emotions. It is the most important physiological aspect that effectively starts to function immediately after birth.

Definition:
Hoarseness of voice is defined as any change in the quality of human voice (lay terms). This term utmost be considered to be a nonspecific one. This term could imply breathiness, roughness, voice breaks or unnatural pitch changes. Dysphonia is the corresponding term used by otolaryngologists to describe this condition.

"Hoarseness is a symptom of utmost significance and calls for a separate consideration as a subject because of the frequency of its occurrence as a distant signal of malignancy and other conditions"

Chevalier Jackson

Causes of hoarseness of voice:
Inflammatory causes including acute injuries
Mucosal disorders of vocal folds
Benign tumors of vocal folds

Malignant tumors of vocal folds
Laryngeal foreign bodies
Neurogenic causes affecting larynx
A useful Mnemonic to remember the causes of hoarseness of voice:

VINDICATE

V - Vascular (thoracic aneurysm)

I - Inflammation

N - Neoplasm

D - Degenerative (Amyotrophic lateral sclerosis)

I - Intoxication (smoking / alcohol)

C - Congenital

A - Allergies (angioneurotic oedema)

T - Trauma / Thyroid surgery

E - Endocrinology (Reidel's struma)

Inflammatory causes:

This is one of the commonest causes of hoarseness of voice. Inflammatory causes could either be acute / chronic.

Acute inflammatory causes: include acute laryngitis, acute epiglottis, acute laryngotracheal bronchitis, diphtheria etc.

Chronic inflammatory causes include: chronic nonspecific laryngitis (due to gastro oesophageal reflux disorders, vocal abuse etc).

Chronic specific laryngitis (tuberculous / syphilitic laryngitis).

Acute vocal cord injuries can lead to vocal fold tears / hematoma causing hoarseness of voice. This condition resolves with complete voice rest.

Mucosal fold disorders:

Laryngeal oedema, Reinke's oedema.

Vocal nodule

Vocal cord polyp

Vocal cord cysts

Benign tumors:

Papilloma

Fibroma

Adenoma

Chondroma

Precancerous lesions:

Hyperkeratosis

Pachydermia

Leukoplakia

Malignant tumors of larynx:

Carcinoma vocal cords

Congenital conditions:

Congenital vocal cord webs

Foreign bodies

Vocal cord paralysis

Possible clinical presentations in patients with hoarseness of voice:

Change in voice
Cough
Fever

Vocal fatigue
Irritation / soreness of throat
Weight loss
Painful vocalization
Difficulty in swallowing
Breathy voice
Neck swelling
Painful swallowing
Heart burn / vomiting
Haemoptysis
Noisy respiration

Diagnosis:

Evaluation of hoarseness of voice should include:

Assessment of

Anatomy

Physiology

Behavioral factors

A complete history should be elicited.

Laryngeal visualization: Indirect laryngoscopy / Direct laryngoscopy

Objective voice assessment: simplest method would be a tape recording of the voice in question. This method should be considered to be subjective.

Acoustic analysis: In this test voice is examined as electrical signals. The term fundamental frequency is used to measure the number of vocal fold vibrations per second. A normal adult male vocal cord vibrates between 100 - 130 Hz, where as in females it vibrates at the rate of 200 - 230 Hz. Abnormally high fundamental frequencies corrected for age and sex changes would indicate hyper contraction of cricothyroid muscle representing a functional pathology or compensatory dysphonia.

Aerodynamic analysis:

The quality of voice is dependent on breath support. Even subtle respiratory problems can lead to changes in voice. Aerodynamic measurements play a role in quantifying airflow during respiration and phonation. Pulmonary function tests may play a role in identifying subtle respiratory problems.

Maximum phonation time: This is a measurement of the amount of time a patient can sustain a vowel sound in one breath.

Normal values range between 15 - 25 seconds. Decreased values indicate incomplete glottic closure or insufficient lung support.

Glottal air flow: This sensitive test captures the amount of air flowing through the glottis during phonation. This is measured in cc/second by dividing the total volume of air flowing through the glottis by the amount of time in seconds. This gives information about the lung capacity and the efficiency of vocal folds. Increased glottal flow is associated with incomplete closure of glottis.

The voice in patients with increased glottal flow is usually breathy / whispering in nature.

Decreased glottal air flow is seen in patients with spasmodic dysphonia due to hyperadduction of vocal folds.

Management:

Absolute voice rest - very useful in acute conditions
Speech therapy - useful in chronic disorders
Antibiotics
Anti inflammatory drugs
Treatment of GERDS
Surgical intervention

Microlaryngeal surgery in the management of papilloma larynx:

Adult Papilloma larynx is a solitary benign tumor (it is multiple when present in pediatric age group). This tumor is caused by HPV infection. Since it is a benign lesion it can be removed under direct and magnified vision by using microlaryngeal surgical procedure.
This surgery is performed under general anesthesia. Patient is placed on the OT table in a recumbent position. Klein sausser self-retaining laryngoscope is used to expose the lesion. The mass can be removed under direct vision using 300 mm objective lens of the microscope to visualize the larynx.

Lasers are being commonly used in this surgery these days. Main advantage of laser being that the tumor can be totally resected with minimal bleeding.

16. Discuss etiology, pathology and management of Carcinoma Larynx.

Etiology:

It has been established that smoking and consumption of alcohol are potent risk factors responsible for laryngeal malignancy. Tobacco itself is a more potent risk factor than alcohol. Both these insults when combined increase the risk of malignancy by nearly 4 times.
Tobacco and Alcohol: These two substances have been proved to play a role in the etiology of Head and Neck malignancies. Of these two substances tobacco undoubtedly plays a vital role in the etiology of squamous cell cancers of head and neck region. Positive association also exists between tobacco usage and malignancy of upper aerodigestive tracts like oesophagus and lungs.

Studies have shown that concomitant exposure to tobacco and alcohol increased the risk of Head and neck malignancy by 4 times. The risk also increases proportionally as the number of cigarettes smoked per day increases. Cessation of smoking led to a reduction in the incidence of malignancy in the head and neck region. The type of tobacco used also plays a vital role in the pathogenesis of malignancy. Air processed dark tobacco increases the risk of malignancy by 2 - 5 times when compared to light colored tobacco.
Role played by tobacco as a carcinogen: Tobacco smoke contains about 4000 different chemicals in different proportions. Out of these about 43 have been positively identified as carcinogens.

These include:
1. Polycyclic hydrocarbons
2. Nitrosamines
3. Radio active polonium - 210

Viruses: Human papilloma virus has been believed to play a role in the aetiology of head and neck cancers. The genotypes of HPV (Human papilloma virus) have been classified in to three categories (risk wise).
1. High risk: (Types 16, 18)
2. Medium: (Types 31, 33)
3. Low: (Types 6, 11)

Occupational factors:
Nickel and chromium refining workers who are constantly exposed to these materials have an increased incidence of laryngeal cancers. Similarly, exposure to asbestos fibers has also shown to play a role in aetiopathogenesis of head and neck malignancies.

Dietary factors: are also known to play a role in the etiopathogenesis of head and neck malignancies. The classic disorder being the Patterson Brown Kelly syndrome which is characterized by iron deficiency anemia, glossitis, koilonychia, and upper oesophageal webs carrying with it a high risk of post cricoid malignancy. The risk diminishes when the patient is nutritionally supported with iron and B complex supplements.
The association between nasopharyngeal carcinoma and salted fish diet has been documented. The traditional salted fish prepared in southern china contain volatile carcinogens like N-nitrosodimethylamine and N-nitrosodiehtylamine. Both these chemicals have been shown to be carcinogenic in nature.
The association of laryngopharyngeal reflux and laryngeal cancers is still being studied. The final word is not yet out at the time of writing this article.
Genetic causes: The development of head and neck cancers involves loss of control over cell proliferation and cell death. In fact there is no control at all. This loss of control at least partly has been attributed to be faulty genes. The regulation of growth and proliferation of all the cell types in the body is known to be controlled by 40 known proteins that regulate the cell cycle. These regulator proteins are known to be products of proto oncogenes and tumor suppressor genes. Under normal conditions these genes work in a coordinated manner creating a balance between cell proliferation and cell death.

Pathology:

WHO classification of Premalignant / Early malignant lesions:
- Hyperplasia
- Keratosis
- Mild dysplasia
- Moderate dysplasia
- Severe dysplasia
- Carcinoma in situ

Malignant lesion of larynx:

Squamous cell carcinoma

Glottic cancer:

Glottic cancer is defined as malignancy involving the true vocal cords / anterior commissure / posterior commissure. Squamous cell carcinoma is the commonest malignant lesion affecting the vocal cord.
Glottic cancers are the most common laryngeal malignancy constituting 56%. The glottic region has very little lymphatic supply. Therefore, there is less risk of early lymphatic spread of malignancy from this area. Early glottic carcinoma is usually confined to the vocal

cord. Breach into the Reinke's space will allow the tumor to involve the entire extent of the vocal cord.

Involvement of vocalis muscle / cricoarytenoid joint by the tumor mass may cause fixation of the cord. Anterior progression of glottic tumor along the Broyle's ligament (anterior commissure tendon), vocalis tendon allows for early invasion of thyroid cartilage at the site of insertion of the ligament. This fact could account for a small glottic cancer to be classified as stage IV tumor because of involvement of thyroid cartilage.

Clinical features of glottic growth:

• Persistent hoarseness of voice. Even small lesions of the cord may cause significant hoarseness of voice, and these patients usually seek medical attention at a very early stage itself.
• Larger lesions of vocal cords may cause stridor.
• Rarely these lesions may cause haemoptysis
• In advanced stages cervical lymph node metastasis could also be seen.
• Indirect laryngoscopic examination will not only help in visualizing the lesion but would also help in assessing the mobility of the vocal cords. A fixed cord always indicates advanced lesion.

Management of glottic malignancy:

1. Securing the airway if the patient is in stridor. This can be achieved by performing a tracheostomy.
2. Confirmation of the diagnosis by performing direct laryngoscopy biopsy.
3. CT scan neck to look for cervical metastasis.
4. X-ray chest

Irradiation:

Irradiation is the primary non surgical treatment modality available for early glottic lesions (T1 and T2 lesions). Standard dose of radiation used in glottic tumors is a total of 60-70 Gy administered in single daily 2 Gy fractions over 6 weeks.
Major advantage of irradiation is that cure rates are excellent with the preservation of voice in early lesions.
Radiation therapy with cetuximab, a monoclonal antibody against the epidermal growth factor receptor, has been shown to be more effective than radiation alone.

Disadvantages of radiotherapy:

1. Long treatment course
2. Potential complications of irradiation

3. Difficulty in diagnosing recurrent lesions after irradiation
Irradiation will really do wonders under the following conditions:
1. Low volume tumors (Tumors less than 3cm3) do the best.
2. Mobile cord.
3. No involvement of ventricle
4. No deep ulceration
5. Lack of supraglottic / Subglottic spread
6. Involvement of only one site
7. Patient should stop smoking

Chemotherapy:

A number of useful drugs are available with excellent activity against squamous cell carcinoma. Commonly used chemotherapeutic agents include: cisplatin, carboplatin, 5-fluorouracil, methotrexate, paclitaxel, docetaxel, and ifosfamide.

Toxicity is greatly reduced when a single agent is used. Major response rates have been achieved in patients treated with cisplatin based combination therapy.

Response rates improve in patients with previously untreated loco regionally advanced disease. Chemotherapy has been largely used as a palliative therapy in patients with advanced laryngeal malignancy.
Induction chemotherapy can be used to identify those patients who would benefit from irradiation.

Surgery is indicated in:

1. Patients suffering from premalignant / carcinoma in situ lesions of vocal cords. Irradiation is contraindicated in these patients because of the risk of malignant transformation. Surgical procedure used in these patients is stripping of vocal cords, micro laryngeal excision of tumor mass etc.
Carbon dioxide lasers can be used to remove these masses. Major advantage of laser therapy is that it causes minimal scarring of vocal folds, hence the voice becomes normal / near normal.
2. Stage III and IV lesions which need to be salvaged by surgery.

17. Describe in detail etiopathognesis, clinical features and management of a patient with nasopharyngeal carcinoma.

Synonyms: NPC, Guangdong tumor

Introduction: This is the most common malignancy involving the nasopharynx. It is common among chinese population. In fact it is so common that it is considered to be endemic disorder in southern china. It is closely associated with Epstein Barr virus infections. Histologically undifferentiated / non keratinizing carcinoma types are common.

Anatomy of nasopharynx: Nasopharynx is at the junction of oropharynx and nasal cavity. It is lined by pseudostratified squamous epithelium. It is open inferiorly. It walls are rigid and bony.

Boundaries of nasopharynx:

. Anterior: Posterior choanae and posterior part of nasal septum

. Floor: Soft palate and part of hard palate

. Roof: It is sloping antero posteriorly. It is formed by basi sphenoid and basi occiput. C1 and C2 vertebrae also contribute.

. Posterior: It communicates with oropharynx. This area is guarded by a ring known as passavant's ridge.

. Lateral: The pharyngeal end of eustachean tube is seen here. Around the pharyngeal end of eustachean tube there is a pad of fat present. This pad of fat is known as Ostman's pad of fat. In malnourished children this pad of fat is lost causing patulous eustachean tube. Fossa of Rosenmuller is seen above and behind the pharyngeal end of eustachean tube. It is about 1.5 cms deep. Its apex is in close relation ship with the carotid canal, and its base is closely related to skull base. Foramen lacerum lies medially. Nasopharyngeal carcinoma commonly arises from fossa of Rosenmuller.

Epidemiology:

1. Highest incidence of nasopharyngeal carcinoma has been reported in southern china. This region accounts for 20% of world's reported cases of nasopharyngeal carcinoma. The incidence rates in India is about 1 per 1 lakh population

2. This disease is three times more common in men than in women.

3. This tumor occurs at a much younger age than other cancers

4. Age wise bimodal distribution is also common. This distribution suggests the influence of different aetiological factors or variations in the host response. In this type of age distribution two peaks are noted, i.e. 1. between ages 15 - 20, 2. the second peak during the 4th and 5th decades. This type of distribution is common in India.

Aetiology:

1. Epstein - Barr virus: E.B. virus infections have been postulated to be the etiological agent responsible for nasopharyngeal carcinoma. The presence of raised antibody titers, and demonstration of viral genome in tumor cells are ample proof.

2. Exposure to chemical agents i.e. tobacco, drugs, and plant products.

3. Dietary factors: Ingestion of salted fish, preserved vegetables, fermented food stuff containing Nitrosamines and nitro precursors.

4. Cooking habits: Household smoke and fumes

5. Religious practices: like incense and joss stick smoke

6. Occupation: Exposure to industrial fumes / chemicals, metal smelting, Formaldehyde, wood dust

7. Other causes: Socioeconomic status, Nutritional deficiencies, weaning habits

8. Genetic susceptibility: Many HLA haplotypes have been associated with increased incidence of nasopharyngeal carcinoma. The loci involved are the HLA-A, B and DR locus situated on the short arm of chromosome 6.

Immunology in nasopharyngeal carcinoma:

Cell mediated immunity: is impaired in patients with nasopharyngeal carcinoma. This can be demonstrated by Mantoux test (in vivo), and Phytohaemagglutinin response of lymphocytes (in vitro). It is possible that this defective specific cell mediated immunity to EB virus allows the virus to be reactivated in the salivary glands. Increased EB virus loads causes increased anti EB virus IgA antibodies.

Association of EB virus with nasopharyngeal carcinoma: EB virus belongs to herpes family. It is lymphotrophic in nature. Its action is restricted to B lymphocytes. EB virus was found in abundance in the lymphoepithelium of the nasopharynx. Primary infection of this virus takes place in childhood and is always accompanied by seroconversion. EB virus is present in dormant state in small numbers of circulating B cells or in saliva. This virus may be reactivated during immunocompromised states.

The association of EB virus with nasopharyngeal carcinoma is supported by:

1. Demonstrable humoral immune response in patients with NPC against EB virus determined antigens (VCA viral capsid antigens, Early antigen EA, and nuclear antigen EBNA).

2. Presence of EB viral markers like EB viral DNA and Nuclear antigen in the tumor cells of nasopharyngeal carcinoma tumor cells.

Serologic markers of Nasopharyngeal carcinoma:

Markers associated with nasopharyngeal carcinoma include:

a. IgA and IgG to viral capsid antigen

b. IgA and IgG to early antigen

c. Antibody to nuclear antigen

d. Antibody dependent cellular cytotoxicity antibodies

Immunoglobulin IgA / VCA, IgG / VCA, and IgA / EA, IgG / EA are useful diagnostic markers of nasopharyngeal carcinoma. Their titers are related to the tumor load and advancing stage of the disease in untreated patients.

Normal values of these titres are:

Anti EB virus VCA / IgG = up to 1 : 160

Anti EB virus EA / IgG = up to 1 : 160

Anti EBV VCA / IgA = below 1 : 5

Anti EBV EA / IgA = below 1: 5

The titres of IgA / VCA and IgA / EA are useful clinical indices for follow up of patients after treatment. Titres may decline to a low level or remain static after successful treatment. The period between detection of raised IgA / VCA and clinical onset of stage I nasopharyngeal carcinoma ranged from 8 - 30 months.

Prognostic serological markers:

1. Prognosis and survival are inversely proportional to the geometrical mean titres of VCA and EA antibodies.

2. Good prognosis is indicated by high antigen dependent cellular cytotoxicity antibodies

Clinical presentation:

The marked invasive and metastatic properties are responsible for its symptomatology. The tumor arising from nasopharynx may spread in the following directions:

1. Anteriorly to nasal cavity, paranasal sinuses, pterygopalatine fossa and orbital apex.

2. Posteriorly to the retropharyngeal space and node of Rouviere, destruction of lateral mass of atlas

3. Laterally into the parapharyngeal space

a. Prestyloid compartment with involvement of mandibular nerve, pterygoid muscles and infiltration of deep lobe of parotid gland.

b. Post styloid compartment causing vascular compression of carotid sheath, invasion of last four cranial nerves and cervical sympathetic nerves

4. Superiorly through the body of sphenoid and sinus involving the parasellar structures and optic nerve, petrous apex and foramen lacerum. Cavernous sinus may be involved along with III, IV, V, and VI. The brain may also be affected by direct spread and not by hematogenous spread.

5. Inferiorly into the oral cavity and retrotonsillar regions.

6. Painless cervical lymphadenopathy because of its tendency for early lymphatic spread. Lateral retropharyngeal node of Rouviere is the first echelon node. The first node to become palpable is the jugulodigastric node / apical node under the sternomastoid muscle. These are second echelon nodes. Ipsilateral and bilateral nodal involvement are common.

7. Epistaxis: is commonly seen in advanced nasopharyngeal carcinoma with or without skull base erosion. It is not torrential in nature but only seen as blood tinged mucous secretion. Nasal obstruction may also be seen in advanced cases. Ozaena may also be a feature of advanced nasopharyngeal carcinoma.

8. Audiological symptoms like tinnitus, otalgia and deafness: These are common symptoms of nasopharyngeal malignancy. This is caused by blockage to the nasopharyngeal end of eustachean tube by the tumor mass.

9. Neurological symptoms like headache, cranial nerve palsy (any cranial nerve can be involved), and Horner's syndrome.

10. Distant metastasis to bone lungs and liver.

Clinical examination of nasopharynx:

Nasopharynx is one of the most difficult areas to examine clinically. Methods of examination of nasopharynx include:

1. Post nasal examination using post nasal mirror

2. Examination using nasopharyngoscope

3. Nasopharyngeal exam using nasal endoscope

Histological classification of nasopharyngeal carcinoma:

WHO classification of nasopharyngeal carcinoma is the most commonly used method. This classification divides nasopharyngeal carcinoma into three histological subtypes on the basis of light microscopic examination.

. Type I squamous cell carcinoma (keratinizing):

- well differentiated

- moderately differentiated

- poorly differentiated

. Type II Nonkeratinizing carcinoma

. Type III Undifferentiated carcinoma

Role of imaging in the diagnosis of nasopharyngeal carcinoma: CT scan is the most preferred imaging modality. It may be needed to identify the site for biopsy of the submucosal lesion. Major role of imaging in nasopharyngeal carcinoma is to help in the staging of the disease.

MRI scanning is useful and the most accurate method of evaluating primary tumor.

PET scanning is useful in diagnosis recurrent / residual lesions following RT.

Biopsy of the lesion is the definitive confirmatory investigation.

Tumor staging:

Modified Ho's classification:

Primary tumor (T)

T1 - Nasopharynx involvement only

T2n - Involvment of nasal cavity in addition

T2o - Involvement of oropharynx in addition

T2p - Involvement of parapharyngeal region

T3a - Bony involvement below the skull base including the floor of sphenoid

T3b - Involvement of skull base

T3c - Cranial nerves involvement

T3d - Orbit, laryngopharynx, infratemporal fossa

T3p - Parapharyngeal region

Regional nodes (N)

N0 - No nodes

N1 - Nodes above skin crease at laryngeal cartilage

N2 - Nodes below the skin crease but above the supraclavicular fossa

N3 - Supraclavicular nodes

Metastasis (M)

Mo - No distant metastasis

M1 - Distant metastasis

Staging

I (T1, T2n,T2o) No Mo

IIa (T2, T2n, T2o) N1, N2 Mo

IIb (T2p,T3, T3p) No Mo

IIIa (T2p, T3, T3p) N1, N2 Mo

IIIb (T1, T2n, T2o) N3 Mo

IVa (T2p, T3, T3p) N3Mo

IVb M1 (any T, any N)

AJCC classification:

Tx - Primary tumor cannot be assessed

To - No evidence of primary tumor

Tis - Carcinoma in situ

T1 - Tumor confined to the nasopharynx

T2 - Tumor extends to soft tissues

T2a - Tumor extends to the oropharynx / nasopharynx without parapharyngeal extension

T2b - Tumor with parapharyngeal extension

T3 - Tumor involves bony structures / paranasal sinuses

T4 - Tumor with intracranial extension / involvement of cranial nerves

Regional node (N)

Nx - Regional nodes cannot be assessed

No - No nodal metastasis

N1 - Unilateral metastasis in lymph nodes 6cms or less in the greatest dimension above the supraclavicular fossa

N2 - Bilateral nodal metastasis 6 cms or less in the greatest dimension above the supraclavicular fossa

N3 - Metastasis in nodes greater than 6 cms

N3a - Extension to supraclavicular fossa

Staging:

Stage 0 - Tis No Mo

Stage I - T1 No Mo

Stage IIa - T2a No Mo

Stage IIb - T1 -2a N1 Mo

Stage III - T1 - 2b N2 Mo

Stage IVa - T4 No -2 Mo

Stage IVb - Any T N3 Mo

Stage IVc - Any T Any N M1

Treatment:

Nasopharyngeal carcinoma is a highly radiosensitive tumor hence irradiation is the preferred modality of treatment.

Megavoltage external radiotherapy is the treatment modality of choice. This is given through two lateral opposing and one anterior fold. Treatment should be delivered without interruption in five sessions per week for six weeks delivering a total dose of 60 Gy.

Role of surgery: is limited to biopsy of the lesion and confirming the diagnosis. If there is nodal metastasis then block neck dissection should be resorted to.

18. Discuss the etiology, spread, clinical presentation and management of juvenile nasopharyngeal angiofibroma.

Synonyms: angiofibroma, juvenile angiofibroma, juvenile nasopharyngeal angiofibroma, (JNA)
Definition: Juvenile nasopharyngeal angiofibroma (JNA) is a histologically benign, but locally invasive neoplasm occurring almost exclusively in adolescent males. These tumors are highly aggressive and are associated with significant morbidity and mortality due to its tendency to bleed.

History: Juvenile nasopharyngeal angiofibroma has been documented since the time of Hippocrates (4 BC). In fact Hippocrates goes on to describe a polyp in the nose which weeped blood. In 1906 Chareau revived the interest in the study of juvenile nasopharyngeal angiofibroma. Shaheen in

1930 reported the first female patient with juvenile nasopharyngeal angiofibroma. Hondousa recorded the youngest JNA patient (8 years). Figi and Davis (1950) emphasised the role of surgery in the management of JNA. They also suggested that subperiosteal dissection of the mass reduced bleeding to a great extent.
Histopathological studies of JNA tissue was extensively done by Harma (1959).

Anatomy of Nasopharynx: Since JNA involves the nasopharynx, a complete understanding of nasopharyngeal anatomy is a must for the safe management of the problem. This area has been considered as an anatomical blind spot (a virtual no man's land) because of its relative inaccessibility. It is very difficult to visualise completely the entire nasopharynx. Cawthorne (1953) remarked "I am never sure whether I will be able to get a really satisfactory view of the whole nasopharynx". The advent of endoscopes have made the task of examination of nasopharynx simpler these days. The recent imaging modalities like CT scan and MRI has further simplified noninvasive ways of examining the nasopharynx.

Location: Nasopharynx is located at the confluence of nasal, aural and pharyngeal air passages. It is an unyielding irregular cuboidal box containing
4 x 3 x 2 cm air space at the base of middle cranial fossa.

Nasopharynx is located immediately behind the nasal cavities and below the body of the sphenoid and basi occiput above the level of soft palate as illustrated in the figure. Posteriorly it is limited by the first two cervical vertebrae. The posterior wall of nasopharynx is about 8 cm from the pyriform aperture along the floor of the nose.

Boundaries of Nasopharynx:

Anterior: Choanal orifice and posterior margin of nasal septum.

Floor: is formed by the upper surface of soft palate in its anterior 2/3 while the posterior 1/3 is formed by nasopharyngeal isthmus.

Roof and posterior wall: is formed by the continuous sloping surface of the body of the sphenoid, the basiocciput and the first two cervical vertebrae up to the level of the soft palate. The upper portion of the posterior wall lies in front of the anterior arch of atlas with a mass of lymphoid tissue embedded in the mucous membrane (adenoid). The prevertebral fascia and muscles separate the adenoid from the vertebrae.

Lateral wall: is dominated by the pharyngeal end of eustachean tube. It is located in the middle of the lateral wall, it is about 1.5 cm equidistant from the roof, posterior wall, choana and the floor. The tubal elevation created by the elastic cartilage of the tube is particularly prominent in its upper and posterior lip. Behind the posterior margin of torus, between it and the posterior wall lies the lateral pharyngeal recess or the fossa of Rosenmuller. Aggregates of lymphoid tissue (Gerlachs tonsil) of variable sizes surround the tubal orifice. This is also known as the tubal tonsil.

Fossa of Rosenmuller is situated at a corner between the lateral and dorsal walls. This recess is not obvious in infants, and can measure up to 1.5 cm in depth in adults. It opens into the nasopharynx at a point below the foramen lacerum related to the internal carotid artery.

Boundaries of Fossa of Rosenmuller:

Anterior: Eustachean tube and levator veli palati muscle.
Posterior: Pharyngeal wall mucosa overlying the pharyngobasilar fascia and retro pharyngeal space, containing the retropharyngeal lymph nodes of Rouviere.
Medial: Nasopharyngeal cavity.
Superior: Foramen lacerum and floor of the carotid canal.
Postero lateral (apex): Carotid canal opening and petrous apex posteriorly, foramen ovale and spinosum laterally.
Lateral: Tensor palati muscle, mandibular nerve and the prestyloid compartment of the para pharyngeal space.

Epithelial lining of nasopharynx: The mucosal lining of nasopharynx is thrown into numerous folds and crypts. The surface area of mucosal lining of nasopharynx is about 50 cm 2 in adults. The mucosa abutting the choanae and immediate nasopharyngeal roof is completely lined by ciliated columnar epithelium. The nasopharyngeal mucosa differs from the rest of the upper respiratory tract in that the subepithelial connective tissue is rich in lymphoid tissue.

To understand the clinical features of mass lesions involving the nasopharynx, a clear understanding of the roof of the fossa of Rosenmuller is important. As already mentioned, the roof of fossa of Rosenmuller is formed by the Foramen Lacerum. Through this foramen lacerum passes:

a. Internal carotid artery
b. Greater superficial petrosal nerve
c. The ascending palatine artery sometimes passes through this foramen.

It is through this foramen tumors from fossa of Rosenmuller invades intracranial structures. The 3rd, 4th, 5th and 6th cranial nerves are in juxtaposition of the foramen. These nerves are commonly involved when tumors invade intracranial structures via the foramen lacerum.

Blood supply of nasopharynx:

a. Ascending pharyngeal artery
b. Ascending palatine branch of facial artery
c. Branches of internal maxillary artery

Veins form a plexus which communicate above with the pterygoid plexus and drains into the common facial and internal jugular veins.

Lymphatics: Nasopharynx is richly endowed with lymphatics. They mainly originate in the pharyngeal tonsil and runs laterally and downwards on the pharyngeal aponeurosis, some of them terminating in the median and lateral veins. The collecting trunks terminate for the most part in the upper nodes of the spinal accessory chain, which lie under the upper end of sternomastoid muscle, and also into the jugulodigastric node of the internal jugular chain. From these nodes efferent branches run down to the middle and lower group of nodes of the internal jugular and spinal accessory chains.

Nerve supply: is derived mainly from the pharyngeal plexus which is formed by branches of the 9th and 10th cranial nerves together with sympathetic fibers.

Aetiopathogenesis of JNA:
This relatively rare tumor occurs in the second decade. Almost exclusively adolescent males are affected. The reported rate of incidence varies from 1/6000 (Harma 1959) to 1/50,000 (Hondousa etal 1954). The exact nature of the tumor and its etiology is not well known. Various theories have been propounded to explain the etiopathogenesis of JNA.

Theories of etiopathogenesis of JNA:

Ringertz theory: This theory was proposed by Ringertz in 1938. He believed that JNA always arose from the periosteum of the skull base.

Som & Neffson (1940): believed that inequalities in the growth of bones forming the skull base resulted in hypertrophy of the underlying periosteum in response to hormonal influence.

Bensch & Ewing (1941): thought that the tumor probably arose from embryoninc fibro cartilage between the basi occiput and basi sphenoid.

Brunner (1942): Suggested an origin from conjoined pharyngobasilar and buccopharyngeal fascia.

Marten et al (1948): Proposed a hormonal theory suggesting that these tumors resulted from deficiency of androgens or over activity of estrogens and that the hormonal stimulation is responsible for angiomatous components seen in JNA tissue.

Sternberg (1954): Proposed that JNA could be a type of haemangioma like a cutaneous haemangioma seen in children which regresses with age.

Osborn (1959): Considered two alternatives to explain the etiology of JNA. They proposed that the swelling could be due to either a hamartoma or residual fetal erectile tissue which were subject to hormonal influences.

Girgis & Fahmy (1973): Observed cell nests of undifferentiated epitheloid cells or "Zell ballen" at the growing edge of angiofibromas. This appearance was more or less similar to that of paraganglioma. They considered JNA to be a paraganglionoma.

The most accepted theory is that JNAs originate from sex steroid–stimulated hamartomatous tissue located in the turbinate cartilage. The proposed hormonal influence may explain why (rarely) some JNAs involute after puberty.

Pathophysiology: The proposed origin of the JNA is located along the posterior-lateral wall in the roof of the nasopharynx, usually in the region of the superior margin of the sphenopalatine foramen and the posterior aspect of the middle turbinate. Fetal histology confirms large areas of endothelial tissue in this region. Rather than invading surrounding tissue, this tumor displaces and distorts, relying on pressure necrosis to destroy and push through its bony confines. Intracranial extension is noted in 10-20% of cases.

JNA are seldom seen in children below the age of 8. The rate of growth of tumor and period of maximum development coincides with rate of erectile tissue of penis, both increasing in size during the period of sexual development.

Pathology:

Macroscopic: Grossly, angiofibromas appear as firm slightly spongy lobulated swelling with presence of nodules. The nodularity increases with age. Their color varies from pink to white. The part which is seen in the nasopharynx and which is covered by mucous membrane is invariably pink, where as those parts which have escaped to adjacent extra pharyngeal areas are often white or grey.

On section the tumor is reticulated, whorled or spongy in appearance lacking a true capsule. The edges of the tumor are however, sharply demarcated and easily distinguishable from the surrounding tissues. Hence to reduce bleeding during surgical excision of the tumor the mass should be peeled off from its attachments and the mass should not be broken into.

Microscopic appearance: Microscopically the picture is of vascular spaces of varying shapes and sizes within a stroma of fibrous tissue. The relative proportions of the vascular and the stromal components change with the age of the swelling. In earlier lesions the vascular component stands out as an all-pervasive feature, whereas in the more long-standing tumors collagen predominates. It could also be seen that, as one strays away from the heart of the tumor the fibrous tissue element overshadows vascular element. The tumor is covered by squamous epithelium. In some cases pseudostratified columnar epithelium is seen side by side with the metaplastic squamous epithelium.
Cellular infiltration is a common feature, particularly in the superficial parts of the tumor, underneath the epithelium. the infiltrates include, plasma cells, lymphocytes, polymorphs and eosinophils in varying proportions.

The structure of the tumor is made up of fibrous tissue elements and vascular channels. The fibrous tissue element is made up of cells which are spindle shaped, oval or round cells. In between these cells, the bundles of collagen fibres are seen running in different directions.
The vascular channels of the tumor may be divided into 2 main types. One type which is seen in all cases is made up of spaces lined with one layer of endothelium and is free of any muscular coating. In the other type the vascular channels are made up of arteries with thick muscular coating.
Mucous glands may be seen in the superficial parts of the tumor underneath the epithelial covering. In some patients nerve bundles could also be seen. In long standing tumors, there is a tendency towards gradual compression of the sinusoids so that the lining endothelial cells are pushed against each other like cords, where in others intravascular thrombosis occur.

Clinical features: Signs and symptoms are present for an average of 6 months prior to the diagnosis, commonly with extension beyond the nasopharynx.
The two cardinal symptoms of angiofibroma are nasal obstruction and intermittent unprovoked epistaxis. Epistaxis may vary in severity from an occasional show to an alarming sometimes threatening torrent. Chronic anaemia is thus a feature of an established JNA.
The nasal obstruction is so complete causing stasis of secretions and sepsis become inevitable. Patients may even have hyposmia or anosmia.

The voice of the patient acquires a nasal intonation. If the swelling enlarges to force the soft palate down, the voice may become plummy.
Blockage of eustachean tube orifice is also common causing deafness and otalgia. Headache is not uncommon in long standing cases. If present it could be attributable to chronic sinusitis in some patients. Intracranial extension of the mass could also be the cause for headache in these patients.

Diplopia may occur secondary to the erosion of the mass into the cranial cavity and causing pressure on the optic chiasma. Failing vision has been attributed by Shaheen to the tenting of the optic nerveby the tumor.

Anterior rhinoscopy shows the presence of abundant purulent nasal secretions together with bowing of nasal septum to the uninvolved side. Posterior rhinosocpy in a cooperative patient shows a pink or red mass filling the nasopharynx. Due to the bulk of the lesion it may not be always possible to ascertain the site of origin accurately.

Gross physical signs become evident when extensive disease involves the nose and infra temporal fossa. The nasal bones become spayed out and there may be obvious swelling in the temple and cheek. Intra oral palpation in the interval between the ascending ramus of the mandible and the side of the maxilla may reveal the tell tale thickening of disease which has crept around the back of the antrum. Impaction of bulky mass in the infra temporal fossa results in extreme signs, such as trismus and bulging of the parotid gland. Proptosis is a definite sign that the orbital fissures have been penetrated. The classic frog face seen in patients with extensive disease is due to massive escape of the disease.

Extension of tumor:

As growth enlarges it has to follow the lines of least resistance.

a. It hangs down in the cavity of nasopharynx and when large enough, it may depress the soft palate and may even peep below it.
b. It can work its way in to the corresponding nasal passage towards the anterior nares. It can cause pressure on the outer wall as well as on the septum bending it to the opposite side. The corresponding turbinates and ethmoidal air cells and the related antral wall may suffer pressure atrophy. The most common deformity referred to as the "frog face" is due to the forward spread involving the ethmoidal region. Lateral spread in to the maxillary sinus may be responsible for the cheek swelling.
c. The mass commonly arises from the sphenopalatine foramen area. It may have two components, one filling the nasopharynx and the other extending out into the pterygopalatine and infratemporal fosse. The central stalk joining the two components occupy the sphenopalatine foramen at the upper end of the vertical plate of palatine bone.
d. It can encroach into the orbit by passing through the infra orbital fissure.
e. It can erode the skull base and cause intracranial problems.

Blood supply of JNA: is from the
1. Enlarged maxillary artery
2. Ascending pharyngeal artery
3. Unnamed branches from internal carotid artery

Staging of the tumor: Staging of the tumor has been done to for prognosis and therapeutic approaches. The currently accepted staging has been devised by Andrews.

Andrews staging classification:

Stage I: Tumor limited to the nasal cavity
Stage II: Tumor extension into the pterygopalatine fossa, or maxillary, sphenoid or ethmoid sinuses.
Stage IIIa: Tumor extension into the orbit without intracranial involvement.
Stage IIIb: Stage IIIa with extradural (parasellar) intracranial involvement
Stage IVa: Intradural without cavernous sinus, pituitary, or optic chiasm involvement
Stage IVb:Involvement of the cavernous sinus, pituitary, or optic chiasm

Surgery is usually recommended for stages up to IVa while for stage IVb radiotherapy is advisable.

Investigations: Standard X-rays of paranasal sinuses may reveal opacity in the nose and sinus areas. Xray skull lateral view may show the mass inside the nasopharynx. CT scan has replaced the preferred imaging modality in these patients. Both plain and contrast CT scans must be performed to clinch the diagnosis. It also reveals the extent of the lesion and also helps in staging of the disease. MRI reveals the precise extent of the mass.

Carotid angiogram may be performed to identify the feeder vessel and also to embolise the feeder to reduce bleeding during surgery.

Differential diagnosis: JNA should be differentiated from:

1. Pyogenic granuloma
2.Choanal polyp
3. Angiomatous polyp
4. Nasopharyngeal cyst
5. Chordoma
6. Carcinoma

Treatment: Depends mainly on the extent of the lesion. Surgery is the preferred modality of treatment for all stages of the mass up to stage IVa while radiotherapy is used for stage IVb. Mainly three lines of treatment are available:
1. Surgery
2. Irradiation
3. Hormonal (purely supportive in nature)

Surgery: Complete excision of an extensive JNA mass is a desirable goal but is a surgical challenge because of the limited field of work, inadequate visualisation and profuse bleeding during surgery. Besides the deformity, scars and adhesions as a result of prior surgery adds to this problem. Currently several approaches are available to access the neoplasm.

I Tumor removal - via naturalis

II Transpalatal approach

III. Lateral rhinotomy approach

IV. Trans hyoid approach

V. Transmandibular approach

VI. Sublabial midfacial degloving approach

Radiotherapy:
Radiotherapy can produce some amount of tumor regression by radiation vasculitis and occlusion of vessels by perivascular fibrosis. Radiotherapy should be reserved for selected patients such as those with inoperable intracranial extensions and recurrent tumors.

Disadvantages of radiotherapy:

a. If the child is exposed to large doses i.e. above 5000-6000 rads, there may be damage to eyes, spinal cord and brain.
b. Small doses are ineffective in reducing the blood supply or the size of the mass.
c. Radiotherapy may cause fibrosis and adhesions of surrounding tissue. Later surgery upon these patients becomes difficult.
d. Sarcomatous changes can occur in the mass as a result of irradiation.

Adjunctive treatment:

Hormonal therapy: Since JNA has been postulated as an endocrine tumor testosterone receptor blocking drugs / estrogens have been tried to reduce the mass. These hormones cause disagreeable side effects such as increased breast size. Hormones could even act as carcinogens.

Embolisation: Was first attempted by Robertson in 1972. This was not meant to be therapeutic measure. After embolisation bleeding is minimised during surgery. It is ideally carried out a few days before surgery. Hence it is a valuable preliminary to surgery. The feeding vessels to the tumor is identified. It is then deliberately occluded by means of materials injected through a selectively placed catheter.
Materials used:
Autologous substances like fat, blood clot, or chopped muscle fragments.
Artificial materials: Gelfoam, Oxidised cellulose, Tantalum powder, glass beads, polyvinyl alcohol etc.
Embolisation should always be preceded by angiography. Subtraction films may be helpful in areas containing complex bony structures.
Immediate complications of embolisation are pain, embolisation of normal vessels, hypersensitivity. Delayed complications include fever, pain and infections.

Cryosurgery and Lasers can also be used during surgery to minimise bleeding.

19. Acquired Immuno Deficiency Syndrome-AIDS in ENT – Discuss.

Almost 45 years have passed since the first description of HIV. It still remains a global pandemic. Ofcourse with the current public awareness and treatment methadology available HIV patients are living longer and lead a normal life. The incidence of HIV is also on the wane.

Nearly 80% of HIV infected patients present in the ENT OPD with otolaryngological manifestations. Among the otolaryngological manifestations, oral disease seems to be the most common, occuring in nearly 50% of these patients. The predisposing factors for HIV related ENT conditions include

CD4+ cell count of less than 200//μL and Plasma HIV-RNA levels greater than 3000 copies/mL. Some of the other predisposing factors in addition to the above stated ones include: xerostomia, poor oral hygiene, and smoking.

Although ENT manifestations may not be diagnostic of HIV infections, they may be heavily suggestive of such an infection. The presence of certain oral manifestations in patients with known HIV disease who are not on treatment could indicate the progression of the disease. The presence of ENT disease in patients on antiretrovial therapy could be the result of an increase in the plasma HIV-RNA and suggest treatment failure.

Oropharyngeal manifestations of HIV infection:

Oral candidiasis also commonly known as thrush, is the most common oral manifestation of HIV infection. Candidal infections can occur in the oropharynx, hypopharynx and larynx. This condition usually causes severe odynophagia and swallowing difficulties.

The prevalence of candidiasis in HIV positive patients is highly variable ranging between 30-90%.

Oral candidiasis in these patients usually present in three forms:

Pseudomembranous candidiasis:

This is the most common fungal infection in HIV patients. The lesions appear are curd like plaques on the buccal mucosa, tongue, and other oral mucosal surfaces. The plaques can be wiped away leaving behind a red / bleeding underlying surface. These lesions are associated with progression of HIV and can also be used as a clinical marker to define the severity of the infection.

The common organism involved is candida albicans; however involvement of non-albican species such as candida glabrata, and candida dubliniensis have also been described.

Erythematous candidiasis:

These lesions present as red, flat, subtle lesion on the dorsal surface of the tongue, or on the hard/soft palate. This lesion often involves two opposing surfaces, i.e. if a lesion is present on the tongue, the palate should also be examined for a matching lesion. These patients usually complain of a burning sensation, especially while eating spicy / salty food. If hypopharynx, esophagus and larynx are affected, symptoms may progress to severe odynophagia and the patient may have swallowing difficulties. This is rather common in children and they may even require hospitalization. These patients should receive intravenous amphotericin B.

Angular chelitis:

This is a condition that causes red, swollen patches in the corners of the mouth where the lips meet and make an angle. There could be irritation and soreness in the corners of the mouth. The corners of the mouth could be:

Cracked

Crusty

Blistered

Painful

Scaly

Swollen

The warmth and moisture in the corners of the mouth creates a perfect condition for fungal proliferation.

Treatment involves using topical antifungal cream directly applied to the affected areas at least 4 times a day for 2 weeks.

Candidiasis can be confirmed in challenging cases from the identification of fungal hyphae in potassium hydroxide preparation (KOH).

Management of mild to moderate cases of erythematous and pseudomembranous candidiasis include clotrimazole torches, nystatin oral suspension. Systemic administration of fluconazole, itraconazole and voriconazole are needed in moderate to severe cases. Voriconazole is reserved for cases of fluconazole resistance. Antifungal therapy should be used for at least 2 weeks for optimal benefit.

Periodontal and gingival disease:

This is frequently seen in patients with HIV. This condition commonly presents as a red band along the gingival margin. This condition is accompanied by occasional bleeding and discomfort. This condition frequently appear at the anterior teeth, but can also extend to the posterior teeth. It can also present on attached and non attached gigiva as petechia like patches.

Treatment of this condition includes debridement by dentist. Mouth rinses with a 0.12% chlorhexidine gluconate suspension twice daily for two weeks. Maintenance of oral hygiene is rather important in these patients.

Necrotizing ulcerative periodontitis is a marker for severe immune suppression. This condition is characterised by severe pain (deep jaw pain), loosening of the teeth, bleeding, fetid odor, ulcerated gingival papillae and rapid loss of bone and soft tissue. This condition is usually managed by curettage and debridement of all involved tissues, and use of topical antiseptic agents such as 0.12% chlorhexidine gluconate or 10% povidone iodine lavage. Severe cases can be managed by a course of metronidazole. Clindamycin and amoxycillin therapy are also recommended.

Oral Herpes simplex virus infection:

This occurs in nearly 10% of adults with HIV. These lesions present as a small crop of vesicles which produce small, painful ulcerations extending onto adjacent skin and may even coalesce to from giant herpetic lesions. These lesions are often bigger in patients with HIV infection. These lesions could be self limiting and sometimes use of antiviral agents like acyclovir may be needed. These lesions oculd become chronic in HIV children needing intravenous administration of acyclovir.

Hairy Leukoplakia of oral cavity:

These are large asymptomatic lesions involving the tongue. These lesions are caused by Epstein Barr virus. These lesions present as a white corrugated lesion on the lateral borders of the tongue. This lesion cannot be removed by the patient. The terminology of this lesion arises from the appearance of elongated filiform papillae which can be accompanied by white plaque like changes. This condition requires no treatment unless cosmetic concerns arise.

Oral human papilloma virus infections has increased in the era of highly active antiretroviral therapy (HAART therapeutic regimens). This could be due to the fact that the drugs used to treat HIV may be a risk factor for oral HPV infection. The most common HPV subtypes seen in the oral cavity are subtypes 16 and 18. These lesions appear as warts, cauliflower like spiked / raised with a flat surface.

Kaposi sarcoma:

This is the most frequent malignant condition seen in the oral cavity. The prevalence of this condition in the mouth is about 0.4% among HIV patients. Oral cavity is commonly affected and is the first site to be involved by Kaposi sarcoma in nearly 20% of cases. This condition involves the skin and viscera in up to 70% of these patients. In oral cavity the hard palate is most frequently involved, followed by gingival and buccal mucosa as well as the dorsum of the tongue.

Kaposi's sarcoma associated herpes virus was proven to be a co-factor in the presentation of Kaposi's sarcoma in patients with HIV. Kaposi's sarcoma can be macular, nodular, or raised and ulcerated. The color of the lesions can range from red to purple. Early lesions tend to be red, flat and asymptomatic. The color of the lesion darkens as the lesion ages. As these lesions progress, they can become symptomatic due to trauma or infection. Biopsy of the lesion, usually under LA is necessary for the diagnosis. Following the diagnosis of Kaposi's sarcoma oral hygiene is necessary.

Management:

Topical injections of chemotherapeutic agents such as vinblastine sulfate, or even surgical removal or radiation therapy can be considered for treatment. Several surgical techniques have been

described including cryotherapy and laser therapy. Systemic chemotherapy is reserved for patients with both oral and extra oral kaposi's sarcoma.

Non-Hodgkin's lymphoma:

This is the second most common malignant condition associated with HIV infection. Lymphomas present as a focal, ulcerated soft tissue mass on the palate or gingival tissues. The affected tissues can be red and inflammed. These lesions can be painful, and progresses rapidly. Suspected lesions can be diagnosed with a biopsy, usually under LA.

Management:

Systemically administered chemotherapy. CHOP regimen (cyclophosphamide, doxorubicin, vincristine and prednisolone) can be considered the standard approach for patients with aggressive NHL in the context of HIV infection. Surgical debulking may be needed for pain relief and improvement of chewing, swallowing and speech in large exophytic or pedunculated lesions. Radiotherapy may be considered for large lesions that cannot be easily accessed.

Neck manifestations of HIV infection:

Cervical adenopathy is the most common manifestation of HIV infection in the neck. In addition to reactive lymphadenitis, cervical adenopathy may result from tuberculosis, lymphoma, or kaposi's sarcoma in HIV patients. The term HIV lymphadenopathy describes the presence of diffuse lymphadenopathy in two or more sites of the neck for longer than three months. This condition can occur in up to 70% of HIV patients within the first few months after seroconversion, even before any other symptoms of HIV infection appear. This condition is also seen in children. The affected nodes are soft and symmetrical ranging from 1-5 cm in diameter. They are frequently observed in the posterior triangle. Histology is suggestive of reactive follicular hyperplasia. FNAC is indicated in cases of asymmetry, rapidly enlarging lymph nodes, or presence of any other suspicious features. Biopsy under local / general may be necessary in cases of high suspicion of lymphoma.

Salivary gland disease is also not uncommon in HIV infected patients. It usually involves he parotid glands and tends to be bilateral. Sometimes they are cystic and can be accompanied by generalized lymphadenopathy. These patients usually present with a history of progressive parotid swelling with minimal tenderness for over several months. Salivary gland enlargement occurs in approximately 30% of adult patients infected with HIV and in up to 20% of affected children. This condition could represent the first clinical manifestation of HIV. Clinical examination should include assessment of the characteristics of the mass (i.e. fixation) and the function of the facial nerve. The three common causes of parotid enlargement in HIV infected patients are reactive hyperplasia of an intraparotid lymph node, benign lymphoepithelial lesions witih ductal metaplasia and benign lymphoepithelial cysts. FNAC is an effective method of distinguishing benign from malignant parotid lumps. The most common FNAC diagnosis include cystic mass / lymphadenitis and chronic inflammation.

Management:

Treatment of salivary gland enlargement in HIV disease still remains controversial. Superficial parotidectomy has been proposed, but has not been widely accepted. Aspiration of these cystic lesions can offer temporary relief, and injections of tetracycline and doxycycline have been shown to be successful, although with limitations due to the presence of multiple cysts. External irradiation can be considered (24 Gy in 1.5 Gy daily fractions).

Otological manifestations of HIV infection:

Otological manifestation spectrum of HIV infections are rather wide. All three portions of the ear (external, middle and inner ears) can be affected both in adults and pediatric age groups.

External ear:

Seborrheic dermatitis has been reported in 90% of HIV infected patients around periauricular area. Otitis externa caused by pseudomonas aeruginosa and candida albicans can also be seen.

Otalgia is a very frequent symptom in HIV patients and this can be attributed to the disproportionately severe inflammatory changes in the mastoid air cells even in otherwise asymptomatic carriers.

Otitis media with effusion secondary to nasopharyngeal lymphoid hyperplasia / presence of other nasopharyngeal masses is also common in HIV positive patients.

Acute otitis media can also occur, but is usually seen in patients with endstage HIV disease. There is also an increased prevalance of Pneumocystis carinii infected aural polypi in these patients.

Sensorineural hearing loss (unilateral/bilateral) are seen in nearly a third of these patients. These patients manifest down-sloping hearing loss, usually moderate in high frequncies. Speech frequency is not significantly not affected. Histological study of organ of corti in these patients dont reveal any abnormality, except for some cystic changes in the spiral ligament and stria vascularis. Probably there is involvement of retrocochlear pathways / cochlear nerve.

Middle ear infections can be managed using broad-spectrum antibiotics, whereas mastoid exploration may be necessary in cases unresponsive to conservative management.

HIV patients also experience significant dysequilibrium, which is also attributed to the central nervous system pathology. Inner ear abnormalities have also been reported. There is also certain amount of inflammatory endolypmphatic precipitations causing Meniere's like manifestation.

Unilateral / Bilateral facial nerve palsy occurs nearly 100 times more in HIV infected patients. Facial nerve neuropathy can occur at any stage of HIV infection. It may even precede the appearance of HIV antibodies, and are known to occur frequently in HIV carriers than in patients with full blown AIDS. Peripheral facial nerve neuropathy are usually self limiting and could be due to idiopathic or due to herpes virus infection (Ramsay Hunt syndrome). Treatment for this condition include acyclovir 800 mg five times a day for seven days and prednisolone 30 mg once a day for 5 days with tapering of the starting dosee in three day intervals. Facial nerve palsy can be seen in end stage patients as an isolated entity or as a part of multiple cranial nerve involvement.

Nasal manifestations of HIV infection:

Nasal manifestations are not uncommon among HIV patients. Rhinosinusitis is known to occur in nearly half of these patients. Despite the fact that cellular immunity is compromised in these patients, studies have revealed excessive production of IgE, which is suggestive of active allergic rhinitis in the absence of parasitic infections. There is a two-fold increase in the incidence of allergic symptoms in HIV infected patients. These patients usually present with clear rhinorrhoea and nasal congestion. Budesonide nasal spray is preferred to fluticasone due to its significantly longer half-life. Monteleukast has also been successfully used in these patients.

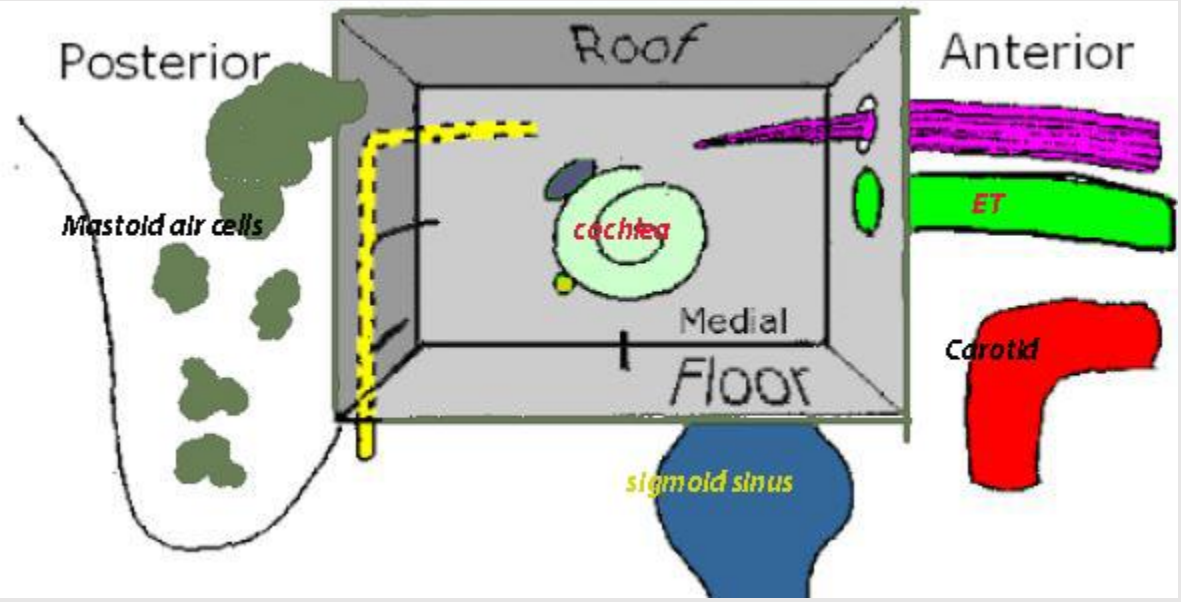
In addition to compromised immunity, there is also evidence of impaired mucociliary clearance in these patients. This accounts for the higher incidence of rhinosinusities in these patients. There is no difference in bacteriology in these patients. Atypical bacteria can be seen in patients with decreased CD4 count. The atypical organism isolated include alternaria alternata, aspergillus, pseudallescheria boydii, cryptococcus, candidia albicans, acanthamoeba castellani, microsporidian and legionella pneumophilia.

These patients can be managed with standard outpatient medical therapy with oral antibiotics for 3 weeks and nasal decongestants. In chronic cases the treatment is to be prolonged for 4-6 weeks. Oral antibiotics administered include amoxicillin, amoxycillin with clavulanic acid, or cefuroxime. It the response to antibiotics is partial then CD4 count should be less than 200. In chronic cases the coverage should be broadened to include pseudomonas, staphylococci, and anaerobic species. These patients can be managed using a combination of fluoroquinolones and clindamycin or metronidazole.

SHORT NOTES

EAR

1. Draw a neat labelled diagram of middle ear cleft



2. Middle ear cleft

The middle ear cleft includes the tympanum (middle ear cavity proper), the eustachean tube, and the mastoid air cell system. The tympanic cavity is an air-filled irregular space contained within the temporal bone. It also contains the three auditory ossicles (malleus, incus and stapes) along with their attached muscles. For the purpose of description, the tympanic cavity may be considered as a box with four walls, a roof and a floor. The corners of this hypothetical box are not sharp.

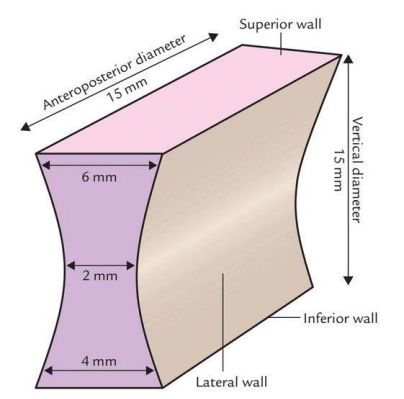
Lateral wall – Formed by the ear drum

Medial wall – Formed by the promontory which is raised by the basal turn of the cochlea

Roof – Is formed by tegmen tympani which separates the middle ear cavity from the brain

Floor - The floor is much narrow. In fact it is narrower than the roof of the middle ear cavity. This portion of the middle ear cavity lies in close relationship with the jugular bulb.

Anterior wall - The anterior wall of the tympanic cavity is very narrow. This is because the medial and lateral walls converge anteriorly. The anterior wall can be divided into two portions; the upper and lower portions. The lower portion of the anterior wall is larger than the upper portion. It has a thin plate of bone which separates this portion from the internal carotid artery as it enters the skull. This plate has two openings for the carotico tympanic nerves. The upper opening transmits the superior carotico tympanic nerve and the inferior opening transmits the inferior carotico tympanic nerve. It is through these nerves that sympathetic nerves reach the tympanic plexus. The upper smaller part of the anterior wall has two tunnels placed one below the other. The upper tunnel transmits the tensor tympani muscle, and the lower tunnel transmits the bony portion of the eustachean tube.



Shape and dimensions of middle ear cavity

Posterior wall - The posterior wall of the middle ear is wider above than below. In its upper part it has an important opening known as the aditus. This aditus helps the middle ear communicate with the mastoid air cell system. Aditus is a large irregular opening connecting the mastoid antrum to the middle ear cavity. Below the aditus is a small depression known as the fossa incudis. Fossa incudis houses the short process of the incus. Below the fossa incudis lies the pyramid.

Pyramid is a small conical projection which is hollow and its apex pointing anteriorly. It contains the stapedius muscle, the tendon of which passes forwards to insert into the neck of the stapes. The canal within the promontory curves downwards and backwards to join the descending portion of the facial nerve canal. Between the promontory and the tympanic annulus is the facial recess. The facial recess is bounded medially by the facial nerve and laterally by the tympanic annulus. Running through the wall between the two with varying degrees of obliquity is the chorda tympani nerve. This nerve always run medial to the tympanic membrane. Drilling over the facial recess area between the facial nerve and the annulus in the angle formed by the chorda tympani nerve can lead into the middle ear cavity. This surgical approach to the middle ear cavity through this area is known as the facial recess approach. This approach is suitable for surgeries involving the round window niche like placement of electrodes during cochlear implant procedures. Hypotympanum can also be approached through this approach.

Contents of middle ear cavity:

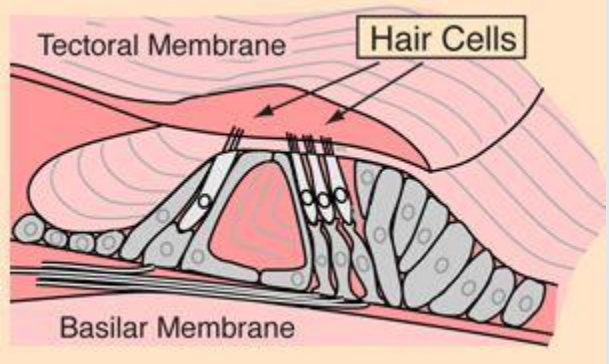
1. Air
2. Malleus / incus / stapes
3. Tensor tympani and stapedius muscle

3. Organ of corti

The organ of Corti is the sensitive element in the inner ear and can be thought of as the body's microphone. It is situated on the basilar membrane in one of the three compartments of the Cochlea. It contains four rows of hair cells which protrude from its surface. Above them is the tectoral membrane which can move in response to pressure variations in the fluid- filled tympanic and vestibular canals. There are some 16,000 -20,000 of the hair cells distributed along the basilar membrane which follows the spiral of the cochlea.

The place along the basilar membrane where maximum excitation of the hair cells occurs determines the perception of pitch according to the place theory. The perception of loudness is also connected with this organ.

Tiny relative movements of the layers of the membrane are sufficient to trigger the hair cells. Like other nerve cells, their response to stimulus is to send a tiny voltage pulse called an "action potential" down the associated nerve



Organ of corti

4. Korner’s septum

Korner's septum (KS) was first described by Cheattle in 1906 and later was named after Korner after the clinical significance of the petrosquamous suture creating a dual antrum. KS is a developmental remnant formed at the junction of mastoid and temporal squama bone, representing the persistence of the petrosquamosal suture. KS identification is associated with retraction pockets or adhesion, retraction of the tympanic membrane than in normal ears. However, various

studies in the literature have shown the very minimal incidence of the presence of KS. However, literature also mentions about the variants of KS. With the advent of high-resolution computed tomography (HRCT) scan of temporal bone it is now possible to identify the presence of KS preoperatively and in turn help in avoiding untoward complications of surgery. The clinical significance of the duality of the antrum and the mastoid process in its relationship to disease of the middle ear and mastoid is clarified by an understanding of KS. During mastoid surgery, it could be taken as a false medial wall of the antrum so that the deeper cells might not be explored.

In general, it is very uncommon to find the persistence of KS in the adult temporal bone as compared to the pediatric temporal bone. However, in our case, KS was seen in chronic otitis media squamosal disease type. A good knowledge of the anatomy of pneumatization of mastoid and HRCT scan of the temporal bone is very essential to avoid complications in micro ear surgery.

5. Physiology of Hearing

The process of hearing begins with the occurrence of a sound. Sound is initiated when an event moves and causes a motion or vibration in air. When this air movement stimulates the ear, a sound is heard.

In the human ear, a sound wave is transmitted through four separate mediums along the auditory system before a sound is perceived: in the outer ear—air, in the middle ear— mechanical, in the inner ear liquid and to the brain—neural.

Sound Transmission through the Outer Ear
Air transmitted sound waves are directed toward the delicate hearing mechanisms with the help of the outer ear, first by the pinna, which gently funnels sound waves into the ear canal, then by the ear canal.

Sound Transmission through the Middle Ear
When air movement strikes the tympanic membrane, the tympanic membrane or eardrum moves. At this point, the energy generated through a sound wave is transferred from a medium of air to that which is solid in the middle ear. The ossicular chain of the middle ear connects to the eardrum via the malleus, so that any motion of the eardrum sets the three little bones of the ossicular chain into motion. Middle ear plays a vital role in impedance matching facilitating transmission of sound from the air filled external auditory canal to the fluid filled inner ear cavity.

Sound Transmission through the Inner Ear
The ossicular chain transfers energy from a solid medium to the fluid medium of the inner ear via the stapes. The stapes is attached to the oval window. Movement of the oval window creates motion in the cochlear fluid and along the Basilar membrane. Motion along the basilar membrane excites frequency specific areas of the Organ of Corti, which in turn stimulates a series of nerve endings.

Sound Transmission to the Brain
With the initiation of the nerve impulses, another change in medium occurs: from fluid to neural. Nerve impulses are relayed through the VIII C.N., through various nuclei along the auditory pathway to areas to the brain. It is the brain that interprets the neural impulses and creates a thought, picture, or other recognized symbol.

6. Optokinetic nystagmus

Optokinetic nystagmus, or OKN for short, is the eye movement elicited by the tracking of a moving field. It differs from smooth pursuit which is the eye movement elicited by tracking of a single distinct target. As moving fields contain within them distinct targets, OKN generally contains within it smooth pursuit. As a consequence, usually OKN performance (gain -- ratio of eye tracking velocity to target velocity), exceeds that of smooth pursuit.

An optokinetic stimulus should present moving targets in both the central and peripheral visual fields. There are several methods of doing this:

light bar
stripe projector
LCD projector or large screen array
physical drum
The stimulus that most closely matches the definition of OKN is to use a full field surround such as is shown above and below. Even these are compromises as individual bars can be tracked, and there are usually distinct objects in the visual field that can be fixated. Accordingly, the "best" OKN stimulus is real, physical surround that rotates, containing a pattern which has no distinguishing features such as a random dot pattern.

Normally, people undergoing OKN testing adopt an eye position that is opposite in direction to the velocity of the OKN drum -- this is called shift of the "beating field". This is presumably adaptive as it allows people to "ride" their gaze evoked nystagmus as well as potentially have a longer period to track. When there are diseases of the fast phases, often beginning with slow saccades, the eye doesn't shift into the "beating field", but rather gets "hung up" in the orbit. This is seen in disorders such as PSP and some of the brainstem related cerebellar degenerations.

This reflex is abnormal in patients with congenital nystagmus. One may observe a paradoxical reversal of the optokinetic nystagmus response.

Patients with horizontal nystagmus with unilateral hemispheric lesions, especially parietal or parietal-occipital lesions, show impaired optokinetic nystagmus when the drum is rotated toward the side of the lesion.

The OKN drum may be used as an estimate of visual acuity. The striped drum is equivalent to a vision of counting fingers when held at a distance of 3-5 feet from the patient. The further the drum is from the patient, the better the visual acuity must be to respond normally to the moving drum.

7. Cerumen

This is also known as ear wax. This is a yellowish waxy substance secreted in the external auditory canal of humans. It protects the skin lining of the external auditory canal from excessive moisture. It also protects the external canal from bacteria, fungi and insects.

Humans are known to secrete two types of cerumen:

Soft and moist
Firm and dry
Persons secreting soft and moist type of ear wax have no problem due to its accumulation. It can easily be extruded by the normal cleansing mechanism of the external auditory canal. This difference in wax secretion has been traced to alterations in C11 gene. Persons secreting frim and dry wax are more prone for impaction of cerumen. Impaction of cerumen causes conductive hearing loss.

Cerumen is usually produced in the outer third of the cartiagenous portion of the external auditory canal. It is composed of:

Viscous secretions from sebaceous glands
Less viscous secretions from modified apocrine sweat glands
Shed layers of skin
Cerumen has been found to have bacterostatic effect. Excessive occlusion of the external canal due to accumulation of cerumen and desquamated epithelial cells associated with migration defect of the lining epithelium can cause keratosis obturans. This is a painful condition which needs to be treated by removing the mass under anesthesia.

Removal of cerumen can be performed using probes / curettes if the consistency is soft. If cerumen is excessively soft then cotton buds can be used for removal.

Firm cerumen should be lubricated by using ceruminolytics / liquid paraffin to soften it up before attempted removal.

Aural syringing is one of the painless way of removing accumulated cerumen.

8. Furuncle of Ear

It is also known as acute localised otitis externa / circumscribed otitis externa.

This is a localised infection usually found to involve the lateral 1/3 of the external canal. It also has a propensity to involve the posterior superior aspect of the external canal. This is caused due to obstruction of the apopilosebaceous units found extensively in this area.

Etiology:

Trauma to skin in this area followed by infection is commonly attributed cause. The organism responsible is commonly staph aureus.

Symptoms:

1. Localised pain

2. Localised itching

3. Purulent discharge if the abscess ruptures

4. If oedema or abscess occludes the external canal hearing loss can occur.

Signs:

1. Erythema of the skin

2. Localised abscess formation

Management:

If the abscess is pointing it can be treated by incision and drainage. Oral antibiotics should be used. The preferred drug of choice is penicillin of first generation cephalosporins. Anti inflammatory drugs can be used to reduce inflammation and pain.

These patients must be advised to cut their nails short and to keep their hands clean, since this is the commonest route of infection.

9. Allergic Otitis externa

External auditory canal is lined by skin. Allergic reaction are known to affect skin lining anywhere in the body. The skin lining of external auditory canal is no exception.

These patients manifest with intense itching of the skin lining of the external canal. It causes diffuse weeping otitis externa. Patient's attempt to scratch the skin with ear buds could lead to secondary infection of the skin lining.

Common allergens include medicated ear drops which is used by the patient.

Management:

Systemic antihistamines will reduce itching of the ear canal. Steroid ointment impregnated cotton wick can be introduced into the external auditory canal to provide relief.

10. Malignant Otitis externa

Malignant otitis externa is a inflammatory disorder involving the external auditory canal caused by pseudomonas organism. Majority of these patients are elderly diabetics. This condition is termed as malignant otitis externa because of its propensity to cause complications. Hence the term malignant must not be construed in a histological sense.

History:

1838 - Toulmousch reported the first case of otitis externa
1959 - Meltzer reported a case of pseudomonas osteomyelitis of temporal bone
1968 - Chandler discussed the various clinical features and described it as a distinct clinical entity

The effectiveness of present-day antibiotics in the management of this condition should provoke the physicians to abandon the term malignant while describing this condition.

Epidemiology:
The typical patient with malignant otitis externa is an elderly diabetic, with males outnumbering females by twice the number. This could be due to the possibility of males being more prone to secrete wax which are more acidic in nature. Malignant otitis externa is very rare in children, if present it will be associated with malnutrition or HIV infection.

Pathophysiology:

Malignant otitis externa is known to affect the external auditory canal and temporal bone. The causative organism being pseudomonas aeruginosa. These patients are invariably elderly diabetics. This disorder usually begins as otitis externa and progresses to involve the temporal bone. Spread of this disease occurs through the fissures of Santorini and osteo cartiagenous junction. This

disorder could be caused by a combination of poor immune response and peculiar characteristics of the offending microbe.
Immunity is reduced in patients with :
1. Diabetis mellitus
2. Blood cancer
3. HIV infections
4. Patients on anticancer drugs

It should also be remembered that diabetic patients have impaired phagocytosis, poor leukocytic response, and impaired intracellular digestion of bacteria. Diabetic patients secrete wax which has less lysozyme content than normal thereby reducing the effectiveness of wax as an antimicrobial agent.

Pseudomonas aeruginosa is a gram-negative aerobe with polar flagella. It is found on the skin. It invariably behaves like an opportunistic pathogen. The pathogenicity of this organism is due to ability to secrete exotoxin and various enzymes like lecithinase, lipase, esterase, protease etc. Since this organism is clothed by a mucoid layer it is resistant to digestion by macrophages.

Clinical features:
The patient gives history of trivial trauma to the ear often by ear buds, followed by pain and swelling involving the external auditory canal. Pain is often the common initial presentation. It is often severe, throbbing and worse during nights. It needs increasing doses of analgesics. On examination granulation tissue may be seen occupying the external canal. It often begins at the bony cartiagenous junction of the external canal. Discharge emanating from the external canal is scanty and foul smelling in nature. When the discharge is foul smelling it indicates the onset of osteomyelitis. Ironically the patient does not have fever or other constitutional symptoms.

Otoscopy: Reveals granulation tissue at the bony cartiagenous junction. The ear drum is usually normal. The external auditory canal skin is soggy and edematous.
Cranial nerve palsies are common when the disease affects the skull base. The facial nerve is the most common nerve affected. As the disease progresses the lower three cranial nerves are affected close to the jugular foramen.
Intracranial complications like meningitis and brain abscess are also known to occur.

Spread of infection:
1. Inferiorly through the stylomastoid foramen to involve the facial nerve.
2. Anteriorly to the parotid
3. Posteriorly to the mastoid and sigmoid sinus
4. Superiorly to the meninges and brain
5. Medially to the sphenoid
6. Spread through vascular channels are also common

Role of imaging:

* Conventional radiology is of no use.
*
* CT scan is useful in assessing bone destruction.
*
* MRI is useful in assessing soft tissue involvement.
*
* Radionucleotide scans with Technetium 99 helps in assessing bone involvement

Imaging alogrithm in these patients are:
1. TC99 scan to seek evidence of bone involvement
2. If this is positive CT scan and MRI scan is a must to rule out bone and soft tissue involvement
3. Serial Ga 67 scans to assess the efficacy of treatment modality.

Levenson's criteria for diagnosis of malignant otitis externa:

* Refractory otitis externa
*
* Severe nocturnal otalgia
*
* Purulent otorrhoea
*
* Granulation tissue in the external canal
*
* Growth of Pseudomonas aeruginosa from external canal
*
* Presence of diabetes and other immunocompromised state

Treatment:

Extensive surgical procedures have failed miserably to cure this condition. The role of surgery is confined to only exclusion of malignancy by biopsy. Wound debridement is a possibility in advanced cases.

Medical management:

Carbenicillin, Pipercillin, Ticarcillin can be used. Third and fourth generation cephalosporins can be used.
Ciprofloxacillin in doses of 1.5 g - 2.5 g /day in divided doses can be administered for a period of 2 weeks.
Gentamycin can also be administered parenterally in doses of 80 mg iv two times a day in adults.

11. Keratosis obturans

Keratosis obturans: is accumulation of desquamated keratin in the external auditory meatus. This should be differentiated from primary auditory canal cholesteatoma which is characterized by invasion of squamous tissue from the external ear canal into a localized area of bone erosion.

Pathology: The keratin plug seen in keratosis obturans appears like a geometrically patterned keratin plug within the lumen of expanded ear canal. These keratin squames are shed from the complete circumference of the deep ear canal forming a lamina. It appears like onion skin.

Etiology: Keratosis obturans is postulated to occur due to abnormal epithelial migration of ear canal skin. The movement of the surface epithelium appears to be reversed in these patients. (The surface epithelium over pars flaccida migrates downwards to the pars tensa and then moves inferiorly across the drum).

Keratosis tympanicum: Is also caused by abnormal migration of squamous epithelium lining the deep portion of the external auditory canal. This condition is also associated with unilateral tinnitus.

Types of keratosis obturans:

a. Inflammatory type: This is caused due to acute inflammation involving the external ear canal. Viral infections commonly cause this problem. The inflammatory reaction involving the ear canal temporarily alters epithelial migration. This condition can only be cured by removal

b. Silent type: In this type there is no predisposing acute infections involved. This condition is postulated to be caused by abnormal separation keratin that persists even after the removal, and will need repeated removals.

c. Primary auditory canal cholesteatoma: Etiology is uncertain. It is commonly thought to be caused by trauma to the bone covering the external canal. This could also be caused by surgical trauma as in patients who have undergone stapedectomy. The piece of exposed bone in the external canal becomes infected and sequests. The lining epithelium migrates into this area causing the formation of cholesteatoma. This condition is characterized by ear pain which is dull and aching in nature. It is not associated with hearing impairment.

Keratosis obturans commonly occur in young patients.

Clinical features:

1. Severe ear pain

2. Mild / moderate conductive hearing loss

3. Associated bronchitis / sinusitis - common

On examination:

The ear canal appears to be widened, making the ear drum stand out. CT scan of temporal bones may reveal canal erosion and widening.

After surgical removal under general anesthesia the specimen must be sent for pathological evaluation to rule out malignancy.

Management:

1. Surgical removal under G.A.

2. Canal plasty is helpful in recurrent cases

3. Mastoidectomy should be performed in cases with primary cholesteatoma of external canal

12. Swimmer's Ear

This is also known as the swimmers ear. This is an inflammatory condition involving the external canal in a diffuse manner. This condition is common in swimmers because of the propensity for the external canal to be exposed to water for long durations. This exposure leads to maceration of the external canal skin, and also lowers the pH of the external canal providing an environment favorable to infections.

Main symptoms:

1. Itching in the external canal

2. Tenderness on palpation

3. Aural fullness rarely occur due to the reduction in size of the external canal lumen due to oedema

4. Rarely stenosis of the external canal may occur causing accumulation of debris and secretions

Common signs:

1. Erythema of the external canal

2. Oedema of external canal

3. Secretions from the external canal (weeping canal)

4. Pain on mastication

5. Pulling of helix in a postero superior direction cause pain

6. In advanced cases fever and lymphadenopathy may occur (pre and post auricular nodes may be involved)

Stages of acute diffuse otitis externa: (Senturia)

Preinflammatory stage: is characterized by intense itching, edema and sensation of fullness in the ear.

Inflammatory stage: may be divided into mild, moderate and severe.

Mild acute inflammatory stage: here the cardinal features are increased itching, pain, mild erythema and oedema of the external canal skin. At later stages exfoliation of skin with minimal amount of cloudy secretions may be seen in the external canal.

Moderate acute inflammatory stage: in this stage the itching and tenderness of the external canal intensifies. The external canal is narrowed due to oedema and accumulation of epithelial debris.

Severe acute inflammatory type: In this stage pain becomes intolerable to such an extent the patient may refuse to eat, the lumen of the external canal becomes totally obliterated due to oedema and accumulated epithelial debris. Otorrhoea may become purulent. In addition, regional nodes may also be involved. Infections from the external canal may involve the parotid gland via the fissures of Santorini.

Common organisms involved: Pseudomonas aeruginosa and staphylococcus aureus are commonly cultured from the external canal of these patients. The normal commensals like staphylococcus epidermidis and corynebacteria are conspicuously absent.

Management:

The aim is twofold:

1. Resolving the infection

2. Promoting the external canal skin's recovery to its original state.

Firstly the canal is cleaned atraumatically by gentle suctioning and debridement under microscope. Topical hydrogen peroxide solution instilled will help the process of debridement.

A cotton wick dipped in I.G. paint can be inserted in to the external canal and allowed to stay for a day. This will reduce the external canal skin oedema and will increase the size of the meatus. Ear drops containing a mixture of neomycin and 1% hydrocortisone may be instilled as ear drops at least three times a day. In addition to the antibiotic and anti-inflammatory effects this drug reduces the pH of the external canal making it more resistant to the organisms.

In severe cases oral antibiotics and anti-inflammatory drugs can be resorted to. Quinolones are commonly used oral antibiotic.

13. Otomycosis

Otomycosis: is superficial mycotic infection of skin lining the external auditory canal. This commonly occurs in humid seasons.

Otomycosis can be classified into primary and secondary otomycosis.

Primary otomycosis: Commonly occurs during humid conditions when there is excessive moisture content in the atmosphere. This excessive moisture makes the external canal skin soggy and oedematous. This predisposes to fungal infections.

Secondary otomycosis: Occurs in immunocompromised individuals and in persons who have preexisting CSOM. Patients with CSOM usually apply broad spectrum antibiotic ear drops. This ear drops not only kills pathogens but also the natural commensals causing secondary fungal infections.

Common fungal species involved:

1. Candida albicans

2. Candida tropicalis

3. Aspergillus niger

4. Aspergillus fumigatus

Of these fungi candida infections cause whitish wet plaques within the ear canal. The plaques may also appear leathery. The aspergillus niger appear as black plaques in the external auditory canal.

Clinical features:

1. Intense itching of external canal

2. Inflammation and scaling of external canal skin

3. White / black plaques seen depending on the type of fungal infection

4. Intense pain in the ear

Treatment:

1. Dry mopping to remove plaques

2. Antifungal ear drops

3. Anti inflammatory drugs in case of acute inflammation

14. Acute otitis media

Definition:

Acute suppurative otitis media is defined as suppurative infection involving the mucosa of the middle ear cleft. By convention it is termed acute if the infection is less than 3 weeks in duration.

Pathophysiology:

Obstruction to the eustachean tube seem to be the most important antecedent event in the pathophysiology of acute suppurative otitis media. Majority of acute suppurative otitis media is triggered by upper respiratory infections which might find its way into the middle ear cavity through the eustachean tube orifice. Infections involving the nasopharynx may find its way into the middle ear through the pharyngeal end of eustachean tube. The infection is initially commonly viral in origin, allergy could also play an important role in the pathogenesis. Later the middle ear mucosa

becomes secondarily infected by pathogenic bacteria. The bacteria commonly implicated in this disorder is S Pneumoniae, H. Influenza, and M Catarrhalis.

The majority of otitis media prone children have a patulous eustachean tube or an hypotonic eustachean tube. Children with neuromuscular disorders or with abnormalities of the first or second arch have a patulous eustachean tube leading on to this problem. To become pathogenic the bacteria must become adherent to the mucosa lining the middle ear cavity, this is made possible by prior infection of the middle ear mucosa by viruses.

Flask model explaining the role of eustachean tube in middle ear infections:

The eustachean tube, middle ear, and mastoid air cell system can be likened to a flask with a long narrow neck. The mouth of the flask represents the nasopharyngeal end, the narrow neck, the isthmus of the eustachean tube, and the bulbous portion, the middle ear and mastoid air chamber. The fluid flow through the neck of the flask would be dependent on the pressure at either end, the radius and length of the neck, and the viscosity of the liquid. When a small amount of liquid is instilled into the mouth of the flask, liquid flow stops somewhere in the narrow neck owing to capillarity within the neck and the relative positive air pressure that develops in the chamber of the flask.

The basic geometry is considered to be critical for the protective function of the eustachean tube - middle ear system. Reflux of liquid into the body of the flask occurs if the neck of the flask is excessively wide, or the length of the neck of the flask is too short as seen in children. Because infants have a shorter eustachean tube than adults, reflux is more likely to occur in the baby. The position of the flask in relation to the liquid is another important factor. In humans, the supine position enhances flow of liquid into the middle ear; thus, infants might be at risk for developing reflux otitis media because they are commonly supine. Reflux of liquid into the vessel can also occur if a hole is made in the bulbous portion of the flask, because this prevents the creation of positive pressure in the bulbous portion. This positive pressure is useful in the prevention of reflux of material from the neck of the flask.

If negative pressure is applied to the bulbous portion of the flask then this pressure is sufficient to cause aspiration of contents from the neck of the flask. This scenario is represented by high negative pressure in middle ear as it occurs in nose blowing, crying, closed nose swallowing, diving or airplane descent. The neck of the eustachean tube is supposed to be compliant hence compliance plays a vital role in prevention of reflux of secretions.

Clinical features:

Acute suppurative otitis media passes through 4 stages:

1. Stage of hyperemia

2. Stage of exudation

3. Stage of suppuration

4. Stage of resolution.

The progression of these stages depends on the virulence of the infecting organisms, resistance of the host, adequacy of antibiotic therapy. If the infecting organism is virulent or if the antibiotic treatment is not sufficient then the disease may progress to a stage of coalescent mastoiditis with its attendant complications.

Stage of hyperemia:

Initial infection by infection results in hyperemia of the mucous membrane causing otalgia, fever and fullness in the affected ear. This stage is characterised by oedema of the mucoperiosteum due to vascular engorgement. Otoscopy show dilated vessels along the handle of malleus and along the rim of the tympanic membrane. Antibiotic therapy during this stage will help in resolution of the disease. Amoxycillin is the drug of choice.

Stage of exudation:

Absence of treatment during the stage of hyperemia leads to the stage of exudation. In this stage there is outpouring of fluid from the dilated vessels of the mucoperiosteum. This fluid is serous in nature containing fibrin, red cells, and polymorphs. This exudate fills the tympanomastoid compartment really fast, and the whole middle ear cavity is under intense pressure due to this retained secretion. Pain is the most prominent feature of this stage. The patients may have fever and fullness in the ear. Otoscopy shows a bulging ear drum with loss of all landmarks. The drum is reddish and bulging in nature. These patients have also coexistent mastoid tenderness due to mastoiditis.

Stage of suppuration:

Failure of treatment during the stage of exudation leads on to stage of suppuration. The exudate present in the middle ear cavity is a very good culture medium and hence there is secondary bacterial infection leading on to suppuration.

Stage of resolution:

is preceded by either rupture of the ear drum leading on to a serous / serosanguinous / purulent disharge from the ear. When the middle ear is free from the exudate / pus the stage of resolution

sets in. The patient has reduction in otalgia, fever subsides. The patient has considerable clinical improvement.

Stage of complication:

If the infection persists beyond a period of 2 weeks then there is associated thickening of the mucoperiosteum especially in the air cells around the peri antral area leading to a block in the drainage from the antral cells. The pent up secretions in the mastoid air cell system causes intense pressure, venous stasis and local acidosis. This acidosis cause dissolution of calcium from the bone causing decalcification and coalescence of the mastoid air cell system. This condition is known as coalescent mastoiditis. This stage is characteristic by emergence of otalgia and low grade fever. Erosion of the outer cortex in the mastoid lead to the formation of abscess under the periosteum of the mastoid cortex. This condition is known as subperiosteal abscess.

Management:

Acute suppurative otitis media is a self-limiting condition. If appropriate antibiotics is started early then it resolves. Amoxycillin is the drug of choice. Cephalosporins may also be started in refractive cases. Anti-inflammatory drugs like ibuprofen is also prescribed in order to alleviate pain. Patients who are refractory to medical management may undergo myringotomy in order to decompress the middle ear cavity. This procedure is done using a myringotome.

Coalescent otitis media and subperiosteal abscess are surgical complications. These patients must be taken up for surgery under adequate antibiotic cover.

15. Secretory otitis media/ Glue ear

Synonyms:

Secretory otitis media, glue ear, serous otitis media, non-purulent otitis media.

Definition:

Otitis media with effusion is defined as chronic accumulation of mucus within the middle ear, and rarely this could involve the mastoid air cell system. This accumulation causes conductive hearing loss.

Histology and histopathology of eustachean tube:

The pseudostratified ciliated columnar epithelium of respiratory tract extends up the eustachean tube as far as the anterior part of the middle ear cavity. These cells are capable of producing mucous. There are also goblet cells seen in their midst. These cells are also capable of secreting mucous material. Otitis media with effusion is caused by inflammation of this epithelium in the

eustachean tube and hypotympanum. In established cases of glue ear, the cuboidal epithelium of middle ear and mastoid air cells gets replaced by thickened pseudostratified columnar epithelium. The cilia of these cells have also been found to be ineffective in propelling the secretions into the nasopharynx. The submucosa is found to be oedematous, inflamed with dilated blood vessels with increased number of macrophages and plasma cells.

Etiology:

1. In many children otitis media with effusion is preceded by an episode of acute otitis media. This is common in children who is more prone for upper respiratory infections. Common being viral infections which damages the eustachean tube epithelium.

2. Craniofacial abnormalities: Children with cleft palate have deficient palatal muscles causing a poor eustachean tube function leading on to Otitis media with effusion. This occurs despite a successful surgical repair of the cleft palate. Children with Down's syndrome are also more prone for OME.

Note: Children with bifid uvula do not appear to have higher incidence of OME

3. Allergy: Previously nasal allergy has been postulated as an important factor in the development of Otitis media with effusion. Studies have been unequivocal.

4. Gastroesophageal reflux: GERDS has been commonly demonstrated radiologically in children with OME. Furthermore, biochemical analysis of middle ear fluid has demonstrated significant amounts of pepsin (in 80% of cases).

5. Parental smoking has been attributed as an important predisposing factor for the development of OME.

Age of occurrence: OME shows classically a bimodal distribution. The first peak occurs around 2 years of age, and the second peak occurs at about 5 years of age. This distribution occurs roughly around the ages when the child goes to preschool and primary school.

Seasonal association: OME commonly occurs during winter season, when there is more likelihood of upper respiratory infections, and also because of the possibility of closer contact with affected children. This is seen in temperate zones. In non-temperate zones it is commonly seen during rainy season.

Clinical features:

A high index of suspicion is necessary to identify this condition. Every child with upper respiratory infection must be otoscopically examined.

Otoscopic findings: The tympanic membrane may be bulging, or retracted with a distorted cone of light. The ear drum may appear yellow, blue or simply clear white. Pneumatic otoscopy will reveal a ear drum which has a restricted mobility.

Microbiology of OME: Commonly middle ear effusions due to glue ear is sterile. Rarely bacteria could be cultured. The incidence of these pathogens are higher in children under the age of 2, and in children with recurrent upper respiratory infections.

Investigations:

Puretone audiometry: Demonstrates mild to moderate conductive deafness.

Tympanograms (Type B) is commonly associated with OME. Type A is infrequently associated while Type C falls somewhere in between. Tympanometry can be used as a screening test to identify patients with OME.

Free field audiometry: Demonstrates deafness.

Management:

1. Antibiotics: Amoxycillin is the drug of choice followed by cephalosporins.

2. Nasal decongestants like oxymetazoline / xylometazoline may help in some cases.

3. Topical nasal steroids can be used in resistant cases.

4. Auto inflation of eustachean tube by performing valsalva maneuver. Balloon blowing may also help.

Surgical management:

1. Adenotonsillectomy

2. Myringotomy and insertion of ventilation tubes

16. Management of Secretory Otitis Media.

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2. Myringotomy and insertion of ventilation tubes

17. Myringotomy

Myringotomy is a surgical procedure of the eardrum or tympanic membrane. The procedure is performed by making a small incision with a myringotomy knife through the layers of tympanic membrane (see the image below). This surgical procedure permits direct access to the middle ear space and allows the release of middle-ear fluid, which is the end product of otitis media with effusion (OME), whether acute or chronic. OME is classified as serous, mucoid, or purulent.

The fluid is suctioned from the middle ear through the incision and, if indicated, sent for bacterial or viral cultures. Currently, bilateral myringotomy is often used in conjunction with placement of middle-ear ventilation tubes, which permits the incised drum to remain open and allows better drainage of middle-ear fluid. This approach facilitates instillation of antibiotic otitic drops, and ultimately results in faster resolution of the OME.

Myringotomy is performed using a myringotomy knife. A radial incision is given in the antero inferior quadrant of the ear drum.

This surgical procedure can be performed either under local / general anesthesia

18. Cholesteatoma

Definition of cholesteatoma: Cholesteatoma is defined as a cystic bag like structure lined by stratified squamous epithelium on a fibrous matrix. This sac contains desquamated squamous epithelium. This sac is present in the attic region. Cholesteatoma is also defined as 'skin in wrong place'. Cholesteatoma is known to contain all the layers of skin epithelium. The basal layer (germinating layer) is present on the outer surface of cholesteatoma sac in contact with the walls of the middle ear cleft.

Theories of bone invasion by cholesteatoma:

1. Pressure theory - states that increase in the pressure caused by enlarging cholesteatoma cause bone erosion. Ischemia has been attributed as the cause in this theory.

2. Enzymatic theory: Inside the cholesteatoma are present multinucleated osteoclasts and histiocytes. These cells release acid phosphatase, collagenase and other proteolytic enzymes. These enzymes are known to cause bone erosion.

3. Pyogenic osteitis: Pyogenic bacteria may release enzymes which could cause bone resorption.

Types of cholesteatoma:

1. Congenital cholesteatoma

2. Primary acquired cholesteatoma

3. Secondary acquired cholesteatoma

Congenital cholesteatoma: is known to arise from embryonic cell rests present in the middle ear cavity and temporal bone. These cell rests are known to commonly occur in cerebello pontine angle and petrous apex. Infact congenital cholesteatoma is seen as a whitish mass behind an intact tympanic membrane.

Derlacki and Clemis laid down the following as criteria to diagnose congenital cholesteatoma:

1. The patient should not have previous episodes of middle ear disease

2. Ear drum must be intact and normal

3. It is purely an incidental finding

4. If discharge and ear drum perforation is present then it should be construed that congenital cholesteatoma has managed to erode the tympanic membrane.

Clinical features: The disorder is an incidental finding. The common location of congenital cholesteatoma is the antero superior quadrant of tympanic membrane, postero superior quadrant being the next common site of involvement. Anteriorly situated congenital cholesteatomas are known to affect the eustachean tube function causing conductive deafness due to middle ear effusion, where as posterior congenital cholesteatoma is known to cause conductive deafness due to impairment of ossicular chain mobility.

Staging of congenital cholesteatoma:

Staging as suggested by Derlacki and Clemis: They were the first to stage congenital cholesteatoma. They classified congenital cholesteatoma into

1. Petrous pyramid cholesteatoma

2. Cholesteatoma involving the mastoid cavity

3. Cholesteatoma involving the middle ear cavity.

Potsic suggested the following staging mechanism:

Stage I : Single quadrant involvement with no ossicular / mastoid involvement.

Stage II : Multiple quadrant involvement with no ossicular / mastoid involvement

Stage III : Ossicular involvement without mastoid involvement

Stage IV : Mastoid extension

Nelson's staging:

Type I : Involvement of mesotympanum without involvement of incus / stapes

Type II : Involvement of mesotympanum / attic along with erosion of ossicles without extension into the mastoid cavity

Type III : Involvement of mesotympanum with mastoid extension

Staging this disease will help in deciding the modality of treatment and in predicting the long term prognosis.

Acquired Cholesteatoma: can be divided into two types, primary acquired and secondary acquired cholesteatomas.

Primary acquired cholesteatoma: In this condition there is no history of preexisting or previous episodes of otitis media or perforation. Lesions just arise from the attic region of the middle ear.

Secondary acquired cholesteatoma: always follows active middle ear infection which manages to destroy the ear drum along with the annulus. This type of destruction is common in acute necrotizing otitis media following exanthematous fevers like measles etc.

Theories to explain pathogenesis of cholesteatoma:

Various theories have been postulated to explain the pathogenesis of cholesteatoma. They are:

1. Cawthrone theory: This theory suggested by cawthrone in 1963 suggested that cholesteatoma always originated from congenital embryonic cell rests present in various areas of the temporal bone.

2. Theory of immigration: This theory was suggested by Tumarkin. He was of the view that cholesteatoma was derived by immigration of squamous epithelium from the deep portion of the external auditory canal into the middle ear cleft through a marginal perforation or a total perforation of the ear drum as seen in acute necrotizing otitis media.

3. Theory of invagination: This theory was suggested by Toss. He theorized that persistent negative pressure in the attic region causes invagination of pars flaccida causing a retraction pocket. This retraction pocket becomes later filled with desquamated epithelial debris which forms a nidus for the infection to occur later. Common organisms known to infect this keratin debris are Pseudomonas, E. coli, B. Proteus etc.

Toss also classified attic retraction pockets into 4 grades:

1. Grade I: The retracted pars flaccida is not in contact with the neck of the malleus.

2. Grade II: The retracted pars flaccida is in contact with the neck of the malleus to such an extent that it seems to clothe the neck of the malleus.

3. Grade III: Here in addition to the retracted pars flaccida being in contact with the neck of the malleus there is also a limited erosion of the outer attic wall or scutum.

4. Grade IV: In this grade in addition to all the above said changes there is severe erosion of the outer attic wall or scutum.

4. Metaplastic theory: This theory was first suggested by Wendt in 1873. He took into consideration the histological changes seen in various portions of the middle ear cavity. The attic area of the middle ear cavity is lined by pavement type of epithelium. This epithelium undergoes metaplastic changes in response to subclinical infection. This metaplastic mucosa is squamous in nature there by forming a nidus for cholesteatoma formation in the attic region.

Of all the above-mentioned theories, the theory of invagination appears to be the most plausible one currently explaining the various pathologic features of cholesteatoma.

Clinical features of acquired cholesteatoma:

Ear discharge: is scanty and foul smelling. In fact the odor is best described as musty in nature. This is due to the presence of saprophytic infection and osteitis.

Hearing loss: is commonly conductive in nature. Some patients may even surprisingly have a normal hearing despite the presence of a huge cholesteatoma. This normal hearing could be attributed to the bridging effects of cholesteatomatous mass.

Sensorineural hearing loss if present could be attributed to the absorption of toxins through the round window membrane, or may be due to use of ototoxic antibiotics topically on a long term basis.

Ear ache: if present could be attributed to the presence of co-existing otitis externa, or presence of extradural abscess.

Tinnitus if present may indicate imminent sensorineural hearing loss.

Vertigo may be present if there is erosion of lateral semicircular canal by the cholesteatomatous matrix. Fistula test if performed is positive in these patients.

Fistula test: This test is positive if there is a third window is present in the labyrinth due to the erosion of the labyrinthine bone. This commonly occurs in the lateral semicircular canal area. This test is performed using a snugly fitting siegles pneumatic speculum and slowly applying pressure by compressing the pneumatic bulb. If labyrinthine fistula is present the patient will feel giddy and will have nystagmus.

Facial palsy may indicate erosion of facial nerve canal with involvement of facial nerve.

On examination:

There is destruction of the outer attic wall, with presence of attic perforation. Cholesteatomatous flakes may be seen through the perforation like cotton wool.

There is associated sagging of the posterior superior meatal wall.

Hearing tests indicate conductive deafness commonly if labyrinth is uninvolved. It may turn out to be sensorineural hearing loss if there is associated erosion of the labyrinth.

X ray mastoids may show sclerosis with presence of cavity.

Management:

Since this is a surgical problem modified radical mastoidectomy is advocated in almost all of these patients.

The aims of the surgical procedure is as follows:

1. To exteriorize the disease

2. To create adequate ventilation to the middle ear cavity

3. To create a permanent skin lined cavity exposed to the exterior.

19. Management of Cholesteatoma

Management:

Since this is a surgical problem modified radical mastoidectomy is advocated in almost all of these patients.

The aims of the surgical procedure is as follows:

1. To exteriorize the disease

2. To create adequate ventilation to the middle ear cavity

3. To create a permanent skin lined cavity exposed to the exterior.

Limited disease where the entire neck of the sac is wide the contents of the sac can be evacuated off its contents by suction clearance. This should be done periodically in order to prevent accumulation of squamous debris within the sac.

Atticotomy can be performed in patients with cholesteatoma limited only to the attic region. In this procedure the outer attic wall is removed and the attic entered via the widened aditus and is cleared of the cholesteatoma debris.

20. Extra cranial complications of chronic suppurative otitis media

The following are extracranial complications of CSOM:

Subperiosteal abscess:

Also known as Luc's abscess after Henri Luc who first described this condition. This is a collection of pus in the subperiosteal region of the mastoid cavity. This condition is commonly seen in Paediatric age group.

This condition is commonly seen in well pneumatized mastoid bones following an episode of coalescent mastoiditis where in the entire mastoid air cell system is inflamed and suppurates. Pus begins to accumulate in the subperiosteal plane causing swelling, pain and tenderness over the mastoid bone.

Clinically the child with subperiosteal abscess is ill, with swelling behind the ear. This swelling pushes the pinna forwards.

This condition can also occur in children with extensive cholesteatoma.

Management:

Incision and drainage which is followed by mastoidectomy as a second sitting procedure.

Labyrinthitis:

Introduction: This is defined as an inflammatory disorder involving the inner ear / labyrinth. Clinically this condition causes disturbances of balance and hearing of varying degrees in the involved ear.
Causes:
1. Bacterial infections
2. Viral infections
3. Autoimmune causes
4. Vascular ischemic causes
Pathophysiology:

Anatomically labyrinth is composed of an outer osseous framework surrounding the delicate membranous labyrinth which contains the peripheral end organs of hearing and balance.
Membranous labyrinth include:
1. Utricle
2. Saccule
3. Semicircular canals
4. Cochlea
The labyrinth lies within the petrous portion of temporal bone. It communicates with the middle ear via the oval and round windows.

Infecting organism may find their way into the inner ear via:

Pre-existing fractures
Oval window
Round window
Congenital dehiscence involving the bony labyrinth
Viral labyrinthitis: Is characterized by sudden unilateral loss of hearing and equilibrium. Vertigo is usually incapacitating and associated with vomiting. These patients are bed ridden. Vertigo usually subsides within 4-6 weeks. Hearing loss is confined to high frequencies and is sensorineural in nature. An attack of upper respiratory tract infection precedes the development of labyrinthitis. This condition should not be compared with vestibular neuronitis which involves only the vestibular nerves and spares the cochlear component. Varicella Zoster oticus is an unique form of viral labyrinthitis caused by reactivation of dormant varicella zoster virus. This reactivated virus is known to attack spiral ganglion.
Common viral causes of labyrinthitis:
1. Mumps
2. Measles
3. Rubella (congenital labyrinthitis)
4. Cytomegalovirus

Bacterial labyrinthitis: can be potentially caused by meningitis / otitis media. This could be caused by direct invasion of membranous labyrinth by the infecting organism (suppurative labyrinthitis) causing permanent destruction of vestibular and cochlear end organs. In patients with meningitis spread of infections can be bilateral since infections can travel via the CSF and involve the inner ear fluids through the internal acoustic meatus / cochlear aqueduct. Bacterial infections involving the middle ear cavity can enter the labyrinth via erosion of lateral canal which is commonly seen in patients with cholesteatoma. Treatment is usually directed against infecting organism and supportive therapy. Suppurative labyrinthitis is usually followed by labyrinthitis ossificans where the whole of the membranous labyrinth gets ossified. Labyrinthitis ossificans indicates a permanently dead labyrinth.

Common bacterial causes of labyrinthitis include:
1. S. pneumonia

2. Haemophilus influenza
3. Streptococcus
4. Staphylococcus
5. Neisseria
6. Bacteroids
7. Proteus
8. Moraxella catarrhalis

Serous labyrinthitis:

This is a potentially reversible disorder caused by diffusion of bacterial toxins into the inner ear via the inflamed round window membrane. Studies have shown that the permeability of the round window membrane is increased when there is inflammation. This may cause diffusion of bacterial toxins and immune mediators into the inner ear causing transient impairment of the inner ear functions.

Autoimmune labyrinthitis:

This is an uncommon cause of sensorineural hearing loss. This may be caused by localized / general autoimmune reactions. Examples of general autoimmune disorders causing labyrinthitis include Wegener's granulomatosis and polyarteritis nodosa.

Bezold's abscess:

Bezold Abscess is a rare deep neck abscess resulting from an intertemporal complication of a coalescent mastoiditis. It was first described by a German Otologist Dr. Friedrich Bezold in 1881. Since the introduction of antibiotics, the number of reported cases of Bezold abscesses have significantly decreased.

Many factors have been shown to play a role in the development of acute, chronic, and suppurative otitis media in children. Factors include the following:

Socioeconomic conditions: there is an increase in the number of the cases in children living in poorer socioeconomic conditions.

Daycare: there appears to be an increase in children attending day care.

Underlying medical conditions that affect the Eustachian tube function: children with cleft palate, craniofacial anomalies, congenital or acquired immune dysfunction, conditions affecting the ciliary function of the Eustachian tube and middle ear mucosa.

The most important contributing factor in the development of a Bezold abscess is the presence of a well-aerated and pneumatized mastoid bone. At birth, the mastoid bone consists mainly of an antral cell. From this antral cell, air cells begin to develop until there is complete pneumatization of the mastoid bone around the age of five. As the process of pneumatization of the mastoid bone occurs, the surrounding walls of the mastoid bone thin, particularly at the tip of the mastoid bone on its medial surface along the incisura digastrica (digastric groove) where the digastric muscle attaches. If the infection is left unchecked, the outer walls of the mastoid tip become involved with the spread of the infection along the sternocleidomastoid muscle, the trapezius, and splenius capitis muscles.

Secretions and bacteria can enter the middle ear through the eustachian tube and from the middle ear can pass directly into the mastoid bone through a small opening the aditus ad antrum. During an inflammatory process (suppurative otitis media), obstruction of the aditus ad antrum occurs. Purulent secretions cannot escape and will accumulate in the mastoid bone. The pressure created by this purulent material with its enzymatic activity within the mastoid bone causes osteitis and osteonecrosis of the fragile pneumatized air cells further thinning the walls of the mastoid bone. This stage is Coalescent Mastoiditis. The inflammatory process can spread in many directions. If it spreads inferiorly towards the mastoid tip, the purulent material will eventually erode the very thin bone along the digastric ridge at the insertion of the digastric muscle. Consequently, a purulent material will spread into the neck between the digastric and sternocleidomastoid muscles.

Zygomatic abscess:

Zygomatic root abscess is a rare extracranial otogenic complication. Atypical otogenic symptoms and lack of awareness are responsible for misdiagnosis.

Since the zygomatic root is in a relatively high position and usually drains well, there is rare chance for an abscess to develop. However, when purulent secretion extends to the highly pneumatized root of the zygomatic process, the pneumatized bone can be eroded and purulent secretion can accumulate within the temporalis fossa once there is penetration through the periosteum. Purulent secretion can even track from the zygomatic root into the cheek to form mumps of the cheek, which usually misleads doctors to make an incorrect diagnosis.

Improper use of antibiotics can mask the signs of otitis media and results in masked mastoiditis, which can also be responsible for the misdiagnosis of zygomatic root abscess. Cultures can guide proper selection of antibiotics. Mastoidectomy is necessary once conservative treatments are found to be ineffective. During the operation, purulent secretion and eroded bone should be removed, clearing the involved air cells of the zygomatic root as well as the mastoid process.

Facial nerve palsy:

This was a common complication of CSOM in the preantibiotic era. This is caused due to oedema of the facial nerve covering. This is seen in patients in whom facial nerve canal is dehiscent. Bacterial toxins and granulation tissue cause inflammation of perineurium causing the nerve to swell. Since it is enclosed inside a bony canal there is no space for expansion of the nerve. This causes block in nerve transmission.

Majority of these patients responded well to the treatment of CSOM which is the causative factor. If mastoid surgery is contemplated then facial nerve canal should be decompressed to allow space for the expansion of the inflamed nerve.

21. Labyrinthitis

Introduction: This is defined as an inflammatory disorder involving the inner ear / labyrinth. Clinically this condition causes disturbances of balance and hearing of varying degrees in the involved ear.

Causes:

1. Bacterial infections
2. Viral infections
3. Autoimmune causes
4. Vascular ischemic causes

Pathophysiology:

Anatomically labyrinth is composed of an outer osseous framework surrounding the delicate membranous labyrinth which contains the peripheral end organs of hearing and balance.

Membranous labyrinth includes:

1. Utricle
2. Saccule
3. Semicircular canals
4. Cochlea

The labyrinth lies within the petrous portion of temporal bone. It communicates with the middle ear via the oval and round windows.

Infecting organism may find their way into the inner ear via:

Pre-existing fractures
Oval window
Round window
Congenital dehiscence involving the bony labyrinth

Viral labyrinthitis: Is characterized by sudden unilateral loss of hearing and equilibrium. Vertigo is usually incapacitating and associated with vomiting. These patients are bed ridden. Vertigo usually subsides within 4-6 weeks. Hearing loss is confined to high frequencies and is sensorineural in nature. An attack of upper respiratory tract infection precedes the development of labyrinthitis. This condition should not be compared with vestibular neuronitis which involves only the vestibular nerves and spares the cochlear component. Varicella Zoster oticus is an unique form of viral labyrinthitis caused by reactivation of dormant varicella zoster virus. This reactivated virus is known to attack spiral ganglion.

Common viral causes of labyrinthitis:

1. Mumps
2. Measles
3. Rubella (congenital labyrinthitis)
4. Cytomegalovirus

Bacterial labyrinthitis: can be potentially caused by meningitis / otitis media. This could be caused by direct invasion of membranous labyrinth by the infecting organism (suppurative labyrinthitis)

causing permanent destruction of vestibular and cochlear end organs. In patients with meningitis spread of infections can be bilateral since infections can travel via the CSF and involve the inner ear fluids through the internal acoustic meatus / cochlear aqueduct. Bacterial infections involving the middle ear cavity can enter the labyrinth via erosion of lateral canal which is commonly seen in patients with cholesteatoma. Treatment is usually directed against infecting organism and supportive therapy. Suppurative labyrinthitis is usually followed by labyrinthitis ossificans where the whole of the membranous labyrinth gets ossified. Labyrinthitis ossificans indicates a permanently dead labyrinth.

Common bacterial causes of labyrinthitis include:
1. S. pnuemoniae
2. Haemophilus influenza
3. Streptococcus
4. Staphylococcus
5. Neisseria
6. Bacteroids
7. Proteus
8. Moraxella catarrhalis

Serous labyrinthitis:
This is a potentially reversible disorder caused by diffusion of bacterial toxins into the inner ear via the inflamed round window membrane. Studies have shown that the permeability of the round window membrane is increased when there is inflammation. This may cause diffusion of bacterial toxins and immune mediators into the inner ear causing transient impairment of the inner ear functions.

Autoimmune labyrinthitis:

This is an uncommon cause of sensorineural hearing loss. This may be caused by localized / general autoimmune reactions. Examples of general autoimmune disorders causing labyrinthitis include Wegener's granulomatosis and polyarteritis nodosa.

22. Lateral Sinus Thrombosis

Thrombophlebitis can develop in any of the veins adjacent to the middle ear cavity. Of these the lateral sinus, which comprise of the sigmoid and transverse sinuses is the largest and most commonly affected. Initially it is usually preceded by the development of an extradural perisinus abscess. The mural thrombus partly fills the sinus. The clot progressively expands and eventually occlude the lumen. The clot may later become organised, and partly broken down and may even be softened by suppuration. During this stage there is a release of infecting organism and infected material into the circulation causing bacteremia, septicemia and septic embolisation.

Extension / propagation of the thrombus upwards may extend to the confluence of the sinuses, and beyond that to the superior sagittal sinus. Invasion of the superior and inferior petrosal sinuses may cause the infection to spread to the cavernous sinus. This spread of venous thrombophlebitis into the brain substance accounts for the very high association of this complication with brain abscess. Downward progression of thrombus into and through the internal jugular vein can reach the subclavian vein.

The harmful effects are caused by the release of infective emboli into the circulation, and also from the haemodynamic disturbances caused to venous drainage from inside the cranial cavity. The use of antibiotics have greatly reduced the incidence of lateral sinus thrombosis these days.

Formerly it was commonly associated with acute otitis media in childhood; now it is commonly seen in patients with chronic ear disease. In the preantibiotic era the commonest infecting organism was beta hemolytic streptococci. This organism was known to cause extensive destruction of red blood cells causing anemia. Now a days the infection is by a mixed flora.

Clinical features:

These patients manifest with severe fever, wasting illness in association with middle ear infection. The fever is high and swinging in nature, when charted it gives an appearance of 'Picket fence'. It is always associated with rigors. The temperature rose rapidly from 39 - 40degree Centigrade. Headache is a common phenomenon, associated with neck pain. The patient appears emaciated and anaemic. When the clot extended down the internal jugular vein, it will be accompanied by perivenous inflammation, with tenderness along the course of the vein. This tenderness descended down the neck along with the clot, and would be accompanied by perivenous oedema or even suppuration of the jugular lymph nodes. Perivenous inflammation around jugular foramen can cause paralysis of the lower three cranial nerves. Raised intracranial pressure produce papilloedema and visual loss. Hydrocephalus could been added complication if the larger or the only lateral sinus is occluded by the thrombus, or if the clot reaches the superior sagittal sinus. Extension to the cavernous sinus can occur via the superior petrosal sinus, and may cause chemosis and proptosis of one eye. If circular sinus is involved it could spread to the other eye. The propagation of the infected emboli may cause infiltrates in the lung fields, and may also spread to joints and other subcutaneous tissues. These distant effects usually developed very late in the disease, these could be the presenting features if the disease is insidious in onset. Masking by antibiotics could be one of the causes. Patients always feel ill, and persisting fever is usual. The patients may have ear ache, in association with mastoid tenderness, and stiffness along the sternomastoid muscle. The presence of anaemia is rare now a days. Papilloedema is still a common finding. Other coexisting intracranial complications must be expected in more than 50 percent of patients. Extension of infected clot along the internal jugular vein is always accompanied by tenderness and oedema along the course of the vein in the neck, and localised oedema over the thrombosed internal jugular vein may still be seen. One rare finding is the presence of pitting oedema over the occipital region, well behind the mastoid process, caused by clotting within a large mastoid emissary vein, this sign is known as the Griesinger's sign. In fact there is no single

pathognomonic sign for lateral sinus thrombosis and a high index of suspicion is a must in diagnosing this condition.

Investigations:

A lumbar puncture must be performed, if papilloedema does not suggest that raised intracranial pressure may precipitate coning. Examination of CSF is the most efficient way of identifying meningitis. In uncomplicated lateral sinus thrombosis the white blood count in the CSF will be low when the cause is chronic middle ear disease, and some what raised in acute otitis media. The CSF pressure is usually normal. The variations in the level of CSF proteins and sugar are not useful.

23. Acute Mastoiditis.

Acute mastoiditis includes all inflammatory processes of the mastoid air cells of the temporal bone. As the mastoid is contiguous to and an extension of the middle ear cleft, virtually every child or adult with acute otitis media (AOM) or chronic middle ear inflammatory disease has mastoiditis. In most cases, the symptomatology of the middle ear predominates (eg, fever, pain, conductive hearing loss), and the disease within the mastoid is not considered a separate entity.

Mastoiditis includes all inflammatory processes of the mastoid air cells of the temporal bone. As the mastoid is contiguous to and an extension of the middle ear cleft, virtually every child or adult with acute otitis media (AOM) or chronic middle ear inflammatory disease has mastoiditis. In most cases, the symptomatology of the middle ear predominates (eg, fever, pain, conductive hearing loss), and the disease within the mastoid is not considered a separate entity.

Mastoiditis progresses in the following 5 stages and may be arrested at any point.

Hyperemia of the mucosal lining of the mastoid air cells

Transudation and exudation of fluid and/or pus within the cells

Necrosis of bone by loss of vascularity of the septa

Cell wall loss with coalescence into abscess cavities

Extension of the inflammatory process to contiguous areas

Etiology:

Host factors include mucosal immunology, temporal bone anatomy, and systemic immunity. Microbial factors include protective coating, antimicrobial resistance, and ability to penetrate local tissue or vessels (ie, invasive strains). As the clearance of the mastoid is dependent upon a patent

antrum, resolution is unlikely unless this anatomical isthmus opens by control of mucosal swelling, which otherwise creates a reservoir for infection.

Reported pathogens in mastoiditis include the following:

Streptococcus pneumoniae – Most frequently isolated pathogen in acute mastoiditis, prevalence of approximately 25%

Group A beta-hemolytic streptococci

Staphylococcus aureus

Streptococcus pyogenes

Moraxella catarrhalis

Haemophilus influenzae

Pseudomonas aeruginosa

Mycobacterium species

Aspergillus fumigatus and other fungi

Nocardia asteroides

Management:

Systemic antibiotics needs to be administered at the earliest. Anti-inflammatory drugs can be administered to alleviate pain.

24. Bezold's abscess

Bezold Abscess is a rare deep neck abscess resulting from an intertemporal complication of a coalescent mastoiditis. It was first described by a German Otologist Dr. Friedrich Bezold in 1881. Since the introduction of antibiotics, the number of reported cases of Bezold abscesses have significantly decreased. According to some authors, since 1967 there have been only 35 reported cases in the English literature, of which only four cases occurred in children under the age of five.

Etiology:

The organisms commonly cultured from Bezold abscesses include gram-positive aerobes: Streptococcus, Staphylococcus, and Enterococcus species; gram-negative aerobes: Klebsiella, Pseudomonas, and Proteus species and anaerobes, particularly Peptostreptococcus and Fusobacterium species.

Epidemiology:

Many factors have been shown to play a role in the development of acute, chronic, and suppurative otitis media in children. Factors include the following:

Socioeconomic conditions: there is an increase in the number of the cases in children living in poorer socioeconomic conditions.

Daycare: there appears to be an increase in children attending day care.

Underlying medical conditions that affect the Eustachian tube function: children with cleft palate, craniofacial anomalies, congenital or acquired immune dysfunction, conditions affecting the ciliary function of the Eustachian tube and middle ear mucosa.

In adults, medical considerations that must be considered are the patient HIV status, history of uncontrolled diabetes, renal failure, and patients who are immunosuppressed.

Pathophysiology:

The most important contributing factor in the development of a Bezold abscess is the presence of a well-aerated and pneumatized mastoid bone. At birth, the mastoid bone consists mainly of an antral cell. From this antral cell, air cells begin to develop until there is complete pneumatization of the mastoid bone around the age of five. As the process of pneumatization of the mastoid bone occurs, the surrounding walls of the mastoid bone thin, particularly at the tip of the mastoid bone on its medial surface along the incisura digastrica (digastric groove) where the digastric muscle attaches.

If the infection is left unchecked, the outer walls of the mastoid tip become involved with the spread of the infection along the sternocleidomastoid muscle, the trapezius, and splenius capitis muscles. Secretions and bacteria can enter the middle ear through the eustachian tube and from the middle ear can pass directly into the mastoid bone through a small opening the aditus ad antrum. During an inflammatory process (suppurative otitis media), obstruction of the aditus ad antrum occurs. Purulent secretions cannot escape and will accumulate in the mastoid bone. The pressure created by this purulent material with its enzymatic activity within the mastoid bone causes osteitis and osteonecrosis of the fragile pneumatized air cells further thinning the walls of the mastoid bone. This stage is Coalescent Mastoiditis. The inflammatory process can spread in many directions. If it spreads inferiorly towards the mastoid tip, the purulent material will eventually erode the very thin bone along the digastric ridge at the insertion of the digastric muscle. Consequently, a purulent material will spread into the neck between the digastric and sternocleidomastoid muscles.

Clinical features:

Bezold abscess may occur in both children and adults with well-developed mastoid bones. Both may have had a history of recurrent otitis media or chronic otitis media with tympanic membrane perforation and a draining ear. Prior mastoid surgery for cholesteatoma normally causes further thinning of the mastoid walls making it easier for infection to spread. An important early clinical sign of a coalescent mastoiditis on physical examination is sagging of the posterior superior external auditory canal. Other common clinical signs include the following:

neck pain

swelling in the lateral neck

post auricular tenderness over the affected mastoid bone

otalgia

otorrhea

hearing loss

If any of the above are documented, and the patient presents with fever with an associated neck mass one should have a high index of suspicion that the infection has spread to adjacent regions within the head and neck. Thus a thorough head and neck evaluation should always be conducted.

In the differential diagnosis, one must always consider an abscess of post-auricular lymph nodes. If CT scanning is not available, then a simple lateral x-ray of the temporal bone may be helpful in ascertaining the status of the mastoid air cell system.

Evaluation:

Laboratory evaluations are often not helpful since the leukocyte count and erythrocyte sedimentation rate may be normal. Contrast computed tomography should be obtained if there is any indication or suspicion of a deep neck abscess, regardless of a lack of evidence of an infection in the mastoid bone since prior antibiotics may have truncated the infection in the mastoid bone while the abscess was still developing in the neck. Recently, in the emergency room setting, a bedside soft-tissue ultrasound with the use of a high-frequency linear array probe to the mastoid was used to detect a complex hypoechoic-anechoic fluid collection in the neck. An intravenous contrast CT scan was used to confirm the ultrasound findings, and both an abscess in the neck and ear pathology were identified.

Treatment:

If a Bezold abscess is present or suspected, practitioners should use intravenous broad-spectrum antibiotics. Once antibiotic therapy is initiated, a contrast CT scan should be obtained. Naturally, cultures are important, particularly if you are dealing with a diabetic or immunocompromised patient since the bacteriology in these cases may be different. If surgical management is indicated, in adults, a post-auricular incision is made, and a simple, complete mastoidectomy should be performed. The objective is to remove as much osteitic bone and granulation tissue present in the mastoid bone. In rare cases, a Bezold abscess may appear in children before there is a complete development of the mastoid air cell system. In these patients, the practitioner must pay particular attention to the position of the facial nerve. Therefore, they should not make a standard post-auricular incision since the facial nerve may be more superficial than anticipated. In this case, a post-auricular superior linear incision is made to drain the purulent material and carefully debride as much granulation tissue as possible. At the same time, the neck abscess should be thoroughly drained. Broad-Spectrum antibiotics should be continued until a final resolution is obtained to prevent further extension of the inflammatory process to adjacent vascular structures.

25. Otogenic brain abscess

Otogenic brain abscess:

Otogenic brain abscess always develop in the temporal lobe or the cerebellum of the same side of the infected ear. Temporal lobe abscess is twice as common as cerebellar abscess. In children nearly 25% of brain abscesses are otogenic in nature, whereas in adults who are more prone to chronic ear infections the percentage rises to 50%. The routes of spread of infection has already been discussed above, the commonest being the direct extension through the eroded tegment plate. Although dura is highly resistant to infection, local pachymeningitis may be followed by thrombophlebitis penetrating the cerebral cortex, sometimes the infection could extent via the Virchow - Robin spaces in to the cerebral white matter. Cerebellar abscess is usually preceded by thrombosis of lateral sinus. Abscess in the cerebellum may involve the lateral lobe of the cerebellum, and it may be adherent to the lateral sinus or to a patch of dura underneath the Trautmann's triangle.

Stages of formation of brain abscess:

Stage of cerebral oedema: This is infact the first stage of brain abscess formation. It starts with an area of cerebral oedema and encephalitis. This oedema increases in size with spreading encephalitis.

Walling off of infection by formation of capsule: Brain attempts to wall off the infected area with the formation of fibrous capsule. This formation of fibrous tissue is dependent on microglial and blood vessel mesodermal response to the inflammatory process. This stage is highly variable. Normally it takes 2 to 3 weeks for this process to be completed.

Liquefaction necrosis: Infected brain within the capsule undergoes liquefactive necrosis with eventual formation of pus. Accumulation of pus cause enlargement of the abscess.

Stage of rupture: Enlargement of the abscess eventually leads to rupture of the capsule containing the abscess and this material finds its way into the cerebrospinal fluid as shown in the above diagram.

Cerebellar abscess which occupy the posterior fossa cause raised intra cranial tension earlier than those above the tentorium. This rapidly raising intra cranial pressure cause coning or impaction of the flocculus or brain stem into the foramen magnum. Coning produces impending death. If the walling off process (development of capsule) is slow, softening of brain around the developing abscess may allow spread of infection into relatively avascular white matter, leading to the formation of seconday abscesses separate from the original or connected to the original by a common stalk. This is how multilocular abscesses are formed. Eventually the abscess may rupture into the ventricular system or subarachnoid space, causing meningitis and death.

The mortality rate of brain abscess is around 40%, early diagnosis after the advent of CT scan has improved the prognosis of this disease considerably.

The bacteriological flora is usually a mixture of aerobes and obligate anaerobes. Anaerobic streptococci are the commonest organisms involved. Pyogenic staphylococci is common in children. Gram negative organism like proteus, E coli and Pseudomonas have also been isolated.

Clinical features:

The earliest stage where the brain tissue is invaded (stage of encephalitis) is marked by the presence of headache, fever, malaise and vomiting. Drowsiness eventually follows. These early features may be masked by the complications such as meningitis or lateral sinus thrombosis. If this stage progresses rapidly to generalized encephalitis before it could be contained by the formation of the capsule, drowsiness may progress to stupor and coma followed by death. Usually the period of local encephalitis is followed by a latent period during which the pus becomes contained within the developing fibrous capsule. During this latent phase the patient may be asymptomatic.

During the next state (stage of expansion) the enlarging abscess first cause clinical features due to the alteration of CSF dynamics, and site specific features may also be seen due to focal neurological impairment. The pulse rate slows with rising intracranial pressure, the temperature may fall to subnormal levels. Drowsiness may alternate with periods of irritability. Papilledema is also found due to elevated CSF pressure.

Clinical features also vary according to the site of involvement. Hence the differences that are seen between the cerebral and cerebellar abscess.

Cerebral (Temporo sphenoidal abscess):

A cerebral abscess in the dominant hemisphere often cause nominal aphasia, where in the patient has difficulty in naming the objects which are in day to day use. He clearly knows the function of these objects. Visual field defects arise from the involvement of optic radiations. Commonly there is quadrantic homonymous hemianopia, affecting the upper part of the temporal visual fields, more rarely it may also involve the lower quadrants. The visual field loss are on the side opposite to that of the lesion. This can be assessed by confrontation method. Upward development affects facial movements on the opposite side, and then progressively paralysis of the upper and lower limbs. If the expansion occurs in inward direction then paralysis first affects the leg, then arm and finally the face.

Cerebellar abscess:

The focal features associated with cerebellar abscess is weakness and muscle incoordination on the same side of the lesion. Ataxia causes the patient to fall towards the side of the lesion. Patient may also manifest intention tremors which may become manifest by the finger nose test. This test is

performed by asking the patient to touch the tip of the nose with the index finger first with the eyes open and then with the eyes closed. The patient may often overshoot the mark when attempted with the eyes closed in case of cerebellar abscess. The patient may also have spontaneous nystagmus. Dysdiadokinesis is also positive in these patients.

Investigations:

CT scan and MRI scans are the present modes of investigation. Scan is ideally performed using contrast media. These scans not only reveal the position and size of the abscess, the presence of localised encephalitis can be distinguished from that of an encapsulated abscess. Associated conditions such as subdural abscess, and lateral sinus thrombosis can also be seen.

Management:

Surgical drainage of the abscess, followed by mastoidectomy to clear the ear disorder.

26. Otosclerosis

Definition: Otosclerosis is a hereditary localised disease of the bone derived from the otic capsule characterised by alternating phases of bone resorption and new bone formation. The mature lamellar bone is removed by osteoclasis and replaced by woven bone of greater thickness, cellularity and vascularity.

History: In 1741 Antonio Valsalva described ankylosis of stapes while doing a postmortem on the body of a deaf patient.
In 1894, Adam Politzer introduced the term "otosclerosis" and described the histopathology of the disease for the first time.
In 1912 Siebenmann introduced the term otospongiosis to denote active otosclerotic foci.

Pathophysiology:

The primary pathological change occurs in the bony labyrinth with secondary effects upon middle ear and inner ear function. The otosclerotic focus may be asymptomatic, or if present in the area of foot plate of stapes it may give rise to ankylosis of foot plate with resultant conductive deafness. Otosclerotic foci may involve other portions of labyrinth causing sensori neural hearing loss and vestibular abnormalities.
A combination of effects are possible in otosclerosis. They are:

Histological otosclerosis: Otosclerotic foci doesnot cause any symptoms and hence known as histological otosclerosis.
Stapedial otosclerosis: is the classical otosclerosis with fixation of stapedial foot plate causing conductive deafness.
Cochlear otosclerosis: The foci involves the cochlea causing sensorineural deafness.
Combined otosclerosis: Here in addition to fixation of foot plate of stapes there is also associated sensorineural hearing loss due to involvement of cochlea.

Otospongiosis: European otologists prefer to use this term to indicate the active phase of otosclerosis.

Incidence: Otosclerosis is common in caucasian races. It is rarely found in Mongoloid and Negro population.

Sex incidence: In clinical practice otosclerosis is seen more often in women than in men. The ratio was found to be 2:1. Nowadays the authors believe the apparent female preponderance may be due to the fact that unilateral otosclerotic deafness is less common in women than in men. Noticeable deterioration in hearing also occur during pregnancy due to hormonal changes. Deafness due to otosclerosis may be initiated or made worse by pregnancy.Causative factors / etiology: Many theories have been proposed to explain the etiological factors of otosclerosis. They are:

1. Metabolic

2. Immune disorders

3. Vascular disease

4. Infection (Measles) currently accepted

5. Trauma : The petrous bone doesnot have regenerative capacity. This is because of the fact that the enzymes released during reparative phase are very toxic to the inner ear hair cells.
Pockets of tissue capable of regeneration may be sequestered in various portions of labyrinthine bone. These tissue could be activated by the presence of regenerative enzymes in the blood following bone fracture elsewhere in the body.

6. Temporal bone abnormalities (congenital)
Genetic factors predisposing to otosclerosis: The tendency for otosclerosis to run in families has been documented. Authors have postulated an autosomal dominant mode of inheritance with varying degrees of penetration.
Otosclerosis is associated with osteogenesis imperfecta in 0.15 % of cases. This is known as Van der Hoeve syndrome or Adair - Dighton syndrome.

Sites affected by otosclerosis: The commonest site for apperance of otosclerotic bone is fissula ante fenestrum. This fissula is constantly seen in the labyrinthine capsule lying in front of the oval window. This area may contain unossified cartilage persisting even in adults. This area was referred to as Cozzolino's zone by Perozzi in memory of his teacher. Otosclerosis may occur in this area due to bony ossification of the cartilage.
Residual cartilage may be present in the following areas of labyrinth:
1. Fissula ante fenestram
2. Fissula post fenestram
3. Intracochlear
4. Round window
5. Semicirular canals
6. Petrosquamous suture
7. Base of styloid process

In normal development the fissula appears as fibrous connective tissue bundle joining the vestibule with the tympanic cavity. This fibrous tissue is encased in primary cartilage which later gets replaced by bone. From the fissula the bone acquires a connective tissue lining which later becomes converted into perichondrium. The fissula is reduced in size by the production of new secondary cartilage from the perichondrium. These changes are completed by birth. The secondary cartilage remains throughout life as a stable, dormant cartilage and hence may even be considered as normal structure. It is only when this secondary cartilage gets ossified otosclerosis occur (Bast & Anson).

Otosclerotic changes may appear as a result of interaction between bone growth promoting substances circulating in the blood stream, and the unstable cartilagenous elements in the capsule of the labyrinth. Otosclerosis is often seen at times when the bone growth promoting substances are circulating in the blood as in pregnancy and following fractures of other bones.

Histopathology of otosclerosis:

The normal endochondral bone of labyrinthine capsule in which otosclerotic focus begins is compact in type. Ultrastructurally, lamellae composed of fine fibrils lying in a ground substance are concentrically disposed around haversian canals containing blood vessels and connective tissue. In otosclerosis there is sharply defined new bone formations that could be differentiated from normal bone by their deep carmine stain and by marked enlargement of bone spaces and haversian canals. The following are the changes which occur in a otosclerotic foci:

1. Focal / diffuse replacement of normal compact bone by irregular, loose cancellous bone with more deeply staining lamellae.
2. There is an associated increase in size of Haversian canals, cell spaces and marrow spaces with corresponding increase in vascularity. The blood vessels are frequently surrounded by a narrow margin of blue staining material that Manassee described first as Blue Mantle zone.
3. Increase in osteocytes, and appearance of osteoblasts and osteoclast cells.

Histologically otosclerosis may be classified into:

1. Early focal otosclerosis
2. Diffuse active otosclerosis
3. Quiescent otosclerosis
4. Cochlear otosclerosis

Early focal otosclerosis: In this type the abnormalities are localised to one or two small areas of an otherwise normal foot plate section. The abnormal areas show an enlarged marrow space surrounded by a blue staining area on H&E staining.

Diffuse active otosclerosis: In this type there is abnormal vascularity with a great increase in size and number of marrow spaces. Most of these spaces are lined by osteoblasts. In places around the circumference of the marrow spaces there is a scalloped appearance where bone has been recently absorbed. The number of osteocytes is greatly increased.

Quiescent otosclerosis: Here even though there may be some increase in the size and number of marrow spaces there is no evidence of bone formation or bone destruction. Osteoblasts and osteoclasts are only occasionally seen. This could be considered as a burnt out phase of the disease spectrum.

Cochlear otosclerosis: This condition causes pure sensorineural deafness without stapes fixation. Otosclerotic foci may occur in the otic capsule without the involvement of stapedial foot plate. The process of bone erosion and new bone formation which occur in otosclerosis releases enzymes like amylase, SGOT, SGPT etc which can enter into the endolymph via the round window membrane. These enzymes are toxic to the sensitive hair cells of the cochlea causing sensorineural hearing loss.

Clinical types of otosclerosis: Classification of various clinical types of otosclerosis is based on microscopic appearances of the diseased foot plate.

Rim fixation: Here the otosclerotic foci starts from the anterior portion of the oval window niche. It gradually expands to involve the anterior portion of the foot plate causing fixation of the anterior portion of the foot plate only leaving the centre of the plate free.

Biscuit foot plate: This type occur less frequently. The focus originates in the foot plate itself and as it expands it gives rise to the biscuit or rice grain foot plate with delineated margins.

Obliterative otosclerosis: Rarely a large mass of otosclerotic new bone fills up the oval window niche obscuring the entire foot plate. This condition is known as obliterative otosclerosis. It is a difficult condition to manage surgically.
Clinical features:

Deafness: Typically deafness in otosclerosis is bilateral and gradually increasing in nature. Unilateral otosclerosis occur in 15% of patients. Frequently it occurs between third and fifth decades of life. In majority of cases the deafness is conductive in nature. The deafness will not be noticed by the patient till the loss reaches 30 dB level. This is the deafness level in which understanding speech becomes difficult. These patients may hear better in noisy environment because the speaker has a tendency to raise his voice because of excessive ambient noise. This phenomenon a feature of otosclerosis is known as Paracusis Willisii.
In cochlear otosclerosis the deafness is purely sensorineural in nature. Some patients may have both conductive and sensorineural hearing loss (mixed deafness) because of the tendency of bone reparative enzymes to damage the inner hair cells.

Patients with otosclerosis have characteristically quiet voice with good tone and the change in speech pattern may be detected only by close relatives.
Tinnitus: is a common symptom and occasionally could be the only presenting feature. The presence of tinnitus should alert the physician about the presence of cochlear otosclerosis. It could also be seen in some patients without cochlear degeneration due to abnormally increased vascularity of the otosclerotic bone. Mostly tinnitus indicates sensorineural degeneration. Tinnitus may be unilateral or bilateral. It is usually roaring in nature.

Vertigo: Transient attacks of vertigo is not uncommon in patients with otoslerosis. This could be due to the action of toxic enzymes released by the lesion into the vestibular labyrinth. These patients may even have coexisting Meniere's disease.

Clinical examination: The ear drum in these patients is normal (mint condition). Rarely during active phase of the disease, the increased vascularity of the promontory may be seen through the ear drum. This sign is known as Flemingo's flush sign or Schwartz's sign. This indicates otospongiosis (active otosclerosis).

Hearing assessment can be done using tuning forks. For detailed description of tuning fork tests read the chapter titled clinical examination of the ear.

Pure tone auditometry will show precisely the amount and type of hearing loss. The presence of Carhart's notch is a classic audiometric feature in these patients. This Carhart's notch is present in bone condition. There is a dip centered around 2000 Hz. This is actually an artifact. In cochlear otosclerosis audiometry reveals sensorineural hearing loss.
Stapes fixation causes an elevation in the bone conduction thresholds of 5dB at 500Hz, 10dB at 1000 Hz, 15 dB at 2000 Hz, and 5 dB at 4000 Hz. In the audiogram this creates a peculiar pattern known as Cookie bite audiogram. The bone conduction audiogram appears like a cookie having been bitten.

Impedence audiometry is an useful investigation to diagnose otosclerosis. Middle ear compliance is often reduced. When stapes is fixed stapedial reflex is absent. The typical impedance curve is As curve.

All these patients with pure conductive deafness have excellent speech discrimination thresholds.

Management:

Medical: The aim of medical management is to convert an active otosclerotic foci into an inactive or quiescent foci. Fluoride is the drug of choice.

Indications of fluoride therapy:

1. Patients with surgically confirmed otosclerosis who show progressive sensorineural deafness disproportionate to age.

2. Patients with pure sensorineural loss with family history, age of onset, audiometric pattern and good auditory discrimination indicate the possibility of cochlear otosclerosis.

3. Patients with radiological demonstration by CT scan of spongiotic changes in the cochlear capsule

4. Patients with positive Schwartz sign.

5. Post op treatment: If patients are found to have an active focus during surgery, fluride therapy is prescribed for 2 years.

Contraindications of fluoride therapy:

1. Patients with chronic nephritis and nitrogen retention

2. Patients with chronic rheumatoid arthritis

3. Patient who are pregnant / lactating

4. In children before skeletal growth has been completed

5. Patients who show allergy for the drug

6. Patients with skeletal fluorosis

Fluorides act on otosclerotic foci by reducing osteoclastic bone resorption with a corresponding increase in osteoblastic bone formation. Fluoride also has antienzymatic action thereby it can neutralize the toxic enzymes released from the otospongiotic foci.

Dose: A daily dose of 50 mg of sodium fluoride is given for a period of 2 years. In patients with positive Schwartz's sign the dose can be increased up to 75 mg per day.

Adverse effects of sodium fluoride therapy:

1. Gastric disturbance

2. Arthritis

3. Skeletal fluorosis

Surgical treatment: Stapedectomy

Hearing aids: These patients will benefit from the use of hearing aids if surgery is not acceptable to the patient or if it is risky. There is always a 1% risk of producing a dead ear during surgery even in the best of hands.

27. Clinical Otosclerosis

This terminology is used to clinically identify cases of otosclerosis. Features that help in clinical diagnosis of otosclerosis include:

1. Bilateral conductive deafness
2. Bilateral conductive deafness in females
3. Normal looking ear drum
4. Paracusis wilisi ability to hear well in noisy environment
5. Low speech modulation
6. Tinnitus is a feature of active otosclerosis

28. Complications of stapedectomy

1. Facial palsy

2. Vertigo in the immediate post op period

3. Vomiting

4. Perilymph gush

5. Floating foot plate

6. Tympanic membrane tear

7. Dead labyrinth

8. Perilymph fistula

9. Labyrinthitis

29. Meniere's disease – clinical features and management

Meniere's disease is a disorder that affects the inner ear. The inner ear is responsible for hearing and balance. The condition causes vertigo, the sensation of spinning. It also leads to hearing problems and a ringing sound in the ear. Meniere's disease usually affects only one ear.

Meniere's disease symptoms tend to come on as "episodes" or "attacks." These symptoms include:

vertigo, with attacks lasting anywhere from a few minutes to 24 hours
loss of hearing in the affected ear
tinnitus, or the sensation of ringing, in the affected ear
aural fullness, or the feeling that the ear is full or plugged
loss of balance
headaches
nausea, vomiting, and sweating caused by severe vertigo

Someone with Meniere’s disease will experience at least two to three of the following symptoms at one time:

vertigo
hearing loss
tinnitus
aural fullness

Medical management of Meniere's disease includes:

1. Dietary management

2. Physiotherapy

3. Psychological support

4. Pharmacologic intervention

Dietary management:

This includes reduction of sodium in the diet. Infact it was Frustenberg in 1934 who introduced a low salt diet for patients with Meniere's disease. Pathophysiology of Meniere's disease is enlargement of membranous labyrinth due to excess accumualtion of endolymphatic fluid. Any attempt to reduce this fluid level will help in alleviate the symptoms of the patient.

Medical managment is mainly used to treat patients during the acute phase of the attack. Vestibular suppresants are commonly used. Drugs used to control attacks of vertigo have varying levels of anticholinergic, antiemetic and sedative properties. Drugs used to alleviate symptoms include phenothiazines (prochlorpherazineand perphenazine), antihistamines like (cinnarizine, cyclizine, dimenhydrinate, and meclizine hydrochloride), benzodiazepines like (lorazepam and diazepam).

Vestibular suppressants:

Diazepam: when used acts as vestibular depressant. It also alleviates the anxiety associated with this disorder. The beneficial effects of diazepam ib vestibular system is presumed to be due to an increase in the cerebellar GABA-ergic system. Stimulation of cerebellar GABA-ergic system mediates inhibition on the vestibular response. This drug is very useful in alleviating vertigo especially when associated with anxiety. Usual dose is 5 mg administered orally every 3 hours. The initial dose may also be administered intravenously.

Antiemetic drugs:

Drugs belonging to this group helps to alleviate vomiting in Meniere's disease.

Anticholinergic drugs:

Glycopyrrolate an anticholinergic drug when combined with diazepam is helpful in controlling inner ear symptoms of nausea and vomiting. In adults it is administered in doses of 1-2 mg. It may also be administered as intramuscular injection (0.1 - 0.2 mg) every 4 hours. Side effects (reversible) of this drug includes dry mouth, distortion of visual acuity, exacerbation of symptoms in patients with prostatic hypertrophy. This drug is contraindicated in patients with glaucoma and prostatic hypertrophy.

Antidopaminergic drugs:

Droperidol: This is an antidopaminergic drug used to alleviate the symptoms of Meniere's disease. This drug is administered in doses of 2.5 - 10 mg orally in adults. If administered intravenously it is given as 5 mg bolus. This drug has fewer incidence of side effects like extrapyramidal symptoms / sedation / hypotension.

Prochlorperazine: This drug belongs to phenothiazine group. It is used as an antiemetic and a potentiator of analgesic and hypnotic drugs. Usual recommended dose is 10 mg given orally or intramuscularly every 4 - 6 hours in adults. This drug has excellent antiemitic effect.

Antihistamines:

Dimenhydrinate: is useful in preventing and treating vertigo associated with Meniere's disease. It is also very effective in controlling nausea and vomiting. Only side effect of this drug is its propensity to cause drowsiness. It is administered as 50 - 100 mg doses thrice a day. This drug can also be administered intramuscularly / intravenously.

Diphenhydramine: This drug is not useful in treating acute vertigo. It may be useful in prevention of vertigo. The usual duration of action is 4-6 hours. Usually this drug is administered as an initial loading dose of 50 mg orally.

Meclizine: This drug is one of the most useful antiemetics to prevent / treat nausea and vomiting associated with vertigo of vestibular origin. It has a slower onset and a longer duration of action (24 hours). For vertigo the usual dose administered in adults is 25 - 100 mg daily in divided doses. Side effects of this drug include: drowsiness, blurred vision, drowsiness.

Promethazine: This drug has pronounced antihistaminic activity in addition to its strong central cholinergic blocking activity. It is effective in the treatment of vertigo and motion sickness. It is administered usually in doses of 25 mg every 4 to 6 hours. One major advantage of this drug is that it can be administered rectally, when severe vomiting prevents its effective oral administration. Most common side effect of this drug is sedation.

Maintenance therapy:

The goal of maintenance therapy is

1. To prevent acute attacks of vertigo

2. To maintain hearing in Meniere's disease

This therapy usually includes dietary modifications combined with pharmacological intervention.

Dietary modifications: The mainstay of diet modifications is to reduce sodium intake. A very low sodium intake or low sodium diet is usually recommended. A strict low sodium diet means a daily allowance of 1500 mg. This is a very stringent diet and patients find it very difficult to comply with this diet. A more practical approach would be to advise the patient to avoid excessively salty food. Restrictions are also imposed on the intake of caffeine, nicotine and alcohol.

Diuretics:

The use of diuretics in the maintenance therapy is based on the supposition that these drugs can alter the fluid balance of inner ear, leading to a depletion of endolymph and a correction of hydrops. In 1934 Furstenburg demonstrated that the symptoms of Meniere's disease were due to retention of sodium. He went on to recommend a low sodium diet / use of diuretics to control Meniere's disease. Boles in 1975 demonstrated that most patients had their vertigo controlled with an 800 - 1000 mg of sodium diet / day.

Hydrochlorthiazide: This diuretic causes natriuresis and kaliuresis by blocking sodium reabsorption in the loop of Henle. Potassium supplementation is required in patients using this drug. Side effects of this drug include: hypokalemia, hyperglycemia, hypotension, and hyperuricemia. It is usually administered as 50 mg tabs orally / day in adults. Potassium supplements is usually required in these patients.

Dyazide: Is a potassium sparing diuretic. It can be conveniently administered as a single daily dose.

Frusemide: This is a loop diuretic. It is a very potent diuretic. It can cause electrolyte and volume depletion more rapidly than other diuretics. It usually causes hypokalemia. Usual adult dose is 10 - 80 mg/day. The duration of action lasts for about 4 hours.

Amiloride: This is a potassium sparing diuretic acting on the distal tube of Henle. Its diuretic potency is highly limited. It is usually used in combination with other diuretics in order to minimize potassium loss.

Carbonic anhydrase inhibitors:

Acetazolamide: Is a carbonic anhydrase inhibitor. It causes a decrease in the sodium - hydrogen exchange in the renal tubule inducing diuresis.

Methazolamied: Is another carbonic anhydrase inhibitor shown to be effective in controlling symptoms of Meniere's disease. This drug is usually administered in doses of 50 mg / day, 5 days a week for 3 months.

Medical ablative therapy:

Aminoglycosides: Ototoxic effects of aminoglycosides are well documented. Streptomycin and gentamycin are predominantly vestibulotoxic. Intramuscular injections of streptomycin administered twice daily for periods of days to weeks have been used in patients with debilitating bilateral disease / unilateral disease in the only hearing ear. Complete ablation causes disabling oscillopsia. Many authors have suggested lower doses and fewer injections to achieve partial ablation, thereby reducing the incidence of severe ataxia. Currently the recommended daily dose is 1 g of streptomycin intramuscularly 5 days a week until vestibular ablation occurs as manifested by absence of ice water caloric test. Intratympanic injections of these drugs have also been used with success.

Vasodilators:

The use of vasodilators is based on the idea that Meniere's disease results from ischemia of the stria vascularis. Betahistine has been used with varying degrees of success. This drug can be used for short term control of vertigo and for maintenance therapy.

Nicotinic acid is another vasodilator which when administered 30 minutes before meals in doses of 50 - 400 mg helps in resolving the acute crisis associated with Meniere's disease.

Calcium channel blockers:

Nimodepine a highly lipophilic drug is very useful in the medical management of Menierie's disease. It readily crosses the blood brain barrier. This drug is useful in patients who have failed diuretic medical therapy.

ACE inhibitors:

These are very effective vasodilators. These drugs block the renin angiotensin aldosterone system. They produce vasodilatation by blocking angiotensin II induced vasoconstriction.

Lipoflavins and vitamins:

Combination of lipoflavins and vitamins have been tried as a management modality with varying degrees of success.

Surgical Management:

Endolymphatic sac decompression

30. BPPV

Benign paroxysmal positional vertigo is the most commonly diagnosed vestibular disorder. This is commonly caused by dysfunction of the posterior semicircular canal. Lateral and superior semicircular canals can also be involved on rare occasions. It is characterized by brief spells of severe vertigo (often lasting for just a few seconds) that are experienced only with specific movements of the head.

Typical features of BPPV as described by Hall & Ruby:

1. Canalithiasis mechanism - This explains the latency of the nystagmus as a result of the time needed for motion of the material within the posterior canal to be initiated by the gravity.

2. Duration of the nystagmus - is correlated with the length of time required for the dense material to reach the lowest part of the posterior canal.

3. The vertical (upbeating) and torsional (superior poles of the eye beating towards the lowermost ear). The nystagmus is more vertical when the patient looks away from the lowermost ear, and more torsional when looking towards the lowermost ear.

4. The reversal of nystagmus when the patient returns to the sitting position is due to retrograde movement of material in the lumen of the posterior canal back towards the ampulla, resulting in ampulo petal deflection of the cupula.

5. The fatiguability of the nystagmus evoked by repeated Dix Hallpike positional testing is explained by dispersion of material within the canal.

Incidence:

BPPV is the most common cause of vertigo constituting 20 - 40% of all patients with peripheral vestibular disease. Mean age of onset ranging between 4th and 5th decades. women outnumbering men by 2:1.

History: Patient c/o severe vertigo associated with change in head position. Symptoms are always sudden in nature, never lasting more than a minute. The patient may even volunteer provocating postures.

On examination: the classic eye movements associated with Dix Hallpike maneuver is seen.

Dix-Hallpike maneuver: The patient is positioned on the examination table in such a way that when he/she is placed supine, the head extends over the edge. The patient is lowered with the head supported and turned 45 degrees to one or the other side. The eyes are carefully observed; if no abnormal eye movements are seen, the patient is returned to the upright position.

This same maneuver is repeated with the head in the opposite direction and the patient's symptoms are noted.

The pattern of response consists of the following:

1. Nystagmus is a combination of vertical upbeating & rotatory (torsional) beating towards the downward eye. Pure vertical nystagmus is not seen in BPPV.

2. There is often a latency of onset of nystagmus

3. Duration is less than a minute

4. Vertiginous symptoms are invariably seen

5. Nystagmus disappears with repeated testing (fatiguability)

6. Symptoms often recur with the nystagmus in opposite direction on return of the head to upright position.

Management:

Medical:

Repositioning maneuver: Currently BPPV is managed by repositioning maneuvers that, in cases of Canalithiasis use gravity to move canalith debris out of the affected semicircular canal and into the vestibule. For posterior canal BPPV the maneuver developed by Epley is effective.

Epley maneuver - This is performed by placing the head of the patient in the Dix Hallpike position that evokes the vertigo. The posterior canal on the affected side is in the earth vertical plane when the head is in this position. After the cessation of initial nystagmus, the head is rolled through 180 degrees, (this is done in two 90 degree increments, stopping in each position until the nystagmus resolves) to the position in which the offending ear is up. The patient is then brought to the upright sitting position. This procedure is likely to be successful when nystagmus of the same direction continues to be elicited in each of the new position (as the debris continues to move away from the cupula). This maneuver is repeated until no nystagmus is elicited. This is successful in 90 % of cases. Posterior canal BPPV can be converted to lateral canal BPPV during Epley maneuver. The lateral canal BPPV resolves in several days. Drugs are usually not prescribed, but low dose meclizine or calmpose can be given 1 hour before the procedure if the patient is anxious or prone to vomiting.

Sermont maneuver - is also effective in posterior canal BPPV, but is most difficult to perform and it has no significant advantages over the Epley maneuver. This is being described here for the sake of completion. In this maneuver the patient is moved quickly in to the position that provokes the vertigo and remains in that position for 4 minutes. The patient is then turned rapidly to the opposite side ear down, and remain in the second position for 4 minutes before slowly getting up.

In both these maneuvers gravity is the stimulus that move the particles within the canal, so there is no need to turn the head on the body, enbloc movement of the head and body as much as possible is the plan.

31. Presbyacusis

Definition: Presbyacusis is defined as a progressive bilateral symmetrical age related sensorineural hearing loss. The hearing loss is confined to higher frequencies.

Presbyacusis is an added problem for the elderly who have a tendency to compensate for their loss of vision through their intact sense of hearing. They even tend to get isolated and become a social recluse due to this problem.

Factors responsible for presbyacusis: Various factors have been postulated as causes of presbyacusis. They are:

1. Hereditary: Features like early aging of the cochlea and susceptiblility of the cochlea for drug insults are genetically determined.

2. Atherosclerosis: May diminish vascularity of the cochlea there by reducing its oxygen supply.

3. Dietary habits: Increased intake of fatty diet may accelerate atherosclerotic changes in old age.

4. Diabetes: May cause vasculitis and endothelial proliferation in the blood vessels of the cochlea there by reducing its blood supply.

5. Noise trauma: Exposure to loud noise on a continuing basis stresses the already hypoxic cochlea hastening the presbyacusis process.

6. Smoking: Is postulated to accentuate atherosclerotic changes in blood vessels aggravating presbyacusis.

7. Hypertension: Causes potent vascular changes, like reduction in blood supply to the cochlea thereby aggravating presbyacusis.

8. Ototoxic drugs: Ingestion of ototoxic drugs like aspirin may hasten the process of presbyacusis.

Cochlear pathology seen in presbyacusis:

Depending on the pathology seen in the cochlea, 4 different types of presbyacusis have been identified. They are Sensory presbyacusis, neural presbyacusis, strial presbyacusis and cochlear conductive presbyacusis. The aging cochlea present disorders that are symmetric in paired ears; but the extent of involvement at the cellular level may be uneven. Hence presbyacusis can occur in 4 differing pathological types, or in combination there of.

A study of pure tone audiograms and cytocochleograms show:

1. Abrupt high frequency hearing loss (attributed to sensory cell pathology (loss).

2. Flat threshold hearing loss (seen in cases with strial atrophy)

3. Diminished speech discrimination (due to loss of cochlear neurons)

4. Gradual descending audiometric pattern (due to inner ear conductive disorder)

Patients with presbyacusis uniformly have poor threshold for frequencies in 8 Khz range. In fact the threshold was as low as 60% in most of the patients.

Sensory Presbyacusis: is caused by loss of hair cells at the basal end of the cochlea. This commonly occurs in an aging cochlea. The area of involvement may extend to involve even the speech frequency area of the cochlea. These changes cause a rapid decrease in the threshold for high freqency sounds.

The earliest changes occurring in the cochlea is the loss of stereocilia, which can be identified only on electronmicroscopy. The second change to occur is distortion or flattening of the organ of corti followed by loss of supporting cells. Finally, the organ of corti appears as an undifferentiated mound of tissue on a basement membrane. There is a gradual reduction in the number of outer hair cells in the elderly more so in the basal area of the cochlea. This occurs to a lesser extent at the apex of the cochlea. The apical loss of outer hair cells is seen only in individuals of more than 70 years of age. The loss of inner hair cells is less marked, but follows the same pattern as the outer hair cells.

The wear and tear pigment lipofuscin is known to accumulate in the apical cytoplasm of the hair cells. The lipofuscin is assumed to be a waste product of lysosomal activity.

Neural presbyacusis: Is caused by a loss in the population of cochlear neurons, but the end organs are still functional causing severe loss in speech discrimination. Pure tone thresholds are nearly normal. Gaeth used the term Phonemic regression to describe this phenomenon. Studies have shown that speech discrimination scores are slightly better in the left ear when compared to the right, this has been attributed to the left cerebral dominance becoming manifest due to the degenerative changes affecting the auditory pathway. The loss of cochlear neurons is the most consistent pathologic change seen in these patients. It has been calculated that loss of cochlear neurons occur at the rate of 2,100 (Schuknecht) neurons every decade. There are roughly about 35,000 cochlear neurons in a normal ear. The loss of cochlear neurons may be genetically determined. The atrophy occurs throughout the cochlea, but is more pronounced in the basal turn of the cochlea.

Strial Presbyacusis:(Also known as Metabolic presbyacusis) Atrophy of stria vascularis is commonly seen in this condition. Hearing loss in these patients is insidious in onset occurring during the 3rd - 6th decades of life. It progresses slowly. The clinical feature that identifies this condition from the other types of presbyacusis is the presence of a flat or a slightly descending audiometric curve. These patients respond well to the amplification produced by hearing aids. This type of presbyacusis carries the best prognosis because of this feature. Takahashi demonstrated two types of atrophy in these patients:

Type I: a patchy type more severe in the apical and extreme basal regions of the cochlea.

Type II: A diffuse type often showing normal strial thickness with large intercellular spaces that may not be visible under light microscopy.

All 3 layers of stria are involved in various degrees. The loss of strial tissue may cause changes in the composition of the endolymphatic fluid causing further damage to the cochlear hair cells.

Pure tone audiometry shows a flat curve because the pathology involves the whole of the cochlea. Speech discrimination is preserved. This type of presbyacusis is considered by many to be familial.

Cochlear conductive (Mechanical) presbyacusis: This type of presbyacusis is differentiated from others by a linear descending audiogram. This is postulated to be caused due to stiffening of the basilar membrane of the cochlea. The thickening has been found to be more severe in the basilar turn of the cochlea where the basilar membrane is thin. Speech discrimination is average for the given frequency.

Mixed presbyacusis: Has been recently introduced to describe conditions which features of either two or all of the types of presbyacusis discussed above. These patients have been clubbed under this mixed category to account for their varied manifestations.

Presbyacusis is a diagnosis of exclusion. All the other causes of sensorineural hearing loss must be ruled out before declaring the patient to be suffering from presbyacusis.

Management: Amplification of the sounds with the use of hearing aids (with proper features) must be considered in all these patients. Administration of placebo drugs like neurotropic vitamins may make the patient feel something is being done to alleviate his problem. In rare patients' cochlear implants may be considered.

32. Ototoxicity

Introduction:

Many drugs used in the management of common ailments are toxic to the inner ear. Any drug which exerts toxic effects on the cochlea, vestibule, semicircular canals, and otoliths are considered to be ototoxic. Drug induced damage may involve the auditory / vestibular / both components of the ear.

Drug induced ototoxicity can result in hearing loss, tinnitus and dizziness. Nearly 100 classes of drugs have been associated with ototoxicity.

The first drug to be identified as ototoxic was streptomycin which was commonly used for the treatment of tuberculosis. It produced irreversible cochlear and vestibular dysfunction in patients who underwent treatment with this drug.

Common groups of ototoxic drugs:

1. Aminoglycosides

2. Platinum based antineoplastic agents

3. Salicylates

4. Quinine

5. Loop diuretics

Features of drug induced ototoxicity:

1. Bilateral high frequency sensorineural hearing loss

2. Tinnitus

3. Positional nystagmus

4. Dysequilibrium

5. Oscillopsia

Even a single dose of these potentially ototoxic drug could lead to ototoxicity

Among all ototoxic drugs aminoglycosides are the most vestibulotoxic. Kanamycin, amikacin, neomycin and dihydrostreptomycin are preferentially cochleotoxic. Gentamycin affects both cochlear and vestibular systems.

Aminoglycosides are cleared rather slowly from the inner ear fluids when compared to that of serum. This enables a latency to exist and hearing loss can progress after cessation of treatment. Patients on ototoxic drugs should be monitored for toxic effects up to 6 months after cessation of treatment. Ototoxicity is considered to be multifactorial.

Risk factors of ototoxicity:

1. Large drug doses

2. High blood levels of drugs

3. Longer duration of therapy

4. Elderly patients

5. Renal insufficiency

Prevention of ototoxicity:

1. Administration of drugs in low doses

2. Co administration of antioxidants like NAC (N acetyl cysteine).

33. Tuning Fork tests

Useful bedside test for hearing is performed using a tuning fork. Ideally 3 frequencies are used 256 Hz, 512 Hz, and 1024 Hz. These three frequencies are used because they fall within speech frequency range. An ideal tuning fork should have the following features:

It should be made of a good alloy.

It should vibrate for one full minute.

It should not produce any over tones.

Tuning fork tests are performed to identify whether the patient is suffering from conductive deafness, sensorineural deafness, or mixed deafness. Three tests are performed towards this end. They are 1. Rinnes test, 2. webers test, 3. Absolute bone conduction test / ABC.

Rinnes test: Ideally 512 tuning fork is used. It should be struck against the elbow or knee of the patient to vibrate. While striking care must be taken that the strike is made at the junction of the upper 1/3 and lower 2/3 of the fork. This is the maximum vibratory area of the tuning fork. It should not be struck against metallic object because it can cause overtones. As soon as the fork starts to vibrate it is placed at the mastoid process of the patient. The patient is advised to signal when he stops hearing the sound. As soon as the patient signals that he is unable to hear the fork anymore the vibrating fork is transferred immediately just close to the external auditory canal and is held in such a way that the vibratory prongs vibrate parallel to the acoustic axis. In patients with normal hearing he should be able to hear the fork as soon as it is transferred to the front of the ear. This result is known as Positive rinne test. (Air conduction is better than bone conduction). In case of conductive deafness the patient will not be able to hear the fork as soon as it is transferred to the front of the ear (Bone conduction is better than air conduction). This is known as negative Rinne. It occurs in conductive deafness. This test is performed in both the ears.

If the patient is suffering from profound unilateral deafness then the sound will still be heard through the opposite ear this condition leads to a false positive rinne.

Weber's test:

Here again 512 Hz tuning fork is used. The vibrating fork is placed over the forehead of the patient and he is asked to indicate on which side he is hearing the sound. Normally when hearing level is equal in both the ears, it is heard in the middle, in patients with conductive deafness the sound is heard in the left ear. This is known as lateralisation of Weber test. If the patient is suffering from sensorineural hearing loss then the sound is lateralised to the normal ear or the better ear. Hence weber's test must always be interpreted along with the Rinne's test. Weber's test is a sensitive test, it can pin point even a 10 dB hearing difference between the ears.

Absolute bone conduction test:

This test is performed to identify sensorinerual hearing loss. In this test the hearing level of the patient is compared to that of the examiner. The examiner's hearing is assumed to be normal. In this test the vibrating fork is placed over the mastoid process of the patient after occluding the external auditory canal. As soon as the patient indicates that he is unable to hear the sound anymore, the fork is transferred to the mastoid process of the examiner after occluding the external canal. In cases of normal hearing the examiner must not be able to hear the fork, but in cases of sensori neural hearing loss the examiner will be able to hear the sound, then the test is interpreted as ABC reduced. It is not reduced in cases with normal hearing.

34. Rinne’s test

Rinnes test: Ideally 512 tuning fork is used. It should be struck against the elbow or knee of the patient to vibrate. While striking care must be taken that the strike is made at the junction of the upper 1/3 and lower 2/3 of the fork. This is the maximum vibratory area of the tuning fork. It should not be struck against metallic object because it can cause overtones. As soon as the fork starts to vibrate it is placed at the mastoid process of the patient. The patient is advised to signal when he stops hearing the sound. As soon as the patient signals that he is unable to hear the fork anymore the vibrating fork is transferred immediately just close to the external auditory canal and is held in such a way that the vibratory prongs vibrate parallel to the acoustic axis. In patients with normal hearing he should be able to hear the fork as soon as it is transferred to the front of the ear. This result is known as Positive rinne test. (Air conduction is better than bone conduction). In case of conductive deafness the patient will not be able to hear the fork as soon as it is transferred to the front of the ear (Bone conduction is better than air conduction). This is known as negative Rinne. It occurs in conductive deafness. This test is performed in both the ears.

35. Absolute bone conduction test

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36. Siegle’s pneumatic speculum

A siegel's pneumatic speculum has an eye piece which has a magnification of 2.5 times. It is a convex lens. The eye piece is connected to a aural speculum. A bulb with a rubber tube is provided to insufflate air via the aural speculum. The advantages of this aural speculum is that it provides a magnified view of the ear drum, the pressure of the external canal can be varied by pressing the bulb thereby the mobility of ear drum can be tested. Since it provides adequate suction effect, it can be used to suck out middle ear secretions in patients with CSOM. Ear drops can be applied into the middle ear by using this speculum. Ear is first filled with ear drops and a snugly fitting siegel's speculum is applied to the external canal. Pressure in the external canal is varied by pressing and releasing the rubber bulb, this displaces the ear drops into the middle ear cavity.

37. Hearing loss

Difficulty in hearing is of three types:

Conductive hearing loss:

Commonest cause for conductive hearing loss is impacted cerumen. Speech discrimination is normal in these patients. Otosclerosis is also known to cause conductive deafness. Conductive deafness is caused due to abnormality in the external ear and middle ear conductive mechanism. Inflammatory diseases of middle ear cavity like ASOM and CSOM can also cause conductive deafness. These candidates are excellent candidates for hearing aids.

Sensorineural hearing loss:

Sensorineural hearing loss is commonly seen in elderly due to normal aging process. This is also known as presbyacusis. Pathologies involving the inner ear are known to cause sensorineural

hearing loss. Speech discrimination scores are rather poor in these patients. These patients would benefit from digital hearing aids.

Mixed hearing loss:

This type of hearing loss involves components of both conductive and sensorineural system. This is common in patients with otosclerosis. Middle ear inflammatory diseases can also cause mixed hearing loss due to effusion of bacterial toxins via round window membrane which shows increased permeability during middle ear infections.

38. Causes and management of sudden sensorineural hearing loss

Sudden sensorineural hearing loss is defined as 20 – 30 dB sensorineural hearing loss in at least 3 contiguous frequencies over a period of less than 3 days. Usually this hearing loss is perceived by the patient suddenly on awakening from overnight sleep. Statistically, this type of deafness constitutes about 1% of all cases of sensorineural hearing loss.

Age incidence: Sudden sensorineural hearing loss may affect persons of any age group. This type of deafness commonly affects patients in their 4th decade.

Associated otological symptoms:

These patients also manifest with associated otological symptoms which include:

Tinnitus – 70% of cases
Vertigo – 40% of cases

Puretone audiogram: Shows the following features:

Downward sloping – High frequency hearing loss (common)
Upward sloping – Low frequency hearing loss
U – shaped hearing loss – Mid frequency loss
Flat curve – Profound sensorineural hearing loss

Management dilemma:

Recovery rates without treatment ranges between 40 – 70%
Recurrence rate with / without treatment – 30%
Pathophysiology is heterogeneous
Incidence is very rare

Causes of sudden sensorineural hearing loss:

Idiopathic – 90% cases
Non idiopathic – 10% cases

It is very important to rule out acoustic neuroma in all these patients as 4% of these patients may suffer from this disorder. Retrocochlear evaluation is a must in all these patients.

Work up: Should include

Pure tone audiometry
Impedance audiometry
MRI of internal acoustic canal
ESR
VDRL / FTA – Abs for syphilis
Serum tested for autoimmune inner ear disease

Idiopathic sudden sensorineural hearing loss: This condition is probably multifactorial in etiology. This factor goes on to explain the high degree of variability in the therapeutic responses shown by these patients.

Two possible theories have been attributed:

Circulatory disturbance
Inflammatory reaction

It should also be stressed that these two theories are not mutually exclusive. Viral insults can cause direct neural injury, direct vascular structure injury, direct injury to red cells causing micro vascular compromise. In addition to all these responses, it can also precipitate inflammatory chain reaction.

Advocated treatment for sudden sensorineural hearing loss:

Steroids
Niacin
Histamine
Lipoflavinoid vitamins

It has been documented that patients with midfrequency hearing loss had complete recovery even without treatment. It has also been shown that 70% of patients with severe sensorineural hearing loss did not respond to therapy.

Patients with moderately severe sensorineural hearing loss responded to steroid therapy. It has been said that they lie in the "Steroid effective zone". It is important to identify patients belonging to this group as they could benefit from steroid administration.

Steroid regimen:

Currently accepted steroid regimen is oral steroid therapy. Prednisolone is administered in doses of 1mg / kg body weight / day for 7 – 10 days. This is followed by a rapid taper of dose. It has also been said that low dose of steroids is ineffective.

Intratympanic steroid administration:

Advantages include:

Drug is directly administered into the site of action
It diffuses through round window membrane into the inner ear
Since inflammation / autoimmunity have been suspected to the causes, steroids could play a role.
There is virtually no systemic side effects

Spinal needle is used for instilling drugs directly into the middle ear cavity. Needle myringotomy is performed at two different sites in the ear drum. Both these holes should be placed in the antero inferior quadrant. They should not be close enough to avoid the complication of causing a large perforation. Drug is instilled through the second hole and the air from the middle ear cavity could be seen bubbling out through the superior opening on being displaced. Dexamethazone preferably in concentration of 10 mg/cc is used. 1ml of it is mixed with 0.1 ml of 1% xylocaine. About 0.5 ml of this solution is injected into the middle ear cavity. Injections are administered twice a week for a period of 6 weeks. Introduction of Silverstein microwick has made the job little bit simpler.

39. Deaf mute

Deaf-mutism is generally due to a loss of hearing before the age of 2 - 3 years which hinders learning to speak. Most cases result from acute infectious diseases such as measles, epidemic meningitis, encephalitis, typhoid, otitis media, toxic effects of drugs, etc. Congenital deaf-mutism is also a possible etiology. Endocrine causes like Pendred syndrome involves deafness along with the presence of goitre. The child's intelligence is normal.

For normal speech to develop in a child, the hearing sensation should be intact. The first three years of life is considered to be crucial for the development of speech. Loss of hearing due to any cause during this crucial period of development causes the development of speech to falter.

Congenitally deaf child is also a mute. In congenital deafness there is failure of normal development of cochlea, leading on to deformities i.e. Mondini defect etc. Congenitally deaf and mute child can be identified by the absence of oculo vestibular reflex. This is the reflex that makes the eye deviate to opposite side when the head is bent to one side. In children with acquired deaf mutism this reflex is intact.

Management:

Rehabilitation is the only way. In the past these children were taught to communicate using sign language. Now a days the children, if they have residual hearing should be provided with hearing amplification devices (hearing aids). They should also undergo a rigorous course of speech therapy.

The recent advances in the field of cochlear implant has added another useful dimension in the management of these patients.

40. Pure tone audiometry

Pure tone audiometry is used to measure the auditory threshold of an individual. The instrument used in the measurement of auditory threshold is known as the audiometer.
Audiometer: An audiometer has been described by the International Electrochemical Commission in 1976 as an instrument used for the measurement of acuity of hearing, and threshold of audibility. There are two types of audiometers widely used. They are:
1. Those that require a subjective response on the part of the patient and
2. Those that require no subjective response from the patient.

Examples include:

1. Pure tone audiometer is the classic example of the first type

2. Impedance audiometer / BERA are examples of the second variety

Components of a Puretone audiometer:

Oscillator: The role of the oscillator in a Puretone audiometer is to generate electronically standardized frequencies within +/- 3% of their nominal value. The frequencies generated are 125, 250, 500,750, 1000, 1500, 2000, 3000, 4000, 6000 and 8000 Hz.

Interrupter switch: The tones presented to the patient should be switched on and off. This feature is important because a continuous tone undergoes decay during a period of time. This switch gives the option of providing the tone in a continuous or an interrupted manner.

Equalization circuit: This circuit contains resistors which helps in equalization of the tones generated. This is because the threshold of human hearing is not uniform, the human ear is most sensitive at frequencies around 2 kHz. It is also insensitive at low or very high frequencies.

Output power amplifier: The signals produced by the oscillator needs to be amplified. The most important characteristic of the amplifier is that it produces very little distortion, and has a good signal to noise ratio. In most audiometers the power amplifier is run at constant high signal output levels.

Hearing level attenuator: The attenuator controls the level of the signal from the audiometer within the range of 110 - 120 dB. The attenuator can be varied in steps of 5 dB. The basic reference point is marked as 'O'. This indicates -5 to -10 dB hearing threshold levels. The attenuator steps should be accurate.

Output transducers: Is of three different types.

1. Ear phones
2. Bone vibrator
3. Loud speaker

The ear phones for audiometers are very special. They cannot be replaced or changed without calibrating the whole equipment. The pre requisites of a good earphones are:

1. It should have a good long-term stability
2. It should have a flat frequency response
3. It should be able to deliver high output sounds.

Bone vibrators: In contrast to ear phones bone vibrators have a limited dynamic frequency range. At low frequencies these vibrators show distortions.

Loud speakers: Are used in testing Paediatric patients. It is used in free field audiometry.

Calibration of the audiometer involves calibration of the audiometer proper, calibration of ear phones, and calibration of bone vibrators. The basic aim of these calibration procedures is to define the audiometric zero for the chosen earphone.
This can be performed using human volunteers or an artificial ear. The calibration of bone vibrators is the same as for earphones except for the measuring device which is different.

Pure tone air conduction testing:

This is a measurement of air conduction thresholds of audibility. Air conduction Threshold is in fact defined as the faintest tone a subject is able to hear via air conduction. In clinical setting pure tone audiogram is performed for two main purposes:

1. To assist in the diagnosis of ear pathology

2. To decide on the appropriate rehabilitation device which can be used to minimize the hearing disability.
Pure tone air conduction threshold is tested using head phones.

Technique of measurement: Some audiologists assess the threshold of air conduction by going from an inaudible to an audible stimulus intensity. This method is known as ascending method of estimation of threshold of hearing, while others assess the threshold of air conduction by going from an audible to an inaudible stimulus intensity. This is known as descending method of threshold estimation.

Instructions to the patient: The patient is instructed to raise the index finger if the sound is heard. The patient should respond even if the sound is faintly heard.

The head phones should be properly seated over the external auditory canal. This step should be performed with care because the patient's pinna comes in various shapes and sizes. Improper placement of head phones will cause threshold variations of even 15 - 20 dB.

The audiometer should be properly checked before performing the test. After the audiometer has had a warm up period, the tester should first place the ear phones on his own ears and listen to various frequencies and intensities of test tones. He should also listen to the masking noise, check for any audible clicks of the interrupter switch.
Before placing the ear phones on the patient, the patient's ear should be examined for the presence of wax. If wax is present it should first be cleaned before the test could be performed. If the ear canal is small or tends to collapse when pinna is pressed, the test could not be valid. If the ear canal tends to collapse when pressure is applied to the pinna, plastic tubes can be inserted into the external canal to prevent such collapses.

To plot the recordings, red color ink is used to plot values of right ear, and blue color ink is used to plot values of left ear.

"Up 5-down 10" method of threshold estimation: This technique is based on Hughson - Westlake ascending technique.

Tones of short duration is used for threshold estimation. This method of threshold estimation involves the following steps:

Step I : The better ear is tested first in order to determine the need for masking.

Step II : Start with a 1000 Hz tone at a level above the threshold to allow easy identification of the tone. This tone is selected because it is an important speech frequency, and the patient is less apt to mistake the frequency. If the patient is suspected to be having a profound hearing loss then the testing should be started with 250Hz frequency. This is because of the fact that the individuals with profound hearing loss often have testable hearing only in the low frequency range.

Step III : The patient's understanding of the listening task should be checked by using both short and long duration test tones. The patient should be instructed to raise the index finger as soon as the sound is heard.

Step IV : During testing, the examiner should vary the interval between tone presentations to avoid telegraphing the stimulus. Tone should not be presented while the attenuator dial is being rotated, because switching artifacts may contaminate the results. As the threshold levels are being reached, a check should be made for the existence of abnormal tone decay. This is done by sustaining the tone for several seconds longer than usual. If the index finger drops before the tone is discontinued, abnormal tone decay should be suspected.

Step V : The starting intensity of the test tone is reduced in 10 dB steps following each positive response, until a hearing threshold level is reached at which the subject fails to respond. Then, the tone is raised by 5 dB, if the subject hears this increment, the tone is reduced by 10 dB; if the tone is not heard then ti is raised by another 5 dB increment. This 5 dB increment is always used if the preceding tone is not heard, and a 10 dB decrement is always used when the sound is heard. The threshold is defined as the faintest tone that can be heard 50% or more of the time, and is established after several threshold crossings.

If there is no response at the maximum output of the audiometer, an arrow pointing downward should be attached to the symbol designating the test ear and placed on the audiogram at the hearing threshold level coinciding with the maximum output for the test frequency. If the tone is heard at the minimum level, the audiogram should be marked in similar fashion but should have the arrow pointing upward.
Step VII : Testing of the second ear should begin with the last frequency used to test the first ear. There is no need to start again with a 1000 Hz tone because if one side of the heard has learned the

listening task, the other side knows it as well. The test is terminated after all desired frequencies have been examined.

Interpretation: With air conduction readings alone, one cannot reliably come to a diagnosis. Even a basic distinction between conductive and sensorineural hearing loss cannot be made with air conduction hearing tests. When there is a hearing loss in air conduction audiogram, it represents a cumulative deficit from the outer, middle, inner ear and retro cochlear system of a subject. Bone conduction audiometry is a must before a classification of the deafness could be made.

Bone conduction audiometry: This is an important measurement of hearing threshold using a bone vibrator. This helps to differentiate conductive from sensori neural hearing loss. The equipment necessary is just a bone vibrator connected to the audiometer. The bone vibrator is placed over the mastoid process of the side to be tested. The auditory threshold is assessed as described for air conduction assessment. The only difference is that the better hearing ear should be masked using a masking tone delivered via a head phone. If the values of bone conduction audiometry is better than that of air conduction, then the patient is said to have conductive deafness. In sensori neural hearing loss both the air and bone conduction curves show a dip. This dip is frequently seen for higher frequencies.
In bone conduction audiometry high frequencies cannot be used for testing. Frequencies above 4000 Hz cannot be used because they are beyond the vibrating capabilities of the bone vibrator.

41. OAE (Otoacoustic Emission)

Definition:

Otoacoustic emissions (OAE) are sounds produced by motile elements of cochlear outer hair cells. These sounds can be recorded easily. These sounds were first identified and reported by Kemp in 1978.

The main function of OAE tests is to assess the function of cochlear hair cells. These tests could be used:

1. To screen children and neonates for hearing disabilities

2. Estimate hearing sensitivity within a limited range of frequencies

3. To differentiate sensory and neural components in sensorineural hearing loss

4. To rule out malingering (functional hearing loss)

Since the measurement of otoacoustic emissions are recorded from the external auditory canal, the integrity of the middle ear and the cochlear function come into play.

Role played by otoacoustic emissions: As explained earlier otoacoustic emissions are generated by the outer hair cells of cochlea. These otoacoustic emissions play the role of cochlear amplifier. In sensori neural hearing loss the cochlear amplification is lost leading to:

a. Reduction in the hearing level

b. Reduction in the clarity of spoken words

Types of Otoacoustic emissions: OAE's are classified according to the stimulus employed to elicit them, or by the mechanism that causes them.

Spontaneous otoacoustic emission: These narrow band continuous signals occur without any stimulus. These signals can be detected in a majority of persons with normal pure tone threshold. The clinical value of this signal is limited, as this is not present in all normal ears. The absence of spontaneous otoacoustic emission does not imply cochlear dysfunction. Synchronized otoacoustic emissions are potentials generated by outer hair cells of cochlea which are synchronized to the external stimuli using time averaging techniques. Measurement of these potentials is difficult and highly cumbersome. They do not have any diagnostic / prognostic value as it is not a consistent feature in all normal ears.

Three types of OAE's have been recorded in response to various stimuli. These are also known as evoked oto acoustic emissions.

Stimulus frequency otoacoustic emission: These potentials are evoked with some kind of acoustic stimulus. The evoking acoustic stimulus is a pure tone one with a low intensity level. Study of these potentials are still in the experimental stage, and hence not widely used.

Transient otoacoustic emission: (TOAES) These potentials are also known as transient evoked otoacoustic emission. Since it was first described by Kemp it is also known as Kemp echos. The time delay between the stimulus and response allows the examiner to isolate these responses. These echos recorded from normal ears always mirrors the spectrum of the stimulating sound impulse. The probe used to record transient otoacoustic emission has two openings, one for the presentation of a single stimulus like a click, and the other opening which is used to record the transient evoked otoacoustic emission. The second opening is connected to a microphone to enable recording to take place.

Clicks are commonly used as stimuli, sometimes tone burst stimuli can also be used. The stimulus used should be 80 - 85dB sound pressure level. The rate of stimuli should be at least 60 / minute. When present TOAES occur at frequencies of 500 - 4000 Hz

Distortion Production Otoacoustic emission: (DPOAEs): These are low intensity signals that occur during stimulation of the ear. A common way to record them is to present the ear with two continuous signals called the primary tones and analyze the spectrum of sounds detected at the external auditory canal. The intensity levels of the signals used are 55 dB and 65 dB respectively.

Prerequisites for obtaining otoacoustic emissions:

1. Unobstructed external auditory canal

2. Perfect seal of external auditory canal with the probe

3. Optimal positioning of the probe

4. Absence of middle ear pathology

5. Functioning cochlear outer hair cells

6. A relatively still patient

7. Quiet recording environment

Interpretation of the recordings made:

Spontaneous otoacoustic emissions: are commonly found in 50% of individuals with normal hearing. It is generally not seen in patients with less than 30 dB hearing level. If spontaneous otoacoustic emissions are elicited in a patient the cochlea could be assumed to be in good health. These emissions are more bilateral than unilateral. They are more commonly recorded in females than in males.

These spontaneous otoacoustic emissions are not associated with tinnitus because the associated cochlear abnormality causes the SOAEs to disappear.

Transient otoacoustic emissions: are usually used to screen neonates for hearing disabilities. These impulses can be recorded only in response to short and transient stimuli. These impulses have a very limited frequency specificity. The presence of these emissions suggests the cochlear sensitivity in the region of 20 - 40 dB or better.

Distortion product otoacoustic emissions: These emissions have greater frequency specificity. These potentials are useful in detection of early detection of cochlear damage i.e. due to noise or drug exposure.

Causes of absent otoacoustic emissions:

Non pathological:

1. Poor probe tip placement

2. Standing waves

3. Cerumen occlusion

4. Vernix caseosa in infants

5. Un cooperative patient

Pathologic causes:

Outer ear: Stenosis, otitis externa, cysts etc.

Tympanic membrane: Perforations. Grommets usually don't complicate recordings

Middle ear: Otosclerosis, ossicular disruption, cholesteatoma, otitis media

Cochlea: Exposure to ototoxic drugs, Noise exposure

Central auditory disorders don't affect otoacoustic emissions.

Conclusion:

Otoacoustic emissions play an important role in screening infants for disorders of hearing.

42. Impedance audiogram

The primary purpose of impedance audiometry is to determine the status of the tympanic membrane and the middle ear. It is also otherwise known as Tympanometry or acoustic immittance test. The secondary purpose of this investigation is to evaluate the acoustic reflex pathway which include the 7th and 8th cranial nerves and the brain stem. This test should not be used to assess the

sensitivity of hearing and the results of this test should always be viewed in conjunction with the results of pure tone audiogram.

Impedance audiometry is a measurement of energy or air pressure which involves the external auditory canal, the ear drum, ossicular chain, stapedius muscle, cochlea, 7th cranial nerve, 8th cranial nerve and the brain stem. This test is affected by the mass, mobility and resistance systems of the external and middle ear cavities.

The following tests have been included under the battery of impedance audiometry:

1. Tympanometry

2. Eustachean tube function

3. Tests to identify perilymph fistula

4. Acoustic reflex threshold

5. Acoustic reflex decay

These tests can be used to identify the following pathologies involving the peripheral and central portions of hearing.

a. Middle ear effusion

b. Ear drum perforations including patency of eustachean tube

c. Tympanosclerosis

d. Hypermobile ear drum

e. Eustachean tube dysfunction

f. Glue ear

g. Otosclerosis

h. Ossicular discontinuity

g. Acoustic neuroma

h. Facial nerve function

i. Hearing loss

j. Brain stem disorders

Tympanometry: Measures the sound reflected from the ear drum while the pressure of the external canal is varied by the operator. It aids in the assessment of outer ear, middle ear and the eustachean tube. This test should not be performed in infants below the age of 7 months because the suppleness of the cartilage of the external canal may produce misleading results.

Procedure: First the probe is inserted into the external auditory canal till a airtight seal is obtained. Probe tone is presented typically at 226Hz into the ear canal while the air pressure of the canal is altered between +200 and - 400 decapascals. The maximum compliance occurs when the pressure of the external auditory canal and the middle ear becomes equal. Only at this pressure maximal acoustic transmission occur through the middle ear. The compliance peak therefore indicates therefore indicates the pressure of the middle ear implying efficacy of the eustachean tube function. The height of the compliance peak indicates the mobility / stiffness of the tympanic membrane or the middle ear cavity.

The term static compliance indicates the height of the tympanogram at its peak, and it is the measurement of the mobility of the whole system.

Classification of tympanograms:

The classification system introduced by Jerger is commonly used to classify various types of tympanograms. Other systems have been proposed, but none of them are in common use.

Type A curve: Suggests normal middle ear function. The compliance peak occurs between -150 - +100 dapa. The value of compliance ranging between 0.2 - 2.5 millimhos. This type of curve is also known to occur in early stages of otosclerosis.

Type As curve: is a shallow curve suggesting a stiffened middle ear system. Compliance peak occurs at -150 - + 100 dapa. The compliance value is less than 0.2 mmhos. This curve is commonly found in patients with glue ear, stiffened ear drum, or otosclerosis.

Type Ad curve: is a deep curve suggests a flaccid ear drum or middle ear system, ossicular disruption. Usually ossicular disruption gives a compliance higher than the recording parameters (in fact the recording goes off chart). The compliance peak occurs between -150 - + 100 dapa. The compliance value is more than 2.5 mmhos.

Type B: is a flat curve with no compliance peak. This Type B curve must always be interpreted in conjunction with the ear canal volume. Average ear canal volume in children ranges between 0.42 - 0.97 ml, while in adults it ranges between 0.63 - 1.46 ml.

Type B curve with normal ear canal volume suggests otitis media.

Type B curve with small canal volume suggests that the ear canal could be occluded by the presence of wax, or the probe of the impedance audiometer has not been properly placed.

Type B curve with large canal volume suggests that there could be perforation of the ear drum. This curve is caused due to a patent pressure equalization system.

Type C curve: This curve suggests a significant negative pressure in the middle ear, or eustachean tube dysfunction. Compliance is recordable but the peak compliance occurs at less than -150 dapa.

Assessing eustachean tube functioning by impedance audiometry:

The function of the eustachean tube can easily be assessed by reading the tympanograms. Type A tympanograms reflect a normal middle ear function which is only possible in the presence of a normally functioning eustachean tube. Similarly Type C tympanograms indicate significant negative pressure in the middle ear implying that the eustachean tube is blocked. If there is tympanic membrane perforation a Type B curve will be produced. In this situation the eustachean tube function cannot be assessed using a tympanogram. However, an indirect assessment of the pressure equalization function of the eustachean tube can be made by increasing the probe pressure in the external ear canal, asking the patient to swallow then assessing whether the eustachean tube is able to clear the increased pressure applied to the external ear canal.

Testing for the presence of absence of perilymph fistula:

This can be indirectly assessed by the presence of intense giddiness along with nystagmus when the external canal pressure in increased by increasing the probe pressure. This sign is also known as the Hennebert's sign. This sign is manifested only in the presence of perilymph fistula.

Eliciting acoustic reflex thresholds: This is a measure of the stapedial muscle reaction to exposure to high intensity sounds. When the stapedius muscle contracts in response to sound it stiffens the ossicles and the ear drum altering the compliance values which can be measured using an impedance audiometer. The recording is ideally made at a single pressure setting i.e. the pressure which shows the maximum compliance. The reflex on the opposite side also is tested since it is a bilateral reflex. The sound frequencies used to test this reflex are 500, 1000, 2000 and 4000Hz. For screening purposes, it is sufficient if 1000Hz is used.

The acoustic reflex cannot be recorded in patients with a type B tympanogram. It also cannot be recorded in patients with severe profound sensorineural hearing loss. The reflex may be attenuated in the presence of conductive deafness. Using this test, it is possible to assess the whole of the

acoustic reflex pathway. If the pathway is affected at central level then ipsilateral recordings will be normal with absent contralateral acoustic reflexes.

43. MERI Index

Introduction:

The term Middle ear risk index is used to predict the success rate of middle ear reconstruction procedures. For accurate prediction of the surgical results of middle ear ossiculoplasty the status of middle ear and its ossicles must be ascertained. Austine Kartush classification has been used as a method to define the pre reconstruction ossicular status.

Austine Kartush classification:

This classification uses middle ear ossicular status. Four different groups have been identified:

Group A - Malleus and stapes present (commonly seen status) because of precarious vascularity of incus

Group B - Malleus and foot plate of stapes present

Group C - Malleus absent and stapes present

Group D - Malleus and stapes suprastructure absent

Kartush added three more classes as a modification of this scheme in include ossicular fixity even when all three ossicles are present.

O - Intact ossicular chain

E - Ossicular head fixation

F - Stapes fixation

Middle ear risk index includes:

1. Austin Kartush classification of ossicular defects

2. Ear drum perforation

3. Cholesteatoma

4. Belluci classification

Weightage is given to these 4 parameters to arrive at the middle ear risk index.

Belluci classification uses otorrhea as an index.

Perforation if present adds a value of 1 to the risk index

Cholesteatoma if present adds a value of 2 to the risk index

Austin Kartush classification

1. M+I+S += 0

2. M+S+=1

3. M-S+ = 3

4. M-S- = 4

5. Ossicular head fixation = 2

6. Stapes fixation = 3

Presence of effusion / granulation in the middle ear adds 2 to the risk index

History of previous surgery adds 2 to the risk factor

History of smoking adds another 2 to the risk factor

Totaling all these factors adds to the middle ear risk index

44. Stapedial reflex

The acoustic reflex, also known as the stapedius reflex refers to an involuntary muscle contraction of the stapedius muscle
in response to a high-intensity sound stimulus. Due to ease of administration and information yielded, the acoustic reflex is
considered one of the most powerful differential diagnostic audiological procedures.

Acoustic reflexes do not measure hearing threshold. Rather, they measure reflected energy which is a function of stapedius

muscle contraction; it allows one to indirectly assess the middle ear, cochlea and neural innervation of the stapedius muscle.
As the acoustic reflex is involuntary and bilateral, it is replicable and provides valuable diagnostic information when comparing the amount of reflected energy according to signal intensity, as well as the
presence of ipsilateral and bilateral acoustic reflexes.

Neural mechanisms mediate the acoustic reflex that results in involuntary stapedius muscle contraction stiffening the stapes
within the middle ear. It is presumed that the physiologic reason for the reflex is that it serves as an inhibitory response to reduce the sound intensities reaching the inner ear by as much as 20 dB evoked when individuals vocalise.

Once a high intensity auditory stimulus is initiated and reaches the cochleae, neural impulses from the auditory nerves (CN VIII) ascend from both cochleae to each ipsilateral ventral cochlear nucleus (VCN). From VCN the reflex has two main neural pathways: one passes from the VCN directly to the ipsilateral facial motor nucleus (CN VII) that directly innervates the stapedius muscle via the facial nerve and its stapedius branch; the other passes from the VCN to the superior olivary complex (SOC) before the impulses cross at the brainstem to innervate both ipsilateral and contralateral facial motor nuclei.

It is important to realize that a bilateral acoustic reflex occurs i.e. stimulating one ear (ipsilateral ear) elicits an acoustic reflex in both the ipsilateral and contralateral ears.

Factors affecting acoustic reflexes:

1. Ipsilateral conductive hearing loss:

Anything that obstructs transmission of a sound signal to the cochlea in the stimulated ear results in reduced or absent contraction of the stapedius muscles. Middle ear disorders therefore easily mask an acoustic reflex. Forty percent of patients with conductive hearing loss of >20 dB HL do not have an acoustic reflex. Once conductive
hearing loss reaches 40 dB HL, about 80% of patients do not have a reflex.

2. Ipsilateral sensorineural hearing loss:
This may obstruct transmission of a signal beyond the cochlea of the stimulated ear and usually causes reduced or absent contraction of the stapedius muscles. However, because of recruitment of loudness in cochlear sensorineural hearing loss, acoustic reflexes may occur even within the expected normal or partially elevated intensity range in the presence of mild or moderate-to-severe sensorineural hearing loss.

3. Stapes fixation: The stapes footplate is more-or-less fixed to the surrounding bone with otosclerosis (and sometimes with tympanosclerosis). This may interfere with the acoustic reflex in two
ways: it causes a conductive hearing loss; and reduced mobility of the stapes prevents stiffening of the tympanic membrane when the stapedius muscle contracts.

4. Ossicular disarticulation: As with otosclerosis, it causes a conductive hearing loss, and if located lateral to the stapes, prevents stiffening of the ossicles and tympanic membrane when
the stapedius muscle contracts.

5. Middle ear effusion: This causes a conductive hearing loss, and reduces compliance of the tympanic membrane and middle ear structures and may mask the presence of an acoustic reflex.

6. Tympanic membrane perforation: This causes a conductive hearing loss. Furthermore, due to the perforation, changes in compliance caused by contraction of the stapedius muscle cannot be measured.

7. Negative/abnormal middle ear pressure: To maximize the likelihood that a reflex will be detected, the pressure on either side of the tympanic membrane has to be equal for the tympanic membrane to be at or near the point of maximum compliance This necessitates one to match the peak pressure of the tympanogram to the actual middle ear pressure so that valid acoustic reflex threshold values may be obtained.

8. Facial nerve dysfunction: The stapedius muscle is innervated by the facial nerve. Contraction of the stapedius muscle has to occur for both ipsi- and contralateral acoustic reflexes.

Interpretation of acoustic reflexes

Acoustic reflexes may be reported as:
- Ipsilateral: Reflex recorded in ear to which auditory stimulus is presented
- Contralateral: Reflex recorded in ear contralateral to which auditory stimulus is presented
- Partially present: Reflex present at some frequencies and absent at others
- Elevated threshold: Reflex thresholds elicited >100 dB HL
- Absent reflex: No reflex elicited

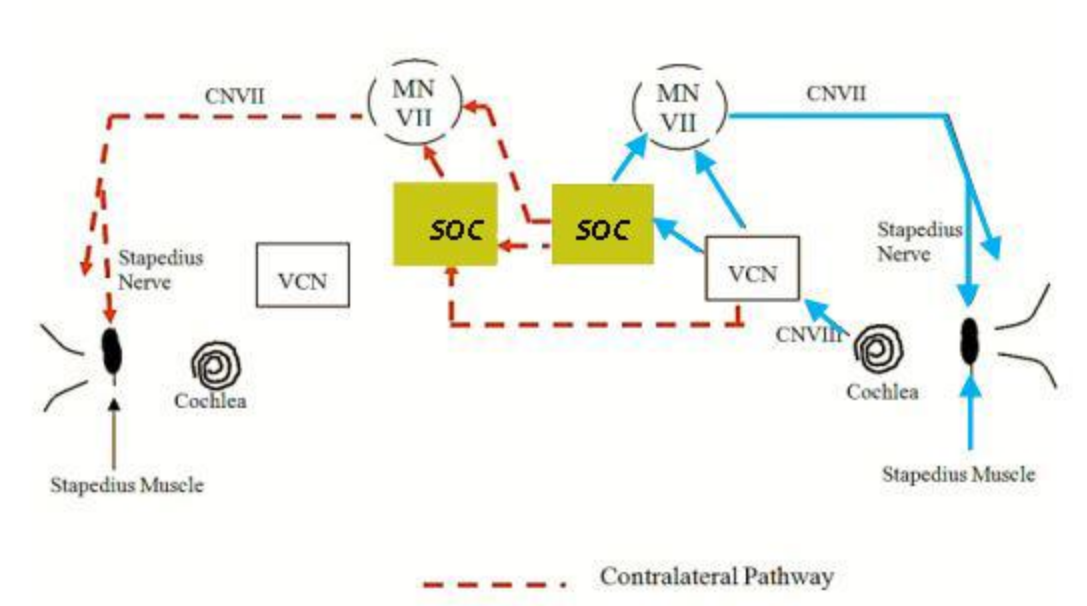

Image showing acoustic reflex pathway

45. Tympanometry

Already provided. Repeat question

46. Kobrak test

This is a quick office procedure where cold water is used for the test. In this test the patient is seated with head tilted 60 degrees backwards to ensure that the horizontal canal is in vertical position. The ear is irrigated with cold water for 60 seconds. The temperature of the irrigated water should be 7 degrees centigrade below the normal body temperature. Initially 5 ml is used. If there is no response then the volume should be increased to 10, 20 and 40 ml if needed. In normal persons the nystagmus starts beating towards the opposite ear when 5 ml is irrigated. If higher volumes of water is needed to elicit nystagmus then the labyrinth is considered to by hypoacitve. If there is no response even after irrigating 40 ml of water then it indicates dead labyrinth.

47. CP angle tumors

Cerebellopontine angle tumors are encountered rather more commonly than anticipated by otolaryngologist. This group of tumors account for nearly 10% of all intracranial tumors. These tumors are rather fatal when not treated. Studies reveal that acoustic neuromas (vestibular schwannomas) constitute nearly 80% of all cerebellopontine angle tumors. Other tumors that could involve this area include:

1. Meningiomas
2. Dermoid tumors
3. Arachnoid cysts
4. Lipoma
5. Metastatic tumors
6. Vascular tumors

Vestibular schwannomas:
These are the most common of CP angle tumors. It constitutes about 75-80% of all CP angle tumors. The overall incidence rate for vestibular schwannomas is rather difficult to ascertain due to the fact that majority of these tumors remain silent as they have a very slow growth rate. Routine autopsy studies have put the estimates to be about 2.5%. With the advent of sensitive imaging modalities more and more of these cases are being picked up at early stages.

These tumors arise from the vestibular segment of the 8th cranial nerve. Studies reveal that its incidence is approximately equal between superior and inferior vestibular nerves. These tumors have been thought to develop from the Schwann cell, close to the glial – Schwann junction. Current knowledge of course refutes this idea. These tumors most commonly arise lateral to this junction close to the internal acoustic meatus. It arises close to the sharpa's ganglion.
This ganglion contains the largest number of Schwann cells. Hence the more appropriate term to describe this disorder is “Vestibular schwannoma”. Rarely these tumors may also develop from the cochlear
division of the 8th nerve.

Clinical features of vestibular schwannomas:

Diagnosis of vestibular schwannomas needs high degree of astuteness and alertness on the part of the physician. Since this is a very slow growing tumor, there is ample time for brain accommodation to kick in.

Symptoms are most commonly vestibular / auditory and is unilateral. The rate of progression of symptoms pertaining to the vestibular schwannoma depends on the rate of growth of the tumor.

Intracanalicular tumors: These tumors are characterized by hearing loss (sensori neural), tinnitus and vestibular dysfunction (giddiness). Tumor growth extending into the cerebellopontine angle: In this stage deafness worsens, disequilibrium becomes more evident. As the tumor grows into the cerebellopontine angle it causes compression in this area causing involvement of trigeminal nerve. These patients present with midfacial hypesthesia. When the tumor expands further it could cause hydrocephalus and vision loss.
According to Cushing the diagnosis of acoustic schwannoma could be made with reasonable degree of certainty only when auditory manifestations definitely precede other symptoms of CP angle tumor.
These patients classically manifest with hearing loss which could be described by the patient as distortion in hearing. This could be evident while the patient attempts to converse through the phone. This distortion could be due to reduced speech discrimination. Some patients may have sudden hearing loss. Even though this is rare the presence of sudden hearing loss does not completely exclude the diagnosis of acoustic schwannoma. Studies reveal that sudden hearing loss in these patients could be caused by acute compression of the auditory nerve.

Ocular symptoms in these patients include:

1. Absent corneal reflex. This usually precedes hypesthesia of one side of the face due to involvement of the trigeminal nerve
2. Nystagmus
3. Diplopia – due to the rare involvement of 6th cranial nerve
4. Visual blurring – is very rare. When present papilledema should be suspected. If untreated it could lead to optic atrophy resulting in tunnel vision. This papilledema is usually due to increased intracranial tension.

Facial weakness is very rare in patients with vestibular schwannomas as the facial nerve tolerates even extensive stretching. Presence of facial weakness should prompt the suspicion of some other tumor in the CP angle rather than schwannoma.
Symptoms caused due to involvement of lower cranial nerves are rather rare and could cause symptoms like dysphagia, hoarseness and aspiration. Symptoms due to cerebellar involvement occur rather late in this disorder.

These symptoms include:
1. In co-ordination / wide based gait.
2. Tendency to fall forward

Presence of intense head ache and vomiting should arouse suspicion of hydrocephalus.

Clinical examination of a patient with acoustic schwannoma:
Patients who are suspected to be suffering from unilateral auditory / vestibular symptoms should be thoroughly examined. During routine otoscopy sensation over the posterior bony meatal wall should

be looked for. Decreased sensation in this area is known as “Hitselberger’s sign” a clear pointer towards the diagnosis of schwannoma. This is due to the involvement of sensory branches of 7th cranial nerve which is more sensitive to compression by schwannomas than its motor counterpart.

This sign could even be present in patients with small tumors in the CP angle area. Eyes should be examined for the presence of nystagmus. Extraocular movements are also accessed.

Tests for facial sensation:

Facial sensation could be blunted in these patients due to the involvement of 5th cranial nerve. Sensation should be tested for both pain and touch. This is usually performed using pin prick / wisp of cotton. It is imperative to test for both these sensations.

Evaluation of corneal sensation:
Corneal sensation can be evaluated using a wisp of cotton and looking for blink reflex. The cornea should be stimulated with a wisp of cotton while the patient is looking up, this is to ensure that the patient is not aware of the wisp of cotton coming.

Checking the integrity of masseter and temporalis muscle:
This is done to rule out the involvement of the motor component of the 5th cranial nerve. The patient is asked to clench the teeth while the masseter and temporalis muscle are palpated. Rigidity of these muscles indicate that the motor component of the 5th cranial nerve is intact.

Audiometric tests:

These tests should help to identify retrocochlear deafness which is commonly seen in these patients.
Previously the following tests were commonly performed to rule out retrocochlear deafness.
1. SISI – Short increment sensitivity index
2. ABLB – Alternate loudness balance test
3. Tone decay test

MRI:
This is actually the gold standard investigation in the diagnosis of vestibular nerve schwannomas. Introduction of gadolinium scanning has in fact increased the accuracy of MRI in the diagnosis of acoustic schwannomas as it is preferentially taken up by schwannomas. Now it is a must that gadolinium be used in all patients before scanning to accurately diagnose the presence of schwannomas in the Cerebello pontine angle. In gadolinium enhanced images the schwannomas enhance brightly in both T1 and T2 weighted MRI images. T1 weighted images are brighter on fat density and T2 weighted images which are brighter in fluid density.

Signs and symptoms of CP angle tumors:

1. Unilateral hearing loss
2. Unilateral tinnitus
3. Aural fullness
4. Lightheadedness
5. Facial hypesthesia
6. Decreased corneal reflex

Goals of treatment of vestibular schwannomas:

1. Primary objective is to preserve life
2. Preservation of facial function
3. If serviceable hearing is present preoperatively preservation of it
4. Complete tumor removal if feasible

Treatment algorithm of CP angle tumors:

1. Observation
2. Surgery – Trans-lab approach, middle cranial fossa approach, retrosigmoid suboccipital approach
3. Stereotactic gamma radiation

48. Glomus Tumor

Definition: Glomus jugulare is defined as a collection of ganglionic tissue within the temporal bone in close relationship with the jugular bulb. The jugular bulb is closely related to the floor of the middle ear cavity (i.e. Hypotympanum).

History: Valentine in 1840 described this condition as ganglia tympanica. Guild recognized its histological relationship with the carotid body. Lattes and Waltner suggested that the ideal generic term for these structures is non-chromaffin paraganglioma.

Paraganglia cells are derived from the neural crest and are found widely distributed in the autonomic nervous system. Paraganglia having negative chromaffin reaction are termed non - chromaffin paraganglia. Guild in his anatomical studies on temporal bones found that on an average three glomus bodies were found in them. They were usually found in close relationship with the tympanic branch of glossopharyngeal nerve or the auricular branch of vagus. These bodies were

supplied with non-medullated sensory fibers from the adjacent nerves. They are supplied by branches from the ascending pharyngeal artery.
Even though the paraganglia cells are closely related to either the tympanic branch of glossopharyngeal nerve or the auricular branch of vagus, their position in the temporal bone is highly variable. Commonly they are found in the adventitial layer of the jugular bulb. In about 25% of cases they may be found over the mucosa of the promontory. Histologically, they resemble carotid body. It contains epithelioid cells interspaced in a highly vascular stroma of capillary and precapillary vessels. The proportion of the cellular and stromal components vary. Guild classified glomus tumors into two types depending on the amount of cellular and stromal components:
1. Cellular glomus bodies - when the cellular component is predominant
2. Vascular glomus bodies - when the vascular stromal component predominates.
Their sizes could be variable, but mostly they are ovoid in shape.

Paragangliomas of the temporal bone are generally divided into those that originate within the middle ear, glomus tympanicum tumors, and those that originate within the jugular fossa, glomus jugulare tumors. This latter term, however, is often used to refer to large tumors where the origin is difficult to determine. The predominance of the paraganglia within the jugular fossa likely accounts for the increased frequency of tumors with this origin. Classification systems that have been developed for temporal bone paragangliomas are used for staging purposes. surgical planning, and comparison among different therapeutic modalities.

Incidence: Glomus jugulare occurs in about 1 in 100000 patients. It is 6 times more common in females when compared to males.

Hereditary pattern: It shows autosomal dominant inheritance with variable penetrance.

Endocrine activity: Even though these tumors are considered non chromaffin paragangliomas with no endocrine activity, some cases with endocrine activity by these tumors have been reported. It is hence important to look for evidence of endocrine activity by urine estimation of VMA (Vanillylmandelic acid).

Glomus tumors sometimes may show multicentric presentation i.e. present in both ears, or in conjunction with other paragangliomas. The carotid body being commonly the second site.

Pathophysiology: Glomus tumors are encapsulated, highly vascular, and locally invasive tumors. Inside the temporal bone they tend to expand along the pathway of least resistance such as air cells, vascular lumen, skull base foramina and eustachean tube. They also invade and erode bone in a lobular fashion. The middle ear ossicles are commonly spared. Initially skull base erosion occurs in the region of jugular fossa and postero inferior part of petrous bone. Later on extension occurs to the mastoid and adjacent occipital bone.
The parenchyma of the paraganglia consists of 2 primary cell types. Type I cells are more common and are typically round with indistinct cell borders. Type II cells are smaller and irregularly shaped.

Presentation: These tumors are slow growing, with very little symptoms. The diagnosis may easily be missed. In fact the average delay between the onset of symptoms and diagnosis varied from 6 years to 15 years. The first symptoms generally follow middle ear involvement is easily overlooked. Pulsatile tinnitus and conductive deafness are the common presenting symptoms. A red mass behind an intact ear drum (rising sun sign) may also be seen. In some 30% of cases cranial nerve palsies are common. Facial nerve is affected most commonly.

Presenting features of Glomus jugulare:
1. Deafness - 69%
2. Middle ear mass - 75%
3. Pulsatile tinnitus - 55%
4. Imbalance - 8%
5. Otorrhoea - 5%
6. Facial palsy - 8%
7. Endocrine syndrome - 3%
8. Cranial nerve deficits
Hoarseness - 16%
Dysphagia - 16%
9. Headache - 15%
10. Visual disturbance - 6%
11. Presence of headache indicates intracranial extension
12. Dural sinuses may be involved may mimic sinus thrombosis

Clinical features: Otoscopic examination reveals a characteristic, pulsatile, reddish-blue tumor behind the tympanic membrane that often is the beginning of more extensive findings (ie, the tip of the iceberg). When the drum is examined under a microscope will show a pulsation of the reddish mass behind the drum. On seigalisation the mass blanches. This sign is known as Brown's sign. This is pathognomonic of glomus tumor.
Audiologic examination reveals mixed conductive and sensorineural hearing loss. The sensorineural component tends to be more significant with larger tumors.

Classification:

Glasscock - Jackson classification of temporal bone paraganglioma:

1. Type I : Small tumor involving the jugular bulb, middle ear and mastoid.
2. Type II: Tumor extending under the internal auditory canal. There may be intracranial extension.
3. Type III: Tumor extending into the petrous apex. There may be intracranial extension.
4. Type IV: Tumor extending beyond the petrous apex into the clivus and infratemporal fossa. There may be intracranial extension.

The Fisch classification of glomus tumors is based on extension of the tumor to surrounding anatomic structures and is closely related to mortality and morbidity.

Fisch classification:

1. Type A tumor - Tumor limited to middle ear (carries the best prognosis)
2. Type B tumor - Tumor limited to the tympanomastoid area with no infralabyrinthine compartment involvement
3. Type C tumor - Tumor involving the infralabyrinthine compartment of temporal bone with extension to petrous apex
This is divided into three types: C1, C2 and C3.
Type C1 - Tumor with limited involvement of the vertical portion of the carotid canal
Type C2 - Tumor invading the vertical portion of the carotid canal
Type C3 - Tumor invasion of the horizontal portion of the carotid canal
4. Type D tumor has 2 types

Type D1 - Tumor with an intracranial extension less than 2 cm in diameter
Type D2 - Tumor with an intracranial extension greater than 2 cm in diameter

Investigations: Radiological investigations help in the diagnosis.

Plain X ray skull: May show enlargement of lateral jugular foramen and jugular fossa.

CT scan and Contrast MRI using Gadolinum enhancement is very helpful in delineating tumor extension.

Treatment:

Treatment is mainly surgical. Complete resection of the mass is curative. Since it is a highly vascular tumor pre op intravascular embolisation may help to reduce bleeding during surgery.
The particular surgical approach used to resect temporal bone paragangliomas depends on the location and extent of the tumor. Paragangliomas originating from the promontory of the middle ear and isolated to the mesotympanum can be resected by elevating the tympanic membrane and removing the tumor using microdissection techniques. If the tumor extends into the hypotympanum or the mastoid, a tympanomastoidectomy is performed and the tumor resected.

In extensive Fisch type 3 tumors the mass can be approached with help from neurosurgeons. The skull base approach ensures better exposure of the mass and facilitates complete resection.

Management of Fisch type 4 tumors is highly controversial. Irradiation of the mass has been tried with very little effect. Considering the slow growth rate of these tumors with a very long doubling time, these patients are best left alone with symptomatic treatment of the complications.

Complications of surgery:

Complications of surgery include death, cranial nerve palsies, bleeding, cerebrospinal fluid (CSF) leak, meningitis, uncontrollable hypotension/hypertension, and tumor regrowth.

49. Myringoplasty

Definition: Myringoplasty is a procedure used to seal a perforated tympanic membrane using a graft material.
Temporalis fascia is the commonly used graft material because:
1. It is an autograft with excellent chance of take
2. It is available close to the site of operation making its harvest easier
3. It has a low basal metabolic rate, brightening its success rate
4. Its thickness is more or less similar to that of tympanic membrane
There are two available methods of performing myringoplasty:
Overlay technique
Under lay technique
Overlay technique: This is a difficult technique to master. Here the graft material is inserted under the squamous (skin layer) of the ear drum. It is a difficult task peeling only the skin layer away from the tympanic membrane, placing the graft over the perforation and redraping the skin layer.
Underlay technique: This is a simpler and commonly used technique. Here the graft is placed under the tympano meatal flap which has been elevated hence the name under lay. The major advantage of this procedure is that it is easy to perform with a good success rate.
Indications of Myringoplasty:
1. Central perforation which has been dry at least for a period of 6 weeks.
2. As a follow up to mastoidectomy procedure to recreate the hearing mechanism

Prerequisites for myringoplasty:
1. Central perforation which has been dry for at least 6 weeks
2. Normal middle ear mucosa
3. Intact ossicular chain
4. Good cochlear reserve

Procedure: Firstly, a temporalis fascia of adequate site must be harvested and allowed to dry.
The surgery is performed under local anesthesia. Temporalis fascia graft is harvested under local anesthesia conventionally and allowed to dry. The external auditory canal is then anesthetized using 2 % xylocaine mixed with 1 in 10,000 adrenaline injection. About 1/2 cc is infiltrated at 3 - o clock, 6 - o clock, 9 - o clock, and 12 - o clock positions about 3mm from the annulus. The patient is premedicated with intramuscular injections of 1 ampule fortwin and 1 ampule Phenergan.

Step I: Freshening the margins of perforation - In this step the margins of the perforation is freshened using a sickle knife of an angled pick. This step is very important because it breaks the

adhesions formed between the squamous margin of the ear drum (outer layer) with that of the middle ear mucosa. These adhesions if left undisturbed will hinder the take up of the neo tympanic graft. This procedure will in fact widen the already present perforation. There is nothing to be alarmed about it.
Step II: This step is otherwise known as elevation of tympano meatal flap. Using a drum knife a curvilinear incision is made about 3 mm lateral to the annulus. This incision ideally extends between the 12 - o clock, 3 - o clock, and 6 - o clock positions in the left ear, and 12 - o clock, 9 - o clock and 6 - o clock positions in the right ear. The skin is slowly elevated away from the bone of the external canal. Pressure should be applied to the bone while elevation. This serves two purposes:
1. It prevents excessive bleeding
2. It prevents tearing of the flap
This step ends when the skin flap is raised up to the level of the annulus.

Step III: Elevation of the annulus and incising the middle ear mucosa. In this step the annulus is gradually lifted from its rim. As soon as the annulus is elevated a sickle knife is used to incise the middle ear mucosal attachment with the tympano meatal flap. This is a very important step because the inner layer of the remnant ear drum is continuous with the middle ear mucosa. As soon as the middle ear mucosa is raised, the flap is pushed anteriorly till the handle of the malleus becomes visible.

Step IV: Freeing the tympano meatal flap from the handle of malleus. In this step the tymano meatal flap is freed from the handle of malleus by sharp dissection of the middle ear mucosa. Sometimes the handle of the malleus may be turned inwards hitching against the promontory. In this scenario, an attempt is made to lateralize the handle of the malleus. If it is not possible to lateralize the handle of the malleus, the small deviated tip portion of the handle can be clipped. The handle of the malleus is freshened and stripped of its mucosal covering.

Step V: Placement of graft (underlay technique). Now a properly dried temporalis fascia graft of appropriate size is introduced through the ear canal. The graft is gently pushed under the tympano meatal flap which has been elevated. The graft is insinuated under the handle of malleus. The tympano meatal flap is repositioned in such a way that it covers the free edge of the graft which has been introduced. Bits of gelfoam is placed around the edges of the raised flap. One gel foam bit is placed over the sealed perforation. This gelfoam has a specific role to play. Due to the suction effect created it pulls the graft against the edges of the perforation thus preventing medialization of the graft material.

50. Cochlear implants

A cochlear implant (CI) is a surgically implanted neuroprosthetic device to provide a person with moderate to profound sensorineural hearing loss a modified sense of sound. CI bypasses the normal acoustic hearing process to replace it with electric signals which directly stimulate the auditory nerve. A person with a cochlear implant receiving intensive auditory training may learn to interpret

those signals as sound and speech. However, one third of deaf children do not develop language if they are on a CI program alone and have no sign language input.

The implant has two main components. The outside component is generally worn behind the ear, but could also be attached to clothing, for example, in young children. This component, the sound processor, contains microphones, electronics that include Digital Signal Processor chips, battery, and a coil which transmits a signal to the implant across the skin. The inside component, the actual implant, has a coil to receive signals, electronics, and an array of electrodes which is placed into the cochlea, which stimulate the cochlear nerve.

The surgical procedure is performed under general anesthesia. Surgical risks are minimal but can include tinnitus, facial nerve bruising and dizziness.

From the early days of implants in the 1970's and the 1980's, speech perception via an implant has steadily increased. Many users of modern implants gain reasonable to good hearing and speech perception skills post-implantation, especially when combined with lipreading. However, for pre-lingually deaf children the risk of not acquiring spoken language even with an implant may be as high as 30%. One of the challenges that remain with these implants is that hearing and speech understanding skills after implantation show a wide range of variation across individual implant users. Factors such as duration and cause of hearing loss, how the implant is situated in the cochlea, the overall health of the cochlear nerve, but also individual capabilities of re-learning are considered to contribute to this variation, yet no certain predictive factors are known.

51. Facial palsy

Severity of nerve injury can be graded using the following mechanism:

1. Neuropraxia - This is a conduction block where flow of axoplasm through the axons is partially obstructed.

2. Axonotmesis - Injury to axons

3. Neurotmesis - Injury to nerve

Sunderland classification:

First degree paralysis:

Partial block to flow of axoplasm; no morphological changes could be seen. Recovery of function is complete (neuropraxia).

Second degree paralysis:

Loss of azons, but endoneural tubes remain intact. During recovery, axons will grow into their respective tubes and the result is good (axonotemesis).

Third degree paralysis:

In this there is injury to endoneurium. During recovery phase, axons of one tube can grow into another. Synkinesis can occur (neurotmesis).

Fourth degree paralysis:

There is injury to perineurium in addition to all of the above. Scarring will impair regeneration of fibres (partial transection).

Fifth degree paralysis:

There is injury to epineurium seen in this type in addition to all the facts stated above (complete transection).

The first three degrees are seen in viral inflammatory disorders, while the fourth and fifth degrees are seen in surgical / accidental trauma to then nerve or in neoplasms.

House Brackmann's classification of facial nerve function grading:

This grading system is used to characterize the degree of facial nerve paralysis. In this grading system, grade I is assigned to normal function, while grade VI would represent complete paralysis. the intermediate grades usually vary according to function at rest and with effort.

Grade I - Normal facial function in all areas

Grade II (mild dysfunction) -

Gross - Sight weakness which is noticeable on close inspection

May have slight synkinesis

At rest, normal symmetry and tone

Motion - Forehead - moderate to good function

Eye - Complete closure with minimal effort

Mouth - Slight asymmetry

Grade III (Moderate dysfunction)

Gross - Obvious but not disfiguring difference between sides

Noticeable (not severe) synkinesis, contracture or hemifacial spasm

Motion - Forehead - Slight to moderate movement

Eye - Complete closure with effort

Mouth - Slightly weak with maximum effort

Grade IV (Moderately severe dysfunction)

Gross - Obvious weakness / disfiguring asymmetry

At rest, normal symmetry and tone

Motion - Forehead - None

Eye - Incomplete closure

Mouth - Asymmetrical with maximum effort

Grade V (Severe dysfunction)

Gross - Only barely perceptible motion

At rest, asymmetry

Motion - Forehead - None

Eye - Incomplete closure

Mouth - Slight movement

Grade VI (Total paralysis)

No movement at all

Causes of facial paralysis:

Causes for facial nerve paralysis could be due to:

Central causes:

1. Brain abscess
2. Pontine gliomas
3. Poliomyelitis
4. Multiple sclerosis

Peripheral causes:

Peripheral lesion could involve the nerve in its intracranial, intratemporal or extratemporal portions. Peripheral lesions are common than central ones. Nearly 2/3 of facial paralysis of them are peripheral lesions and of idiopathic in nature.

Intracranial portion of facial nerve:

1. Acoustic neuroma
2. Meningioma
3. Congenital cholesteatoma
4. Metastatic carcinoma
5. Meningitis

Intratemporal part of facial nerve:

- Idiopathic causes
  - Bell's palsy
  - Melkersson Rosenthal syndrome
- Infections
  - ASOM
  - CSOM
  - Ramsay Hunt syndrome
  - Malignant otitis externa
- Trauma
  - Surgical - Mastoidectomy and stapedectomy
  - Accidents - Fracture temporal bone
- Neoplasms
  - Malignancies of external & middle ear
  - Glomus jugulare tumor
  - Facial nerve neuroma
  - Metastasis of temporal bone (breast cancer, bronchus and prostrate)

Extracranial portion of facial nerve

- Malignancy parotid
- Surgery of parotid

Accidental injury in parotid region

Neonatal facial injury (forceps delivery)

Systemic diseases

Diabetes mellitus

Hypothyroidism

Uraemia

Polyarteritis nodosa

Wegener's granulomatosis

Sarcoidosis

Leprosy

Leukemia

Demyelinating diseases

Electrodiagnostic tests:

Electrodiagnositc tests are very useful in differentiating neuropraxia from degeneration of the nerve. These tests are also of immense prognostic value and could indicate the time for surgical decompression of the nerve.

Minimal nerve excitability test:

In this test the nerve is stimulated at steadily increasing intensity till the facial twitch is just noticeable. This is compared with that of the normal side. In conduction block there is no difference between the normal and paralyzed side.

In injuries associated with degeneration nerve excitability is lost gradually. If the difference between the sides exceed 3.5 m amp the test should be considered as positive for degeneration. It should be noted that degeneration of nerve fibers cannot be detected before 48-72 hours of its commencement.

Maximal stimulation test:

This is similar to the minimal nerve excitability test, but instead of measuring the minimal nerve excitability threshold the current level which elicits maximal facial movement is determined and is compared with that of the opposite side. Response is visually graded as equal, decreased or absent. Reduced or absent response with maximal stimulation indicates degeneration and in these cases recovery is incomplete.

Electroneuronography (ENoG):

This is a type of evoked electromyography. The facial nerve is stimulated at the level of stylomastoid foramen and the compound muscle action potentials are picked up by surface electrodes and recorded. Supramaximal stimulation is used to obtain maximal action potentials. The responses of action potentials of the paralyzed side are compared with that of the normal side. Percentage of degenerating fibers can be calculated. Faster the rate of degeneration within the first two weeks poorer is the prognosis.

Electromyography:

This test assesses the motor activity of facial muscles by direct insertion of needle electrodes into the orbicularis oculi and orbicularis oris muscles. Recordings are made during rest and voluntary contraction of muscle. In normal resting muscle, biphasic or triphasic potentials are seen every 30-50 ms. In denervated muscle spontaneous involuntary action potentials known as fibrillation potentials are seen. These potentials appear within 14-21 days after denervation of the muscle. This test is useful in planning reanimation procedures. Preservation of these potentials after 1 year of injury indicates that reinnervation is taking place. Electroneuronography and electromyography are complementary and helps in prognosis of these cases. It also helps in decision making regarding the procedure for reanimation.

Localization of lesions involving the facial nerve:

Central lesions causing facial paralysis can be caused by cerebrovascular accidents (hemorrhage, thrombosis or embolism), tumor or abscess. These lesions cause paralysis only in the lower half of face on the contralateral side. Forehead movements are retained due to bilateral innervation of frontalis muscle. Involuntary emotional movements and the tone of the facial muscles are also retained.

Peripheral lesions causing facial paralysis involve all muscles of the face, and on the involved side these muscles are paralyzed. These patients are unable to frown, close the eye, purse the lips or whistle.

Lesions at the level of nucleus can be identified by associated paralysis of 6th cranial nerve.

CP angle lesions can be identified by the presence of vestibular and auditory defects. Other cranial nerves like 5th, 9th, 10th and 11th cranial nerves could also be affected.

Level of lesions of facial nerve from the internal acoustic meatus up to the stylomastoid foramen can be localised by Topognostic tests. Lesions involving the facial nerve outside the temporal bone, i.e. in the parotid area affects only the motor functions of the nerve. Facial nerve paralysis could be incomplete as some branches of the nerve could survive carcinoma involvement.

Topognostic tests for lesions involving facial nerve in its Intratemporal portion:

Schirmer test:

This test is basically prepared to quantitate tear production. This test is performed by placing strips of white filter paper at the junction of the middle and lateral thirds of the lower eyelids after administration of a topical anesthetic agent. The tear production is measured with the eyes closed. Produced tears will wet the filter paper. The length of the filter paper which becomes wet is assessed at the end of 5 minutes. Normal test result is between 10mm and 30 mm of wet filter paper. Normally it should not exceed 30 mm. A value of more than 30 mm is considered to be epiphora. A value of less than 10 mm is considered to be dry eye (hyposecretion).

Stapedial reflex:

This reflex is lost in lesions involving Intratemporal portion of the facial nerve (above the level of the nerve to stapedius). This can be tested by tympanometry.

Taste test:

Taste can be measured by a pinch of salt or sugar solution placed on one side of the protruded tongue, or by electrogustometry. Impairment of taste indicates lesion above the chorda tympani nerve.

Submandibular salivary flow test:

This test measures the function of chorda tympani nerve. Polythene tubes are passed into both Wharton ducts and drops of saliva is counted during one minute period. Decreased salivation indicates injury to facial nerve above the level of chorda.

Complications of facial paralysis:

Incomplete recovery - Facial asymmetry persists. Eye cannot be closed resulting in epiphora. A weak oral sphincter causes drooling and difficulty in taking food.

Exposure keratitis - When eye cannot be closed tear film from the cornea evaporates causing dryness, exposure keratitis and corneal ulcer. This condition worsens in the presence of decreased lacrimation which is a feature of facial paralysis. This condition can be prevented by use of artificial tears (methylcellulose eye drops) every 1-2 hours. Temporary tarsorraphy is also indicated. Eye closure can be improved by using gold weight implant sutured to the tarsal plate deep to levator palpebrae muscle.

Synkinesis - This occurs when the patient wishes to close the eye, the corner of the mouth also twitches. This is due to cross innervation of fibers.

Tics and spasms - This condition is caused as a result of faulty regeneration of fibers. Involuntary movements are seen on the affected side of the face.

Contractures - This is due to fibrosis of atrophied muscles or fixed contraction of a group of muscles. They affect movements of face but facial symmetry at rest is good.

Crocodile tears - There is unilateral lacrimation when the patient masticates. This is due to faulty regeneration of parasympathetic fibers which supply the lacrimal glands instead of salivary glands. This condition can be treated by sectioning of greater superficial petrosal nerve or tympanic neurectomy.

Frey's syndrome (gustatory sweating) - In this condition, there is flushing and sweating of skin over parotid area during mastication. This results from parotid surgery.

Hyperkinetic disorders of facial nerve:

Hemifacial spasm - This is characterized by repeated, uncontrollable twitching's of facial muscles on one side. There are two types of hemifacial spasm.

Essential (idiopathic) the cause is unknown. This condition can be treated by selective sectioning of the branches of facial nerve in the parotid or by puncturing the facial nerve with a needle in its tympanic segment. Botulium toxin injection can be used in the affected muscle. It blocks the neuromuscular transmission by preventing release of acetylcholine.

Secondary hemifacial spasm - This is caused by acoustic neuroma, congenital cholesteatoma, or glomus tumor. Majority of hemifacial spasm is caused by irritation of the nerve because of the presence of vascular loop at the CP angle. Microvascular decompression through posterior fossa craniotomy has produced high success rates.

Blepharospasm - In this condition twitching's and spasm are limited to orbicularis oculi muscles on both sides. The eyes are closed due to muscle spasms causing functional blindness. The cause is uncertain but could be due to lesions involving basal ganglia. Botulinum A toxin can be injected into the periorbital muscles would provide relief for 3-6 months. Injections can be repeated if needed.

Surgical management of facial nerve paralysis:

1. Decompression - The facial nerve may be compressed by oedema. Removing its bony covering and by excising its sheath would allow space for its expansion and would provide relief.

2. END to END anastomosis - This can be done if the gap between the severed ends of the nerve is just a few millimeters and suturing of the cut ends without undue tension is possible.

3. Nerve graft (cable graft) - When the gap between the severed ends of the nerve cannot be closed by end to end anastomosis, then nerve grafting can be performed. Greater auricular nerve, or lateral cutaneous nerve of the thigh, or sural nerve can be used for this purpose.

4. Hypoglossal facial anastomoses - In this procedure hypoglossal nerve could be anastomosed to the severed end of the facial nerve. This procedure improves facial muscle tone and also permits some movements of facial musculature. The tongue muscles atrophy on the same side.

52. Bell's palsy

Bell's palsy is defined as idiopathic lower motor neuron type of facial nerve paralysis. This is in fact the most common type of facial palsy. This condition was first described by Sir Charles Bell one century ago.

This condition is mostly unilateral, and rarely bilateral. Bell's palsy is a diagnosis of exclusion, which must be made only after excluding all the known causes of facial nerve paralysis.

Pathophysiology: Etiology and pathophysiology is highly controversial. The patient gives history of going to bed normally, and waking up with facial palsy, or there is a history or bus / train travel with the patient seated close to the window.

1. Exposure to cold air has been postulated as one of the causes

2. Viral infections involving the nerve sheath

There is inflammation of the facial nerve causing it to swell up. Since it is enclosed inside a rigid bony canal it has virtually no space to expand causing the damage to the nerve. The labyrinthine segment of the facial canal is the narrowest portion of the whole facial canal (about 0.6mm).

Clinical features:

The patient wakes up with lower motor neuron type of facial paralysis.

1. Inability to close the ipsilateral eye

2. Reduction of tearing in the ipsilateral eye

3. Deviation of the angle of the mouth to the opposite side

4. Drooling of saliva

5. Metallic taste in the tongue

6. Inability to wrinkle the forehead

7. Bell's phenomenon (rolling of eyeball upwards)

This condition is very rare in pregnant women, and if present it tends to be very severe with poor recovery.

Prognosis is excellent. 99% of patients recovering completely.

Management:

1. Eye care: The patient should wear glasses to protect cornea. (Black glasses are preferable), use of artificial tears.

2. Regular physiotherapy (Balloon blowing)

3. Cheek / eye massage

4. Steroids: Very useful in early stages of the disease

5. Antiviral drugs like acyclovir has been tried with varying degrees of success

6. Facial nerve decompression can be considered in patients who don't show signs of recovery within 6 months

53. Frey’s syndrome

Frey's syndrome (also known as Baillarger's syndrome, Dupuy’s syndrome, auriculotemporal syndrome,or Frey-Baillarger syndrome) is a rare neurological disorder resulting from damage to or near the parotid glands responsible for making saliva, and from damage to the auriculotemporal nerve often from surgery.

The symptoms of Frey's syndrome are redness and sweating on the cheek area adjacent to the ear (see focal hyperhidrosis). They can appear when the affected person eats, sees, dreams, thinks about or talks about certain kinds of food which produce strong salivation.Observing sweating in the region after eating a lemon wedge may be diagnostic.

Signs and symptoms include erythema (redness/flushing) and sweating in the cutaneous distribution of the auriculotemporal nerve, usually in response to gustatory stimuli. There is sometimes pain in the same area, often of a burning nature. Between attacks of pain there is sometimes numbness or other altered sensations (anesthesia or paresthesia). This is sometimes termed "gustatory neuralgia".

Frey's syndrome often results as a side effect of surgeries of or near the parotid gland or due to injury to the auriculotemporal nerve, which passes through the parotid gland in the early part of its course. The auriculotemporal branch of the mandibular (V3) branch of the trigeminal nerve carries parasympathetic fibers to the parotid salivary gland and sympathetic fibers to the sweat glands of the scalp. As a result of severance and inappropriate regeneration, the parasympathetic nerve fibers may switch course to a sympathetic response, resulting in "gustatory sweating" or sweating in the anticipation of eating, instead of the normal salivatory response.It is often seen with patients who have undergone endoscopic thoracic sympathectomy, a surgical procedure wherein part of the sympathetic trunk is cut or clamped to treat sweating of the hands or blushing. The subsequent regeneration or nerve sprouting leads to abnormal sweating and salivating. It can also include discharge from the nose when smelling certain food.

Rarely, Frey's syndrome can result from causes other than surgery, including accidental trauma, local infections, sympathetic dysfunction and pathologic lesions within the parotid gland.An example of such rare trauma or localized infection can be seen in situations where a hair follicle has become ingrown, and is causing trauma or localized infection near or over one of the branches of the auriculotemporal nerve.

Diagnosis is made based on clinical signs and symptoms and a starch iodine test, called the Minor Iodine-Starch test. The affected area of the face is painted with iodine which is allowed to dry, then

dry corn starch is applied to the face. The starch turns blue on exposure to iodine in the presence of sweat.

Management:

Injection of botulinum toxin A
Surgical transection of the nerve fibers (only a temporary treatment)
Application of an ointment containing an anticholinergic drug such as scopolamine

NOSE

1. Lateral wall of Nasal cavity

Already provided in the essay question key

2. Sphenoid sinus.

Sphenoid sinus:

Is located in the skull base at the junction of the anterior and middle cranial fossa. Pneumatization of sphenoid starts during the 4th year of childhood and gets completed by the 17th year. The sphenoid sinuses vary in size and may be asymmetric.

They drain through the superior meatus via a small ostium about 4mm in diameter located disadvantageously 20mm above the sinus floor.

This sinus is related to several important vital structures. They are:

1. Pituitary gland lies above the sphenoid sinus.

2. Optic nerve and internal carotid arteries traverse its lateral wall.

3. The nerve of pterygoid canal lie in the floor of the sinus.

Hence infections of sphenoid sinus may involve the optic nerve if the canal of the optic nerve is dehiscent.

Functions of para nasal sinuses:

1. They lighten the skull.

2. They add resonance to speech

3. They play a role in conditioning the inspired air.

3. Middle meatus

   Situated between the middle and inferior concha, the middle meatus extends from the starting to the finishing point of the latter. It has a bony projection known as the ethmoidal bulla, which contains the middle ethmoidal cells. The uncinate process, a horn-shaped bony projection, is located beneath this bulla. The ethmoidal infundibulum, containing the fronto-nasal duct leads to this process while the duct is connected with the frontal sinus, which empties into the middle meatus.

   The half moon shaped semi-lunar hiatus is located between the ethmoidal bulla and uncinate process, with the frontal sinus, maxillary sinus and the anterior and middle ethmoid air cells opening into it.

Uncinate process: actually, forms the first layer or lamella of the middle meatus. The uncinate process is a wing or boomerang shaped piece of bone. It attaches anteriorly to the posterior edge of the lacrimal bone, and inferiorly to the superior edge of the inferior turbinate. Superior attachment of the uncinate process is highly variable, may be attached to the lamina papyracea, or the roof of the ethmoidal sinus, or sometimes to the middle turbinate. The configuration of the ethmoidal infundibulum and its relationship to the frontal recess depends largely on the behavior of the uncinate process. The uncinate process can be classified into 3 types depending on its superior attachment. The anterior insertion of the uncinate process cannot be identified clearly because it is covered with mucosa which is continuous with that of the lateral nasal wall. Sometimes a small groove is visible over the area where the uncinate attaches itself to the lateral nasal wall.

Type I uncinate: Here the uncinate process bends laterally in its upper most portion and inserts into the lamina papyracea. Here the ethmoidal infundibulum is closed superiorly by a blind pouch called the recessus terminalis (terminal recess). In this case the ethmoidal infundibulum and the frontal recess are separated from each other so that the frontal recess opens in to the middle meatus medial to the ethmoidal infundibulum, between the uncinate process and the middle turbinate. The route of drainage and ventilation of the frontal sinus run medial to the ethmoidal infundibulum.

Type II uncinate: Here the uncinate process extends superiorly to the roof of the ethmoid. The frontal sinus opens directly into the ethmoidal infundibulum. In these cases a disease in the frontal recess may spread to involve the ethmoidal infundibulum and the maxillary sinus secondarily. Sometimes the superior end of the uncinate process may get divided into three branches one getting attached to the roof of the ethmoid, one getting attached to the lamina papyracea, and the last getting attached to the middle turbinate.

Type III uncinate process: Here the superior end of the uncinate process turns medially to get attached to the middle turbinate. Here also the frontal sinus drains directly into the ethmoidal infundibulum.

Rarely the uncinate process itself may be heavily pneumatised causing obstruction to the infundibulum.

Ethmoidal infundibulum: is a cleft like space, which is three dimensional in the lateral wall of the nose. This structure belongs to the anterior ethmoid. This space is bounded medially by the uncinate process and the mucosa covering it. Major portion of its lateral wall is bounded by the lamina papyracea, and the frontal process of maxilla to a lesser extent. Defects in the medial wall of the infundibulum is covered with dense connective tissue and periosteum. These defects are known as anterior and posterior fontanelles. Anteriorly the ethmoidal infundibulum ends blindly in an acute angle.

Hiatus semilunaris: Lies between the anterior wall of the Bulla and the free posterior margin of the uncinate process. This is in fact a two-dimensional space. Through this hiatus a cleft like space can be entered. This is known as the ethmoidal infundibulum. This ethmoidal infundibulum is bounded medially along its entire length by the uncinate process and its lining mucosa. The lateral wall is formed by the lamina papyracea of the orbit, with participation from the frontal process of the maxilla and the lacrimal bone. The anterior group of sinuses drain into this area. In fact this area acts as a cess pool for all the secretions from the anterior group of sinuses.

Osteomeatal complex: This term is used by the surgeon to indicate the area bounded by the middle turbinate medially, the lamina papyracea laterally, and the basal lamella superiorly and posteriorly. The inferior and anterior borders of the Osteomeatal complex are open. The contents of this space are the agger nasi, nasofrontal recess (frontal recess), infundibulum, bulla ethmoidalis and the anterior group of ethmoidal air cells.

4. Hiatus semilunaris

Hiatus semilunaris: Lies between the anterior wall of the Bulla and the free posterior margin of the uncinate process. This is in fact a two-dimensional space. Through this hiatus a cleft like space can be entered. This is known as the ethmoidal infundibulum. This ethmoidal infundibulum is bounded medially along its entire length by the uncinate process and its lining mucosa. The lateral wall is formed by the lamina papyracea of the orbit, with participation from the frontal process of the maxilla and the lacrimal bone. The anterior group of sinuses drain into this area. In fact this area acts as a cess pool for all the secretions from the anterior group of sinuses.

5. Physiology of nose

Physiology of paranasal sinuses have undergone rapid advancement since the days of Galen. It was Galen who described the anatomy of paranasal sinuses as "Porosity of skull" in the second century AD. It was Leonardo da vinci who made a detailed anatomical illustration and description of the paranasal sinuses. His illustrations contained description of frontal sinuses and maxillary sinus ostium.

The physiological role played by paranasal sinuses have always been conjectural. Several hypotheses have been made. For easier understanding these hypotheses have been grouped under three main heads:

1. Structural theories

2. Evolutionary theories

3. Functional theories

Structural theory:

Fallopio's theory: Fallopio in 1600 hypothesized that these para nasal sinuses are present to make the skull bone lighter thereby reducing the load on neck musculature which supports the head.

Another theory belonging to this group suggests that these sinuses contribute to the maintenance of equilibrium and the positioning of the head by lightening the anterior portion of the cranium.

Proetz theory: suggests that the paranasal sinuses play a role in remodeling of facial bones.

Evolutionary theory:

This theory is very innovative. It considers the paranasal sinuses as the evolutionary response of anthropomorphic monkeys to shift from terrestrial environment to the aquatic one. This theory was proposed by Hardy. According to him the African monkeys were forced to take to water when the whole of Africa was surrounded by sea about 6.5 million years ago. The necessity to cross large stretches of water enabled them to develop these air-filled sinus cavities which helped them to keep afloat for long hours in water. This occurred due to an evolutionary process known as Natural selection.

Functional theories:
Bartholini's theory:

Bartholin considered these cavities as organs of resonance which added quality and resonance to the voice.

Cloquet's theory:

Cloquet hypothesized that paranasal sinuses contained olfactory epithelium aiding in the function of smell. This theory has been disproved as olfactory epithelium has not been demonstrated in the sinus cavity.

The most acceptable theory is that these sinuses improves nasal function. The fact that these paranasal sinuses embryologically originates as invaginations from the nose and its histological continuity with the nasal mucosa leads credence to this theory. These sinuses have been shown to

strengthen the defense function through additional secretion of lytic enzymes and immunoglobulins.

Ventilatory function:

Since the mucosal lining of the paranasal sinuses are in continuity with that of nasal mucosa they play an important role in ventilatory function. Gaseous exchange are known to occur between the inspired air and blood supply of the sinus mucosal lining. Gaseous exchanges are determined by active and passive phenomena. During respiration the nasal valve transforms the inspired air into a laminar flow. When this laminar flow reaches the middle meatus area turbulence starts to occur. This turbulence causes uniform mixing of air. There is a pressure gradient between the nasal cavity and para nasal sinuses causing airflow in to the sinus cavities. Air enters the sinuses at the end of every inspiration and at the beginning of the following expiration. This is because only during these times a positive pressure exists in the nasal cavities driving air into the sinuses.

It has been estimated that only 1/1000 of the air volume inside the sinuses are exchanged through a single respiratory act. The patency of the ostium is verify important for the gaseous exchange to take place.

Mucociliary clearance:

The nose and paranasal sinuses form the first line of defense for the lower airways. They protect the lower airways from noxious substances and microbes. They play a vital role in specific and nonspecific modes of defense. Among the nonspecific protective mechanisms, Mucociliary clearance play an important role. This mechanism depends on the integrity of the Mucociliary system which includes the ciliated cells, goblet cells and the quality of the mucous secreted. Goblet cells are more numerous in maxillary sinus than other sinuses. The muco ciliary transport mechanism needs continuous supply of oxygen. This mechanism clears the nasal and sinus cavity. The microbes and particulate matter if any gets entrapped in the secretions and are transported out.

Immune defense:

This defense mechanism can be divided into nonspecific (phylogenetically older) and specific (phylogenetically newer) mechanisms. The older nonspecific mechanism is also known as natural immunity. This is brought about by factors like:

1. Lysozyme - which destroys the bacterial cell wall

2. Interferon

3. Complement

4. Enzymes

Specific immunity is provided by macrophages and immunoglobin secreting lymphocytes. This immunity is highly specific against microbes invading the nasal cavity. Nose associated local immune tissue (NALT) has been attributed with this function.

6. Nasal cycle

In 1927, Heetderks described the alternating turgescence of the inferior turbinates in 80% of a normal population. According to Heetderks, the cycle is the result of alternating congestion and decongestion of the nasal conchae or turbinates, predominantly the inferior turbinates, which are by far the largest of the turbinates in each nasal fossa. Turbinates consist of bony projections covered by erectile tissue, much like the tissues of the penis and clitoris. The turbinates in one fossa fill up with blood while the opposite turbinates decongest by shunting blood away. This cycle, which is controlled by the autonomic nervous system, has a mean duration of two and a half hours. He further observed and documented that the turbinates in the dependent nasal fossa fill when the patient is in the lateral decubitus (lying down on your side) position. Some postulate that this alternating positional obstruction has the purpose of causing a person to turn from one side to the other while sleeping in order to prevent bedsores. Others note that the asymmetric airflow may have some benefit to overall olfactory sensitivity. The nasal cycle is an alternation in both time and between left and right sides, with the total resistance in the nose remaining constant. In patients with a fixed septal deviation and intermittent nasal obstruction, the interplay of the nasal cycle becomes evident; the sensation of obstruction frequently mirrors the congestion phase.

It is possible that the nasal cycle may exacerbate the nasal congestion caused by the common cold, as the lack of motility of the cilia in one half of the nose may lead to an uncomfortable sensation of not being able to shift mucus by blowing the nose.

It has been shown that the cilia of the congested side suspend their motility until that side decongests. Thus, the cycle ensures that one side of the nose is always moist, to facilitate humidification, which is one of the three functions of the nose, the other two being filtration and warming of inspired air prior to its entering the lungs.

Some odor chemicals bind with olfactory receptors easily, even under conditions of high airflow, and other odors need more time, under low airflow conditions, to bind with receptors. With high airflow on one side and low airflow on the other side, the olfactory center detects a greater range of smells.

The nasal cycle should not be confused with pathological nasal congestion: individuals with normal nasal breathing usually do not realize their breathing is asymmetric unless there is underlying nasal obstruction. In pathological conditions, however, the nasal cycle may influence the symptoms.

7. Mucociliary clearance mechanism.

The extensive epithelial surface of the respiratory tract between the nose and the alveoli is exposed daily to viral and bacterial pathogens, particulates, and gaseous material with potentially harmful effects. In response to these challenges, humans have developed a series of defense mechanisms to protect the airways from these insults, thereby maintaining the lungs in a nearly sterile condition. Lung defense involves cough, anatomical barriers, aerodynamic changes, and immune mechanisms; however, the primary defense mechanism is mucociliary clearance (MCC). Healthy airway surfaces are lined by ciliated epithelial cells and covered with an airway surface layer (ASL), which has two components, a mucus layer that entraps inhaled particles and foreign pathogens, and a low viscosity periciliary layer (PCL) that lubricates airway surfaces and facilitates ciliary beating for efficient mucus clearance.

Mucociliary clearance mechanism forms the basis of FESS. The ciliated columnar epithelium of maxillary sinus clears secretions from maxillary sinus by beating towards the natural ostium. Discharge from maxillary sinus cannot be cleared by inferior meatal antrostomy as the secretions are pushed via the natural ostium.

Proper ciliary function is absolutely required for effective MCC. Cilia are specialized organelles that provide the force necessary to transport foreign materials in the respiratory tract toward the mouth where they can be swallowed or expectorated. To accomplish this crucial function, the cilia beat in coordinated metachronal waves at a beat frequency that has multiple physiological regulators. Much of what we know about cilia structure and function has been derived from studies performed using Chlamydomonas as a model, while the importance of cilia in maintaining airway clearance was revealed by clinical and pathological studies of genetic and acquired forms of chronic airway diseases, including primary ciliary dyskinesia (PCD), cystic fibrosis (CF), asthma, and chronic obstructive pulmonary disease (COPD). In this review, we summarize the current knowledge of the components of MCC, with an emphasis on the role of cilia in normal mucociliary transport. We then briefly describe the health effects of impaired MCC caused by genetic defects in cilia in patients with PCD.

8. Potato nose

Introduction:

Rhinophyma is a progressive disfiguring soft tissue hypertrophy of the tip of nose. This condition is also known as potato nose. This is usually caused by hyperplasia of the cells of sebaceous glands. It has been accepted as the end stage of chronic acne rosacea. In addition to the obvious cosmetic disfigurement it can also cause obstruction to vision. It can also become infected and may bleed.

Surgical management:

This involves shaving of the lesion with cauterization of the bleeding vessels. Surgery should aim at removing the diseased tissue while conserving the normal underlying sebaceous glands which could facilitate in normal re-epithelization.

Microdebriders have been used with varying degrees of success in removing the diseased tissue. The bleeders if any can be controlled using flow seal.

Carbon dioxide lasers have been used to manage this condition. Major advantage of laser is that the depth of excision is really precise and excess tissue removal is rare. There is very minimal risk of scar tissue formation.

Currently radiofrequency devices like coblators have been used in the management of this condition. Using this device there is very little risk of thermal damage to underlying tissue.

Numerous treatment modalities have been attempted to manage this condition. Oral antibiotics has shown excellent promise during early phases of this disease. Surgery will have to be resorted to in order to manage advanced rhinophymas.

9. Epistaxis.

Epistaxis is defined as bleeding from the nasal cavity. This is a Greek word meaning nose bleed. Since it is a very common problem its true incidence is very difficult to predict.

The nose has a rich supply of blood vessels with good contribution from both external and internal carotid systems. The general rule of the thumb is that the area of nasal cavity below the level of middle turbinate has rich blood supply from the external carotid system, whereas the area above the middle turbinate receives extensive supply from the internal carotid artery. Anastomosis occur between the external and internal carotid system throughout the nasal cavity.

External carotid system: Blood from the external carotid system reaches the nasal cavity via the facial and the internal maxillary arteries which are branches of the external carotid artery. The artery of epistaxis is the sphenopalatine branch of internal maxillary artery. This is called so because this vessel supplies the major portion of the nasal cavity. It enters the nasal cavity at the posterior end of the middle turbinate to supply the lateral nasal wall, it also gives off a septal branch which supplies the nasal septum.

Facial artery: the superior labial branch of the facial artery is one of its terminal branches. It supplies the anterior nasal floor and anterior portion of the nasal septum through its septal branch.

Internal maxillary artery: after entering into the pterygopalatine fossa this vessel gives rise to 6 branches. These branches are posterior superior alveolar artery, descending palatine artery, infra orbital artery, sphenopalatine artery, pterygoid artery, and pharyngeal artery. The descending palatine artery enters the nasal cavity through the greater palatine canal to supply the lateral wall of the nose, it also contributes blood supply to the nasal septum through its septal branch.

Internal carotid system: the internal carotid artery supplies the nasal cavity via its ophthalmic artery. It enters the orbit via the superior orbital fissure and divides into many branches. The posterior ethmoid artery one of the branches of ophthalmic artery exits the orbit via the posterior ethmoidal foramen located 2-9 mm anterior to the optic canal. The anterior ethmoidal artery which is larger leaves the orbit through the anterior ethmoidal foramen. Both these vessels cross the roof of the ethmoid and descends into the nasal cavity through the cribriform plate. It is here that these vessels divide into lateral and septal branches to supply the nose.

Little's area: This area is located in the anterior part of the cartiagenous portion of the nasal septum. Here there is extensive submucous anastomosis of blood vessels both from the external and the internal carotid systems. Bleeding commonly occurs from this area since it is highly vascular and is also exposed to the exterior. Anastomosis occur between the septal branches of sphenopalatine artery, greater palatine artery, superior labial artery and the anterior ethmoidal artery. This plexus is also known as Keisselbach's plexus. Bleeding from this area is common because mucosal drying occurs commonly here and this area is easily accessible to nose picking. Among the vessels taking part in the anastomosis the anterior ethmoidal artery is from the internal carotid system while the other vessels are from the external carotid system. Bleeding from this area is clearly seen and easily accessible and flows through the anterior nasal cavity hence it is known as anterior bleed.

Woodruff's plexus: is responsible for posterior bleeds. This area is located over the posterior end of the middle turbinate. The anastomosis here is made up of branches from the internal maxillary artery namely its sphenopalatine and ascending pharyngeal branches. The maxillary sinus ostium forms the dividing line between the anterior and posterior nasal bleeds. Posterior nasal bleeds are difficult to treat because bleeding area is not easily accessible. Bleeding from Woodruff's plexus commonly occur in patients with extremely high blood pressure. In fact this plexus acts as a safety valve in reducing the blood pressure in these patients, lest they will bleed intracranially causing

more problems. In patients with posterior bleeds it is difficult to access the amount of blood loss because most of the blood is swallowed by the patient.

Etiology: The etiology of epistaxis is not just simple or straight forward. It is commonly multifactorial, needing careful history taking and physical examination skill to identify the cause. For purposes of clear understanding the etiology of epistaxis can be classified under two broad heads, i.e. local and systemic causes.

Local factors causing epistaxis: include vascular anomalies, infections and inflammatory states of the nasal cavity, trauma, iatrogenic injuries, neoplasms and foreign bodies. Among these causes the commonest local factors involved in epistaxis is infection and inflammation. Infections and inflammation of the nasal mucous membrane may damage the mucosa leading on to bleeding from the underlying exposed plexus of blood vessels. Chronic granulomatous lesions like rhinosporidiosis can cause extensive epistaxis.

Aneurysms involving the internal carotid artery may occur following head injury, injury sustained during surgical procedures. These extradural aneurysm and aneurysms involving the cavernous sinus may extend into the sphenoid sinus wait for the opportune moment to rupture. It can cause sudden fatal epistaxis, or blindness. Urgent embolization is the preferred mode of management of this condition.

Trauma is one of the common local causes of epistaxis. It is commonly caused by the act of nose picking in the Little's area of the nose. This is commonly seen in young children. Acute facial trauma may also lead to epistaxis. Patients undergoing nasal surgeries may have temporary episodes of epistaxis.

Irritation of the nasal mucous membrane: any disruption of normal nasal physiology can cause intense drying and irritation to the nasal mucosa causing epistaxis. These episodes are common during extremes of temperature when the nasal mucosa is stressed to perform its airconditioning role of the inspired air. In these conditions there is extensive drying of nasal mucosa causes oedema of the nasal mucous membrane. This oedema is caused due to venous stasis. Ultimately the mucosa breaches exposing the underlying plexus of blood vessels causing epistaxis.

Anatomical abnormalities: Common anatomical abnormality causing epistaxis is gross septal deviation. Gross deviations of nasal septum cause disruption to the normal nasal airflow. This disruption leads to desiccation / drying of the local mucosa. The dry mucosa cracks and bleeds.

Septal perforations: Chronic non healing septal perforations can cause bleeding from the granulation tissue around the perforation.

Neoplasms: involving the nose and paranasal sinuses can cause epistaxis. Neoplasms include benign vascular tumors like hemangioma, juvenile nasopharyngeal angiofibroma, and malignant

neoplasms like squamous cell carcinoma. If epistaxis occurs along with secretory otitis media then nasopharyngeal carcinoma should be the prime suspect.

Systemic causes for epistaxis:

Hypertension is one of the common systemic causes of epistaxis. Accumulation of atherosclerotic plaques in the blood vessels of these patients replaces the muscular wall. This replacement of muscular wall reduces the ability of the blood vessels to constrict facilitating epistaxis. This is one of the common causes of posterior nasal bleeds. It commonly arises from the Woodruff's plexus found close the posterior end of the middle turbinate.

Hereditary hemorrhagic telangiectasia is another systemic disorder known to affect the blood vessels of the nose. This disease causes loss of contractile elements within the blood vessels causing dilated venules, capillaries and small arteriovenous malformations known as telangiectasia. These changes can occur in the skin, mucosal lining the whole of the respiratory passage and urogenital passage. Bleeding from this telangiectasia is difficult to control. Bleeding invariably starts when the patient reaches puberty. Common cause of mortality in these patients is gastrointestinal bleed.

Systemic diseases like syphilis, tuberculosis & wegner's granulomatosis cause epistaxis because of their propensity to cause ulceration of the nasal mucous membrane.

Blood dyscrasias can also cause epistaxis. A low platelet count is one common cause of nasal bleed in this category. In thrombocytopenia the platelet count is less than 1 lakh. Epistaxis can start when the platelet count reduces to 50,000. Platelet deficiency can be caused by ingestion of drugs like aspirin, indomethacin etc. Hypersplenism can cause thrombocytopenia in idiopathic thrombocytopenic purpura. These patients need to be transfused fresh blood in adequate quantities. Only when thee platelet count increases will the nasal bleed stop.

Incidence: The incidence of epistaxis is known to be slightly higher in males. It also has a bimodal distribution affecting young children and old people.

Evaluation: While evaluating a patient with epistaxis it is absolutely necessary to assess the quantum of blood loss. The blood pressure and pulse rate of these patients must be constantly monitored. These patients will have tachycardia. Infusion of fluid must be started immediately. Initially ringer lactate solution will suffice. If the patient has suffered blood loss of more than 30% of their blood volume (about 1.5 liters) then blood transfusion becomes a must. Further examination should be started only after the patient's general condition stabilizes.

History: Careful history taking is a must. History taking should cover the following points:

1. History regarding the frequency, severity and side of the nasal bleed.

2. Aggravating and relieving factors must be carefully sought.

3. History of drug intake must be sought.

4. History of systemic disorders like hypertension and diabetes mellitus must be sought.

Physical examination:

The nasal pack if any must be removed. Anterior nasal examination should be done, first attempted without the use of nasal decongestants. If visualization is difficult due to oedema of the nasal mucosa then nasal decongestants can be used to shrink the nasal mucosa. The solution used for anesthetizing the decongesting the nose is a mixture of 4% xylocaine and xylometazoline.

Nasal endoscopy can be performed under local anesthesia to localised posterior bleeds.

Investigations:

If bleeding is minimal no investigation is necessary.

If bleeding is more than a complete blood work up to rule out blood dyscrasias is a must. It includes bleeding time, clotting time, platelet count and partial thromboplastin time.

Imaging studies like CT scan of the para nasal sinuses must be done to rule out local nasal conditions of epistaxis. Imaging must be done only after 24 hours of removing the nasal packing. Scans done with the nasal pack or immediately after removing the nasal pack may not be informative.

In difficult and intractable cases angiography can be done and the internal maxillary artery can be embolized in the same sitting. This procedure should be reserved only for cases of intractable nasal bleeding.

Management:

Conservative:

Nasal packing: Anterior nasal packing using roller gauze impregnated with liquid paraffin is sufficient to manage a majority of anterior nasal bleeds. The liquid paraffin acts as a lubricant, and as a moistening agent. The tamponing effect of a nasal pack is sufficient to stop nasal bleeding. This type of roller gauzes can be kept inside the nasal cavity only up to 48 hours after which it has to be removed and changed. The newer packs like the BIPP (Bismuth Iodine paraffin paste) packs can be left safely in place for more than a week.

To manage post nasal bleed a post nasal pack is a must. Post nasal packing can be done in 2 ways:

Post nasal packing (conventional): A gauze roll about the size of the patient's naso pharynx is used here. Three silk threads must be tied to the gauze roll. One at each end and the other one at the middle. The patient should be in a recumbent position. After anesthetizing the nasal cavity with 4% xylocaine the mouth is held open. Two nasal catheters are passed through the nasal cavities till they reach just below the soft palate. These lower ends of the catheters are grasped with forceps and pulled out through the mouth. The silk tied to the ends of the gauze is tied to the nasal catheters. The post nasal pack is introduced through the mouth and gradually pushed into the nasopharynx, at the same time the nasal catheters on both sides of the nose must be pulled out. When the pack snugly sits inside the nasopharynx, the two silk threads tied to its end would have reached the anterior nares along with the free end of the nasal suction catheter.

The two silk threads tied to the suction catheters are untied. The catheters are removed from the nose. The silk thread is used to secure the pack in place by tying both the ends to the columella of the nose. The silk tied to the middle portion of the gauze pack is delivered out through the oral cavity and taped to the angle of the cheek. This middle portion silk will help in removal of the nasal pack. In addition to the postnasal pack anterior nasal packing must also be done in these patients.

Postnasal pack using balloon catheters: Specially designed balloon catheters are available. This can be used to perform the post nasal pack. Foleys catheter can be used to pack the post nasal space. Foley's catheter is introduced through the nose and slid up to the nasopharynx. The bulb of the catheter is inflated using air through the side portal of the catheter. Air is used to inflate the bulb because even if the bulb ruptures accidentally there is absolutely no danger of aspiration into the lungs. After the foleys catheter is inflated the free end is knotted and anchored at the level of the anterior nares.

Surgical management:

Endoscopic cauterization can be tried if the bleeders are localised and accessible. If not accessible, ligation of the internal maxillary artery can be done through Caldwell Luc approach. Sphenopalatine artery clipping can be done endoscopically. It is accessible close to the posterior end of the middle turbinate. In rare cases external carotid artery ligation at the neck can be resorted to. External carotid artery is differentiated from the internal carotid in the neck by the fact that internal carotid artery does not give rise to branches in the neck, while the external carotid artery does so.

Ethmoidal artery ligation: If epistaxis occurs high in the nasal vault, anterior and posterior ethmoidal arteries may be ligated using ligaclips. These arteries can be accessed using an external ethmoidectomy incision. The anterior ethmoidal artery is usually found 22mm from the anterior lacrimal crest. If ligation of the anterior ethmoidal artery does not stop bleeding then posterior

ethmoidal artery should also be ligated. The posterior ethmoidal artery can be found 12mm posterior to the anterior ethmoidal vessel.

Epistaxis caused by the presence of tumors both benign and malignant calls for definitive treatment of the tumor parse.

10. Anterior nasal packing

Anterior nasal packing using roller gauze impregnated with liquid paraffin is sufficient to manage a majority of anterior nasal bleeds. The liquid paraffin acts as a lubricant, and as a moistening agent. The tamponing effect of a nasal pack is sufficient to stop nasal bleeding. This type of roller gauzes can be kept inside the nasal cavity only up to 48 hours after which it has to be removed and changed. The newer packs like the BIPP (Bismuth Iodine paraffin paste) packs can be left safely in place for more than a week.

Anterior nasal packing will require the following equipment and materials:

A hospital bed that has a back that can be positioned to 90 degrees
Headlamp
Otoscope
Suction canister with either wall suction or portable suction
Frazier suction tip
Yankauer suction tip
Nasal speculum
Bayonet forceps
Tongue depressor
4x4 inch gauze
2x2 inch gauze
Dental roll
Sterile lubricant
Antibiotic ointment
Intranasal vasoconstrictor
Topical anesthetic
Nasal tampon or intra-nasal device
Personal protective equipment:
Goggles
Face mask
Gown
Gloves

Nasal packs are available made out of polyvinyl alcohol (PVA) in the shape of a nasal tampon with a string at the base that expands when it comes in contact with moisture and is available in a variety of sizes. Some devices are made of expandable foam that comes inside an applicator shaped as a nasal tampon that expands when coming in contact with moisture. The foam is in an expandable balloon that is layered with carboxymethylcellulose (CMC) that serves a double purpose of applying direct pressure as well as platelet aggregation as a function of the CMC once it comes into contact with a liquid. Initial steps for nasal packing with any of the agents are nearly identical.

Depending on the device selected, soak in water for 30 seconds.
Achieve anesthesia via vasoconstrictive agents (e.g. cocaine 4%, lidocaine 1 to 4% with epinephrine 1 per 100000, or oxymetazoline 0.05%).
Generously coat either antibiotic ointment or petroleum jelly onto the tampon.
Insert tampon into affected naris by applying quick, steady pressure directed along the floor of the nose, which is parallel to the ground (be cautious not to insert in the superior direction).
Once inserted, the packing will likely already have started expanding secondary to contact with blood. However, injecting approximately 10 mL of normal saline or oxymetazoline 0.05% or tranexamic acid (TXA) will expand the tampon and may help in achieving hemostasis.
Based on manufacturer recommendations, inflate the balloon using a 20mL syringe filled with air.
Monitor the patient for another ten to thirty minutes to ensure that hemostasis is achieved.
The packing will likely turn pink, which is normally secondary to a bit of oozing. If the patient continues to have active bleeding turning the packing bright red, or blood visualized dripping out past the packing, or if the patient is still swallowing blood, consider packing to have failed and moved on to further intervention.

11. Little’s Area

This area is located in the anterior part of the cartiagenous portion of the nasal septum. Here there is extensive submucous anastomosis of blood vessels both from the external and the internal carotid systems. Bleeding commonly occurs from this area since it is highly vascular and is also exposed to the exterior. Anastomosis occur between the septal branches of sphenopalatine artery, greater palatine artery, superior labial artery and the anterior ethmoidal artery. This plexus is also known as Keisselbach's plexus. Bleeding from this area is common because mucosal drying occurs

commonly here and this area is easily accessible to nose picking. Among the vessels taking part in the anastomosis the anterior ethmoidal artery is from the internal carotid system while the other vessels are from the external carotid system. Bleeding from this area is clearly seen and easily accessible and flows through the anterior nasal cavity hence it is known as anterior bleed.

12. Deviated nasal septum

Septal deviations are pretty common occurrence. In fact a dot central nasal septum is a clinical curiosity. Even though septal deviations are common they are usually not severe enough to cause symptoms.

Aetiology:

Direct trauma - Many septal deviations are a result of direct trauma and this is frequently associated with damage to other parts of the nose such as fractures of nasal bone.

Birth moulding theory - Many patients with septal deviation do not give history of trauma. Birth moulding theory was propounded by Gray. According to him abnormal intrauterine posture may result in compression forces acting on the nose and upper jaws. Displacement of septum can occur in these patients due to torsion forces that occur during parturition. Dislocations are more common in primipara and when the second stage of labor lasted for more than 15 minutes. Dislocations are generally to the right in the case of left occipitoanterior presentations and to the left with right occipitoanterior presentations. Subsequent growth of nose accentuates these asymmetries.

Differential growth between nasal septum and palate - This is the most acceptable theory today. When the nasal septum grows faster in certain individuals than the palate then the nasal septum starts to buckle under pressure.

Pathophysiology:

Deformity of nasal septum may be classified into:

1. Spurs

2. Deviations

3. Dislocations

Spurs - These are sharp angulations seen in the nasal septum occurring at the junction of the vomer below, with the septal cartilage and / or ethmoid bone above. This type of deformity is the result of vertical compression forces. Fractures that occur through nasal septum during injury to the nose

may also produce sharp angulations. These fractures heal by fibrosis that extend to the adjacent mucoperichondrium. This increases the difficulty of flap elevation in this area.

Deviations - May be C shaped or S shaped. These can occur in either vertical or horizontal plane. It may also involve both cartilage and bone.

Dislocations - In this the lower border of the septal cartilage is displaced from its medial position and projects into one of the nostrils.

In patients with septal deviation a compensatory hypertrophy of the turbinates and bulla may occur on the side opposite to the deviation. If compression forces are involved the septal deviations are often asymmetrical and may also involve the maxilla, producing flattening of the cheek, elevation of the floor of the affected nasal cavity, distortion of the palate and associated orthodontic abnormalities. The maxillary sinus is usually slightly smaller on the affected side.

Anterior septal deviations are often associated with deviations in the external nasal pyramid. Deviations may affect any of the three vertical components of the nose causing:

1. Cartilaginous deviations

2. The C deviation

3. The S deviation.

Cartilaginous deviations:

In these patients the upper bony septum and the bony pyramid are central, but there is a dislocation / deviation of the cartiagenous septum and vault.

The C deviation:

Here there is displacement of the upper bony septum and the pyramid to one side and the whole of the cartiagenous septum and vault to the opposite side.

The S deviation:

Here the deviation of the middle third (the upper cartiagenous vault and associated septum) is opposite to that of the upper and lower thirds. With deviations of the nose, the dominant factor is the position of the nasal septum, hence the adage 'as the septum goes, so goes the nose'. The first step, therefore in treating the twisted nose is to straighten the septum, and if this objective is not achieved, there is no hope of successfully straightening the external pyramid.

Effects of septal deviation:

Nasal obstruction - This is always found on the side of the deviation, and can also be present on the opposite side as a result of hypertrophic changes of the turbinates.

Mucosal changes - The inspiratory air currents are abnormally displaced and frequently gets concentrated on small areas of nasal mucosa, producing excessive drying effect. Crusting will occur and the separation of the crusts often produces ulceration and bleeding. Since the protective mucous layer is lost the resistance to infection is reduced. The mucosa around a septal deviation may become oedematous as a result of Bernouilli's phenomenon. This oedema further increases nasal obstruction.

Neurological changes - Pressure may be exerted by septal deviations on adjacent sensory nerves can produce pain. This was first explained by Sluder and the resultant condition became known as 'the anterior ethmoidal nerve syndrome'. In addition to these direct neurological effects, reflex changes perhaps may result from septal deformities which affect the nasopulmonary and nasal reflexes.

Symptoms:

The symptoms caused by septal deviations are entirely the result of their effects on nasal function. The dominant symptom being nasal obstruction, but this is rarely severe enough to cause anosmia.

Signs:

Septal deviations are evident on anterior rhinoscopy. This should be done without the use of nasal speculum because the insertion of speculum is sufficient to straighten the nasal septum. When the tip of the nose is lifted septal deviation become evident. Nasal obstruction may also be present on the opposite side (paradoxical nasal obstruction). This is due to the presence of hypertrophied turbinates. If the hypertrophy is limited to turbinate mucosa alone then it will shrink when decongestant drugs are used in the nasal cavity. If the hypertrophy is bony then decongestant drops is useless.

Septal deviations in the region of the nasal valve area cause the greatest obstruction, since this is the narrowest part of the nasal cavity. This can be identified by the Cottle test. A positive Cottle test will confirm the fact that narrowing is present in the nasal valve area. This is done by asking the patient to pull the cheek outwards and this maneuver is supposed to open up the area thus reducing the block. The septum should not be considered in isolation and it is necessary to do a careful examination of the lateral wall of the nasal cavity. Whenever sinus complications like sinusitis is suspected due to obstruction to the drainage channel of the sinuses by the deviation Xray sinus must be taken.

Septal deviation in new born is associated with asymmetry of the nostrils, an oblique columella and tip which points in the direction which is opposite to the deviation. Most of these patients are

diagnosed by the use of Gray's struts. These struts are 4mm wide and 2mm thick and after lubrication, are inserted into the nostrils and then gently pushed backwards along the floor of the nasal cavity, hugging the nasal septum. Normally these struts can be introduced for a distance of 4 - 5 cms, but in cases of septal deviation a frank obstruction is encountered, usually 1 - 2 cms from the nostril.

Cottle has classified septal deviations into three types:

Simple deviations: Here there is mild deviation of nasal septum, there is no nasal obstruction. This is the commonest condition encountered. It needs no treatment.

Obstruction: There is more severe deviation of the nasal septum, which may touch the lateral wall of the nose, but on vasoconstriction the turbinates shrink away from the septum. Hence surgery is not indicated even in these cases.

Impaction: There is marked angulation of the septum with a spur which lies in contact with lateral nasal wall. The space is not increased even on vasoconstriction. Surgery is indicated in these patients.

Indications for submucous resection of nasal septum:

1. Marked septal deviation occurring behind the vertical line passing between the nasal processes of the frontal and maxillary bones. This deviation must be the cause for the patient's symptoms.

2. Closure of septal perforations

3. Source of grafting material

4. To obtain surgical access in hypophysectomy, and Vidian neurectomy

Deviated nasal septum can be corrected by:

SMR

Septoplasty

13. Cottle’s test

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careful examination of the lateral wall of the nasal cavity. Whenever sinus complications like sinusitis is suspected due to obstruction to the drainage channel of the sinuses by the deviation Xray sinus must be taken.

14. Discuss the difference between SMR and septoplasty

Septoplasty	SMR
Freer incision	Killian incision
Conservative procedure	Radical procedure
Complications like septal perforation less	Complications are common
Flap elevated only on one side of nasal septum	Flap elevated on both sides of nasal septum

15. Nasal Septal perforation

Septal perforation happens to the common disorder of nasal septum. In most patients septal perforations are incidental finding without any symptoms. Septal perforations may cause crusting, whistling sound during inspiration, epistaxis, nasal obstruction and pain. Whistling will be more when the perforation is small.
These symptoms depend on the location of perforation, size, shape and conditon of the margins of the perforation. Surgical closure happens to be the best option, but a prudent decision will have to be made taking into consideration all the above said parameters as well as the competancy of the surgeon involved.

Classification of septal perforation:

Septal perforations can be classified according to the following parameters:
1. Cause
2. Symptoms
3. Location
4. Size
5. Shape
6. Condition of margins
7. Presence / absence of cartilage or bone around the defect

Causes of septal perforation:
1. Septal surgery (commonest cause) – Cartilage / bone around the perforation is usually missing
2. Repeated nose picking – Cartilage / bone around the perforation is usually present
3. Repeated bilateral coagulation for epistaxis (in pts with hereditary teleangiectasis)

4. Trauma
5. Cocaine abuse
6. Septal abscess – Cartilaginous portion of nasal septum is totally destroyed
7. Granulomatous infections of nasal septum. In Wegener's granulomatosis conservative management is preferred over surgery
8. Tumor surgeries of nose
9. Exposure of caustics – Poor quality of mucosa surrounding the perforation should be borne in mind before embarking on surgery

Symptoms:

1. Asymptomatic
2. Severe crusting
3. Epistaxis
4. Whistling sound during inspiration – common in small perforations. Larger the perforation lesser becomes the whistling
5. Perforations involving the anterior portion of the septum is more symptomatic than those involving the posterior portion
6. Symptoms are severe in small perforations than larger ones

Location of perforation: Location of the perforation plays a vital role in the symptomatology. It should also be considered before deciding the management modality. Anterior perforations cause more problems and are also amenable to surgical closure since it is easily accessible and can be managed endonasally. Posterior perforations are asymptomatic and are difficult to manage surgically because of poor access. Endonasal approach is virtually ruled out for repairing posterior perforations. The ideal route of surgical approach is mid facial degloving, as this would provide good visualization and access to the perforated area.

Treatment:

1. No treatment: May be difficult for a surgeon to digest, a significant number of septal perforations do well without repair. Sometimes a failed repair will make the patient worse.
2. Conservative management: Application of bland ointments / Vaseline over the edges of perforation using a cotton applicator will cause symptomatic relief and prevent excessive crusting. Regular nasal douching using alkaline saline will prevent infection. If the mucosal edges around the perforation are infected then antibiotic steroid ointment can be applied.
3. Surgical closure: of the perforation is still the best option. The results are of course dependent on the size of the perforation. Mucosal margins should be healthy for closure of perforation to be successful. Any attempt to close a septal perforation caused by repeated cauterization of septum may fail because of the poor quality of mucosa lining the edges of the perforation. Similarly it is surgically difficult to close an irregularly shaped perforation when compared to a regular oval shaped one. Anterior perforations can be closed easily when compared to posterior ones due to better access. Four methods of surgical closure have been accepted. They include: Direct closure, Rotation flap technique, Bridge flap technique and Bucco gingival flap technique.

Direct closure: This method is ideal in cases of immediate repair of septal mucosal laceration. This can be performed only when the size of the perforation is small, and there is also associated septal deviation. By correcting the deviation of nasal septum sufficient slack can be achieved to facilitate tension free primary closure of mucosa. For direct closure the nasal septal mucosa should be undermined. The undermined mucosa should be sufficient for tension free closure of the perforation.

Rotation flap technique: This method is very useful in closing anterior based perforation which is less than 2 cms. The margins should be of good quality, and there is adequate bone / cartilage around the perforation. Two pedicled mucosal flaps are created. One flap (upper septal flap) on one side and another one from the floor on the opposite side are created. The upper septal flap is based cranio posteriorly and is supplied by anterior and posterior ethmoidal arteries. The lower septal flap is pedicled caudo posteriorly and is supplied by the palatine artery.

16. Septal Hematoma

Septal hematoma is collection of blood between the perichondrium of nasal septum and the septal cartilage.

Nose is the most prominent part of the face and hence is more prone for injuries resulting in a hematoma formation in the nasal septum.

Pathophysiology:
When the nasal septum is subjected to sharp buckling stress, the submucosal blood vessels are frequently damaged, and if the mucosa remain intact, will result in the formation of hematoma. If the trauma is severe enough to fracture the septal cartilage, the blood will seep to the opposite side causing bilateral septal hematoma. This bilateral septal hematoma is dangerous because it compromises the nutrition of the septal cartilage the most and cause dissolution of the whole cartiagenous septum itself. Since the nutrition of the cartilage is dependent on the intact perichondrium, elevation of the perichondrium away from the cartilage causes necrosis of the cartilage. Avascular cartilage can remain viable only for 3 days after compromise of the perichondrium. Cartilage absorption can occur with alarming rapidity.
If the hematoma is small and unilateral it may not cause necrosis of the cartilage, but may be absorbed causing permanent thickening of the nasal septum and gross fibrosis.

Signs & symptoms:
The dominant symptom is nasal obstruction. If hematoma is unilateral then obstruction is also unilateral, if hematoma is bilateral then obstruction is also bilateral. Examination must be carried out without a nasal speculum. It will reveal a smooth rounded unilateral / bilateral swelling often extending up to the lateral nasal wall causing severe obstruction.

Treatment:

It has been shown that early surgical drainage of the hematoma reduces the risk of cartilage necrosis, and hence is always indicated. A hemitransfixation incision (incision made at the lower border of the nasal septal cartilage) is used, since the perichondrium is already lifted off the cartilage the accumulated blood and infected material is aspirated. The state of the cartilage is assessed and if there is any defect it is advisable to support the defect with homograft cartilage. These cartilage grafts can be used even if abscess formation has occurred thus effectively preventing saddle nose deformities. The homograft cartilage can be harvested from patients who have under gone submucosal resection of the nasal septum. These harvested cartilages can be stored in 0.1% sodium mecurothiosalicylate.

Complications:

External deformity of the nose: The cartiagenous dorsum of the nose is supported by the septal cartilage and if this is lost then dorsal saddling can occur causing pig snout deformity (Pig nose like). If this injury occurs during childhood, it may also affect the development of the whole of the middle third of the face causing resultant maxillary hypoplasia.

Septal abscess: Hematoma is a good culture medium and hence may become infected causing abscess formation. This complication is always associated with severe pain, together with manifestations of toxemia, such as increased pulse rate.

Septal deviation:
Minor hematomas especially the unilateral ones may get absorbed and appear as thickened areas in the nasal septum with extensive fibrosis leading on to deviation of nasal septum to that side due to contracture caused by fibrosis.

17. Diagnostic Nasal Endoscopy

Introduction: Examination of nose has been revolutionized by the advent of nasal endoscopes. These endoscopes are nothing but miniature telescope. It comes in the following sizes 2.7mm, and 4mm. It comes in various angulations namely 0 degrees, 30 degrees, 45 degrees, and 70 degrees. The 2.7 mm endoscope is used for diagnostic nasal endoscopy and in children. For diagnostic nasal

endoscopy it is better to use a 2.7 mm 30-degree nasal endoscope if available. A 4mm 30-degree nasal endoscope can also be used for diagnostic nasal endoscopy in adults.

Indications of diagnostic nasal endoscopy:

1. To evaluate why a patient is not responding to medication.
2. To determine whether surgical management is necessary.
3. To examine the results of sinus surgery
4. To determine the effects of conditions such as severe allergies, immune deficiencies and Mucociliary disorders (disorders that affect mucous membranes and cilia)
5. To determine whether a nasal obstruction (e.g., polyps, tumor) is present in the nasal cavity
6. To determine whether any foreign bodies (e.g., small object inserted by a child) are lodged in the nasal cavity
7. To remove a nasal obstruction or foreign material from the nasal cavity
8. To determine whether an infection has moved beyond the sinuses
9. To diagnose chronic recurrent sinusitis in children with asthma
10. To diagnose reason for anosmia (loss of smell).
11. To evaluate any discharges from the nasal cavities like CSF.
12. To diagnose reason for facial pain / headaches.

Procedure: Topical anesthetic 4% xylocaine is used to anesthetize the nasal cavity before the procedure. About 7 ml of 4% xylocaine is mixed with 10 drops of xylometazoline. Cotton pledgets are dipped in the solution, squeezed dry and used to pack the nasal cavity. Pledgets are packed in the inferior, middle and superior meati. Packs are left in place for full 5 minutes. Diagnostic endoscopy is performed using a 30-degree nasal endoscope. If 2.7 mm scope is available it is preferred because it can reach the smallest crevices of the nose. 4mm endoscope is sufficient to examine adult nasal cavities.

The process of examination can be divided into three passes:

1. First pass / inferior pass
2. Second pass
3. Third pass.

First pass: In this the endoscope is introduced along the floor of the nasal cavity. Middle turbinate is the first structure to come into view. Its superior attachment is studied. Inferior surface of the middle turbinate is studied. As the endoscope is slid posteriorly the adenoid tissue comes into view. On the lateral surface of the nasopharynx the pharyngeal end of eustachean tube can be identified. Its function can be assessed by asking the patient to swallow. The endoscope is now turned 90 degrees in the opposite direction, the uvula and soft palate comes into view. The endoscope is again rotated by 90 degrees in the same direction, the opposite side pharyngeal end of eustachean tube is visualized. In this field both eustachean tubes become visible.

Second pass: After the first pass is over, the scope is gently withdrawn out and slide medial to the middle turbinate. The relationship between the middle turbinate and nasal septum is studied. This relationship is classified as TS1, TS2, and TS3. It depends on whether, after application of decongestant both the medial and lateral surfaces of the middle turbinate is visible (TS1), part of the middle turbinate is obscured by septal deviation (TS2), or the septal deviation is completely obscures the middle turbinate (TS3). The scope is gently slipped medial to the middle turbinate. The sphenoid ostium comes into view. Secretions if any from the ostium is noted.

Third pass: Is the most important of all the three passes. This pass studies the crucial middle turbinate area. The middle turbinate is evaluated for its shape and size as well as its relationship to the lateral nasal wall and septum. A bulge just above and anterior to the attachment of the middle turbinate suggests an enlarged agger nasi cells. Sometimes the anterior tip of the middle turbinate may be triangular. This shape has no significance unless it causes obstruction to the middle meatus. A middle turbinate that is concave medially rather than laterally is considered paradoxical. But paradoxical turbinate which is symptomatic needs to be treated. If the middle turbinate is enlarged due to the presence of a large air cell inside the middle turbinate it is known as concha bullosa. The middle turbinate is gently medialized using its plasticity. The middle meatus comes into view. The attachment of the uncinate process is carefully noted. Discharge if any from this area is also recorded. If accessory ostium is present it comes into view now. Accessory ostium is present more posteriorly. Normal ostium is actually not visible during diagnostic nasal endoscopy. Accessory ostium is spherical in shape and oriented anteroposteriorly, while the natural ostium of maxillary sinus is oval in shape and oriented transversely.

18. Functional Endoscopic sinus surgery

FESS is the acronym for Functional Endoscopic Sinus Surgery. This procedure has revolutionized the management of sinus infections to such an extent the hitherto commonly performed antral lavage has been relegated to history.

Middle meatus area: This is a crucial area for the drainage of anterior group of sinuses. Any pathology in this area could effectively compromise this rather critical drainage process. The success of FESS depends on how effectively this area is cleared.

Stamberger's hypothesis:

Stamberger proved that drainage from the maxillary sinuses always occurred through the natural ostium. He also demonstrated that the cilia of the epithelium covering the maxillary sinus cavity always beat towards the natural ostium propelling the mucous and secretions through the ostium. He also demonstrated that a more dependent inferior meatal antral opening had no role in this clearance because the cilia always pushed the secretions towards the natural ostium. So he found there is no logic in performing inferior meatal antrostomy to clear the pent up secretions.

Pathology affecting middle meatus:

1. Gross deviated nasal septum

2. Concha bullosa of middle turbinate obstructing the middle meatus

3. Infections involving the anterior ethmoidal air cells

Aim of FESS:

1. Disease clearance

2. Improvement of drainage

Instruments:

1. Nasal endoscope

2. Camera (endo)

3. Monitor

4. Surgical instruments

Procedure: Could be performed both under local / G.A.

1. Uncinectomy

2. Bullectomy

3. Identification of natural ostium

4. Widening the natural ostium

19. Orbital complications of sinusitis.

Classification of orbital complications of sinusitis will help otolaryngologist in devising effective treatment modalities. Any classification system devised will have to take into consideration the anatomy of the orbit and the mechanism causing it. Hubert was the first person to embark on

classifying these complications. He studied clinical data from about 114 patients during preantibiotic era. He based his classification on the anatomy of orbit, perceived progression of infection, responsiveness to treatment and general prognosis. Chandler fine-tuned this classification system and made it more acceptable.

Chandler's classification system:

Chandler grouped his patients under 5 heads:

Group I – Preseptal cellulitis
Group II – Orbital cellulitis
Group III – Subperiosteal abscess
Group IV – Orbital abscess
Group V – Cavernous sinus thrombosis

Group I (Preseptal cellulitis): This is actually inflammatory oedema anterior to orbital septum causing the eyelids to swell. This condition is caused due to restricted venous drainage. The eyelids though swollen are not tender. Since the inflammation doesn't involve postseptal structures there is no chemosis, Extraocular muscle movement limitations and vision impairment. Proptosis may be present to a mild degree.

Group II: Orbital cellulitis causes pronounced oedema and inflammation of orbital contents without abscess formation. It is imperative to look for signs of proptosis and reduced ocular mobility as these are reliable signs of orbital cellulitis. Chemosis is usually present in this group. Loss of vision is very rare in this group, but vision should be constantly monitored.

Group III: In this group abscess develops in the space between the bone and periosteum. Orbital contents may be displaced in an inferolateral direction due to the mass effect of accumulating pus. Chemosis and proptosis are usually present. Decreased ocular mobility and loss of vision is rare in this group.

Group IV: Orbital abscess usually involves collection of purulent material within the orbital contents. This could be caused due to relentless progression of orbital cellulitis or rupture of orbital abscess. Severe proptosis, complete ophthalmoplegia, and loss of vision are commonly seen in this group of patients.

Group V: Cavernous sinus thrombosis – Development of bilateral ocular signs is the classic feature of patients belonging to this group. These patients classically manifest with fever, headache, photophobia, proptosis, ophthalmoplegia and loss of vision. Cranial nerve palsies involving III, IV, V1, V2 and VI are common.

Schramm's modification of Chandler's classification:

Schramm after studying his patients classified those patients with preseptal cellulitis with chemosis as a separate entity. Prognostically he placed these patients between Chandler's group I and group III patients. Schramm considered these patients as a separate entity as they did not consistently improve with antibiotics and surgery needs to be advocated.

Moloney's modification of Chandler's classification:

Moloney modified Chandler's classification by according lower priority to preseptal orbital infections. In a nutshell he divided orbital complications into preseptal and post septal complications.

Signs indicating post septal complications are:

Proptosis
Gaze restriction
Decreased visual acuity
Color vision defects

Afferent pupillary defect

20. Oroantral fistula

This is a fistulous communication between the floor of the maxillary sinus to the oral cavity. This commonly occurs following dental extraction of infected upper molar and premolar tooth.

The upper lateral teeth when removed has a tendency to form blood clots. Fibrosis sets in within the clot material aiding the healing process. Fibrosis inside the clot is the most critical stage in the healing process. During this process of healing the air pocket within the maxillary sinus could keep constantly extruding hampering the healing process. This eventually leads to the formation of oroantral fistula. In order to prevent this fistula formation, the mucosal flaps after extraction of upper lateral teeth should be sutured.

Clinical features:

1. Patients manifest with signs and symptoms of maxillary sinus infections.

2. Purulent discharge could be seen from the middle meatus

3. History of dental extraction - positive

4. Fistulous communication could be seen within the oral cavity through which pus could be seen extruding

Valsalva test: This is confirmatory test for the presence of oroantral fistula. This test is performed by asking the patient to blow air through the nose after pinching the nose closed. The patient must keep the mouth open. The air could be heard hissing out of the fistula. This test could be negative in some patients in whom oedematous middle ear mucosa occludes the fistula (false negative).

Probe test: using a blunt probe, an attempt should be made to probe the suspected fistulous area.

Radiology:

1. X ray para nasal sinuses water's view shows haziness of the involved maxillary antra.

2. CT scan of para nasal sinuses is diagnostic. The defect can be clearly seen ion the bone window cuts.

CT scan showing hazy antrum with bone defect showing the fistula

Management:

1. Wait and watch approach: A significant amount of these fistulas tend to heal spontaneously. This is more so if the size of the fistula is 2 mm or less. If the size is 3 mm or more then spontaneous healing is hampered because of sinus infection in the periodontal area.

2. Caldwell Luc procedure: This surgery aims at creating a more permanent drainage via the antrostomy performed through the inferior meatus. This helps in spontaneous healing of the fistula.

3. Direct closure of the fistula can be attempted using palatal flaps.

21. Frontal mucocele

These are epithelial lined, mucus containing sac which completely fills the sinus cavity. They are also capable of expansion. This condition is different from that of a blocked sinus cavity which simply contains mucus.

Sites of occurrence: The fronto ethmoidal region is the most common site, followed by sphenoid sinus. Mucoceles involving the maxillary sinus are pretty rare. The fronto ethmoidal region is commonly involved because of its complex drainage pattern when compared to that of maxillary and sphenoid sinuses.

Etiology: Mucoceles are fairly uncommon, and in 1/3 of cases occur without any predisposing factors. It is so slow growing that there may be a considerable time lag between the initiating factor and the clinical presentation of mucocele. In the case of surgery or trauma the average time lag could exceed two decades. Acute infections have a somewhat lesser time lag (2 years). Mucoceles are thought to arise as a consequence of obstruction and inflammation.

Pathogenesis: Three main theories of pathogenesis are available. They are:

1. Pressure erosion

2. Cystic degeneration of glandular tissue

3. Active bone resorption and regeneration

Mucoceles are lined by pseudostratified columnar epithelium with squamous metaplasia. There may also be associated goblet cell hyperplasia. The cellular infiltrate present within the lining mucosa include components of both acute and chronic inflammation.

Clinical features:

Patients with frontoethmoidal mucoceles are first seen by the ophthalmologist because of proptosis. CT scan paranasal sinuses is diagnostic.

Xray paranasal sinuses water's view will show exapansile mass inside the frontal sinus with loss of frontal sinus hausterations. This is one of the important diagnostic features of frontoethmoidal mucocele.

Diagnostic nasal endoscopy may reveal an expansile mass presenting inside the nasal cavity. Mucoceles of maxillary sinus may expand into the nasal cavity producing nasal obstruction, or may erode the anterior wall of maxilla at the level of canine fossa causing a cheek swelling.

Sphenoidal mucoceles due to its intimate relationship with orbital apex and cavernous sinus may present with signs of visual disturbances.

Frontoethmoidal mucoceles will present as swelling of the orbit, causing proptosis. It may also erode the outer table of the frontal sinus causing a swelling over the forehead region.

Management:

Endoscopic sinus surgery is almost curative in all these cases.

Tip:

Whatever be the type of approach used, there is no necessity to reconstruct the areas where bone resorption has occurred. As long as the mucosal lining is intact, restitution of contour occurs rapidly. In young patients reossification can also occur.

22. Fungal sinusitis.

Fungi are eukaryotic organisms comprising of moulds, yeasts, mushrooms and other similar organisms. Among this group of organisms only about 0.1% are human pathogens. The term mycosis is used to define diseases caused by fungi.

Mycosis can be classified under 4 heads based on the portal of entry and site of infection.

Types of mycosis:

Type	*Pathophysiology*	*Route*	*Example*
Superficial	*Limited to keratinized tissues*	*Topical*	*T. pedis*
Subcutaneous	*Localised to subcutaneous tissues*	*Broken skin*	*Rhinosporidiosis*
Systemic	*Disseminated widely*	*Inhalation*	*Histoplasmosis*
Opportunistic	*Local / Disseminated*	*Cell mediated immunity compromise*	*Candida / Mucormycosis*

Fungal infections of nose and sinuses are getting common these days. With increasing incidence of HIV and other diseases like diabetes which compromise host immunity it is no wonder that the incidence of fungal infections involving nose and para nasal sinuses is on the rise. In India the incidence of fungal sinusitis in immuno competent patients is also showing a rise. This particular fact need to be studied further.
Classification of fungal sinusitis: There are 5 different types of fungal sinusitis with differing pathophysiology and clinical presentation. They include:

1. Acute fulminant invasive sinusitis

2. Chronic invasive fungal sinusitis

3. Granulomatous invasive fungal sinusitis

4. Fungal ball

5. Allergic fungal rhino sinusitis

Recently one more group is added i.e. Eosinophilic fungal rhinosinusitis.
Acute fulminant invasive sinusitis: The whole duration of illness in these patients is just less than 4 weeks. These patients are mostly immunocompromised individuals. The reduced immunity could very well be a result of

a. Diabetes mellitus

b. AIDS

c. Immunosuppressive medicines

d. Malignancy causing immune suppression

The fungus commonly associated with this infection belongs to Mucoraceae family or Aspergillus family. If Mucor is involved then the lesion is Angio invasive and destroys both bone and soft tissue. Patients belonging to this group have high mortality rate. Extensive surgical debridement of the lesion with removal of necrotic tissue should be performed and must be followed with intravenous antifungal medication i.e. amphotericin B. If possible, the underlying cause for immune compromise should also be addressed. Since granulocytes are essential to combat this condition, granulocyte infusions have been tried out with success after surgical debridement.

Chronic invasive fungal sinusitis: This condition is also known as Non granulomatous chronic invasive fungal sinusitis. This condition commonly afflicts patients with diabetes mellitus. This disorder is characterized by low grade inflammation with tissue necrosis. There is very little vascular invasion. Granuloma formation which is common in these patients requires an appropriate cell mediated immune response which is common in diabetics.
The duration of this disease is longer than 4 weeks (more than 6 weeks in some cases). Orbital extension is common, causing proptosis. These patients can be managed by wide surgical debridement, followed by systemic intravenous antifungal drugs like amphotericin B.

Granulomatous invasive fungal sinusitis: This condition is also known as Indolent fungal sinusitis. These patients have an intact cell mediated immune response. Clinically it is virtually impossible to distinguish it from non-granulomatous fungal sinusitis. Histopathology will clinch the diagnosis. The intact immune system of the patient limits the invasion to the superficial mucosa. Granulomas could be seen surrounding the fungal elements thus preventing their deeper invasion. The granulomas could be seen surrounded by multinucleated giant cells, eosinophils etc. Surgical debridement alone is sufficient to cure these patients. Their intact immune system takes over from now on.

Fungal Ball: These mycetoma's commonly present as unilateral opacification of maxillary or sphenoid sinus. The ethmoids and frontal sinuses are very rarely involved. These patients are usually immunocompetent without evidence of atopy. The fungal ball is composed of tightly packed hyphae often from Aspergillus, Alternaria and P. boydii. Treatment is mostly surgical removal of the fungal mass combined with widening of ostium there by increasing the ventilation of the sinuses. Antifungal drugs are not indicated in these patients.

Allergic fungal sinusitis: These patients have a combination of nasal polyposis, crust formation associated with positive sinus cultures for aspergillus. Robson (1989) introduced the term allergic

fungal sinusitis to describe the findings associated with this disease. These patients consistently demonstrate allergic reactions to aspergillus proteins.
Bent's criteria for the diagnosis of allergic fungal sinusitis:
1. Demonstrable type I hypersensitivity to fungi
2. Nasal polyposis
3. Radiological findings (Heterodense mass lesion)
4. Presence of eosinophilic mucin mixed with non invasive fungus
5. Positive fungal stain / fungal culture

These patients present with progressive nasal obstruction, crusting, rhinorrhea, and chronic rhinosinusitis. These patients can also come with dramatic symptoms like visual loss and total nasal obstruction.

Classically radiology shows unilaterally expansile lesion of the sinuses associated with bony erosion. The mass appears as heterodense due to the presence of metallic elements in the fungal hyphae.

The mechanism of causation of allergic fungal sinusitis is IgE mediated hypersensitivity to fungal proteins especially to aspergillus.

Eosinophilic fungal sinusitis: This terminology was first suggested by Ponikau et al to explain the pathogenesis of not only allergic fungal sinusitis but also of chronic rhinosinusitis. This disorder is usually bilateral. Using sensitive techniques, he was able to demonstrate the presence of fungal hyphae in 97% of cases with chronic rhinosinusitis. Since he failed to demonstrate typical type I allergic features like elevated IgE levels in sera he classified these patients under this head since he was not able to include them in the allergic fungal sinusitis group. He hence suggested an alternative theory saying that chronic sinusitis belonging to this group is caused by abnormal cell mediated response to fungal proteins.

Pathophysiology of eosinophilic fungal sinusitis:

In response to exposure to fungal proteins allergic mucin is formed in the mucosa. This allergic mucin attracts eosinophils from blood vessels. These eosinophils surround fungal hyphae and secrete substances like Major basic proteins and various interleukins.

Management of eosinophilic fungal sinusitis include surgical removal of polypi with creation of good antrostomy to improve ventilation of the sinuses. Oral steroids are indicated in these patients to prevent recurrence. Prednisolone is administered in doses of 0.1mg/kg/day in cycles of four days and is gradually tapered after 6 weeks. After tapering of oral steroids, topical nasal steroids can be administered for a period of 6 - 9 months.

23. Fungal polyposis

In this condition the colonizing fungi elicits allergic mucosal inflammation without features of invasion. The protein components of fungi elicit IgE mediated allergic mucosal inflammation.

History / physical findings: Presentation of AFRS may range from dramatic to subtle. Dramatic features include: acute visual loss, gross facial dysmorphia or complete nasal obstruction.

Subtle features are more common and include:

gradual nasal airway obstruction – may be very gradual that the patient may even be unaware
presence of semisolid nasal crusts containing allergic fungal mucin
Pain is usually not present, if presence concomitant bacterial rhinosinusitis should be suspected

Physical findings include:

Signs of intranasal inflammation
Polyposis
Gross facial disfigurement
Orbital / ocular abnormalities

Diagnostic criteria:

Patients with AFRS demonstrate five characteristics:

Gross production of Eosinophilic mucin containing noninvasive fungal hyphae
Nasal polyposis
Radiological findings
Immunocompetence
Allergy to fungi
Radiologic features:

Slow accumulation of allergic fungal mucin provides unique characteristics to the disease. Usually allergic fungal mucin is sequestered within involved paranasal sinus cavities. Its accumulation leads to the classic radiologic findings seen in this disease.

Even though it is a bilateral condition, there is a certain degree of asymmetry seen in radiographs / CT
Bone erosion and extension of the disease into adjacent areas can be seen
Expansion, remodeling and thinning of sinus walls are commonly seen – this is due to the expansile nature of accumulating mucin
Heterogeneous areas of signal intensities within paranasal sinuses filled with allergic fungal mucin are frequently seen in CT scans – this is due to the accumulation of heavy metals (iron / manganese) and calcium salt precipitation within inspissated allergic fungal mucin.
Desiccation of sinus contents contributes to the hyper dense areas seen on CT scans

MRI features: The high protein and low water concentration of allergic fungal mucin coupled with high water content within surrounding oedematous sinus mucosa gives rise to specific MRI characteristics.

T1 – Involved sinus cavities demonstrate varying signal intensities. There is enhancement of periphery of the involved sinuses due to mucosal Oedema

T2 – Hypo intensity of signal within involved sinuses – due to dehydrated state of mucin

Enhancement of periphery of the involved sinus due to mucosal oedema

Investigations:

Estimation of IgE – Total IgE values is elevated in AFRS. A value of more than 1000 IU/ml is an indicator of AFRS activity.

RAST / ELISA Test – Positive for bipolaris specific IgE and IgG antibodies. These patients show positive evidence of fungal allergy.

Histologic characteristics of fungal mucin:

The production of allergic mucin is pathognomonic of AFRS. Grossly allergic mucin is thick, tenacious and viscous in consistency. Its color may vary from brown to dark green. It is only the mucin rather than sinus mucosa that provides the relevant histological evidence necessary to make the diagnosis of AFRS. Examination of nasal mucosa / polypi shows evidence of chronic inflammation. Eosinophils are also seen in abundance. Pathologic examination of these tissues is done to establish that fungal invasion is not present.

Histology of allergic fungal mucin reveals the characteristic ranching non invasive fungal hyphae within sheets of eosinophils and Charcot – Layden crystals. Classically H&E stains accentuate the mucin and cellular components of allergic mucin but fail to stain the fungal hyphae. Silver stains are specifically used to stain fungal hyphae. Silver stains color fungi black / dark brown.

Fungal culture: These tests atmost provide supportive evidence. Diagnosis of AFRS is not based on positive fungal cultures from mucin.

Treatment: Still evolving. Previously it used to be radial surgery. Now a combination of conservative surgery in combination with adjunct medical therapy is becoming popular.

The goal of any surgical procedure is to eradicate all allergic mucin while providing permanent drainage and ventilation for the affected tissues.

Even in the best of hands the incidence of AFRS recidivism is very high when treated with surgery alone. Adjunctive medical therapy should also be tried to get over this problem. Fungal immunotherapy and immunomodulation is becoming popular these days.

Role of steroids is limited only to postpone the surgery.

24. Antrochoanal polyp

Definition: Antrochoanal polyp is a benign solitary polypoidal lesion arising from the maxillary sinus antrum causing opacification and enlargement of antrum radiologically without any evidence of bone destruction. It exits the antrum through the accessory ostium reaches the nasal cavity, expands posteriorly to exit through the choana into the post nasal space.

Incidence: It commonly affects children and young adults.

Etiopathogenesis: This disease is commonly seen only in non atopic persons. Its etiology is still unknown. In fact this disorder is not associated with nasal allergy.
Proetz theory: Proetz suggested that this disease could be due to faulty development of the maxillary sinus ostium, since it was always been found to be large in these patients. Hypertrophic mucosa of maxillary antrum sprouts out through this enlarged maxillary sinus ostium to get into the nasal cavity. The growth of the polyp is due to impediment to the venous return from the polyp. This impediment occur at the level of the maxillary sinus ostium. This venous stasis increases the oedema of the polypoid mucosa thereby increasing its size.

Bernoulli's phenomenon: Pressure drop next to a constriction causes a suction effect pulling the sinus mucosa into the nose.

Mucopolysaccharide changes: Jakson postulated that changes in mucopolysaccharides of the ground substance could cause nasal polyp.

Infections: Recurrent nasal infections have also been postulated as the cause for nasal polyp

Vasomotor imbalance theory: This theory attributes polyp formation due to autonomic imbalance

Polypoidal tissue from the maxillary antrum exits out through the accessory maxillary sinus ostium according to some workers. This accessory sinus ostium is placed posteriorly, which could be the reason for the polyp to present posteriorly. The accessory sinus ostium widens progressively, ultimately at one stage merging with the natural ostium of the maxillary sinus forming one huge opening into the maxillary antrum.

Possible reasons for migration of Antrochoanal polyp in to the post nasal space:

1. The accessory ostium through which the polyp gets out of the maxillary antrum is present posteriorly.

2. The inspiratory air current is more powerful than the expiratory air current thereby pushes the polyp posteriorly.

3. The natural slope of the nasal cavity is directed posteriorly, hence the polyp always slips posteriorly.

4. The cilia of the ciliated columnar epithelial cells lining the nasal cavity always beats anteroposteriorly pushing the polyp behind.

Histology: Shows respiratory epithelium over normal basement membrane. The interstitial layer is grossly oedematous, with no eosinophils. The interstial layer contains other inflammatory cells.

Clinical features: Since the disorder is unilateral (commonly) the patient always present with

1. Unilateral nasal obstruction
2. Unilateral nasal discharge
3. Headache (mostly unilateral)
4. Epistaxis
5. Sleep apnoea
6. Rhinolalia clausa due to presence of polyp in the post nasal space
7. Difficulty in swallowing if the polyp extends into the oropharynx

Anterior rhinoscopy may show the polyp as glistening polypoidal structures. They will be insensitive to touch. this feature helps to differentiate it from a hypertrophied nasal turbinate.

Postnasal examination will show the polyp if extending posteriorly at the level of choana. If it fills up the nasopharynx it will be visible there.

Xray paranasal sinuses will show a hazy maxillary antrum.

CT scan of paranasal sinuses is diagnostic. It will show the polyp filling the maxillary antrum and exiting out through the accessory ostium into the nasal cavity.

The Antrochoanal polyp is dumb bell shaped with three components i.e. antral, nasal and nasopharyngeal.

Treatment:

This is a surgical problem. Formerly it was treated by avulsion of the polyp trans nasally. This method led to recurrences. A Caldwell luc approach was preferred in patients with recurrences. In Caldwell luc procedure in addition to the polypectomy, the maxillary antrum is entered via the canine fossa and the antral component is completely excised.

Endoscopic approach: With the advent of nasal endoscope this approach is the preferred one. Using an endoscope, it is always easy to completely remove the polypoid tissue. The uncinate process

must also be completely excised. Endoscopic approach has the advantage of a complete surgical excision with negligible recurrence rates.

25. Ethmoidal polyposis

Polyp is defined as simple oedematous hypertrophic mucosa. Ethmoidal polypi arise from ethmoidal air cell lining mucosa. This condition is bilateral. In anterior rhinoscopy ethmoidal polypi appears as a bunch of grapes.

Ethmoidal polypi is caused by allergy.

Management:

It responds well to systemic / topical steroids

This is actually a medical problem.

Polypectomy is indicated when the entire nasal cavity is occluded by the presence of ethmoidal polypi.

26. Difference between Antro choanal polyp and Ethmoidal polyp.

Ethmoidal polypi	Antrochoanal polyp
Seen in adults	Seen in children & adolescents
Allergy is the common cause	Infection is the common cause
Multiple and appears like a bunch of grapes	Single
Arises from ethmoidal labyrinth	Arises from maxillary antrum
Easily seen in anterior rhinoscopy	Seen commonly in post nasal examination
X-ray PNS may show hazy ethmoids and normal maxillary sinus	X-ray PNS shows hazy maxillary sinus antrum
Bilateral	Usually unilateral
Recurrence is common	Recurrence is uncommon
Polypectomy is done	Caldwell Luc surgery is needed in recurrent cases

27. Nasal Polyposis

Nasal polyp: Polyp is a Latin word meaning polypous (many footed).

Definition:

Polyp is defined as simple oedematous hypertrophied mucosa of the nasal cavity. It can be unilateral / bilateral.

Causes of nasal polyp:

1. Infections

2. Allergy

3. Polyp due to mucovisidosis

Classification:

Nasal polyp can be classified as:

1. Simple polyp

. Ethmoidal polypi

. Antrochoanal polyp

2. Fungal polyp

3. Malignant polyp

28. Nasal cholesteatoma

This is another name for Rhinitis caseosa. The term “Rhinitis caseosa” was coined by Duplay in 1868. Eggston and Wolff after a detailed study in 1947 concluded that this condition could occur secondarily following pent up secretions in the sinus cavities. Their studies revealed that this condition is more common in patients with extensive bilateral ethmoidal polyposis. The presence of polypi in the nasal cavities caused obstruction to the normal drainage mechanism of the paranasal sinuses. This led to accumulation of secretions. Whitish to yellow cheesy material were found within the nasal cavities of these patients behind the nasal polypi. This cheesy material also caused expansion of sinus cavities, erosion of bone and extension into orbit.

Eggston used the term pseudocholesteatoma to describe this condition which resembled cholesteatoma only morphologically. Histologically squamous elements could not be identified in them. Histologically this tissue comprises of inflammatory tissue, granulation mixed with mucoid debris.

Presently allergic fungal sinusitis also resembles this condition and hence included under this category.

Clinical features:

Nasal obstruction
Presence of nasal polypi
Telecanthus
Swelling over medial canthal region
Proptosis with the eye being pushed downwards and laterally
These patients may present with loss of vision due to involvement of optic nerve
Intracranial extension due to erosion of anterior cranial fossa skull base is also seen
This disease could be considered to be end stage manifestation of sinus inflammation. Hence the use of the term “Rhinitis caseosa syndrome” makes lot of sense.

Pinus theory of nasal cholesteatoma:

According to Pinus a majority of patients suffering from nasal cholesteatoma also suffered from dental cysts which obliterated the entire maxillary sinus cavity.

Management:

Rhinitis caseosa can be managed by debridement followed by regular douching using normal saline.

29. RhinoCerebral-Phycomycosis

Rhinocerebral mucormycosis is a rare opportunistic infection of the sinuses, nasal passages, oral cavity, and brain caused by saprophytic fungi. The infection can rapidly result in death. Rhinocerebral mucormycosis commonly affects individuals with diabetes and those in immunocompromised states.

The pathogens that cause rhinocerebral mucormycosis are prevalent in nature but may be more prone to cause infection in moist, temperate climates.

Saprophytic aerobic fungi of the class Phycomycetes (order Mucorales) cause rhinocerebral mucormycosis, also known as phycomycosis. The 3 genera responsible for most cases are Rhizopus, Absidia, and Mucor. Researchers have also reported cases of rhinocerebral mucormycosis caused by Rhizomucor, Saksenaea, Apophysomyces, and Cunninghamella species.

Phycomycetes are ubiquitous in nature, being commonly found in decaying vegetation, soil, and bread mold. They grow rapidly and can release large numbers of airborne spores. Thus, they are frequently found colonizing the oral mucosa, nose, paranasal sinuses, and throat. Phycomycetes do not generally cause disease in immunocompetent individuals who are able to generate phagocytic containment of the organisms. Persons at risk for infection (ie, immunocompromised individuals) typically also have decreased phagocytic activity because of an impaired glutathione pathway.

In individuals who are immunocompromised, germination and hyphae formation occur, and this allows the organism to invade the patient's blood vessels. Mucormycosis is described almost exclusively in patients with compromised immune systems or metabolic abnormalities.

Rhizopus species have an active ketone reductase system that enables them to thrive in an acidic pH and glucose-rich medium. Hyperglycemia enhances fungal growth and impairs neutrophil chemotaxis; therefore, individuals with diabetic ketoacidosis are commonly affected. Rhizopus species also favor an iron-rich environment and are frequently isolated in patients receiving deferoxamine therapy (an iron-chelating agent).

Risk factors

Seventy percent of mucormycosis cases occur in patients with diabetes mellitus, although this percentage is declining with the use of chemotherapy and with increasing frequency of various types of immunocompromised states. An underlying risk factor is recognized in more than 96% of mucormycosis cases. Risk factors for rhinocerebral mucormycosis include the following:

Diabetes mellitus

Iron overload

Burns

Human immunodeficiency virus (HIV) and acquired immunodeficiency syndrome (AIDS)

Blood dyscrasias

Transplantation

Immunosuppression (ie, prednisone therapy)

Chemotherapy

Intravenous drug use - Embolic to brain

Disease states treated with high-dose steroids

Diabetes mellitus

General symptoms of rhinocerebral mucormycosis include the following:

Headache

Nausea

Fever

Lethargy

Facial symptoms of the disease include the following:

Weakness

Numbness

Pain

Nasal symptoms of rhinocerebral mucormycosis include:

Purulent drainage

Stuffiness and rhinorrhea

Epistaxis

Nasal hypoesthesia

Ocular symptoms include the following:

Periorbital or retro-orbital pain

Diplopia and blurred vision

Amaurosis (unilateral or bilateral)

Possible rapid progress to blindness

Treatment of rhinocerebral mucormycosis includes the following:

Reversing underlying immunocompromised states

Administering systemic antifungals

Performing urgent surgical debridement

30. Rhinosporidiosis

Rhinosporidium seeberi: was initially believed to be a sporozoan, but it is now considered to be a fungus and has been provisionally placed under the family Olipidiaceae, order chritridiales of phycomyetes by Ashworth. More recent classification puts it under DRIP'S clade. Even after extensive studies there is no consensus on where Rhinosporidium must be placed in the Taxonomic classification. It has not been possible to demonstrate fungal proteins in Rhinosporidium even after performing sensitive tests like Polymerase chain reactions.

Of all the reported cases 95 % were from India and Srilanka. An all India survey conducted in 1957 revealed that this disease is unknown in states of Jummu & Kashmir, Himachal pradesh, Punjab, Haryana, and North Eastern states of India. In the state of TamilNadu 4 endemic areas have been identified in the survey, (Madurai, Ramnad, Rajapalayam, and Sivaganga). The common denominator in these areas is the habit of people taking bath in common ponds.

Theories of mode of spread:

Demellow's theory of direct transmission
Autoinoculation theory of Karunarathnae (responsible for satellite lesions)
Haematogenous spread - to distant sites
Lymphatic spread - causing lymphadenitis (rarity)
Demellow's theory of direct transmission - This theory propounded by Demellow had its acceptance for quite sometime. He postulated that infection always occured as a result of direct transmission of the organsim. When nasal mucosa comes into contact with infected material while bathing in common ponds, infection found its way into the nasal mucosa.

Karunarathnae accounted for satellite lesions in skin and conjunctival mucosa as a result of auto inoculation.

Rhinosporidiosis affecting distant sites could be accounted for only through haematogenous spread.

Karunarathnae also postulated that Rhinosporidium existed in a dimorphic state. It existed as a saprophyte in soil and water and it took a yeast form when it reached inside the tissues. This dimorphic capability helped it to survive hostile environments for a long period of time.

Reasons for endemicity of Rhinosporidiosis:

It has to be explained why this disease is endemic in certain parts of South India and in the dry zone of Srilanka. If stagnant water could be the reason then the chemical and physical characteristics of the water needs to be defined. In addition other aquatic organisms may also be playing an important synergistic reaction. This aspect need to be elucidated. Text book of microbilogy is

replete with examples of such synergism i.e. lactobacillus with trichomonas, and Wolbachia with filarial nematodes.

Host factors responsible for endemicity: Even though quite a large number of people living in the endemic areas take bath in common ponds only a few develop the disease. This indicates a predisposing, though obscure factors in the host. Blood group studies indicate that rhinosporidiosis is common in patient's with group O (70%), the next high incidence was in group AB. Jain reported that blood group distribution is too variable to draw any conclusion. Larger series must be studied for any meaningful analysis. HLA typing also must be studied. The possibility of non-specific immune reactivity especially macrophages in protecting the individual from Rhinosporidium seeberi must be considered.

Life cycle: (Ashworth) Spore is the ultimate infecting unit. It measures about 7 microns, about the size of a red cell. It is also known as a spherule. It has a clear cytoplasm with 15 - 20 vacuoles filled with food matter. It is enclosed in a chitinous membrane. This membrane protects the spore from hostile environment. It is found only in connective tissue spaces and is rarely intracellular.

The spore increases in size, and when it reaches 50 - 60 microns in size granules starts to appear, its nucleus prepares for cell division. Mitosis occurs and 4, 8, 16, 32 and 64 nuclei are formed. By the time 7th division occurs it becomes 100 microns in size. A fully mature sporangia measures 150 - 250 microns. Mature spores are found at the centre and immature spores are found in the periphery. The full cycle is completed within the human body.

Life cycle (recent): Since rhinosporidium seeberi has defied all efforts to culture it, any detail regarding its life cycle will have to be taken with a pinch of salt. This life cycle has been postulated by studying the various forms of rhinosporidium seen in infected tissue.

Trophozoite / Juvenile sporangium - It is 6 - 100 microns in diameter, unilamellar, stains positive with PAS, it has a single large nucleus, (6micron stage), or multiple nuclei (100 microns stage), lipid granules are present.

Intermediate sporangium - 100 - 150 microns in diameter. It has a bilamellar wall, outer chitinous and inner cellulose. It contains mucin. There is no organized nucleus, lipid globules are seen. Immature spores are seen within the cytoplasm. There are no mature spores.

Mature sporangium - 100 - 400 microns in diameter, with a thin bilamellar cell wall. Inside the cytoplasm immature and mature spores are seen. They are found embedded in a mucoid matrix. Electron dense bodies are seen in the cytoplasm. The bilamellar cell wall has one weak spot known as the operculum. Maturation of spores occur in both centrifugal and centripetal fashion. This spot does not have chitinous lining, but is lined only by a cellulose wall. The mature spores find their way out through this operculum on rupture. The mature spores on rupture are surrounded by mucoid matrix giving it a comet appearance. It is hence known as the comet of Beattee

Mature spores give rise to electron dense bodies which are the ultimate infective unit.

Clinical classification of Rhinosporidiosis:

Nasal
Nasopharyngeal
Mixed
Bizzarre (ocular and genital)
Malignant rhinosporidiosis (cutaneous rhinosporidiosis

Common sites affected:

Nose - 78%
Nasopharynx - 68%
Tonsil - 3%
Eye - 1%
Skin - very rare
Gross features of rhinosporidiosis:

Lesions in the nose can be polypoidal, reddish and granular masses. They could be multiple pedunculated and friable. They are highly vascular and bleed easily. Their surface is studded with whitish dots (sporangia). They can be clearly seen with a hand lens. The whole mass is covered by mucoid secretion. The rhinosporidium in the nose is restricted to the nasal mucous membrane and does not cross the muco cutaneous barrier.

Histopathology of nasal rhinosporidiosis:

There is papillomatous hyperplasia of nasal mucous membrane with rugae formation. The epithelium over the sporangia is thinned out, foreign body giant cells can be seen. Accumulation of mucous in the crypts seen with increased vascularity. The increased vascularity is responsible for excessive bleeding during surgery. Increased vascularity is due to the release of angiognenesis factor from the rhinosporidial mass. Rhinosporidial spores stain with sudan black, Bromphenol blue etc.

Endosporulation:

Endospores represent asexual spores of Rhinosporidium seeberi. After nuclear division in the juvenile sporangia, endospores are formed by condensation of cytoplasm around the nuclei with the formation of cell walls. This process is known as endosporulation. These endospores have been postulated to develop from the inner sporangial wall. Endospores are liberated from the sporangium by being shot out from the sporangium after its rupture (as suggested by Beattee), or through the operculum as suggested by Ashworth, or by osmotic mechanism as suggested by Demello. Endospores are thick walled measuring about 7 microns in diameter, round in shape and

stains with PAS. It has a vesicular nucleus and a granular cytoplasm. The peripheral cytoplasm is vacuolated containing deeply staining bodies called as spherules. These bodies give the spore a morullated appearance and hence the term spore morullae.

Features of rhinosporidiosis:

The cardinal features of rhinosporidiosis are 1. chronicity, 2. recurrence and 3. dissemination.

The reasons for chronicity are

1. Antigen sequestration - The chitinous wall and thick cellulose inner wall surrounding the endospores is impervious to the exit of endosporal antigens from inside, and is also impermeable to immune destruction. However, this sequestered antigen may be released after phagocytosis.

2. Antigenic variation - Rhinosporidial spores express varying antigens thereby confusing the whole immune system of the body.

3. Immune suppression - ? possible release of immuno suppressor agents

4. Immune distraction - Studies of immune cell infiltration pattern have shown that immune cell infiltration has occurred in areas where there are no spores, suggesting that these infiltrates reached the area in response to free antigen released by the spores. This serves as a distraction.

5. Immune deviation

6. Binding of host immunoglobins

Treatment:

Surgery is the treatment of choice. Rhinosporidial mass can be removed intranasally, the only problem being bleeding. Post operatively the patient is started on T. Dapsone in dose of 100 mg / day for a period of 6 months.

Unsolved problems:

Habitat - Breeds in ponds (highly theoretical, spores have not been isolated from ponds even on intense effort)
Lifecycle - In the absence of viable ways to culture the organism the life cycle remains highly speculative
Pathogenicity - does not fulfill any of the 4 criterial laid down by Koch regarding the infectivity.

31. Rhino scleroma

This is a progressive granulomatous lesion beginning in the nose and eventually extending into the Nasopharynx and oropharynx. Rarely larynx, trachea and lower air way may also be involved.

Scleroma may occur at any age. Both sexes may equally be affected.
This disease is common in central and south eastern Europe, North Africa, Pakistan and Indonesia.
One common factor seen in these patients is – poor standard of life and oral hygiene

Pathology: Organism causing this disorder is Klebsiella Rhinoscleromatis. This organism could be a secondary invader following a viral infection.
Granulomatous tissue infiltrates the submucosa and is characterized by the presence of an accumulation of plasma cells, lymphocytes and eosinophils among which are scattered large foam cells (Mikulicz cells). These foam cells have a central nucleus, and a vacuolated cytoplasm containing the bacilli.
Russell bodies have also been demonstrated. These cells resemble plasma cells and have eccentric nucleus with deep staining cytoplasm.

Investigations:
Levin test: This is a complement fixation test, based on the reaction of the patient's serum with suspensions of K. Rhinoscleromatis.
High titres of antibodies against K. Rhinoscleromatis has been demonstrated. This indicates humoral immunity to be intact in these patients.

Clinical features:

Three different stages have been documented:
1. Atrophic stage: In this stage changes appear in the nasal mucous membrane.
These changes resemble that of atrophic rhinitis. Foul smelling crusts are seen.
2. Granulation / nodular stage: Nodules are non ulcerative in nature. Initially these nodules are bluish red and rubbery. Later these nodes become a little paler and harder.

3. Cicatrizing stage: Adhesions and stenosis distort the normal nasal anatomy. The shape and contour of the nose changes causing a condition known as "Tapir's nose". The disease may extend to involve the maxillary sinus, naso lacrimal duct, Nasopharynx, trachea and bronchi. Lymphatic spread is uncommon because of extensive fibrous tissue deposition. This deposition blocks the lymphatics.

Treatment:

Diagnosis is confirmed by biopsy. Bactericidal antibiotics should be administered in large doses. It should be administered at least for a period of 4-6 weeks.
Surgical debridement should be considered when extensive cicatrization is present.

32. Allergic Rhinitis

Rhinitis, which occurs most commonly as allergic rhinitis, is an inflammation of the nasal membranes that is characterized by sneezing, nasal congestion, nasal itching, and rhinorrhea, in any combination. Although allergic rhinitis itself is not life-threatening (unless accompanied by severe asthma or anaphylaxis), morbidity from the condition can be significant.

Signs and Symptoms
History

Signs and symptoms of allergic rhinitis include the following:

Sneezing

Itching: Nose, eyes, ears, palate

Rhinorrhea

Postnasal drip

Congestion

Anosmia

Headache

Earache

Tearing

Red eyes

Eye swelling

Fatigue

Drowsiness

Malaise

Complications of this allergic rhinitis include the following:

Acute or chronic sinusitis

Otitis media

Sleep disturbance or apnea

Dental problems (overbite): Caused by excessive breathing through the mouth

Palatal abnormalities

Eustachian tube dysfunction

Physical examination

Nasal features of allergic rhinitis can include the following:

Nasal crease: A horizontal crease across the lower half of the bridge of the nose; caused by repeated upward rubbing of the tip of the nose by the palm of the hand

Thin, watery nasal secretions

Deviation or perforation of the nasal septum: May be associated with chronic rhinitis, although there can be other, unrelated causes

Manifestations of allergic rhinitis affecting the ears, eyes, and oropharynx include the following:

Ears: Retraction and abnormal flexibility of the tympanic membrane

Eyes: Injection and swelling of the palpebral conjunctivae, with excess tear production; Dennie-Morgan lines (prominent creases below the inferior eyelid); and dark circles around the eyes ("allergic shiners"), which are related to vasodilation or nasal congestion

Oropharynx: "Cobblestoning," that is, streaks of lymphoid tissue on the posterior pharynx; tonsillar hypertrophy; and malocclusion (overbite) and a high-arched palate.

Diagnosis
Laboratory tests used in the diagnosis of allergic rhinitis include the following:

Allergy skin tests (immediate hypersensitivity testing): An in vivo method of determining immediate (IgE-mediated) hypersensitivity to specific allergens

Fluorescence enzyme immunoassay (FEIA): Indirectly measures the quantity of immunoglobulin E (IgE) serving as an antibody to a particular antigen

Total serum IgE: Neither sensitive nor specific for allergic rhinitis, but the results can be helpful in some cases when combined with other factors

Total blood eosinophil count: Neither sensitive nor specific for the diagnosis, but, as with total serum IgE, can sometimes be helpful when combined with other factors.

Management
The management of allergic rhinitis consists of the following 3 major treatment strategies:

Environmental control measures and allergen avoidance: These include keeping exposure to allergens such as pollen, dust mites, and mold to a minimum

Pharmacologic management: Patients are often successfully treated with oral antihistamines, decongestants, or both; regular use of an intranasal steroid spray may be more appropriate for patients with chronic symptoms

Immunotherapy: This treatment may be considered more strongly with severe disease, poor response to other management options, and the presence of comorbid conditions or complications; immunotherapy is often combined with pharmacotherapy and environmental control.

33. Allergic Fungal Sinusitis

In this condition the colonizing fungi elicits allergic mucosal inflammation without features of invasion. The protein components of fungi elicit IgE mediated allergic mucosal inflammation.

History / physical findings: Presentation of AFRS may range from dramatic to subtle. Dramatic features include: acute visual loss, gross facial dysmorphia or complete nasal obstruction.

Subtle features are more common and include:

gradual nasal airway obstruction – may be very gradual that the patient may even be unaware
presence of semisolid nasal crusts containing allergic fungal mucin
Pain is usually not present, if presence concomitant bacterial rhinosinusitis should be suspected

Physical findings include:

Signs of intranasal inflammation
Polyposis
Gross facial disfigurement
Orbital / ocular abnormalities

Diagnostic criteria:

Patients with AFRS demonstrate five characteristics:

Gross production of Eosinophilic mucin containing noninvasive fungal hyphae
Nasal polyposis
Radiological findings
Immunocompetence
Allergy to fungi
Radiologic features:

Slow accumulation of allergic fungal mucin provides unique characteristics to the disease. Usually allergic fungal mucin is sequestered within involved paranasal sinus cavities. Its accumulation leads to the classic radiologic findings seen in this disease.

Even though it is a bilateral condition, there is a certain degree of asymmetry seen in radiographs / CT
Bone erosion and extension of the disease into adjacent areas can be seen
Expansion, remodeling and thinning of sinus walls are commonly seen – this is due to the expansile nature of accumulating mucin.
Heterogeneous areas of signal intensities within paranasal sinuses filled with allergic fungal mucin are frequently seen in CT scans – this is due to the accumulation of heavy metals (iron / manganese) and calcium salt precipitation within inspissated allergic fungal mucin.
Desiccation of sinus contents contributes to the hyper dense areas seen on CT scans.

MRI features: The high protein and low water concentration of allergic fungal mucin coupled with high water content within surrounding oedematous sinus mucosa gives rise to specific MRI characteristics.

T1 – Involved sinus cavities demonstrate varying signal intensities. There is enhancement of periphery of the involved sinuses due to mucosal Oedema

T2 – Hypo intensity of signal within involved sinuses – due to dehydrated state of mucin

Enhancement of periphery of the involved sinus due to mucosal oedema

Investigations:

Estimation of IgE – Total IgE values is elevated in AFRS. A value of more than 1000 IU/ml is an indicator of AFRS activity.

RAST / ELISA Test – Positive for bipolaris specific IgE and IgG antibodies. These patients show positive evidence of fungal allergy.

Histologic characteristics of fungal mucin:

The production of allergic mucin is pathognomonic of AFRS. Grossly allergic mucin is thick, tenacious and viscous in consistency. Its color may vary from brown to dark green. It is only the mucin rather than sinus mucosa that provides the relevant histological evidence necessary to make the diagnosis of AFRS. Examination of nasal mucosa / polypi shows evidence of chronic inflammation. Eosinophils are also seen in abundance. Pathologic examination of these tissues is done to establish that fungal invasion is not present.

Histology of allergic fungal mucin reveals the characteristic ranching non invasive fungal hyphae within sheets of eosinophils and Charcot – Layden crystals. Classically H&E stains accentuate the

mucin and cellular components of allergic mucin but fail to stain the fungal hyphae. Silver stains are specifically used to stain fungal hyphae. Silver stains color fungi black / dark brown.

Fungal culture: These tests at most provides supportive evidence. Diagnosis of AFRS is not based on positive fungal cultures from mucin.

Treatment: Still evolving. Previously it used to be radial surgery. Now a combination of conservative surgery in combination with adjunct medical therapy is becoming popular.

The goal of any surgical procedure is to eradicate all allergic mucin while providing permanent drainage and ventilation for the affected tissues.

Even in the best of hands the incidence of AFRS recidivism is very high when treated with surgery alone. Adjunctive medical therapy should also be tried to get over this problem. Fungal immunotherapy and immunomodulation is becoming popular these days.

Role of steroids is limited only to postpone the surgery.

34. Atrophic rhinitis

Atrophic rhinitis is defined as a chronic nasal disease characterised by progressive atrophy of the nasal mucosa along with the underlying bones of turbinates. There is also associated presence of viscid secretion which rapidly dries up forming foul smelling crusts. This fetid odor is also known as ozaena. The nasal cavity is also abnormally patent. The patient is fortunately unaware of the stench emitting from the nose as this disorder is associated with merciful anosmia.

Aetiology:

The etiology of this problem still remains obscure. Numerous pathogens have been associated with this condition, the most important of them are

1. Coccobacillus,

2. Bacillus mucosus

3. Coccobacillus foetidus ozaenae

4. Diptheroid bacilli

5. Klebsiella ozaenae.

These organisms despite being isolated from the nose of diseased patients have not categorically been proved as the cause for the same.

Other possible factors which could predispose to this disease are:
1. Chronic sinusitis
2. Excessive surgical destruction of the nasal mucosa and turbiantes
3. Nutritional deficiencies
4. Syphilis.
5. Endocrine imbalances (Disease is known to worsen with pregnancy / menstruation)
6. Heredity (Autosomal dominent pattern of inheritance identified)
7. Autoimmune disease

The triad of atrophic rhinitis as described by Dr. Bernhard Fraenkel are 1. Fetor, 2. crusting and 3. atrophy.

Age of onset: Usually commences at puberty.

Females are commonly affected than males. Heredity is known to be an important factor as there appears to be increased susceptibility among yellow races, latin races and American negro races. Poor nurtrition could also be a factor. Bernat (1965) postulated iron deficiency could be a cause of this disorder.

Recently immunologists have considered atrophic rhinitis to be an autoimmune disorder. Fouad confirmed that there was altered cellular reactivity, loss of tolerance to nasal tissues. This according to him could be caused / precipitated by virus infection, malnutrition, immunodeficiency.

Pathology:

1. Metaplasia of ciliated columnar nasal epithelium into squamous epithelium.
2. There is a decrease in the number and size of compound alveolar glands
3. Dilated capillaries are also seen

Pathologically atrophic rhinitis has been divided into two types:
Type I: is characterised by the presence of endarteritis and periarteritis of the terminal arterioles. This could be caused by chronic infections. These patients benefit from the vasodilator effects of oestrogen therapy.
Type II: is characterised by vasodilatation of the capillaries, these patients may worsen with estrogen therapy. The endothelial cells lining the dilated capillaries have been demonstrated to contain more cytoplasm than those of normal capillaries and they also showed a positive reaction for alkaline phosphatase suggesting the presence of active bone resorption. It has also been demonstrated that a majority of patients with atrophic rhinitis belong to type I category.

Once the diagnosis of atrophic rhinitis is made then the etiology should be sought. Atrophic rhinitis can be divided in to two types clinically:
1. Primary atrophic rhinitis - the classic form which is supposed to arise denovo. This diagnosis is made by a process of exclusion. This type of disease is still common in middle east and India. All the known causes of atrophic rhinitis must be excluded before coming to this diagnosis. Causative organisms in these patients have always be Klebsiella ozenae.

2. Secondary atrophic rhinitis: Is the most common form seen in developed countries. The most common causes for this problem could be:
1. Extensive destruction of nasal mucosa and turbinates during nasal surgery
2. Following irradiation
3. Granulomatous infections like leprosy, syphilis, tuberculosis etc

Clinical features:
The presenting symptoms are commonly nasal obstruction and epistaxis. Anosmia i.e. merciful may be present making the patient unaware of the smell emanating from the nose. These patients may also have pharyngitis sicca. Choking attacks may also be seen due to slippage of detached crusts from the nasopharynx into the oropharynx. These patients also appear to be dejected and depressed psychologically.
Clinical examination of these patients show that their nasal cavities filled with foul smelling greenish, yellow or black crusts, the nasal cavity appear to be enormously roomy. When these crusts are removed bleeding starts to occur.

Why nasal obstruction even in the presence of roomy nasal cavity?
This interesting question must be answered. The nasal cavity is filled with sensory nerve endings close to the nasal valve area. These receptors sense the flow of air through this area thus giving a sense of freeness in the nasal cavity. These nerve endings are destroyed in patients with atrophic rhinitis thus depriving the patient of this sensation. In the absence of these sensation the nose feels blocked.

Radiographic findings:
Are more or less the same in both primary and secondary atrophic rhinitis. Plain xrays show lateral bowing of nasal walls, thin or absent turbinates and hypoplastic maxillary sinuses.

CT scan findings:
1. Mucoperiosteal thickening of paranasal sinuses
2. Loss of definition of osteomeatal complex due to resorption of ethmoidal bulla and uncinate process
3. Hypoplastic maxillary sinuses
4. Enlargement of nasal cavity with erosion of the lateral nasal wall
5. Atrophy of inferior and middle turbinates

Management:
Conservative:
Nasal douching - The patient must be asked to douche the nose atleast twice a day with a solution prepared with:

Sodium bicarbonate - 28.4 g
Sodium diborate - 28.4 g
Sodium chloride - 56.7 g
mixed in 280 ml of luke warm water.

The crusts may be removed by forceps or suction. 25% glucose in glycerin drops can be applied to the nose thus inhibiting the growth of proteolytic organism.
In patients with histological type I atrophic rhinitis oestradiol in arachis oil 10,000 units/ml can be used as nasal drops.

Kemecetine antiozaena solution - is prepared with chloramphenicol 90mg, oestradiol dipropionate 0.64mg, vitamin D2 900 IU and propylene glycol in 1 ml of saline.

Potassium iodide can be prescribed orally to the patient in an attempt to increase the nasal secretion.
Systemic use of placental extracts have been attempted with varying degrees of success.

Surgical management:

1. Submucous injections of paraffin, and operations aimed at displacing the lateral nasal wall medially. This surgical procedure is known as Lautenslauger's operation.
2. Recently teflon strips, and autogenous cartilages have been inserted along the floor and lateral nasal wall after elevation of flaps.
3. Wilson's operation - Submucosal injection of 50% Teflon in glycerin paste.
4. Repeated stellate ganglion blocks have also been employed with some success
5. Young's operation - This surgery aims at closure of one or both nasal cavities by plastic surgery. Young's method is to raise folds of skin inside the nostril and suturing these folds together thus closing the nasal cavities. After a period of 6 to 9 months when these flaps are opened up the mucosa of the nasal cavities have found to be healed. This can be verified by postnasal examination before revision surgery is performed. Modifications of this procedure has been suggested (modified Young's operation) where a 3mm hole is left while closing the flaps in the nasal vestibule. This enables the patient to breathe through the nasal cavities. It is better if these surgical procedures are done in a staged manner, while waiting for one nose to heal before attempting on the other side.

35. Surgical management of Atrophic rhinitis.

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36. Management of Atrophic rhinitis.

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Sodium bicarbonate - 28.4 g
Sodium diborate - 28.4 g
Sodium chloride - 56.7 g
mixed in 280 ml of luke warm water.

The crusts may be removed by forceps or suction. 25% glucose in glycerin drops can be applied to the nose thus inhibiting the growth of proteolytic organism.
In patients with histological type I atrophic rhinitis oestradiol in arachis oil 10,000 units/ml can be used as nasal drops.

Kemecetine antiozaena solution - is prepared with chloramphenicol 90mg, oestradiol dipropionate 0.64mg, vitamin D2 900 IU and propylene glycol in 1 ml of saline.

Potassium iodide can be prescribed orally to the patient in an attempt to increase the nasal secretion.
Systemic use of placental extracts have been attempted with varying degrees of success.

Surgical management:

1. Submucous injections of paraffin, and operations aimed at displacing the lateral nasal wall medially. This surgical procedure is known as Lautenslauger's operation.
2. Recently teflon strips, and autogenous cartilages have been inserted along the floor and lateral nasal wall after elevation of flaps.
3. Wilson's operation - Submucosal injection of 50% Teflon in glycerin paste.
4. Repeated stellate ganglion blocks have also been employed with some success
5. Young's operation - This surgery aims at closure of one or both nasal cavities by plastic surgery. Young's method is to raise folds of skin inside the nostril and suturing these folds together thus closing the nasal cavities. After a period of 6 to 9 months when these flaps are opened up the mucosa of the nasal cavities have found to be healed. This can be verified by postnasal examination before revision surgery is performed. Modifications of this procedure has been suggested (modified Young's operation) where a 3mm hole is left while closing the flaps in the nasal vestibule. This enables the patient to breathe through the nasal cavities. It is better if these surgical procedures are done in a staged manner, while waiting for one nose to heal before attempting on the other side.

37. Allergic rhinitis

Already described

38. Seasonal allergic rhinitis

Already described

39. Myiasis of nose

Myiasis narium; larva in the nose; Peenash (Indian term)

This is a common problem affecting patients in India. The predisposing factors being:

1. Poorly nourished patient with poor hygiene

2. Patient's with atrophic rhinitis

3. Patient's with leprosy in the nose

4. Diabetics with purulent sinus infections

5. Midline granulomatous lesions involving the nose

6. Malignancy involving the nose.

7. Syphilitic disease involving the nose

Etiology: This condition is brought about by the development inside the nasal cavity of larvae hatched from the eggs already laid by certain flies. The commonest of them being Lucilia hominivora or Sacrophaga Georginia. These flies get attracted by the foul-smelling stench emanating from the nasal cavity and lays their eggs inside the nose of the patient. In addition to the stench these patients have a relatively insensitive nose because the sensory nerves are already damaged.

Symptoms:

There is a tickling sensation and sensation of something moving inside the nasal cavity. It is almost always associated with sneezing, foul smelling sero sanguineous discharge. The patients may have secondary infection of the nasal mucosa causing intense head ache. Oedema of eye lids are also common. The patient may go in for septicemia. Worms can be seen coming out of the nasal cavity. If the patient has septicemia then there may be associated fever with chills. The patient may even become toxic and delirious. The mucous membrane of the nasal cavity is destroyed, in addition the cartilages and bone also undergo necrosis.

Treatment:

The use of various germicides inside the nasal cavity must be condemned because of their effects on the nasal mucosa. Liquid paraffin may be used to stifle the worm. Liquid paraffin blocks or chokes the lung of the maggots thereby killing them. The killed worms can easily be removed. Chloroform can also be used to kill the migrating worm making their manual removal easy. With the advent of the nasal endoscope complete removal of the worms is a possibility.

40. Rhinolith.

Synonyms: Nasal calculi; Concretions in the nose.

Rhinoliths are stone like calcareous deposits found inside the nasal cavity. These are of two types:

Exogenous rhinolith and Endogenous rhinolith. These concretions are common in the nasal cavities of females than in men. They may also occur although rarely in the naso pharynx. They are almost always single and unilateral. These masses are more or less irregularly spherical, they may also show prolongations according to their directions of growth.

The surface of a rhinolith is mulberry like, may be grey or brownish pink in color. Rhinoliths are friable, and they crumble readily under pressure. They are chiefly made of phosphates and carbonates of calcium. Sometimes phosphate of magnesia, chloride of sodium and carbonates of magnesia are also seen. These salts originate from the nasal mucous secretions, tears, and inflammatory exudates.

These salts have been found to be deposited around a nucleus which could be inspissated mucous, blood clot or a small foreign body. If concretions occur around a foreign body then the rhinolith is known to be exogenous in nature and if if forms around a blood clot or inspissated mucous plug then it is known to be endogenous in nature. Gauze swabs inadvertently left in the nose can also act as a nidus for the formation of rhinolith.

Symptoms:

These patients have unilateral nasal discharge, which may be serosanguinous in nature. As the rhinolith increases in size, the symptoms of nasal obstruction become more pronounced and the patient may manifest with unilateral purulent nasal discharge. Swelling of the nose, face epiphora could be some of the symptoms.

On probing the presence of a stony hard structure could be identified. It is common in the inferior meatus

Management:

Complete removal of the offending mass is the dictum. If the rhinolith is reasonably small it can be removed per via naturalis without incision. If it is large attempt must be made to break it to manageable pieces to facilitate per via naturalis removal. If attempts to break the mass fail then lateral rhinotomy should be resorted to for complete removal of the mass.

Once successfully removed it does not recur.

41. Choanal atresia

Choanae are otherwise known as posterior nasal apertures. Air which is breathed through the nose finds its way into the lungs through this aperture. In some children the choanae may be congenitally closed. This condition goes by the name choanal atresia. Choanal atresia may be either unilateral or bilateral. Unilateral choanal atresia is invariably not identified early, and it needs constant suspicion for its identification. On the contrary bilateral choanal atresia is a medical emergency, wherein the pateint's breathlessness increases when the child starts to cry. This condition common affects one in 8000 live births. Female children are affected twice as common as male children.

In bilateral choanal atresia the child is in acute respiratory distress which improves when the child starts to cry, since it takes in air through the mouth by passing the obstructed choanal airway.

Neonates are obligate nasal breathers for the first 6 weeks. When bilateral choanal atresia is present in a neonate, emergency

Types of choanal atresia:

1. Bony - 90%

2. Membranous - 10%

This atretic plate of bone / membrane are generally situated just in front of the posterior end of nasal septum. The congenital choanal atresia should not be considered as an isolated plate of bone but as one component of a skull base anomaly developing between the 4th and 12th weeks of gestation.

Embryology:

Nose proper develops from neural crest cells. These cells commence their caudal migration to reach the midface by the 4th week of gestation. Two nasal placodes develop inferiorly, and they are divided into the medial and lateral nasal processes by the presence of nasal pits. The medial nasal process give rise to the nasal septum, philtrum and premaxilla of the nose, whereas the lateral

processes form the sides of the nose. Inferior to the nasal complex, the stomodeum, or future mouth, forms. Nasobuccal membrane separates the oral cavity inferiorly from the nasal cavity superiorly. As the olfactory pits deepen, the choanae are formed. Primitive choanae form initially, but with continued posterior development, the secondary or permanent choanae develop. By 10 weeks, differentiation into muscle, cartilage, and bony elements occurs. Failure of these carefully orchestrated events in early facial embryogenesis may result in multiple potential anomalies, including choanal atresia, medial or lateral nasal clefts, nasal aplasia, and polyrrhinia.

Four theories for the development of choanal atresia:

1. Persistence of a buccopharyngeal membrane from the foregut.

2. Persistence of the nasobuccal membrane of Hochstetter - most commonly accepted theory.

3. The abnormal persistence or location of mesodermal adhesions in the choanal region.

4. A misdirection of mesodermal flow secondary to local genetic factors better explains the popular theory of persistent nasobuccal membrane

Boundaries of the atretic plate:

1. Superior - Under surface of the body of sphenoid

2. Lateral - Medial pterygoid lamina

3. Medial - vomer

4. Inferior - Horizontal plate of palatine bone

Additional anomalies seen are:

1. The palatal arch is accentuated

2. Lateral and posterior nasal walls sweep inwards

3. The naso pharynx is narrowed

Associated other anomalies:

CHARGE association - (C- coloboma; H- congenital heart disease; A- atresia choanae; R- retarded growth and development; G- genital anomalies
in males; E-ear anomalies and deafness). 60% of these patients have bilateral choanal atresia while the rest present with unilateral atresia.

Symptoms:

In patients with bilateral choanal atresia, mouth breathing is seen. The patient is unable to clear the nasal cavity of its secretions. There is also associated loss of sensation of smell. Patient's with unilateral atresia has c/o unilateral nasal block associated with thick tenacious secretions which cannot be cleared fully. These patients commonly have foul smelling breath either due to mouth breathing and its attendant drying effects, or due to the inability to clear the nasal cavity of its secretions. These patients also have associated change in voice due to loss of normal nasal intonation i.e. Rhinolalia clausa. The respiratory obstruction is cyclic - as the child falls asleep the mouth closes and a progressive obstruction starting with stridor
followed by increased respiratory effort and cyanosis. Either the observer opens the child's mouth or the child cries and the obstruction is
cleared. Child with bilateral atresia has difficulty in sucking milk.

Clinical examination:

1. Failure to pass a # 6 to 8 French plastic catheter through the nares into the pharynx. (a typical solid feeling will be encountered at
the level of the posterior choana approx. 3-3.5 cm from the alar rim). If obstruction is encountered within 1 - 2 cms from the nasal rim it is probably due to traumatic deflection of nasal septum during delivery. If obstruction is due to mucosal oedema it can be shrinked using nasal decongestants like oxymetazoline / xylometazoline.

2. Wisps of cotton may be placed in front of the nasal cavity and the movement of air flow can be ascertained.

3. Placing methylene blue in the nares and not visualizing it within the pharynx.
Investigations:

The current investigation of choice is CT and gives information whether the obstruction is membranous or bony and the actual structures involved and its thickness. It demonstrates thickening of the vomer, bowing of lateral wall of the nasal cavity and fusion of bony elements in choanal region. Congenital unilateral atresia is always associated with deviation of nasal septum and thickening of the vomer bone.

Management:

In bilateral atresia securing the airway takes the first place. An oral airway may be introduced to tide over the immediate crisis.

Intraoral nipple - a large nipple can be modified by having its end cut off and then ties are attached to the nipple and placed around the occiput. This type of airway is called a McGovern nipple and provides an airway through which the baby can breathe. A very small feeding tube can then we passed either through another hole in the nipple or alongside the nipple for gavage feeding. This is the preferred method of establishing an oral airway.

Role of tracheostomy:

This is controversial. This is one way that must be considered if the patient is unable to maintain the oral airway.

Surgical management:

Transnasal approach: (using endoscopes): The surgery is performed under general anesthesia. A self retaining nasal speculum is used to expose the nasal cavity and the atretic plate. If the atresia is membranous in nature a simple perforation of the same under endoscopic guidance would suffice. The nasal cavity is decongested using 4% xylocaine with adrenaline in the concentration of 1 in 10,000 concentration. Under endoscopic guidance a mucosal incision is made and the mucosal flaps are elevated exposing the posterior vomer and lateral pterygoid lamina. A diamond burr on an angled hand piece is used to drill the atretic bony plate. It is perforated at the junction of the hard palate and the vomer. Incidentally this is the thinnest part of the atretic plate. This procedure was first described by Stankiewicz. To improve visualization the inferior turbinate can be out fractured or even be trimmed. After drilling care is taken to preserve the mucosal flaps. A silastic stent is placed into each nostril passing through the drilled neo choana. This helps in reducing the incidence of restenosis. Stent is kept in place for at least 6 weeks.

Caution:

While performing this procedure caution must be taken not to injure the sphenopalatine vessels behind the middle turbinate.

Advantages of this procedure:

1. This process is faster and easier

2. Blood loss is minimal

3. Can be performed in children of all ages who do not have associated external nasal deformities

4. Child can be immediately breast fed

5. Child can be discharged on the 3rd day itself

Disadvantages:

1. Vision is highly limited especially in the new born

2.Inability to adequately remove enough of the posterior vomerine septal bone and prevent restenosis

3. Longer stenting time

4. Endoscopes do not offer binocular vision

5. Cannot be done safely and with good results on patients with multiple nasal and nasopharyngeal anomalies.

Transpalatal approach:

This procedure is performed under general anesthesia. A Dingman-Denhardt mouth gag with the infant tongue blade is used. The palate is injected with 0.5% lidocaine with 1:200,000 epinephrine in the area of the mucosal incision. a Owens type(U-shaped) mucosal incision is made beginning just behind the maxillary tuberosity on one side and then continued medial to the alveolar ridge up to the canine region and then angled back to the nasopalatine foramen. A likewise incision is made on the opposite side and the mucosal flap is elevated taking care not to damage the greater palatine arteries. Mucosa of the nose and nasopharynx is elevated and preserved. Then the palatine bones posterior to the greater palatine foramina, the atresia plates and the posterior vomer are carefully drilled away using a diamond burr. Two 14 or 16 French catheters are passed simultaneously into each nostril to check the patency of the newly created choanae.

The preserved mucosa is then used to cover the superior and inferior surfaces of the newly formed choanae and then sutured in place to
cover the bone. Stents are left in place for 4 weeks.

Advantages:

1. Better visualization and exposure

2. Both hands are free

3. Less stenting period (a portex endotracheal tube can be cut and used as a stent)

4. Less failure rate

Disadvantages:

1. The incisions, which are identical to those for a cleft palate repair, may have a banding effect on maxillary growth due to scar formation. (Therefore, most surgeons prefer to wait to use this approach until some teeth are in occlusion - at approx.12-18 months).

2. Palatal growth can be stunted in 50 % of individuals

3. Increased blood loss

4. Increased risk of development of palatal fistulas post operatively

Care of the post op patient:

1. The parents must be taught to maintain the stents with frequent suction and a saline-moistened pipe cleaner or cotton applicator 3 to 6 times per day.

2. Antibiotics and decongestants are prescribed if there is evidence of rhinitis

3. Patients must be followed up regularly till the stents are removed.

42. CSF rhinorrhea

CSF discharging from the nose is known as CSF rhinorrhea. Cerebrospinal fluid is a clear colorless fluid that bathes the brain and spinal cord, cushioning them against trauma. In fact in literal terms the brain and spinal cord floats in the cerebrospinal fluid. The specific gravity of brain is only 4% of that of CSF, hence it could float easily in the CSF.

CSF Leaks always occur when the barrier that retains the Cerebrospinal fluid is breached. These barriers are skin, galeal periosteum, skull, dura and arachnoid. The mucosal lining of the nasal cavity also forms its last line of defense.

Causes of CSF rhinorrhea:

Traumatic:

a. Accidental: Acute / Delayed

b. Iatrogenic: Acute / Delayed

Non traumatic:

a. High pressure

i. Tumors: Direct / Indirect

ii. Hydrocephalus

b. Normal pressure:

i. congenital anomalies

ii. Focal atrophy - Olfactory / Sellar

iii. Osteomyelitic erosion

iv. Idiopathic

Types of CSF rhinorrhea:

I. Traumatic

II. Non traumatic (spontaneous) - a. High pressure leaks (always associated with concomitant hydrocephalus)

The high-pressure leaks are commonly encountered in the cribriform area. This is due to the fragility and unique anatomy in this area i.e. (prolongation of the subarachnoid space along the olfactory filaments). The leak during these conditions functions as a safety valve alleviating the increased intracranial pressure. These high-pressure leaks are associated with slow growing tumors and 1/4 of them have hydrocephalus. Pituitary neoplasms are the most common type of intracranial tumor found, next common are the posterior cranial fossa lesions. Direct invasion of the skull base is not the usual mechanism of this leak. Closure of these leaks may worsen the condition of the patient if the causative lesion is left untreated.

b. Normal pressure leaks - These leaks are associated with congenital dehiscence or thin bone along the skull base. Commonly this type of leaks occur in the ethmoidal sinus adjacent to the cribriform plate. Potential leak pathways include the prolongation of the subarachnoid space along the olfactory nerves and stalk of the hypophysis. Minor degrees of maldevelopment of the cribriform plate or the diaphragma sella may allow further extension of the subarachnoid space through the foramina of the cribriform plate or around the hypophysis (empty sella). The former is common and accounts for the majority of normal pressure leaks.

Cerebrospinal leaks need to be treated because of the impending threat of meningeal infections.
Spontaneous CSF rhinorrhea:

True spontaneous leaks are really rare. There is almost always some antecedent traumatic event.

Nuss postulated the various causes of spontaneous CSF rhinorrhea. He named them as "4 P's".

1. Increased intracranial pressure

2. Brain pulsations which continuously occur along the skull base

3. Degree of pneumatization of the paranasal sinuses

4. Arachnoid pits / villi exist normally along the skull base. Continued transmission of pulsation, erodes the bone until the arachnoid communicates with a pneumatised space with the potential to develop fistula.

Trauma: is the commonest etiology

Iatrogenic: Surgery involving skull base and paranasal sinuses may cause CSF rhinorrhea due to breach in the skull base.

Congenital: Meningocele and Meningoencephalocele

Neoplasia

Bedside tests for detecting CSF rhinorrhea:

Reservoir sign: This test is ideally performed immediately on rising from the bed. The patient is asked to place the chin over their chest. The patient must stay in that position for one full minute. Clear fluid dripping from the nose is CSF.

Handkerchief test: Discharge from the nose is blown into a handkerchief and is allowed to dry. If the discharge is CSF the handkerchief will not stiffen, if the discharge is secretions from the nose the handkerchief stiffens due to the presence of mucin in the nasal secretions.

The most sensitive laboratory test is to look for Beta 2 transferrin in the nasal secretions. In CSF Beta 2 transferrin is present, and it is absent in normal nasal secretions.

The most sensitive test to detect CSF leak is intrathecal radionucleotide test.

Tests that help to localize the CSF leak:

1. Flow sensitive MRI is useful. The leak must be active at the time of the scan for visualising the site. The advantage of this test is that it is non invasive.

2. Intra thecal administration of non ionic contrast with high resolution CT scan. Intra thecal administration of low quantities of flurescein can also be used. If the leak is present it can be viewed in the nasal cavity with a 490 nm light generated by a special optical filter. Dye injection is done using Barbolage technique in which 1 - 2 drops of 5 % Fluoresate is diluted with the patients own CSF, and then injected partially, then CSF is withdrawn further diluting the dye and then reinjecting the dye.

Management:

1. Bed rest

2. Elevation of the head end of the bed

3. Stool softeners

4. Short course of azetazolamide

5. Continuous / daily intermittent lumbar spinal drainage helps to reduce the fistula

6. Antibiotic prophylaxis to prevent meningitis.

Surgical management:

CSF leaks into the nose can be approached by two routes:

1. Intracranial repair

2. Extracranial repair

Intracranial repair: till recently this was the commonest approach adopted to repair CSF rhinorrhea. Leaks from the anterior defects can be repaired by frontal anterior fossa craniotomy. A middle cranial fossa craniotomy or posterior fossa craniotomy can be used to manage leaks from these areas. Leaks from the sphenoid sinus area are difficult to approach via the intracranial route. The repair techniques involve use of a pedicled periosteal or dural flaps, muscle plugs can be used to plug the defects, mobilized portions of falx cerebri or other facial grafts can be utilized. Fibrin glue can be used to stabilize the grafts used in case of large leaks.

The advantages of this approach are

1. The adjacent brain tissue can be directly inspected

2. Direct visualization of the dural defect

3. The repair can also be done even under conditions of increased intracranial tension

4. Even if efforts to localize the leak fails blind repair is possible in this approach. The areas covered with grafts must include the cribriform plate and the sphenoid sinus.

Disadvantages of this approach:

1. Increased morbidity

2. Increased risk of permanent anosmia

3. Trauma related to brain retraction (hematoma, oedema, seizures, cognitive dysfunction etc)

4. Longer hospital stay

Extracranial repair: This can be divided into external approaches and endoscopic techniques

External approach:

This include anterior osteoplastic approach via Bicoronal or eyebrow incision, external ethmoidectomy, transethmoidal sphenoidectomy, and transeptal sphenoidectomy. Graft materials used could range from fascia lata, temproalis fascia, septal cartilage, turbinate mucosa, muscle and fat. For cribriform plate, or fovea leaks a transnasal ethmoidectomy is performed. For sphenoid leaks sphenoidectomy is performed.

Disadvantages of this procedure include:

1. Inability to repair associated intracranial abnormalities

2. Ineffective in repairing high pressure leaks

3. Ineffective in repairing frontal and sphenoid sinus leaks when they have prominent lateral extensions

Endoscopic techniques:

This method has several advantages including better visualization and magnification. Other advantages include the ability to clean the mucosa adjacent to the leak, and in accurate positioning of the graft to plug the leak. There is no threat of anosmia, and this procedure has low morbidity.

43. Antral wash

Anatomy of inferior meatus:
Inferior meatus is the largest of the three meatuses of the nasal cavity. This is actually the space between the inferior turbinate and the lateral nasal wall. It extends almost the entire length of the lateral wall of the nose. It is broader in front than behind which makes it easy for accessing the lateral nasal wall from here. Anteriorly the nasolacrimal duct opens here.
Inferior turbinate is a separate bone unlike the superior and middle turbinates which are components of ethmoid bone. Inferior concha / inferior turbinate matures via endochondral ossification.
Articulations of inferior turbinate:
Anterior – Frontal process of maxilla
Anteromedial – Articulates with the uncinate process of ethmoid bone and lacrimal bone
Posteromedial – Perpendicular plate of palatine bone
Indications for antral lavage:
1. Acute bacterial maxillary sinusitis causing pressure symptoms in middle of face
2. Feeling of numbness of teeth / symptoms that does not resolve with medical management
3. Patients with maxillary sinusitis who are not fit for general anesthesia to perform functional endoscopic sinus surgery
4. Patients on assisted mechanical ventilation who commonly develop sinusitis (nearly 40% of them develop). Lavage in these patients can be performed as a bedside procedure under local anesthesia to clear the pent-up secretions from the maxillary sinuses.
5. In patients with permanent disability of muco ciliary clearance mechanism like kartagener's syndrome and Young's syndrome. In these patients FESS is almost useless and only inferior meatal antrostomy could salvage them.

Contraindications:
1. In young children in whom maxillary sinus is not fully developed. Maxillary sinus completes its development only after the age of 9.
2. Blow out fracture of orbit / history of blow out fracture of orbit because irrigated fluid from the sinus could infuse into the orbit via the fracture line causing orbital problems
3. Patients who have undergone previous surgeries involving the lateral nasal wall as the needle could enter through the posterior wall of maxillary sinus into the pterygopalatine fossa

4. In patients with atrophic rhinitis because the lateral nasal wall will be pretty thick in these patients making the procedure rather difficult. It may require a chisel and gouge to create inferior meatal opening in these patients. Simple trocar and cannula would not do.

Procedure:
This procedure involves introduction of a canula into the maxillary sinus cavity via an opening made in the inferior meatus. This procedure is rather outdated these days because the maxillary sinus drainage in the presence of normal muco ciliary clearance mechanism is not dependent on gravity. The beating cilia always propels the secretions from the sinus cavity towards the natural ostium which is situated slightly above. There is no point in expecting gravity to work against the natural muco ciliary clearance mechanism.
This surgery is performed under local anesthesia. Topical anesthesia is produced by using 4% xylocaine soaked nasal pledgets. Topical anesthesia lasts about 45 minutes which is more than sufficient for completion of the procedure. While using 4% xylocaine topical anesthesia it should be ensured that the maximum volume of drug used should not exceed 7ml. A reasonable dose of xylocaine that is safe for topical use is 4mg/kg body weight. By mixing xylocaine with adrenaline, the effect of the drug can be prolonged plus the added benefit of vasoconstriction which reduces bleeding. Ideal is to mix I ampule of adrenaline to one 30 ml bottle of 4% xylocaine. This will ensure that adrenaline concentration is about 1 in 10000 units. Cottonoids if available are preferred to pledgets.
Each nasal cavity should be packed with 3 packs soaked with 4% xylocaine with 1in 10000 units adrenaline. Before packing the pack should be squeezed to remove excess xylocaine.
The first pack is placed over the floor of the nasal cavity, the second one is placed in the inferior meatus. The third pack is placed in the middle meatus area. Surgeon should be aware that the posterior pharyngeal wall mucosa would also be anesthetized by xylocaine trickling into that area. This could cause the patient to aspirate because the sensation is lost. The surgeon should be conscious about this problem while performing the procedure.
The patient should be instructed not to sniff while nasal packing is done as it would promote drug to trickle into the posterior pharyngeal wall.
A short description of innervation of nose and nasal cavity would not be out of place. Nasal innervation can be simplified by dividing it into internal (mucosal) innervation and external (innervation involving the skin of the nose).

Innervation of external nose:
The external nose is innervated by the ophthalmic division of 5th cranial nerve, and maxillary division of 5th cranial nerve. The superior aspect of the nose including the tip is supplied by Infratrochlear nerve. The supratrochlear nerve and external nasal branch of anterior ethmoidal nerves also supply this area. The infraorbital nerve supplies the inferior and lateral aspects of the nose extending up to the lower eyelids.

The patient is comfortably seated in a chair with adequate back support. Eye pad should be used to blind the patient. This will reduce the anxiety level of the patient.

The Tilley Lichwitz trocar and canula is passed under the attachment of inferior turbinate and is directed towards the outer canthus of the ipsilateral eye. With a firm turn the inferior meatus is punctured. While introducing index finger of the surgeon should be placed at the junction of anterior 1/3 and posterior 2/3 of the trocar canula assembly. This will help in ensuring the safe penetration depth. The trocar is gently removed leaving the canula in position. A syringe is connected to the cannula and aspiration is attempted. If it is inside the maxillary sinus secretions could be aspirated. If the sinus is empty then air will be aspirated. If gross blood is aspirated then it should be construed that the canula is not inside the maxillary sinus cavity. A Higginson's syringe which contains a bulb and a one-way valve is connected to the canula and the other end of the syringe is placed inside a vessel containing water at body temperature. Flushing can be performed by squeezing the bulb of Higginson syringe. Dilute potassium permanganate wash can also given. Three successive washes should be given. A kidney tray should be held under the patient's mouth. The patient can be asked to hold the tray so that their mind will be diverted from the actual procedure. When the antrum is being flushed the patient should be asked to keep the mouth open so that fluid used for irrigation will drain through the patient's mouth.

Complications:
1. Bleeding
2. Orbital damage. Perforation of orbital floor will cause proptosis and pain
3. Cheek swelling: This is caused by breaching the soft tissue of the cheek and the anterior wall of the sinus.
4. Air embolism due to injury to veins
5. Infection of maxillary sinus
6. Vaso vagal shock

44. Lefort's Fracture

Le Fort I fracture:

Fracture runs above and parallel to the palate. It crosses the lower part of nasal septum, maxillary antra and the pterygoid plates.

Le Fort II (Pyramidal fracture):

Fracture passes through the root of the nose, lacrimal bone, floor of orbit, upper part of maxillary sinus, and pterygoid plates. This fracture has some features common with zygomatic process.

Le Fort III (Craniofacial dysfunction):

In this type there is complete separation of facial bones from the cranial bones. The fracture line passes through the root of the nose, ethmofrontal junction, superior orbital fissure, lateral wall of orbit, frontozygomatic and Temporo zygomatic sutures and the upper part of pterygoid plates.

Clinical features:

1. Malocclusion of teeth with anterior open bite

2. Midface elongation

3. Mobility in the maxilla

4. CSF rhinorrhea. Cribriform plate is injured in Le fort II and III fractures.

CT imaging is diagnostic.

45. Nasal bone fracture

Introduction: Nose is the most prominent part of the face; hence it is likely to be the most common structure to be injured in the face. Although fractures involving the nasal bones are very common, it is often ignored by the patient.
Patients with fractures of nasal bone will have deformity, tenderness, hemorrhage, edema, ecchymosis, instability, and crepitation. These features may be present in varying combinations.

Pathophysiology:

1. Nasal bones and underlying cartilage are susceptible for fracture because of their more prominent and central position in the face.
2. These structures are also pretty brittle and poorly withstands force of impact.
3. The ease with which the nose is broken may help protect the integrity of the neck, eyes, and brain. Thus, it acts as a protective mechanism.
4. Nasal fractures occur in one of two main patterns- from a lateral impact or from a head-on impact. In lateral trauma, the nose is displaced away from the midline on the side of the injury, in head-on trauma, the nasal bones are pushed up and splayed so that the upper nose (bridge) appears broad, but the height of the nose is collapsed (saddle-nose deformity). In both cases, the septum is often fractured and displaced.
5. The nasal bone is composed of two parts: A thick superior portion and a thin inferior portion. The intercanthal line demarcates these two portions. Fractures commonly occur below this line.

Types of nasal bone fractures: Fractures involving nasal bones are divided into three categories depending on the degree of damage, and its management.

Class I fractures: Very little force is sufficient to cause a fracture of nasal bone. It has been estimated to be as little as 25-75 pounds / sq inch. Class I fractures are mostly depressed fractures of nasal bones. The fracture line runs parallel to the dorsum of the nose and naso maxillary suture and joins at a point where the nasal bone becomes thicker. This point is about 2/3 of the way along its length. The fractured segment usually regains its position because of its attachment along its lower border to the upper lateral cartilage. The nasal septum is not involved in this particular injury. Class I fractures do not cause gross lateral displacement of nasal bones, though a persistent depressed fragment may give it the appearance. In children these fractures could be of green stick variety and a significant nasal deformity may develop subsequently during puberty when nasal growth accelerates.
Clinically this fracture will present as a depression over the nasal bone area. There may be tenderness and crepitus over the affected nasal bone. Radiological evidence may or may not be present. In fact class I fracture of nasal bone is purely a clinical diagnosis.

Class II fractures: These fractures cause a significant amount of cosmetic deformity. In this group not only the nasal bones are fractured, the underlying frontonasal process of the maxilla is also fractured. The fracture line also involves the nasal septum. This condition must be recognised clinically because for a successful result both the nasal bones as well as the septum will have to be reduced. Since both the nasal bones and the fronto nasal process of maxilla would have absorbed a considerable amount of force, the ethmoidal labyrinth and the adjacent orbit should be intact.
The precise nature of the deformity depends on the direction of the blow sustained. A frontal impact may cause comminuted fracture of nasal bones causing gross flattening and widening of the dorsum of the nose. A lateral blow of similar magnitude is likely to produce a high deviation of the nasal skeleton. The perpendicular plate of ethmoid is invariably involved in these fractures, and is characteristically C shaped (Jarjaway fracture of nasal septum).

Class III fractures: Are the most severe nasal injuries encountered. This is caused by high velocity trauma. It is also known as naso orbital fracture / naso ethmoidal fracture. Recent term to describe this class (Naso orbito ethmoid fracture) indicates the clinical importance of orbital component in these injuries. These fractures are always associated with Le Fort fracture of the upper face involving the maxilla also. In these fractures the nasal bone along with the buttressing fronto nasal process of maxilla fractures, telescoping into the ethmoidal labyrinth. Two types of naso ethmoidal fractures have been recognized:

Type I: In this group the anterior skull base, posterior wall of the frontal sinus and optic canal remain intact. The perpendicular plate of ethmoid is rotated and the quadrilateral cartilage is rotated backwards causing a pig snout deformity of the nose. The nose appears foreshortened with anterior facing nostrils. The space between the eyes increase (Telecanthus), the medial canthal ligament may be disrupted from the lacrimal crest.

Type II: Here the posterior wall of the frontal sinus is disrupted with multiple fractures involving the roof of ethmoid and orbit. Sphenoid and parasellar regions may sometimes be involved. Since the dura is adherent to the roof of ethmoid fractures in this region causes tear in the dura causing csf rhinorrhea. Pneumocranium and cerebral herniation may complicate this type of injury.

Management:

If fractures of nasal bones are left uncorrected it could lead to loss of structural integrity and the soft tissue changes that follow may lead to both unfavorable appearance and function. The management of nasal fractures is based solely on the clinical assessment of function and appearance; therefore, a thorough physical examination of a decongested nose is paramount. Patients with fractures involving nose will have intense bleeding from nose making assessment a little difficult. Bleeding must first be controlled by nasal packing. These patients also have considerable amount of swelling involving the dorsum of the nose, making assessment difficult. These patients must be conservatively managed for at least 3 weeks for the oedema to subside to enable precise assessment of bony injury. According to Cummins Fracture reduction should be accomplished when accurate evaluation and manipulation of the mobile nasal bones can be performed; this is usually within 5-10 days in adults and 3-7 days in children.

Radiological investigations:

1. Plain Xray nasal bones
2. Xray paranasal sinuses water's view
3. CT scan paranasal sinuses - This is a must in all cases of class II and class III fractures of nasal bones for precise estimation of damage.

Most class I fractures can be managed by closed reduction and immobilization by application of POP. Digital pressure alone commonly does the job.

If the fractured fragments are impacted then a Welsham's forceps will have to be used to disimpact and reduce the fractured nasal bone.

In the event of using Welsham's forceps to disimpact the nasal bone, there will be extensive trauma to the nasal mucosa causing epistaxis. The nasal cavity of these patients must be packed with roller gauze, with application of an external splint to stabilize the bone.

Class II septal fractures:

Closed reduction in these cases do not give optimal results because the septal fracture is not corrected. Since the fractured fragments of the perpendicular plate of ethmoid and the septal cartilage fragments are not repositioned the results of closed reduction are not satisfactory. In

these patients closed manipulation of nasal bones should always be accompanied by open correction of septal deformity.

Class III fractures: Must be treated with open reduction and internal fixation. The problem here is even though the nasal bones can be reduced the adjacent supporting bones (components of the ethmoidal labyrinth) do not support the nasal bones because of their brittleness. It is always better to reconstruct and stabilize the anterior table of the frontal bone so that other parts of nasal skeleton can derive support from it. Formerly transnasal wires were used to fix the nasal bones, but with the advent of plates and screws the whole scenario has undergone a dramatic change.

Ellis procedure of management of Class III fractures:

Aims of the procedure include:

1. Provision of adequate surgical exposure to provide an unobstructed view of all components of the fracture.

2. The medial canthal ligament should be identified. This is rarely avulsed and is usually attached to a large fragment of bone. Once identified the ligament should be reattached and secured to the lacrimal crest. This step will avoid the future development of telecanthus.

3. Reduction and reconstruction of medial orbital rim. This can be achieved by use of transnasal 26-gauge wires. If plates are used they should be very thin otherwise they will become conspicuous once the wound has healed.

4. Reconstruction of medial orbital wall and floor with bone grafts

5. Realignment of nasal septum

6. Augmentation of dorsum of the nose by the use of bone grafts

7. Accurate soft tissue readaptation should be encouraged by placing splints.

Complications of nasal bone fracture:

1. Cosmetic deformity (saddle nose, pig snout deformity)
2. Persistent septal deviation
3. CSF leak
4. Orbital oedema / complications
5. Nasal block / compromise of nasal functions

46. Classification of carcinoma maxilla

Lederman's classification:

Two horizontal lines of sebileau pass through floors of orbits and maxillary sinus thereby producing:

Supra structure:

Ethmoid, sphenoid and frontal sinuses and olfactory area of nose

Mesostructure:

Maxillary sinus, respiratory part of nose

Infrastructure:

Alveolar process.

Growth involving infrastructure and Mesostructure carried better prognosis than those involving suprastructure.

Ohngren's classification:

An imaginary line is drawn extending between medial canthus of the eye and the angle of the mandible. Growth above this line have a poorer prognosis than those situated below this line.

Histological classification:

Well differentiated squamous cell carcinoma

Moderately differentiated squamous cell carcinoma

Poorly differentiated carcinoma

1. Tubal tonsil

The tubal tonsil also known as Gerlach tonsil is one of the four main tonsil groups comprising Waldeyer's tonsillar ring, which also includes the palatine tonsils, the lingual tonsils, and the pharyngeal tonsils.

The tubal tonsil is very close to the torus tubarius, which is why this tonsil is sometimes also called the tonsil of (the) torus tubarius. Equating the torus with its tonsil however might be seen as incorrect or imprecise.

It is located posterior to the opening of the Eustachian tube on the lateral wall of the nasopharynx.

Inflammation of tubal tonsil can cause eustachean block leading on to the development of secretory otitis media. Ear symptoms that follows adenoiditis is actually due to involvement of tubal tonsil by the infection / inflammation of adenoid tissue.

2. Lingual tonsil

The lingual tonsils are a collection of lymphatic tissue located in the lamina propria of the root of the tongue. This lymphatic tissue consists of the lymphatic nodules rich in cells of the immune system (immunocytes). The immunocytes initiate the immune response when the lingual tonsils get in contact with invading microorganisms (pathogenic bacteria, viruses or parasites).

Lingual tonsils are covered externally by stratified squamous nonkeratinized epithelium that invaginates inward forming crypts. Beneath the epithelium is a layer of lymphoid nodules containing lymphocytes. Mucous glands located at the root of tongue are drained through several ducts into the crypt of lingual tonsils. Secretions of these mucous glands keep the crypt clean and free of any debris.

Blood supply:

Lingual artery, branch of external carotid artery
Tonsillar branch of facial artery
Ascending and descending palatine arteries
Ascending pharyngeal branch of external carotid artery

Like other lymphatic tissues, the function of lingual tonsils is to prevent infections. These tonsils contain B and T lymphocytes which get activated when harmful bacteria and viruses come in contact with tonsils. B lymphocytes kill pathogens by producing antibodies against them, while T

lymphocytes directly kill them by engulfing them or indirectly by stimulating other cells of the immune system.

3. Killian’s dehiscence

Killian's dehiscence: Is the junction between thyropharyngeus and cricopharyngeus muscles. This is a potentially weak area not supported by other constrictor muscles. The cricopharyngeus muscle is thicker and bulkier than the thyropharyneal component of inferior constrictor. The pressure generated by the constriction of cricopharyngeus muscle is sufficient to cause prolapse of mucosal lining through this potentially weak area.

Pharyngeal pouch develops from this area of weakness.

4. Waldeyer’s Ring

Waldeyer's tonsillar ring (pharyngeal lymphoid ring, Waldeyer's lymphatic ring, or tonsillar ring) is a ringed arrangement of lymphoid organs in the pharynx. Waldeyer's ring surrounds the naso- and oropharynx, with some of its tonsillar tissue located above and some below the soft palate (and to the back of the mouth cavity).

The ring consists of the (from top to bottom):

1 pharyngeal tonsil (or "adenoid"), located on the roof of the nasopharynx, under the sphenoid bone.
2 tubal tonsils on each side, where each auditory tube opens into the nasopharynx
2 palatine tonsils (commonly called "the tonsils") located in the oropharynx
lingual tonsils, a collection of lymphatic tissue located on the back part of the tongue

Inflammation of components of waldayer's ring cause:

Tonsillitis

Adenoiditis

Pharyngitis

5. Parapharyngeal space

Lateral pharyngeal space: (Para pharyngeal space)

This space is situated lateral to the fascia covering the constrictor muscles of the pharynx (buccopharyngeal fascia). Lateral to this space lie the pterygoid muscle, mandible and carotid sheath.

Superiorly it extends up to the skull base while inferiorly it ends at the level of hyoid bone because of the attachment of the submandibular gland sheath to the sheaths of the stylohyoid muscle and the posterior belly of digastric muscle.

The carotid sheath lies close to the posterolateral wall of this space.

Postero medially this space communicates with the retropharyngeal space.

Anteriorly and inferiorly this space communicates with the spaces associated with the floor of the mouth.

This space is most commonly involved in neck space infections. Infections from this space can easily spread to the carotid and retropharyngeal spaces.

Common routes of infections of parapharyngeal space:

1. Lingual infections
2. Submandibular gland infections
3. Infections involving the parotid space
4. Spread from peritonsillar abscess

6. Pyriform fossa

Is a potential space that lie on either side of the larynx. They are two in number. It is shaped like a pyramid with the base pointing above and the apex below. They belong to the hypopharyngeal area of the pharynx. It has two parts; the shallow upper part and a deeper lower part.

Boundaries: The pyriform fossa is bounded laterally by the mucosa covering the lamina of the thyroid cartilage. Medially it is bounded by the aryepiglottic fold and arytenoid cartilages above and the cricoid cartilage below. Superiorly it is bounded by the lateral glosso epilglottic fold (Pharyngoepiglottic fold), inferiorly it continues with the oesophagus.

Deep to the mucous membrane of the lateral wall of the pyriform fossa lies the internal laryngeal branch of the superior laryngeal nerve. It supplies sensori fibres to this area.

Clinical importance of pyriform fossa:

1. Anatomically it is a hidden area. Any malignancy in this area will initially cause fewer symptoms and has a tendency to present very late.

2. This area is richly endowed with lymphatics. They drain into the upper deep cervical group of lymph nodes. Any malignancy in this area has a tendency for nodal metastasis.

3. Foreign bodies in the throat commonly gets lodged here.

4. Since superior laryngeal nerve lies superficially in this area, it can be topically blocked by placing cotton pledgets soaked in 4% xylocaine in this area. This is known as the pyriform fossa block.
Examination of the pyriform fossa:

The superficial shallow portion of the pyriform fossa is easily visible in a laryngeal mirror. This portion will be visible in the indirect laryngoscopy examination. The deeper portion of the pyriform fossa is hidden and is not visible to the IDL mirror. Only a direct examination using a upper oesophageal speculum will reveal this portion.

Tumors involving the pyriform fossa commonly arise from its deep portion. This may escape detection during a IDL scopy examination. But if one looks for pooling of saliva in the involved pyriform fossa the underlying growth can be suspected. Hence pooling of saliva is an important clinical sign indicating a under lying tumor in the deep portion of the pyriform fossa, or the presence of a foreign body can also be suspected by this sign.

Causes for pooling of saliva in the pyriform fossa:

Pooling of saliva in the pyriform fossa is not only caused by growth affecting this area causing obstruction to saliva being swallowed, but also due to intense cricopharyngeal muscle spasm.

1. Malignant growth involving the deep portion of the pyriform fossa

2. Foreign body being lodged in the pyriform fossa.

3. Growth involving the crico pharynx or upper oesophagus can also cause pooling of saliva.

7. Functions of Larynx

Functions of larynx:

1. Airway protection

2. Respiration

3. Swallowing

4. Phonation

Airway protection: is the most important function of human larynx. Larynx in fact acts as a sphincter protecting the lower airway from secretions of the oropharynx. It also protects the airway from spillage of food during deglutition. Larynx acts as a three-tier protective mechanism. These are from above downwards: Aryepiglottic fold, ventricular band and vocal cords. These three structures when contracted can effectively seal the lower airway from the contents of the oropharynx.

Contraction occurs from below upwards, first the vocal cord adducts, followed by ventricular bands. Finally, the aryepiglottic fold adducts sealing the lower airway completely.

Respiration: Another important function of larynx is to keep the airway open during respiration. This is done by gentle abduction of the vocal cords while the ventricular bands and aryepiglottic folds are fully abducted.

Swallowing: During swallowing the sphincters of larynx stay contracted preventing aspiration of food into the air passage. During the pharyngeal stage of swallowing the larynx is elevated towards the lower jaw, this elevation opens up the cricopharyngeal sphincter thus facilitating swallowing. The hyoid bone rotates in such a way that the greater cornua becomes horizontal, producing a backward tilting of the epiglottis towards the posterior pharyngeal wall. This movement of hyoid bone effectively closes the laryngeal inlet.

Phonation: The larynx acts as a transducer during phonation converting the aerodynamic forces generated by the lungs, diaphragm, chest and abdominal muscles into acoustic energy. This energy transduction precisely at the space between the two vocal folds. However subglottic and supra glottic pressures also play a role in this transformation of aerodynamic energy into sound energy.

The requirements of normal phonation are as follows:

1. Active respiratory support

2. Adequate glottic closure

3. Normal mucosal covering of the vocal cord

4. Adequate control of vocal fold length and tension.

The vibrations of the vocal folds are complex in nature and are known as the glottic cycle. This cycle involves glottic opening and closing at set frequencies determined by the subglottic air pressure. Normal vocal folds produce three typical vibratory patterns:

1. Falsetto

2. Modal voice

3. Glottal fry

In falsetto or (light voice) the glottic closure is not complete, and only the upper edge of the vocal fold vibrates.

In Modal voice complete glottic closure occurs. This occurs in a majority of mid frequency range voice. During this modal voice production, the vocal fold mucosa vibrates independently from the underlying vocalis muscle. This is the basic frequency at which a person phonates. The modal frequency in adult males is 120 Hz while in adult females it is 200 Hz.

Glottal fry is also known as low frequency phonation is characterized by closed phase. This closed phase is long when compared to the open phase. The vocal cord mucosa and vocalis muscle vibrate in unison.

During phonation two vibratory phases occur i.e. open and closed phases. The open phase denotes the phase during which the glottis is at least partially open, while the closed phase denotes the phase when the vocal folds completely occlude the glottic chink.

The open phase can be further divided into opening and closing phases. The opening phase is defined as the phase during which the vocal folds move away from one another, while during the closing phase the vocal folds move together in unison.

One important physiologic parameter which must be noted during phonation is the mucosal wave. The mucosal wave is an undulation which occur over the vocal fold mucosa. This wave travels in an infero superior direction. The speed of mucosal wave ranges from 0.5 - 1 m/sec. The symmetry of these mucosal waves must also be taken into consideration while studying the physiology of voice production. Any mild asymmetry between the two vocal folds must be considered as pathological.

The function of vocal folds is to produce sound varying in intensity and pitch. This sound is then modified by various resonating chambers present above and below the larynx and are converted into words by the articulating action of the pharynx, tongue, palate, teeth and lips.

The consonants of speech can be associated with particular anatomical sites responsible for their generation i.e. 'p' and 'b' are labials, 't' and 'd' are dentals and 'm' and 'n' are nasals.

8. Physiology of swallowing

The process of swallowing is known as deglutition. The act of swallowing can be divided into three stages for easy understanding.

1. Oral stage: This is the only voluntary stage in the act of swallowing. It consists of:

Mastication of food making it into a bolus fit to be swallowed. The main muscles involved in the act of chewing are:

a. Masseter

b. Temporalis

c. Pterygoids

These muscles of mastication are supplied by the mandibular division of the trigeminal nerve.

The tongue plays a vital role in this phase and is supplied by the hypoglossal nerve.

The facial nerve also plays a vital role by supplying the Buccinator and orbicularis oris. These muscles prevent drooling of food from the mouth and keeps the contents inside the oral cavity by their contraction. In patients with facial palsy the buccinator and orbicularis oris are paralyzed causing drooling on that side of the mouth.

2. Pharyngeal stage: Is involuntary and is very complex. It passes the masticated food through the oropharynx, through the cricopharynx into the upper oesophagus. This passage is facilitated by relaxation of cricopharyngeus muscle.

During this phase the nasopharynx is shut off from the oropharynx by the contraction of muscles of soft palate and posterior pharyngeal wall, thus preventing nasal regurgitation of masticated food.

During this phase the laryngeal musculature should also constrict to prevent aspiration.

This stage relies on the coordination of many muscle groups including the muscles of the soft palate which is supplied by the 5th, 7th, 9th and 12th cranial nerves.

The pharyngeal muscles also play a vital role in propelling the food through the cricopharynx. These muscles are supplied by the 9th and 10th cranial nerves. The cricopharyngeus muscle is supplied by the 10th cranial nerve and sympathetic nerves.

Neurological disorders affecting the pharynx impair swallowing by altering motor and / or sensory functions in the oral and pharyngeal stages.

3. Oesophageal stage: This stage is again involuntary. It propels the food bolus down the oesophagus. This phase is mediated by the 10th and sympathetic nerves. This phase is dependent on the peristalsis of the oesophageal musculature. During swallowing, peristaltic waves pass down the oesophagus with waves of positive pressure reaching (50-100mmHg). When liquids and semisolids are swallowed, there is an initial negative wave caused by elevation of the larynx drawing on the cervical oesophagus. This is followed by a abrupt positive wave, which coincides with the entry of the bolus into the oesophagus. This wave is known as the primary peristaltic wave. This wave is followed by a smaller positive wave known as the stripping wave, which clears the leftover food material from the oesophagus.

Secondary peristaltic waves are generated in the oesophagus in response to dilatation of oesophagus

Tertiary peristaltic waves are irregular, non-propulsive contractions involving large segments of oesophagus. This occurs during emotional stress.

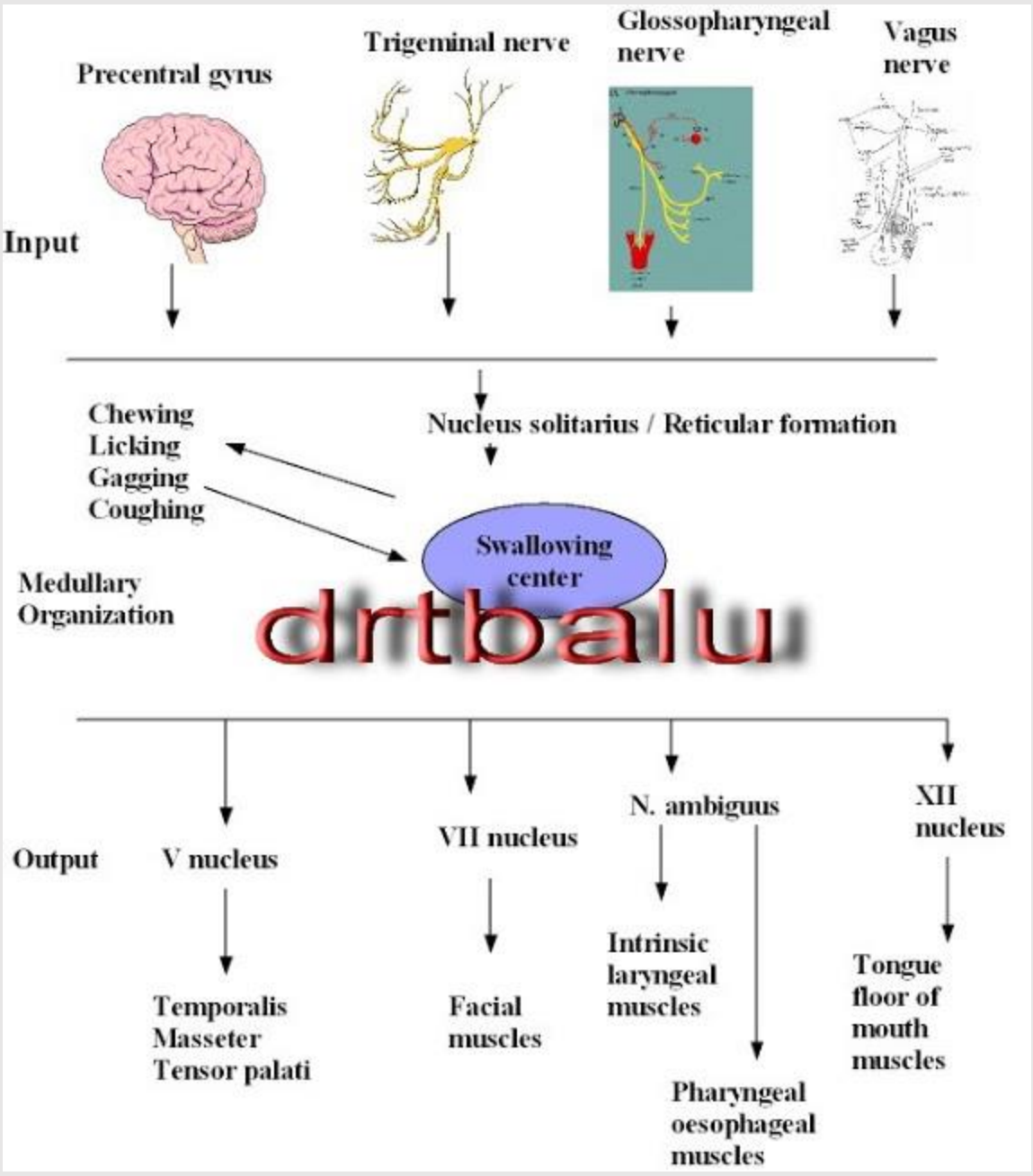
When fluid is swallowed, it may be projected from the pharynx to the oesophago gastric junction in about 1 second flat (when the subject is standing). This occurs well ahead of the peristaltic wave. Thus it should be borne in mind that this fact causes burns due to swallowing of corrosive fluids more at the distal end of oesophagus.

The rate of progression of peristaltic wave varies in different portions of oesophagus:

1. In the upper part of oesophagus the peristaltic wave progresses rapidly.

2. The waves are more sluggish in the lower third of oesophagus.

These differences are due to the fact that the musculature is striated in the upper portion of oesophagus and smooth at the lower 1/3 of oesophagus.


Precentral gyrus
Trigeminal nerve
Glossopharyngeal nerve
Vagus nerve
Input
Chewing
Licking
Gagging
Coughing
Nucleus solitarius / Reticular formation
Swallowing center
Medullary Organization
drtbalu
Output
V nucleus
VII nucleus
N. ambiguus
XII nucleus
Temporalis
Masseter
Tensor palati
Facial muscles
Intrinsic laryngeal muscles
Pharyngeal oesophageal muscles
Tongue floor of mouth muscles

9. Indirect laryngoscopy

Indirect laryngoscopy: is a procedure which is used to view the larynx and vocal cords using mirrors and reflected light. It was first performed by Manual Garcia who was a singer. He in fact visualized his vocal cords in a barber shop through a set of mirrors placed on the wall.

1. The mirror used is plane mirror with a long handle.

2. It is held like a pen in the dominant hand with the mirror pointing downwards.

3. The mirror is warmed with a spirit lamp, the temperature is tested on the back of the hand. This is done to prevent fogging of the mirror.

4. The patient is asked to protrude the tongue and it is held with a gauze.

5. The mirror is introduced into the mouth and gently slide under the uvula.

6. The mirror is tilted to get good view of the larynx.

7. The patient is asked to say eee.

8. The mobility of the vocal cord can be tested.

The image of larynx visualized is reversed one, the near parts appear the farthest (antero posterior inversion). The right and left structures are not actually reversed, the right-hand structures are seen in the observers left.

To examine the anterior commissure of the vocal cords, the patient is made to crouch / kneel with the head tilted upwards and extended, the examiner stands and holds the mirror nearly vertical over the posterior pharyngeal wall. This position is known as reverse killian's position.

10. Obstructive sleep apnoea.

Snoring is defined as a rough rattling noise made on inspiration during sleep by vibration of the soft palate and the uvula. On inspiration, air on its way to the lungs travels by the tongue, soft palate, the uvula and the tonsils. In awake persons the muscles at the back of the throat tightens to hold these structures in place and prevents them from collapsing and vibrating in the airway. During sleep, the soft palate and uvula may vibrate causing the sounds of snoring. Snoring occurs nearly in a third of adults.

Sleep disordered breathing an overview:

This condition encompasses a spectrum of disorders with implications in many fields of medicine. The following are the spectrum in the order of increasing significance:

Primary snoring

Upper airway resistance syndrome

Obstructive sleep apnea hypopnea syndrome

Apnea:

This is defined as a cessation of breathing for 10 seconds. In obstructive sleep apnea, the apneas accompanied by observed ventilatory effort (rise and fall of chest).

Sleep disordered breathing is represented by snoring. Rarely snoring could be totally benign, occurring as a consequence of a removable cause like nasal congestion, excessive fatigue, abnormal sleep position, CNS depressants etc.

It should be stressed that snoring indicates an underlying pathological change that is indeed more significant than the auditory annoyance of one's bed partner.

Terminologies:

Snoring:

This is a respiratory sound, typically occurring during inspiration or expiration, and is generated in the upper airway during sleep. Simple snoring, noted by the bed partner has no clinical sequelae. These patients don’t experience daytime somnolence. These patients usually benefit from weight reduction, positional modification during sleep or by using an oral appliance.

If a patient with snoring presents with comorbid hypertension / daytime excessive sleepiness then it is an indication for further investigation.

RERA:

Also known as respiratory effort related arousal. This occurs when upper airway narrowing has led to an increased respiratory effort. This extra effort could stimulate arousal. This can be identified from the EEG of a polysomnogram. This is assumed to impact daytime sleepiness. When patients are noted to have several RERAs with clinically insignificant Apnoea-Hypopnea index then Upper airway resistance syndrome (UARS) should be suspected.

UARS:

Upper airways resistance syndrome indicates a middle ground between obstructive sleep apnea and primary snoring. These patients don't have overt sleep apnoea and may not even snore, but still can't get a good night's sleep.

These patients may demonstrate anthropomorphic abnormalities with decreased posterior airspace with retrodisplacement of the tongue. Air flow is limited, but subsequent apnea / hypopnea is minor and don't cause concomitant oxygen desaturation.

This condition is also associated with esophageal pressure changes as per the sleep study conducted with esophageal pressure monitor. Daytime sleepiness is a consequence of disturbed, but not apneic sleep.

Apnea:

This is defined as a cessation of breathing for 10 seconds. In obstructive sleep apnoea, the apnea is accompanied by observed ventilatory effort (chest rise and fall). In central apnea no ventilatory effort is seen. Pure central apnea is rather rare.

A mixed apnea is a disordered breathing that begins as a disordered breathing event that begins as a central apnea and ends as an obstructive one.

Hypopnea episode is a partial reduction in ventilation with continued effort for at least 10 seconds. The criteria for hypopnea can vary between laboratories, a general definition is a reduction of 30% in airflow from baseline level plus a 4% or greater decrease in oxygen saturation.

AHI also known as Respiratory disturbance index (RDI):

Also known as Apnoea-Hypopnea index is the cornerstone which helps to place a patient on the sleep apnoea spectrum. AHI is actually the sum of apneas plus hypopneas in 1 hour of sleep. The severity of apnea is not measured by AHI alone (other factors like clinical presentation, daytime sleepiness, hypoxemia, sleep fragmentation, and presence of arrhythmias should be taken into account).

Classification of OSA severity:

1. AHI of less than 5 is normal

2. AHI of 5-14 is classified as mild apnea

3. AHI of more than 30 is classified as severe apnea

Even though respiratory disturbance index is similar to AHI there are certain minor differences. AHI measures total sleep time and averages the number of apneas and hypopneas per hour. RDI uses total recording time (which includes awake time in the sleep lab). RDI may also include RERA which AHI strictly excludes.

AHI does not factor the degree of oxygen desaturation or the number of arousals throughout the night.

Arousal:

Is defined as transient awakening from sleep as a result of apnoea or respiratory efforts.

Arousal index:

It is the number of arousal events in one hour. Less than four is normal.

Multiple sleep latency test or nap study:

Patient is given 4-5 scheduled naps usually in the daytime. Latency period from wakefulness to the onset of sleep and rolling eye movement sleep are measured. This test is performed when narcolepsy is suspected or daytime sleepiness is evaluated objectively.

Mechanism of snoring:

During the muscles of pharynx are relaxed and cause partial obstruction. Breathing against obstruction causes vibrations of soft palate, tonsillar pillars and tongue base producing the sound. Sound intensity could be as high as 90dB.

Snoring could be primary (without association with OSA). Primary snoring is not associated with excessive daytime sleepiness and has apnoea-hypnoea index of less than 5.

Causes of snoring:

1. Adenotonsillar enlargement (common in children)

2. Deviated nasal septum

3. Turbinate hypertrophy

4. Nasal valve collapse

5. Nasal polypi / tumors

6. Elongated soft palate

7. Elongated uvula

8. Tonsillar enlargement

9. Macroglossia

10. Retrognathia

11. Laryngeal stenosis

12. Omega shaped epiglottis

13. Obesity with a thick neck

14. Use of alcohol, sedatives and hypnotics

Sites of snoring:

Sites affected in snoring could be soft palate, tonsillar pillars or hypopharynx. The site could vary from patient to patient and even in the same patient multiple sites can be involved.

Physiology of sleep:

Normal healthy adult sleeps for 7-8 hours a day. Sleep is known to occur in two phases i.e. non-REM and REM. These two phases occur in semiregular cycles, each cycle lasting for about 90-120 mins. There are thus three to four cycles of sleep.

NON-REM sleep:

This is also known as non-rapid eye movement sleep. It constitutes about 80% of sleep time.

StageI :

This involves transition from wakefulness to sleep. It constitutes 2-5% of sleep. EEG shows decrease of alpha and increase of theta waves. There is loss of muscle tone. A person can be easily aroused from this stage.

Stage II:

This stage is characterized by sleep spindles or 'K' complexes and decrease in muscle tone. It constitutes about 50% of sleep

Stage III:

This stage forms 3% of sleep and is characterized by delta waves. It is deep sleep.

Stage IV:

This forms 10% of sleep characterized by delta waves. It is deep and most restful sleep.

REM sleep:

This forms 20-25% of total sleep. This is characterized by rapid eye movements, increased autonomic activity with erratic cardiac and respiratory movements. Dreaming occurs in this stage, muscular activity is decreased.

Obstructive sleep apnea syndrome:

This is defined as a chronic respiratory sleep disorder typified by recurrent episodes of partial or complete upper airway obstruction during sleep that cause cessation of airflow in the presence of respiratory effort. These episodes cause repeated arousals and fragmented sleep and could be due to various anatomic and physiologic dysfunctions.

Nocturnal symptoms:

1. Restless sleep and snoring (this is observed in nearly 90% of these patients)

2. Sleep disruptions

3. Choking

4. Esophageal reflux

5. Nocturia

6. Heavy sweating

Day time symptoms:

1. Excessive daytime somnolence. This symptom is observed in 70% of patients. Motor vehicle accidents are a significant concern in patients with OSA.

2. Morning headaches

3. Sexual dysfunction

4. Hearing loss

5. Automatic behavior

6. Short term memory loss

7. Hypnogenic hallucinations

Clinical evaluation:

History:

Patient's bed partner gives more reliable information than the patient. History should include snoring during sleep, restless disturbed sleep, gasping, choking or apnoeic events and sweating.

In the daytime there is history of excessive sleepiness and fatigue as measured by Epworth sleepiness scale, irritability, morning headaches, memory loss and impotence. The body position of the patient during sleep should also be elicited, intake for alcohol, and sedatives should be sought.

Epworth sleepiness scale:

Situation Score (0-3)

Sitting & reading

Watching TV

Sitting inactive in a public place

Passenger in a car for 1 hour

Lying down to rest in the afternoon

Sitting and talking to someone

Sitting quietly after a lunch without alcohol

Sitting in a car while stopped in traffic for a few minutes

0 - Never doze off

1 - Slight chance of dozing off

2 - Moderate chance of dozing off

3 - High chance of dozing off

Examination should include:

Body mass index:

This is calculated by dividing the body weight in kilograms by height in meters squared.

18.5-24.9 - Normal BMI

25-29% - Overweight

30-34.9 - Obese

Obese patients should be advised to reduce their body weight.

Collar size:

Neck circumference at the level of cricothyroid membrane is measured. Collar size should not be more than 42 cm in males ad 37.5 cm in females.

Complete head and neck examination:

Examination should look out for tonsillar hypertrophy, retrognathia, macroglossia, elongated soft palate and uvula, base of tongue tumors, septal deviation, nasal polypi, turbinate hypertrophy and nasal valve collapse. Nasopharynx and larynx should also be examined.

Muller's maneuver:

This should be performed in all suspected patients with OSA. A flexible endoscope is passed through nose and the patient is asked to inspire vigorously with nose and mouth completely closed. Collapse of soft tissues at the level of tongue base and just above the soft palate should be looked out for. Level of pharyngeal obstruction can be elicited.

Systemic examination:

Hypertension, congestive cardiac failure, pedal oedema, truncal obesity and any sign of hypothyroidism.

Cephalometric radiographs:

Should be taken for craniofacial anomalies and tongue base obstruction.

Polysomnography:

This investigation is the corner stone of diagnosis and evaluation of OSA. Polysomnography is an overnight study that records a multitude of physiologic factors, which includes:

Brain waves

Muscle tone

Eye movements

Respiratory effort

Oxygen saturation

Individual patient studies are performed in a sleep lab where the state, quality of sleep in each stage, and the apnea-hypopnea index can be determined.

The following studies need to be performed:

EEG - To assess the brain waves

EMG - To assess the muscle tone

EOG - To assess eye movements

Measurement of airflow, chest and abdominal efforts - To assess respiratory effort

ECG - To assess heart rate rhythm and activity

Pulse oximetry - To assess oxygen saturation

Sleep apnea worsens during REM sleep when the muscle tone of hypopharyngeal and oropharyngeal musculature are at their lowest resulting in an upper airway collapse. Airway obstruction, oxygen desaturation and in severe cases carbon dioxide retention can cause arousals from sleep.

Polysomnography can differentiate between primary snoring, pure OSA and central sleep apnoea.

Split night polysomnography:

In this study the first part of the night is used in usual polysomnography while the second part of night is used in titration of pressures for continuous positive airway pressure. This procedure is not recommended because episodes of sleep apnoea occur more often in second half of night and are thus missed. Titration of pressures for CPAP should ideally be done on a second night.

Sleep physiology:

Physiologic changes of sleep affect multiple systems including CVS, CNS, RS, GI, Thermoregulatory and Endocrine systems.

CVS changes:

CVS changes are controlled by autonomic nervous system. Generally vagal tone increases and sympathetic input decreases throughout the night causing the below stated changes:

1. Heart rate and blood pressure decreases during NREM sleep.

2. Further decreases in HR and BP occurs during tonic REM sleep.

3. HR and BP increase during phasic REM

4. Cardiac dysrhythmias may diminish / disappear during sleep, premature ventricular contractions increase during REM sleep.

CNS changes:

1. Blood flow to the brain increases during sleep and is higher during REM sleep

2. Intracranial pressure and temperature increase during REM sleep and decrease during NREM sleep. These differences indicate variations in the metabolic activity of the brain.

Pulmonary system:

1. Respiration becomes fully involuntary

2. Ventilatory control becomes mainly driven by carbon dioxide levels. The partial pressure of carbon dioxide rises by 4 mm of Hg during sleep. In a healthy person this increase is physiologic; but however, in a patient with lung disease, this increase may result in significant oxygen desaturation. Chemical responses to hypoxia and hypercapnia decrease in NREM and decrease still further in REM sleep. This is the reason why OSA is most severe during REM sleep.

3. Decrease in tidal volume causing overall decrease in ventilation

4. In NREM sleep, respiratory rate, tidal volume, and minute ventilation decrease, leading to an increase in end-tidal carbon dioxide and a decrease in oxygen saturation.

5. During REM sleep, respiration may be rapid and irregular

6. Upper airway resistance increases by up to 7 times that of waking levels, Muscle tone is lost, especially in the intercostal and pharyngeal muscles; however, the diaphragm maintains its tone

7. Mucociliary clearance of alveolar oxygen tension and arterial oxygen tension decreases. The decrease in Pao2 is normally less than 2%.

8. Patients with sleep disturbances experience arousals due to labored breathing. The exact stimulus for arousal is unknown, but mechanoreceptors in the upper / lower airway or the diaphragm may be responsible.

GI changes:

1. Gastric acid secretion generally increases during sleep and peaks during REM sleep

2. Swallowing and esophageal motility deceases during sleep

Thermoregulatory system changes:

1. During REM sleep thermoregulation and perspiration are absent. Body becomes cold blooded.

2. Overall body temperature decreases during sleep

Endocrine system changes:

1. Growth hormone levels peak during early hours of sleep and gradually decline

2. Prolactin levels are affected by sleep and increase during both nocturnal and daytime sleep

3. TSH levels tend to decrease during sleep coinciding with the decreased metabolic needs off the body during sleep.

4. Melatonin and cortisol levels are affected by circadian rhythms but not by sleep itself.

Risk factors of sleep apnoea:

1. Obesity - 10% increase in weight is associated with 6 fold risk of developing sleep disordered breathing.

2. Sex - OSA affects men commonly. The incidence in women increases after menopause

3. Obstruction - Upper airway may be obstructed at any level. Mallampati classification scale can be used to help in classifying this category

4. Neck circumference - A neck circumference of more than 43 cm is a predictor for an increased apnea-hypopnea index

5. Pulmonary disease - COPD and restrictive / neuromuscular diseases of lungs can cause airway problems

6. CNS depressants like alcohol and sleeping pills cause more relaxed airway with propensity to collapse

7. Tobacco use

8. Hypothyroidism

9. Acromegaly (due to macroglossia)

10. Supine position during sleep

11. Craniofacial anomalies and previous trauma that affects upper airway function / patency

Apnoea clusters and oxygen desaturation:

OSA are often known to occur in clusters. Oxygen desaturation occurs with each apnea. The end of the apnea sequence typically ends with a brief (less than 3 seconds) EEG arousal. In patients with severe OSA, the cluster of apneas occurs throughout sleep. The desaturation from the first apnoea is typically associated with a higher desaturation percentage change than subsequent apneas in the series.

Management:

1. Life style changes - Patients with mild disease and minimal symptoms can be treated with weight loss and dietary restrictions.

2. Alcohol use should be avoided

3. Smoking to be avoided

4. Positional therapy - Patient should sleep on the side as supine position may cause obstructive apnoea. A rubber ball can be fixed to the back of shirt to prevent the patient from assuming supine position.

5. Intraoral devices- These devices alter the position of the mandible / tongue to open the airway and relieve snoring and sleep apnoea. Mandible advancement device keeps the mandible forward while tongue retaining device keeps tongue in anterior position during sleep. Mandibular advancement device is useful in retrognathic patients.

6. CPAP - Provides pneumatic splint to airway and increases its caliber. Optimal airway pressure for the device to open the airway is determined during sleep study and is usually keptat 5-20 cm of water. About 40% of patients find the use of CPAP device cumbersome and difficult to carry with them when travelling.

If CPAP is not tolerated, a BiPAP (bilevel positive airway pressure) device can be used. It delivers positive pressure at two fixed levels - a higher inspiratory and a lower expiratory pressure.

Surgery:

Is indicated for failed conservative therapy.

Tracheostomy:

This is the gold standard of treatment but is not accepted socially because of its complications

Adenotonsillectomy

Septoplasty

Polypectomy

Turbinectomy

Uvulopalatopharyngoplasty

Advancement genioplasty with hyoid suspension

Tongue base radiofrequency ablation

Maxillomandibular advancement osteotomy

Static & dynamic factors involved in the pathophysiology of sleep disorders:

Both static & dynamic factors are involved in the development of OSA. Static factors include:

1. Surface adhesive forces

2. Neck & jaw posture

3. Tracheal tug

4. Gravity - Gravitational forces can be felt simply by tilting the head back to where the retro position of the tongue and soft palate reduces the pharyngeal space.

Any anatomic feature that decreases the size of pharynx (eg. retrognathia) increases the chance of development of OSA. For most patients OSA worsens in the supine sleeping position.

An important static factor that has been found in patients with OSA is the reduced diameter of the pharyngeal airway in wakefulness in OSA patients compared with that of non-OSA patients. In the absence of craniofacial abnormalities, the soft palate, tongue, parapharyngeal fat pads, and lateral pharyngeal walls are enlarged in OSA patients.

Dynamic factors:

1. Nasal / Pharyngeal airway resistance

2. Bernoulli effect

3. Dynamic adherence

Bernoulli effect plays an important role (dynamic) in OSA pathophysiology. According to this effect, when the airflow velocity increases at the site of stricture in the airway, the pressure on the lateral wall decreases causing airway collapse in that region. This effect is exaggerated in areas where the airway is most compliant. This effect helps partially to explain why obese patients (with excessive fat deposition) around the neck are most likely to have OSA.

The cross-sectional area of the airway in patients with OSA is smaller that of people without OSA.

11. Polysomnography

Polysomnography:

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12. Nasopharyngeal Angiofibroma

Definition: Juvenile nasopharyngeal angiofibroma (JNA) is a histologically benign , but locally invasive neoplasm occurring almost exclusively in adolescent males. These tumors are highly aggressive and are associated with significant morbidity and mortality due to its tendency to bleed.

Aetiopathogenesis of JNA:
This relatively rare tumor occurs in the second decade. Almost exclusively adolescent males are affected. The reported rate of incidence varies from 1/6000 (Harma 1959) to 1/50,000 (Hondousa etal 1954). The exact nature of the tumor and its etiology is not well known. Various theories have been propounded to explain the etiopathogenesis of JNA.

Theories of etiopathogenesis of JNA:

Ringertz theory: This theory was proposed by Ringertz in 1938. He believed that JNA always arose from the periosteum of the skull base.

Som & Neffson (1940): believed that inequalities in the growth of bones forming the skull base resulted in hypertrophy of the underlying periosteum in response to hormonal influence.

Bensch & Ewing (1941): thought that the tumor probably arose from embryonic fibro cartilage between the basi occiput and basi sphenoid.

Brunner (1942): Suggested an origin from conjoined pharyngobasilar and buccopharyngeal fascia.

Marten et al (1948): Proposed a hormonal theory suggesting that these tumors resulted from deficiency of androgens or over activity of estrogens and that the hormonal stimulation is responsible for angiomatous components seen in JNA tissue.

Sternberg (1954): Proposed that JNA could be a type of hemangioma like a cutaneous hemangioma seen in children which regresses with age.

Osborn (1959): Considered two alternatives to explain the etiology of JNA. They proposed that the swelling could be due to either a hamartoma or residual fetal erectile tissue which were subject to hormonal influences.

Girgis & Fahmy (1973): Observed cell nests of undifferentiated epithelioid cells or "Zell ballen" at the growing edge of angiofibromas. This appearance was more or less similar to that of paraganglioma. They considered JNA to be a paraganglioma.

The most accepted theory is that JNAs originate from sex steroid–stimulated hamartomata's tissue located in the turbinate cartilage. The proposed hormonal influence may explain why (rarely) some JNAs involute after puberty.

Pathophysiology: The proposed origin of the JNA is located along the posterior-lateral wall in the roof of the nasopharynx, usually in the region of the superior margin of the sphenopalatine foramen and the posterior aspect of the middle turbinate. Fetal histology confirms large areas of endothelial tissue in this region. Rather than invading surrounding tissue, this tumor displaces and distorts, relying on pressure necrosis to destroy and push through its bony confines. Intracranial extension is noted in 10-20% of cases.

Extension of tumor:

As growth enlarges it has to follow the lines of least resistance.

a. It hangs down in the cavity of nasopharynx and when large enough, it may depress the soft palate and may even peep below it.
b. It can work its way in to the corresponding nasal passage towards the anterior nares. It can cause pressure on the outer wall as well as on the septum bending it to the opposite side. The corresponding turbinates and ethmoidal air cells and the related antral wall may suffer pressure atrophy. The most common deformity referred to as the "frog face" is due to the forward spread involving the ethmoidal region. Lateral spread in to the maxillary sinus may be responsible for the cheek swelling.
c. The mass commonly arises from the sphenopalatine foramen area. It may have two components, one filling the nasopharynx and the other extending out into the pterygopalatine and infratemporal fosse. The central stalk joining the two components occupy the sphenopalatine foramen at the upper end of the vertical plate of palatine bone.
d. It can encroach into the orbit by passing through the infra orbital fissure.

e. It can erode the skull base and cause intracranial problems.

Blood supply of JNA: is from the

1. Enlarged maxillary artery
2. Ascending pharyngeal artery
3. Unnamed branches from internal carotid artery

Management:

Surgical removal

13. Juvenile Nasopharyngeal angiofibroma modes of spread and clinical features

Extension of tumor:

As growth enlarges it has to follow the lines of least resistance.

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d. It can encroach into the orbit by passing through the infra orbital fissure.
e. It can erode the skull base and cause intracranial problems.

Clinical features: Signs and symptoms are present for an average of 6 months prior to the diagnosis, commonly with extension beyond the nasopharynx.
The two cardinal symptoms of angiofibroma are nasal obstruction and intermittent unprovoked epistaxis. Epistaxis may vary in severity from an occasional show to an alarming sometimes threatening torrent. Chronic anaemia is thus a feature of an established JNA.
The nasal obstruction is so complete causing stasis of secretions and sepsis become inevitable. Patients may even have hyposmia or anosmia.

The voice of the patient acquires a nasal intonation. If the swelling enlarges to force the soft palate down, the voice may become plummy.
Blockage of eustachean tube orifice is also common causing deafness and otalgia. Headache is not uncommon in long standing cases. If present it could be attributable to chronic sinusitis in some patients. Intracranial extension of the mass could also be the cause for headache in these patients.

Diplopia may occur secondary to the erosion of the mass into the cranial cavity and causing pressure on the optic chiasma. Failing vision has been attributed by Shaheen to the tenting of the optic nerve by the tumor.

Anterior rhinoscopy shows the presence of abundant purulent nasal secretions together with bowing of nasal septum to the uninvolved side. Posterior rhinoscopy in a cooperative patient shows a pink or red mass filling the nasopharynx. Due to the bulk of the lesion it may not be always possible to ascertain the site of origin accurately.

Gross physical signs become evident when extensive disease involves the nose and infra temporal fossa. The nasal bones become spayed out and there may be obvious swelling in the temple and cheek. Intra oral palpation in the interval between the ascending ramus of the mandible and the side of the maxilla may reveal the telltale thickening of disease which has crept around the back of the antrum. Impaction of bulky mass in the infra temporal fossa results in extreme signs, such as trismus and bulging of the parotid gland. Proptosis is a definite sign that the orbital fissures have been penetrated. The classic frog face seen in patients with extensive disease is due to massive escape of the disease.

14. Management of Juvenile Nasopharyngeal Angiofibroma

Treatment: Depends mainly on the extent of the lesion. Surgery is the preferred modality of treatment for all stages of the mass up to stage IVa while radiotherapy is used for stage IVb. Mainly three lines of treatment are available:
1. Surgery
2. Irradiation
3. Hormonal (purely supportive in nature)

Surgery: Complete excision of an extensive JNA mass is a desirable goal but is a surgical challenge because of the limited field of work, inadequate visualization and profuse bleeding during surgery. Besides the deformity, scars and adhesions as a result of prior surgery adds to this problem. Currently several approaches are available to access the neoplasm. They are:

I Tumor removal - via naturalis:

This approach is preferred for very small tumors confined to nasopharynx. The tumor can be removed by subperiosteal dissection after soft palate retraction. Access is limited in this approach.

II Transpalatal approach:
Wilson in 1951 described this approach. This approach gives exposure to nasopharynx as well as extensions into the sphenoid sinus and choana. It gives no visible scar and post op healing is good. This approach is useful in dealing with masses in the nasopharynx with minimal extension into the choana and sphenoid sinus.

Procedure: Patient is put in tonsillectomy position. A forward curved incision is made just in front of the junction of hard and soft palate. Mucoperiosteum is separated either way. Posterior spine of the hard palate is removed. Incision is extended laterally and downwards on either side along the pterygomandibular raphe. The mucosa of the lateral pharyngeal wall is not divided and care is taken not to damage the greater palatine vessels. A good view of nasopharynx is achieved in this procedure. The mucous membrane on the side of the growth is incised with a blunt knife. Thus, with blunt dissection the periosteum is elevated, growth is separated and finally avulsed in one piece.

III. Lateral rhinotomy approach: This approach is suited for smaller growth restricted to the nasal cavity. It is contraindicated for larger masses and whose extensions and attachments cannot be ascertained.

IV. Trans hyoid approach: This is suitable for tumors localised to nasopharynx without any extension into the surrounding structures. The major disadvantage is that it requires a temporary tracheostomy.

V. Transmandibular approach: (Kermen)
Incision is made vertically Infront of the ear and carried down the neck anterior to the sternomastoid muscle. Dissection is started in the neck by exposing the carotid bifurcation at which level the external carotid artery is ligated. Lower pole of parotid is dissected free. The insertions of masseter muscle is severed from the mandible. Lateral aspect of the mandible is exposed by elevation of the periosteum. Transection of mandible is done with a Gigli saw at a point 1 cm below the notch formed by coronoid and condyloid processes. Separation of mandible exposes the tube muscular wall of nasopharynx which is incised longitudinally on its lateral wall so that its lumen is entered. Tumor is exposed and dissected out.

VI. Sublabial midfacial degloving approach (Conley 1979): It is a bilateral extended trans nasal maxillary approach. There is no visible scarring, adequate exposure of nasal complex, nasopharynx and middle third of the face is obtained. Sublabial incision is performed from one maxillary tuberosity to the other. Intercartilagenous incisions are given to separate soft tissue of the nose from the upper lateral cartilages. Incisions along the pyriform aperture connects the circumferential septal vestibular incisions to the sublabial incision. This allows total mid facial degloving up to the roof of the nose and infra orbital foramen. The complication of this procedure is vestibular stenosis.

VII. Transzygomatic approach (Sami & Girgis 1965): This approach is useful for removal of tumor involving the temporal and infra temporal regions.

Combined approaches:
Depending on the size and extent of the mass a combination of various approaches can be attempted to extirpate the mass in toto.

I. Transpalatal sublabial approach (Saldana 1965): This approach is useful for tumors extending into pterygoid and infratemporal fossa. A sublabial incision 'S' shaped is made extending to the maxillary tuberosity. After elevation of mucoperiosteal flap, the greater palatine neurovascular pedicle is preserved. The surgeon's index finger is inserted into the pterygopalatine fossa and blunt dissection is used to free the tumor from its lateral attachment. The mass can be delivered via the nasopharynx.

II. Combined transpalatal and lateral rhinotomy approach: This approach is indicated for larger mass, recurrent JNA, and when attachment and extensions of the tumor are not predetermined. This approach gives excellent exposure and so the chances of recurrence are minimal.

III. Triple approach of Hiranandani (1968): In this approach Transpalatal and lateral rhinotomy are combined along with Caldwell luc. Complete exposure of pterygopalatine fossa is possible by removal of posterior wall of the maxillary antrum, after opening the antrum through Caldwell luc incision. Chances of recurrence are minimized. Ligation of internal maxillary artery is done to reduce the bleeding.

Radiotherapy:
Radiotherapy can produce some amount of tumor regression by radiation vasculitis and occlusion of vessels by perivascular fibrosis. Radiotherapy should be reserved for selected patients such as those with inoperable intracranial extensions and recurrent tumors.

Disadvantages of radiotherapy:

a. If the child is exposed to large doses i.e. above 5000-6000 rads, there may be damage to eyes, spinal cord and brain.
b. Small doses are ineffective in reducing the blood supply or the size of the mass.
c. Radiotherapy may cause fibrosis and adhesions of surrounding tissue. Later surgery upon these patients becomes difficult.
d. Sarcomatous changes can occur in the mass as a result of irradiation.

Adjunctive treatment:

Hormonal therapy: Since JNA has been postulated as an endocrine tumor testosterone receptor blocking drugs / estrogens have been tried to reduce the mass. These hormones cause disagreeable side effects such as increased breast size. Hormones could even act as carcinogens.

Embolization: Was first attempted by Robertson in 1972. This was not meant to be therapeutic measure. After embolization bleeding is minimized during surgery. It is ideally carried out a few days before surgery. Hence it is a valuable preliminary to surgery. The feeding vessels to the tumor is identified. It is then deliberately occluded by means of materials injected through a selectively placed catheter.
Materials used:
Autologous substances like fat, blood clot, or chopped muscle fragments.
Artificial materials: Gelfoam, Oxidized cellulose, Tantalum powder, glass beads, polyvinyl alcohol etc.
Embolization should always be preceded by angiography. Subtraction films may be helpful in areas containing complex bony structures.
Immediate complications of embolization are pain, embolization of normal vessels, hypersensitivity. Delayed complications include fever, pain and infections.

Cryosurgery and Lasers can also be used during surgery to minimize bleeding.

15. Faucial diphtheria

Faucial diphtheria: is caused by Corynebacterium diphtheria. This is a very rare condition these days considering the effectiveness of the universally administered vaccinations under the immunization schedule.

The disease is characterized by membranous exudate at the site of infection. This is followed by distant toxic effects.

Age of occurrence: Occurs in the age group between 2 and 10 years. Below 2 years the passive immunity provided by the mother is still persistent. After the age of 10 the child gains immunity from exposure to community infections or immunization.

Pathogenesis: Organisms multiply in the throat, producing toxins. The necrosis of mucosa along with collection of polymorphs and fibrin leads to false membrane formation. This is a pseudo membrane because it contains layers of necrotic mucosa, while a true membrane will be found superimposed on intact mucosa.

Incubation period: ranges from 2 - 10 days

Clinical features:

1. The child is abnormally quiet and refuses to eat

2. Malaise and headache

3. Toxemia could be present

4. Pulse rate increased out of proportion to fever

5. Presence of massive cervical lymphadenopathy (Bull's neck)

6. Toxic myocarditis is common

Investigations:

1. Throat swab reveals the organisms

2.Schick test - positive

Treatment:

1. Antibiotics - Penicillin group

2. Antidiphtheritic serum to neutralize the toxin. This is administered as follows: Mild case 20,000 units, moderate to severe cases 40,000 to 80,000 units. Half of this dose is administered intravenously and the other half intramuscularly. Of course, test dose should be administered.

3. If airway is compromised tracheostomy should be done

3. Complete isolation - 2 weeks

16. Vincent's angina

Vincent angina goes by many other names including acute necrotizing ulcerative gingivitis (ANUG), acute membranous gingivitis, fusospirillary gingivitis, fusospirillosis, fusospirochetosis, fusospirochetal gingivitis,
necrotizing gingivitis, phagedenic gingivitis, ulcerative gingivitis, Vincent stomatitis, Vincent gingivitis Vincent infection, anaerobic pharyngitis and trench mouth. This condition is so called after the French physician Henri Vincent (1862-1950). The word "angina" comes from the Latin "angere" meaning "to choke or throttle." Poor oral hygiene coupled with physical or emotional stress, nutritional deficiencies, blood dyscrasias, debilitating diseases, and insufficient rest predispose to this disease. It rarely occurs in nonsmokers.
Etiology:
It is a mixed infection of spirochetes (Treponema vincenti) and anaerobic fusiforms (Fusobacterium necrophorum). T. vincentii is 5-10 microns long, has 3-8 irregular spirals, stains uniformly and is strict anaerobe.

Fusobacteria are thin, curved rods measuring 10-14 microns, fusiform shaped with central portion deeply stained than ends.
Clinical features:
It is a progressive painful infection with ulceration, swelling and sloughing off of dead tissue from the mouth and throat due to the spread of infection from the gums. The chief manifestations are acutely painful, bleeding gingivae, salivation, and overwhelming fetor oris. The condition is characterized by the presence of grayish membrane and foul odor to the breath. The ulcerations, usually limited to the marginal gingiva and interdental papillae, have a characteristic punched-out appearance. They are covered by a grayish membrane and bleed with slight pressure or irritation. Swallowing and talking may be painful. Regional lymphadenopathy is often present. Loss of the gingival papillae is generally permanent. Differential diagnoses include streptococcal pharyngitis and diphtheria.

Laboratory diagnosis:
Throat swabs should be collected for smear and culture. Gram stained smear shows the presence of gram negative fusiform bacilli and spirochete. Since the pathogens are anaerobes, they are difficult to culture.
Additionally, both spirochetes and fusobacteria are normally found in oral cavity and their isolation in culture is not always conclusive.

Treatment:
Treatment of Vincent's angina involves hydrogen peroxide mouthwash, debridement and antibiotic penicillin.
Alternatives to penicillin are Ampicillin/sulbactam IV or amoxicillin/clavulanate oral, Penicillin IV plus metronidazole and Clindamycin oral or IV

17. Quinsy

Quinsy otherwise also known as peritonsillar abscess is a collection of pus in the peritonsillar space between the superior constrictor and capsule of the tonsil. It is usually unilateral, and commonly affects adolescent males.
Pathophysiology:
Infection usually starts in the crypta magna from where it spreads beyond the confines of the capsule causing peri tonsillitis initially, and peritonsillar abscess later.

Another proposed mechanism is necrosis and pus formation in the capsular area, which then obstructs the weber glands, which then swell, and the abscess forms.

Weber's glands:

These are mucous (minor) salivary glands present in the space superior to the tonsil, in the soft palate. There are 20 - 25 such glands in this area. These glands are connected to the surface of the

tonsil by ducts. The glands clear the tonsillar area of debris and assist with the digestion of food particles trapped in the tonsillar crypts. If Weber's glands become inflamed, local cellulitis can develop. Inflammation causes these glands to swell up causing tissue necrosis and pus formation i.e. the classic features of quinsy. These abscesses generally form in the area of the soft palate, just above the superior pole of the tonsil, in the location of Weber's glands.

The occurrence of peritonsillar abscesses in patients who have undergone tonsillectomy further supports the theory that Weber's glands have a role in the pathogenesis.
Aetiology:
Recurrent attacks of tonsillitis cause obstruction and obliteration of intra tonsillar clefts and the infection spreads to peritonsillar area causing suppuration.

Smoking and chronic periodontal disease could also cause quinsy.
Clinical features:
1. Patient looks very ill and febrile
2. Odynophagia (painful swallowing)
3. Dribbling of saliva
4. Inability to open mouth
5. Muffled / Hot potato voice otherwise known as rhinolalia clausa
On examination:
The tonsil is found pushed downwards and medially; it blanches on slight pressure. The uvula is edematous and is pushed to the opposite side. Tonsillar pillars are congested. Patient also has halitosis (bad breath), trismus and tender enlarged jugulodigastric nodes.

Medical management:

1. Broad spectrum antibiotics. The anti-bacterial spectrum should ideally include gram positive, gram negative and anaerobes. Commonly used drugs are broad spectrum penicillins like ampicillin / amoxycillin, in addition to which metronidazole or clindamycin can be combined to take care of anaerobes.
2. Anti-inflammatory drugs like Ibuprofen and antipyretics like paracetamol.

Surgical management:
Incision and drainage: This is performed with patient in sitting position to prevent aspiration of pus into the larynx. First the oral cavity and throat of the patient is sprayed with 4 % topical xylocaine spray to anaesthetize the mucosa.
A Saint claire Thompson quinsy forceps, or a guarded 11 blade can be used. The 11 blade is gaurded to prevent the blade from penetrating the tonsillar substance deeply and damaging underlying vital structures like internal carotid artery.
Site of incision:
Is commonly over the point of maximum bulge. It can also be made at the junction between a horizontal imaginary line drawn from the base of the uvula to the anterior pillar and a vertical

imaginary line drawn along the anterior pillar. After incision is made a sinus forceps is introduced to complete the drainage procedure.

Six weeks after I&D tonsillectomy is performed in this patient to prevent further recurrence. This is known as interval tonsillectomy.
Some authors prefer to do tonsillectomy immediately on a quinsy patient. This is known as Hot tonsillectomy. But this method is fraught with danger because of excessive bleeding and impending risk of thromboembolism.

18. Retro pharyngeal abscess

Retropharyngeal abscess is a collection of pus between the posterior pharyngeal wall and the fascia and muscles covering the cervical vertebrae. It occurs in two forms - 1. The acute primary retropharyngeal abscess which is common in infants, and 2. Chronic retropharyngeal abscess which is common in adults. These two types of abscesses differ in their etiology and management.
Acute primary retropharyngeal abscess: Is the more dangerous type occurring in infants. It is common between the age group of 3 months to 3 years. The predisposing factors are malnutrition, gastroenteritis, poor hygiene etc.

Etiology: Abscesses may follow general debilitating illnesses like scarlet fever, measles etc. Infections from tonsils, adenoid and naso pharynx may even lead to the formation of retropharyngeal abscess. Rarely foreign bodies like bone pieces and pins may also cause retropharyngeal abscess.

Pathology: The disease consists of suppurative lymphadenitis of the retropharyngeal nodes of Henle, situated on either side of midline between the posterior pharyngeal wall and the aponeurosis over the bodies of the second and third cervical vertebrae. These glands receive the lymphatics of the post nasal space, pharynx, nose, eustachean tube and middle ear. These nodes atrophy between the 3rd and 5th year of life hence acute retropharyngeal abscess is uncommon in children above the age of 4.

The Henle's node when infected from the lymphatics, there is first adenitis, then peri adenitis and abscess formation occur. The suppuration is usually one sided, and most prominent in the oro pharynx. If not evacuated in time or when it does not rupture, pus may spread along the esophagus or burst in different directions - towards the larynx, the angle to the jaw or even in to the external auditory canal. The pus is generally foul smelling yellow or whitish in color. It usually contains streptococci, and more rarely staphylococci and pneumococci.

Chronic retropharyngeal abscess: Is commonly known to occur in adults. This is usually caused by tuberculosis. The tuberculous foci occur in the bodies of the cervical vertebrae (Pott's disease) which later spread into the retropharyngeal space. Primary syphilis of the mouth and pharynx may also cause retropharyngeal abscess. This abscess usually is present in midline and is free to spread to either side also.

Symptoms: These patients have excruciating pain while swallowing (odynophagia). Young infants with retropharyngeal abscess will refuse feed, may have extensive drooling. In adults the head may be held straight. Torticollis is also common in these patients. These patients may have difficulty in breathing (stridor), in which case tracheostomy must be considered to secure the airway in the first place. Constitutional symptoms like fever / toxicity is very common in acute retropharyngeal abscess.

Investigations:

Complete blood count show leucocytosis. Blood cultures can also be performed to ascertain the appropriate antibiotics to be used.

C reactive proteins are also found to increased in these patients

Xray soft tissue neck - A.P. and lateral views.

These pictures show prevertebral soft tissue widening. This can be ascertained by estimating the size of the prevertebral soft tissue which is normally half the size of the body of the corresponding vertebra. If the widening is more than half the body size of the corresponding vertebra then retropharyngeal abscess must be considered. The cervical spine are straightened with loss of the normal lordosis (Ram Rod spine). Above the prevertebral shadow air shadow is seen in almost all cases of retropharyngeal abscesses. This gas shadow is caused by entrapped air which occur during breathing. Some bacteria esp. Clostridium are known to form gases which may be entrapped in the prevertebral space.

C.T. scan neck or MRI study of neck will also help in clinching the diagnosis. This must ideally be performed using intravenous contrast agents. It appears as a hypodense lesion in the retropharyngeal space with ring enhancement. Other effects that could be seen are soft tissue swelling, and obliteration of normal fat planes.

C.T. scan is really helpful in differentiating cellulitis from abscess.

Management:

In majority of cases incision and drainage is done and the pus is immediately aspirated out using suction. The incision is made with 11 blade knife over the most prominent portion of the swelling. The I&D is done under local anesthesia. In the case of infants, it is preferable that the patient is held upside down while the surgery is being performed to prevent aspiration of pus into the lungs. When general anesthesia is preferred a cuffed endotracheal tube must be used to minimize the hazard of aspiration of pus into the lungs. The patient must be put in Rose position (tonsillectomy position) while the I&D is being done to reduce the threat of aspiration.

When the abscess points towards the neck then it should be opened through an incision over the neck, preferably along the posterior border of sternomastoid muscle. The dissection is carried out behind the great vessels of the neck and in front of the prevertebral muscles. The surgery is followed by a course of antibiotics mostly cephalosporin group. Clinamycin in dose of 600-900mg intravenously 8th hourly can be administered in adults. Injection penicillin G in doses of 24 million units per day as continuous infusion along with metronidazole injection in doses of 500mg three times a day can also be considered. Metronidazole is highly effective against anaerobes.

If tuberculosis is suspected to be the cause then surgery is deferred. Anti tuberculous treatment is initiated.

Complications:

1. Mediastinitis

2. Airway obstruction

3. Atlanto occipital dislocation

4. Jugular vein thrombosis

5. Cranial nerve deficits especially the lower three ones

6. Hemorrhage secondary to involvement of the carotid artery

19. Potential Neck Spaces, Acute Retro Pharyngeal Abscess

Already discussed.

20. Ludwig's angina.

Ludwigs angina is described as rapidly spreading cellulitis involving the floor of the mouth. It was first described by Wilhelm Friedrich von Ludwig in 1836. This disorder has a potential for airway obstruction.

Synonyms: Cynanche, Carbuculus gangraenosus, Morbus strangulatorius, and Angina maligna.

Anatomy:

This infection involves the submandibular space. The submandibular space can be divided into two spaces: submaxillary and sublingual space. These two spaces are separated from each other by the

mylohyoid muscle. These two spaces are connected posteriorly through a cleft known as the mylohoid cleft. The mylohyoid cleft contains the following structures:

1. Tail of submandibular gland

2. wharton's duct

3. Lingual nerve

4. Hypoglossal nerve

5. Lymphatics

6. Arteries and veins

The floor of the submandibular space is formed by the superficial layer of deep cervical fascia. It is attached from the hyoid bone to the mandible. This space communicates across the midline with that of the space on the opposite side.

Boundaries of submandibular space:

The submandibular space is bounded by the oral mucosa and tongue superiorly and medially, the mandible superiorly, the superficial layer of deep cervical fascia with its tight attachment to the mandible and hyoid bone laterally, and the hyoid bone inferiorly.

Since the mandible and superficial layer of deep cervical fascia provide unyielding barriers superiorly and laterally, the tongue is forced upward and posteriorly giving rise to airway obstruction. This is the most important danger in Ludwig's angina.

Pathophysiology:

Commonest cause of Ludwig's angina is dental infections. One important factor to be considered is the relationship of mandibular dentition to the attachment of mylohoid muscle (mylohyoid ridge). The anterior teeth and first molars regularly attach superior to this line, and infections arising from these roots commonly result in a limited sublingual abscess. The second and third molar roots are attached routinely below this line. Infections involving these roots cause infections of submaxillary space. One other important relationship is that the roots of the anterior teeth and first molar approximate the lateral mandibular surface, whereas the second and third molar roots approach the lingual surface of the mandible.

Criteria for diagnosing Ludwig's angina:

To diagnose Ludwig's angina the following features should be present:

1. Rapidly spreading cellulitis with no specific tendency to form abscess.

2. Involvement of both submaxillary and sublingual spaces, usually bilaterally

3. Spread by direct extension along facial planes and not through lymphatics

4. Involvement of muscle and fascia but not submandibular gland or lymph nodes

5. Originates in the submaxillary space with progression to involve the sublingual space and floor of the mouth.

Etiology:

1. Ludwigs angina is commonly caused as a sequlae to dental infections. In fact it is very common in young adults with periodontal disease. Dental causes account for 75% to 80% of these cases.

2. Penetrating injuries involving the floor of the mouth (stab wounds, gun shot wounds etc)

3. Mandibular fractures

Bacteriology of Ludwig's angina:

Since a majority of cases of Ludwig's angina are caused by dental infections, cultures from this infected area show oral cavity flora. The most common aerobes isolated are alpha haemolytic streptococci followed by staphylococci. Anaerobic cultures are difficult to interpret. The anaerobes isolated are Pepto streptococcus, peptococcus, fusobacterium nucleatum, and bacteroids. The combination of aerobic and anaerobic organisms has a synergistic effect due to production of endotoxins like collagenase, hyaluranidase, and proteases. These endotoxins contribute to the rapidly spreading cellulitis.

Clinical features:

1. Patient has c/o increasing oral cavity and neck pain.

2. These patients have poor oral hygiene

3. Symptoms are at first unilateral but soon become bilateral

4. The soft tissues of the floor of the mouth swells

5. Tongue gets pushed posteriorly causing air way obstruction

6. These patients are usually febrile

On examination:

These patients have tachycardia, fever, and variable degrees of respiratory obstruction with dysphagia and drooling. The submandibular and submental regions are tense, swollen and tender. The floor of the mouth may become tense swollen and indurated. Fluctuation is not present. The tongue is seen to be pushed backwards.

Diagnosis of Ludwigs angina is based on the clinical features enumerated above. These patients may show leukocytosis. X ray soft tissue neck may show soft tissue oedema. CT scan neck is to be considered in all persistent cases to rule out complications. Xray chest must also be considered to rule out mediastinitis.

Management:

Airway management: Since the airway is threatened insertion of oral airway is to be considered. If the patient does not tolerate an oral airway then tracheostomy is to be considered.

Intravenous antibiotics with broad spectrum features (chloramphenicol)may be administered. The drug of choice is amoxycillin with clavulanic acid. Metronidazole must also be administered. Clindamycin can be administered in resistant cases.

Role of surgical drainage: Wide decompression of the supra hyoid region may be considered. The approach is through a median horizontal incision three to four finger breadths below the mandibular margin. The mylohyoid muscle is split in the midline, and drainage is established both medially and laterally. Pus is very rarely encountered during this procedure, but starts to drain several days after the procedure.

Complications:

1. Airway compromise

2. Extension to mediastinum causing mediastinitis. This can be suspected if there is persistent swelling in the neck with pain, spiking fever and persistent leukocytosis.

3. Extension into the carotid sheath and retropharyngeal space.

21. Chronic retropharyngeal abscess

Retropharyngeal abscess is a collection of pus between the posterior pharyngeal wall and the fascia and muscles covering the cervical vertebrae. It occurs in two forms - 1. The acute primary retropharyngeal abscess which is common in infants, and 2. Chronic retropharyngeal abscess which is common in adults. These two types of abscesses differ in their etiology and management.

Chronic retropharyngeal abscess commonly known to occur in adults. This is usually caused by tuberculosis. The tuberculous foci occur in the bodies of the cervical vertebrae(Pott's disease) which later spread into the retropharyngeal space. Primary syphilis of the mouth and pharynx may also cause retropharyngeal abscess. This abscess usually is present in midline and is free to spread to either side also. Rarely infections can spread from tonsils to involve this space.

Symptoms: These patients have excruciating pain while swallowing (odynophagia). Young infants with retropharyngeal abscess will refuse feed, may have extensive drooling. In adults the head may be held straight. Torticollis is also common in these patients. These patients may have difficulty in breathing (stridor), in which case tracheostomy must be considered to secure the airway in the first place. Constitutional symptoms like fever / toxicity is very common in acute retropharyngeal abscess.

Investigations:

Complete blood count show leukocytosis. Blood cultures can also be performed to ascertain the appropriate antibiotics to be used.

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If tuberculosis is considered to be the cause of chronic retropharyngeal abscess then surgery is contraindicated. Anti-tuberculous therapy is initiated.

Patients with non-tuberculous chronic retropharyngeal abscess need to undergo incision and drainage under local anesthesia. Local anesthesia is preferred in order to prevent aspiration.

22. Scarlet Fever

Scarlet fever also known as scarlatina is a syndrome characterized by:

1. Exudative tonsillitis

2. Exudative pharyngitis

3. Fever

4. Bright red exanthematous skin rashes.

This syndrome is caused by exotoxins produced by group A beta hemolytic streptococci. These organism can be found in abundance in discharge from nose, throat and skin. Food borne out breaks are also known.

Scarlet fever is known to evolve from tonsillar / pharyngeal focal infections. Skin rashes are known to develop in about 10% of these patients. The site of bacterial replication is usually inconspicuous when compared with the dramatic effects of released toxin. It can be lethal if streptococcal toxic syndrome develops.

Scarlet fever can also occur following streptococcal infection of skin, soft tissue and even surgical wounds.

Pathophysiology:

Group A streptococci are normal inhabitants of throat and nasopharynx Group A streptococci can cause pharyngitis, and skin infections, pneumonia, bacteremia and lymphadenitis also. Majority of streptococci are known to produce toxins and the erythrogenic toxin produced by them have been implicated in scarlet fever. This toxin was first discovered by Dick in 1924.

It is known to affect school going children because of crowded class rooms and lack of hygiene.

The incubation period for scarlet fever is about 12 hours to 7 days. These patients are usually contagious during acute phase as well as during subclinical phase also.

It is known to affect children between ages 1 -10. It can rarely occur in older children and adults also. Children on reaching the age of 10 would have developed life long protective antibodies against streptococcal pyrogenic exotoxins. This condition is rare in children under 1 year because of the presence of maternal anti exotoxin antibodies.

This disease was dreadful during preantibiotic era. With the advent of antibiotics and improved immune status scarlet fever are not so disastrous these days.

Complications:

1. Septicemia

2. Vasculitis

3. Hepatitis

4. Rheumatic fever

Whenever scarlet fever has been determined to be due to soft tissue infection or near the bone, evaluation for bony involvement should be considered.

Clinical findings:

Fever

Tachycardia

Cervical adenopathy

Mucous membrane lesions (bright red petechiae could be seen over tonsils, posterior pharyngeal wall and soft palate.

Heavily coated tongue

Oedematous red papilla seen in the tongue (strawberry tongue). This occurs between days 4 and 5 after the whitish membrane sloughs off. Skin exanthem usually develop within 48 hours after the

onset of fever. It usually begins as erythematous patches below the ears, on the neck, chest and axilla. The skin after these eruptions feels rough like coarse sand paper. These erythematous lesions are known to blanch on pressure. The skin usually feel itchy but these rashes are not painful. Spread to the trunk and extremities takes about one more day to occur.

Capillary fragility is found to be increased and frequent capillary ruptures are also common. Transverse areas of hyperpigmentation with linear petechial lesions in the axillary, antecubital and inguinal areas are also seen. These arrays can also be present even 48 hours after the rashes vanish.

The cutaneous rashes lasts for 4-5 days and is usually followed by desquamation. This desquamation is one of the features of scarlet fever rashes. This peeling phase occur in about a week and begins from the face. Peeling from the palms and fingers occur one more week later (during the second week).

Complications:

1. Cervical lymphadenitis

2. Otitis media / acute mastoiditis

3. Ethmoiditis

4. Peritonsillar abscess

5. Sinusitis

6. Bronchopneumonia

7. Meningitis

8. Brain abscess

9. Intracranial venous thrombosis

10. Septicemia, meningitis, osteomyelitis and septic arthritis

11. Hepatitis

12. Vasculitis

13. Uveitis

14. Myocarditis

15. Streptococcal toxic syndrome

Common among these are otitis media, pneumonia, septicemia, osteomyelitis, rheumatic fever and acute glomerulo nephritis.

This condition should be differentiated from other viral exanthematous fevers like:

Rubella

Infectious mononucleosis

Enteroviral infections

HIV infections

Streptobacillus moniliformis (rat bite fever)

Diagnosis:

Based mostly on clinical presentation.

Investigations:

Throat / nasal culture

Rapid streptococcal test

Anti deoxyribonuclease B, ASO titres

Antibodies to streptococcal extracellular products

Antihyaluronidase

Antifibrinolysin

Complete blood count: Leukocytosis Eosinophilia up to 20% can occur during the second week.

Urine analysis and liver function tests should be performed

Direct antigen detection kits (rapid antigen tests) - These tests allow immediate diagnosis and prompt administration of antibiotics.

Antideoxyribonuclease B and ASO titres:

These tests are used to confirm previous group A streptococcal infections. An increase in ASO titres may be observed but could be a late finding.

Streptococcal antibody tests provide confirmatory evidence of recent infection. This of course is of no value in acute infection and is not indicated in acute infections. This test could be of value in patients with suspected acute renal failure / acute glomerulonephritis.

Goals of treatment:

1. To prevent acute rheumatic fever

2. To reduce spread of infection

3. To prevent post streptococcal sequel like AGN

4. To prevent other suppurative sequel like adenitis, mastoiditis, ethmoiditis, abscess and cellulitis

5. To shorten the course of illness

Antibiotic therapy should be initiated without delay. Penicillin / amoxicillin are the drug of choice. In the presence of odynophagia, the patient should be admitted for administration of parental antibiotics.

23. Cardinal features of chronic tonsillitis

Duration of complaint of more than 3 weeks duration.

Enlarged tonsil tissue on both sides

Tonsillar enlargement can be graded under 4 groups:

Grade 0: The tonsils are fully inside the pillars.
Grade 1: Tonsils found to be enlarged and out of its pillars
Grade 2: Tonsillar enlargement extends just up to half the distance of the uvula
Grade 3: Tonsillar enlargement up to the level of the uvula.
Grade 4: Tonsillar enlargement is so huge that they are virtually in contact with each other i.e. Kissing tonsil.

Anterior pillar congestion

Enlarged tender jugulodigastric node (Wood's node)

Squeeze test positive

Types of chronic tonsillitis:
Chronic follicular tonsillitis:
In these patients the tonsillar enlargement is associated with the presence of prominent inflamed follicles. Whitish material can be seen extruding from the follicles when the anterior pillars are pressed with a tongue depressor. This is known as the squeeze test. A positive squeeze test always indicates the diagnosis of ch follicular tonsillitis. Inflammation and blockage of crypta magna in these patients lead on to the formation of Quincy or peritonsillar abscess.
Chronic parenchymatous tonsillitis:
In these patients' tonsils are enlarged but the follicles are not prominent. Infection is found within the substance of the tonsil.
Infection in patients with chronic tonsillitis is always poly microbial with a predominance of gram negative and anaerobic organisms. Surgery is commonly indicated in these patients.

24. Tonsillolith

Tonsillolith is a rare dystrophic calcification occurring in the tonsil as a result of chronic inflammation. Most commonly tonsilloliths are Intratonsillar and are asymptomatic. They have been identified incidentally. Commonly patients with tonsillolith complain of foul breath and throat pain. Throat pain is usually very intense during acts of swallowing. Also known as tonsil concretions or tonsillar stones.

Deposition of calcium salts (Calcium phosphate) occurs normally in the skeleton. When calcification occurs in soft tissues in an unorganized fashion it is known as heterotopic calcification. Calcium salts (Calcium phosphate) occurs normally in the skeleton. When calcification occurs in soft tissues in an unorganized fashion it is known as heterotopic calcification. This heterotopic calcification can be further subdivided into three categories:

Metastatic calcification: This calcification occurs in normal tissues due to deposition of calcium. This is the result of higher than normal levels of serum calcium as in the case of hyperparathyroidism or higher levels of serum phosphate as in patients with chronic renal failure. Metastatic calcification usually occurs bilaterally and symmetrically.

Idiopathic calcification: This condition refers to deposition of calcium in normal tissue despite normal serum levels of calcium and phosphate. Examples include chondrocalcinosis and phleboliths.

Dystrophic calcification: Is pathologic and usually occurs in degenerative and dead tissues. This calcification occurs despite normal serum calcium and phosphate levels.

Pathogenesis: Largely remains unknown. It has been commonly attributed to be due to recurrent infections involving the tonsillar tissue. It can also occur due to obstruction to the largest tonsillar crypt (Crypta magna) causing inspissated secretions to accumulate within the crypt causing it to undergo calcification. Another possibility could be due to obstruction to the ducts of accessory salivary glands (Weber's glands) causing it to calcify. This obstruction is possible due to the formation of scar tissue following tonsillectomy or infection.

Clinical features:

1. Commonly asymptomatic

2. Halitosis

3. Pain in the throat

4. Cervical adenopathy usually involving the upper deep cervical nodes

Tonsillolith may occur in any age group. Tonsilloliths are usually hard in consistency, may be single or multiple, may be round or oval, cylindrical or irregular pyramidal shape.

Management:

Usually unilateral tonsillectomy is indicated in these patients. Spontaneous extrusion of tonsilloliths have also been reported.

You can view the surgical clipping from here.

Histopathology:

Microscopic examination of the tonsillolith shows necrotic debris, ghost cells, calcifications and

inflammatory cells.

Composition of tonsillolith:

Usually tonsillolith contains minerals like carbonates and phosphonates of calcium. Other minerals like magnesium, sodium, silica, potassium, ammonia have been reported.

25. Differential diagnosis of membrane on the tonsil

Differential diagnosis of membrane over the tonsil:

The following are different conditions that present with membrane in the tonsillar region.

1. Membranous tonsillitis:

It occurs due to pyogenic organisms. An exudative membrane forms over the medial surface of tonsils along with features of acute tonsillitis.

2. Diphtheria:

Unlike acute tonsillitis which is abrupt in onset, diphtheria is slower in onset with less local discomfort. The membrane in diphtheria extends beyond the tonsils on to the soft palate. The membrane is dirty white in color. This membrane is adherent and its removal would eventually leave a bleeding surface. Urine from these patients would show albumin. Smear and culture from throat swab will reveal corynebacterium diphtheriae.

3. Vincent angina:

This condition is caused by the presence of fusiform bacilli and spirochetes. This condition is insidious in onset with less fever and less discomfort in throat. Membrane forms over one tonsil and can be easily removed revealing an irregular ulcer on the tonsil. Throat swab will reveal the two organisms.

4. Infectious mononucleosis:

This condition affects young adults. Both tonsils are enlarged, congested and covered with membrane. There is also marked local discomfort. Lymph nodes in the posterior triangle of neck is enlarged and there is also splenomegaly. This disease is suspected because of failure of antibiotic treatment. Blood smear may demonstrate more than 50% lymphocytes of which about 10% are atypical. Paul Bunnel test is positive.

5. Agranulocytosis:

This condition presents with ulcerative necrotic lesions on the tonsils and oropharynx. These patients are severely ill. In its acute fulminating form the total leukocyte count is decreased to less than 2000/cubic mm. This could even to down to as low as 50/cubic mm.

6. Leukemia:

This condition also causes membranes to appear over tonsil. 75% of leukemias in children are acute lymphoblastic and 25% acute myelogenous. In adults 20% of acute leukemias are lymphocytic and 80% nonlymphocytic.

7. Aphthous ulcers:

This condition may involve any portion of the oral cavity or oropharynx. Sometimes it is solitary and may involve tonsils and their pillars. It may be small or large and could cause lot of pain.

8. Malignancy tonsil

10. Candida infection of tonsil

26. Mention the causes for a white patch over the tonsil

Differential diagnosis of membrane over the tonsil:

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27. Complications of tonsillectomy

Complications can be classified in to immediate, intermediate and delayed.

Immediate complications:

Mostly encountered on the table during surgery. The most common of them being the complications of general anaesthesia. Next is troublesome intra operative bleeding. This is common in poorly prepared tonsillectomies (i.e. patients who have been taken up for surgery without a pre op course of antibiotics), hot tonsillectomy (i.e. quinsy tonsillectomy). Bleeding can be controlled by proper dissection, staying in the correct plane (i.e. sub capsular plane) during dissection, ligation of bleeders, using bipolar cautery to coagulate the bleeding vessels.

Trauma to the anterior and posterior pillars. Trauma to posterior pillar causes nasal regrugitatin whenever the patient attempts to drink fluids after surgery. It may also cause undesirable changes in the voice i.e. Rhinolalia aperta.

Teeth must be taken care when mouth gag is bing applied. Any loose tooth, dentures must be removed before intubation because the loose teeth can easily be dislodged and be aspirated.

Trauma to the lips and gums: can be avoided by using the right sized tongue blade. The size of the blade can be measured by placing it between the mentum and the angle of the mandible.

Intermediate complications:

Are mostly haemorrhage. Haemorrhage during immediate post op period is also known as reactionary haemorrhage. This is caused due to

1. Wearing off of the hypotensive effect of the anaesthesia during the immediate post op period.

2. Slipping of ligature

These patients must be taken to the operation theatre, reanaesthetised and the bleeders must be ligated or cauterised.

If bleeding is diffuse and uncontrollable pillar suturing can be resorted to. This is done by suturing both the anterior and posterior pillars after placing a gauze or gelfoam in the tonsillar fossa. If gauze is used to pack the tonsillar fossa, silk is used to suture the pillars and these sutures must be removed after 48 hours and the gauze is removed. On the other hand, if absorbable material like gel foam is used the pillars can be sutured with chromic cat gut and the sutures need not be removed.

Delayed complications:

Are mostly due to infections. These commonly occur a week after the surgery. Bleeding during this period is known as secondary hemorrhage. Antibiotics are used to control infections.

28. Premalignant conditions of oral cavity

Premalignant Lesions

Leukoplakia

The term leukoplakia describes a white patch or plaque that cannot be characterized clinically or pathologically as any other disease. The precise definition of leukoplakia continues to undergo refinement in an attempt to distinguish benign from premalignant lesions, and leukoplakia remains a clinical diagnosis of exclusion. Leukoplakia occurs most often in middle-aged and older men and arises most frequently on the buccal mucosa, alveolar mucosa, and lower lip.

However, note that lesions arising on the floor of mouth, lateral tongue, and lower lip are the most likely to harbor dysplasia or progress to malignancy. The rate of progression to malignancy has been reported to be between 3.6% and 17.5%, and as many as 19.9% of leukoplakic lesions may demonstrate some degree of dysplasia, with 3.1% showing frank carcinoma.

A literature review by Warnakulasuriya and Ariyawardana suggested that risk factors for malignant transformation of oral leukoplakia include advanced age, female sex, lesions of more than 200 mm2, nonhomogeneous lesions, and higher-grade dysplasia. Please see the Medscape Drugs & Diseases article Oral Leukoplakia for further discussion.

Erythroplakia

Erythroplakia is a clinical term used to describe a fiery red patch that cannot be clinically or pathologically distinguished as any other definable disease. Similar to leukoplakia, the erythroplakic lesion is considered as a diagnosis of exclusion because numerous other disease entities must be excluded before erythroplakia is considered as the diagnosis. The clinical appearance of erythroplakia is described as a red macule or patch with a soft, velvety texture most often occurring on the floor of mouth, lateral tongue, retromolar pad, and soft palate.

Although far less common than leukoplakia, erythroplakia is a worrisome clinical condition that often harbors dysplasia. Upon histological analysis, 51% of erythroplakic lesions have been shown to demonstrate invasive squamous cell carcinoma (SCC), with 40% demonstrating carcinoma in situ, and 9% exhibiting mild-moderate dysplasia.

Proliferative verrucous leukoplakia

Proliferative verrucous leukoplakia (PVL) is a unique form of aggressive disease considered to be within the continuum of leukoplakia and erythroplakia. [29] Most patients with PVL are women, and many do not have a history of tobacco use. PVL generally appears on the oral mucosa as an irregular white patch or plaque with a varying surface. The disease is often characterized by resistance to treatment, recurrence, multifocal proliferation, and a progression to carcinoma in up to 87% of patients.

Palatal lesion of reverse smokers

The palatal lesion of reverse smokers is unique to individuals who place the lit end of a cigarette inside the mouth. The resulting palatal lesion may appear clinically as a red, white, melanotic patch or papule. Up to 84% of palatal lesions have been demonstrated to harbor dysplasia upon histologic analysis.

Oral submucous fibrosis

Oral submucous fibrosis (OSF) is a chronic progressive condition found predominantly in people of Asian decent. OSF is considered to be the result of the use of the Areca nut product with resultant disruption of the extracellular matrix. The disease often manifests with diffuse involvement of the oral cavity, pharynx, and upper esophagus that appears clinically as whitish mucosa lacking elasticity. Epithelial dysplasia has been described in 7-26% of OSF tissues, and long-term studies suggest a malignant transformation rate in approximately 7% of these lesions.

Lichen planus, discoid lupus erythematous, and epidermolysis bullosa

Although classified as potentially malignant conditions, the data regarding progression to malignancy for these conditions is controversial. Because of the difficulty in classifying and clinically distinguishing the varied lesions associated with these conditions, the potential for malignant transformation remains unclear.

29. Leukoplakia.

Leukoplakia
The term leukoplakia describes a white patch or plaque that cannot be characterized clinically or pathologically as any other disease. The precise definition of leukoplakia continues to undergo refinement in an attempt to distinguish benign from premalignant lesions, and leukoplakia remains a clinical diagnosis of exclusion. Leukoplakia occurs most often in middle-aged and older men and arises most frequently on the buccal mucosa, alveolar mucosa, and lower lip.

However, note that lesions arising on the floor of mouth, lateral tongue, and lower lip are the most likely to harbor dysplasia or progress to malignancy. The rate of progression to malignancy has been reported to be between 3.6% and 17.5%, and as many as 19.9% of leukoplakic lesions may demonstrate some degree of dysplasia, with 3.1% showing frank carcinoma.

Erythroplakia
Erythroplakia is a clinical term used to describe a fiery red patch that cannot be clinically or pathologically distinguished as any other definable disease. Similar to leukoplakia, the erythroplakic lesion is considered as a diagnosis of exclusion because numerous other disease entities must be excluded before erythroplakia is considered as the diagnosis. The clinical appearance of erythroplakia is described as a red macule or patch with a soft, velvety texture most often occurring on the floor of mouth, lateral tongue, retromolar pad, and soft palate.

Although far less common than leukoplakia, erythroplakia is a worrisome clinical condition that often harbors dysplasia. Upon histological analysis, 51% of erythroplakic lesions have been shown to demonstrate invasive squamous cell carcinoma (SCC), with 40% demonstrating carcinoma in situ, and 9% exhibiting mild-moderate dysplasia.

Proliferative verrucous leukoplakia
Proliferative verrucous leukoplakia (PVL) is a unique form of aggressive disease considered to be within the continuum of leukoplakia and erythroplakia. Most patients with PVL are women, and many do not have a history of tobacco use. PVL generally appears on the oral mucosa as an irregular white patch or plaque with a varying surface. The disease is often characterized by resistance to treatment, recurrence, multifocal proliferation, and a progression to carcinoma in up to 87% of patients.

30. Leucoplakia of palate

Leukoplakia of soft palate:

Is a whitish patch seen over the soft palate. This patch cannot be easily scrapped off.

Histology:

Leukoplakia demonstrates a thickened surface layer of parakeratin, sometimes orthokeratin. Basilar cells and keratinocytes in the lower portions of the epithelium usually show no evidence of dysplasia, besides a mild basilar hyperplasia, but about 10% of cases will be dysplastic and these have an elevated risk of malignant transformation.

Causative factors:

1. Smoking with the burning end of cigar inside the mouth

2. Tobacco chewing

3. Ill fitting dentures

4. Ultraviolet radiation

5. Presence of torus palatinus

6. Alcoholism

Predominantly common in males.

Leukoplakias begin as thin gray or gray/white plaques which may appear somewhat translucent, are sometimes fissured or wrinkled, and are typically soft and flat. They usually have sharply demarcated borders but occasionally blend gradually into normal mucosa..

When leukoplakia becomes red, it is known as erythroplakia. This is again a premalignant condition.

Staging of leukoplakia:

Phase I leukoplakia: is very thin patch of leukoplakia. The lesion is so thin that the underlying mucosa can be clearly seen. This stage of leukoplakia can regress in due course of time.

Phase II leukoplakia: These patches are homogeneous or thick, smooth, perhaps fissured. Leukoplakia can remain in this stage indefinitely or it can progress to phase III.

Phase III leukoplakia: These patches have surface irregularities of a nodular or granular nature, hence are referred to as granular or nodular leukoplakia. Phase III leukoplakias may become dysplastic, even invasive, with no change whatsoever in the clinical appearance.

Phase IV leukoplakia: Are also known as erythroleukoplakia, speckled leukoplakia, nonhomogeneous leukoplakia. These lesions are the ones that may undergo malignant transformation.

31. CROUP

This clinical syndrome is characterized by Hoarseness of voice, stridor (inspiratory / biphasic), barking cough.

This is caused by mucosal oedema of larynx and trachea. These patients will give h/o symptoms of upper respiratory tract infections associated with fever and malaise. This condition is classically caused by parainfluenza type I virus. Other viruses that can cause this condition include Parainfluenza type II, Respiratory syncytial virus and influenza A and B viruses.

Children between 6 months and 3 years of age are affected. Peak incidence occurs in 2 year old infants.

This is actually a self limiting disease and most of the children would improve within the first 24 hours of illness. Complete recovery occurs within 4 days even without treatment.

Acute air way obstruction would need hospital admission. If the affected children have coexistent bronchopneumonia / Measles prognosis is really poor.

Investigations:

X-ray chest PA view is diagnostic. Characteristic narrowing could be seen at the level of subglottis. This narrowing is seen as a Steeple / pencil tip.

Stridor in these patients is caused by oedema of subglottic region. This region is the narrowest portion of a child's airway.

Some children are more prone for complications than others. Children with pre-existing tracheal narrowing / chronic lung disease / BA are at risk. In infants with recurrent croup congenital / acquired subglottic stenosis should be considered.

Westley croup score: This allows the severity of symptoms to be classified. Maximum score possible is 17. A score of 2-3 indicates mild croup, a score of 4-7 indicate moderate croup and a score of above 8 indicates severe croup.

Treatment:

In patients with mild croup it is sufficient if supportive therapy is given. The child should not be sedated as this will reduce the respiratory drive. In moderately severe cases, the patient can be nebulized with epinephrine (1 ml of i in 1000 epinephrine which is diluted with 3 ml of 0.9% saline). Epinephrine is known to cause reduction of mucosal oedema.

Corticosteroids should be administered in patients with severe croup. Oral dexamethazone is administered in doses of 0.6mg/kg.

Budesonide 2mg may be used as nebulization.

Oxygenation can be provided with face mask in moderate croup cases. In patients with severe degree of croup with altered sensorium intubation may have to be resorted to ventilate the patient.

32. Acute Epiglottitis (Acute supraglottitis)

This is an inflammation involving the epiglottis. This condition is commonly seen in children. It is commonly caused by H. Influenza. In adults it can be caused by allergy, trauma to epiglottis due to foreign body, GERD etc.

Clinical features:

1. Excessive throat pain

2. Hot potato voice

3. Difficulty in swallowing

4. Difficulty in breathing is common in children. These patients may need tracheostomy.

Indirect laryngoscopic examination shows: Oedematous reddish epiglottis. The aryepiglottic folds may also appear edematous.

Treatment:

1. Rest

2. Voice rest

3. Antibiotics

4. Systemic steroids (Hydrocortisone in doses of 100 mg 6th hourly can be administered)

5. Tracheostomy if there is respiratory distress

33. Benign tumors of the vocal folds

The following conditions are included under Benign vocal fold mucosal disorders:

1. Vocal nodules

2. Laryngeal polyps

3. Mucosal hemorrhage

4. Intracordal cysts

5. Mucosal bridges

6. Glottic sulci

If the affected patient happens to be a singer, they may seek professional help because of voice limitation during singing, usually in the upper range. If the patient is a non singer then the patient may seek help with a little advanced lesion with a change in sound capabilities of the speaking voice.

34. Laryngeal papilloma

Papilloma larynx are glistening whitish irregular mass seen in the larynx. It involves the vocal cords, false cords, and epiglottis. These masses are friable and bleed on touch.

It occurs in two forms: 1. Juvenile papilloma and 2. Adult papilloma.

Juvenile papilloma: Occurs in infants and young children. It is multiple, and show aggressive behavior. These lesions are known to recur even after successful surgery. It is caused by Human papilloma virus type 6 or type 1. The affected children are known to get their infection from infected mother's genitals during delivery.

These lesions appear as multiple white glistening irregular, friable masses over the true / false vocal cords. It can also involve epiglottis. These lesions have a predilection to involve the squamocolumnar junction. They show a very aggressive behavior. Recurrence even after successful surgical removal is common.

The affected patients have hoarseness of voice. The child may have difficulty especially while crying. If the mass reaches a size large enough to obstruct the laryngeal inlet, patients develop stridor.

Adult papilloma: Is solitary in nature, known to involve the true vocal cord at the junction of the anterior 1/3 and posterior 2/3. This is the mobile portion of the vocal cord. Males are affected twice as common as females. These lesions are not aggressive and hence surgical management is always successful.

Management:

Tracheostomy: to secure the airway if the patient manifests with stridor.

Microlaryngeal excision of the masses - Bleeding and airway complications are common during this procedure.

Cryosurgery - Bleeding is minimized during this procedure

Laser surgery - Is best suited for juvenile papillomas. In this procedure the bleeding is minimal, and scarring is also minimal.

Recurrence - In juvenile papilloma can be reduced by postoperative treatment with interferon. Herbal drung (TUJA) has been tried with varying degrees of success.

35. Vocal cord nodule /Singers Nodules

This disorder frequently affects children and adults. In children it appears as spindle shaped thickenings of the edges of the vocal cords, whereas in adults they appear as more localised thickenings, varying from small points - nodules. These nodules typically appear at the junction of the anterior and middle 1/3 of the vocal cords. They appear almost aways symmetrically.

Pathophysiology:

Are caused by a combination of overtaxing and incorrect use of the voice. This is also aggravated by the presence of infections in the para nasal sinuses, tonsils, and adenoids. Patients with habitual dysphonia frequently encounter this condition. This condition can be effectively prevented or cured by voice rest or by using the voice properly. Infact the nodules can appear and disappear in a matter of weeks. If the aggravating factors persist for a long time then these nodules become permanent.

Stages of vocal nodule formation:

Stage of transudation:

Oedema occurs in the submucosal plane in this stage. This occur during the acute phase of the disorder. This stage is reversible in nature and may become normal on giving voice rest.

Stage of ingrowth of vessels:

In this stage neovascularization of the area occur. This phase is also reversible, but takes a long time to become normal.

Stage of fibrous organisation:

In this stage the transudate in the submucosal area is replaced by fibrinous material. This stage is more or less resistant to conservative line of management.

These stages can be clearly observed by laryngoscopy under stroboscopic light. Local oedematous swelling of recent onset vibrates in phase with the whole vocal fold, whereas an older and more fibrous swelling can impede the vibrations so much that only a part of the cord is seen to vibrate. The improvement in the vibration pattern or signs of recovery are picked up early during stroboscopic examination.

Clinical features:

1. Change in voice

2. Fatiguability of voice

3. Decreased pitch range

Management:

1. Voice rest plays a sheet anchor role in the management of vocal nodule. This may range from complete voice rest to partial rest.

2. Speech therapy will help patients with habitual dysphonia from developing vocal nodule.

3. Treatment of sinus infections, tonsillitis and adenoiditis must not be overlooked.

4. If the vocal nodule become permanent then microlaryngeal removal is advocated.

36. Functional aphonia

Functional aphonia is defined as loss of speech suffered by the patient without any attributable organic cause. Patient afflicted by this condition suddenly loses voice / sometimes voice gets reduced to a whisper. These patients don't suffer from hoarseness of voice.

Aetiology:

This condition is caused by sudden psychological trauma / emotional shock.

Affected patients are mostly more than 15 years of age.

Females are commonly affected.

Symptoms:

Aphonia: Patient suddenly loses voice / sometimes voice gets reduced to a whisper. There is no hoarseness of voice. Importantly these patients are able to cough normally.

These patients normally have associated psychological problems. These patients may have pain in the neck.

On examination:

Vocal cords are normal. On phonation they don't come into contact with each other. These cords normally adduct during coughing.

This condition should be differentiated from other organic causes of aphonia.

Treatment:

1. Reassurance

2. Psychotherapy

3. Speech therapy

37. Retro pharyngeal abscess

Already discussed.

38. Vocal cord paralysis

Vocal cord paralysis is caused by paralysis of intrinsic muscles of larynx. This is a symptom of an underlying disorder and not a disease by itself. The intrinsic muscles of the vocal cord are supplied by the vagus nerve. The term vagus means "wanderer" which is the apt term to describe this nerve because of its long anatomical course.

Unilateral vocal fold paralysis occurs due to dysfunction of recurrent laryngeal or vagus nerve causes a breathy voice. The breathiness of voice is caused by glottic chink which allows air to escape when the patient attempts to speak. Normal voice production is dependent on proper glottal closure resulting from bilateral adduction of the vocal cords. This adduction of vocal folds combined with subglottic air pressure causes the vocal folds to vibrate causing phonation.

Pathophysiology of vocal cord paralysis:

The physiologic function of larynx is adversely affected by vocal fold paralysis. Interference with the protection of the tracheobronchial tree and respiration are more serious and life threatening than interference with voice production. In recurrent laryngeal nerve paralysis the vocal folds may assume a number of positions. Six positions have been described. They are median, paramedian, cadaveric (intermediate), gentle abduction and full abduction. The various positions of the vocal cords cannot be recorded precisely.
Various theories have been proposed to explain the various positions assumed by a paralysed vocal cord.

Semon's law: This theory proposed by Rosenbach and Semon in 1881, depends on the concept that abductor fibres in the recurrent laryngeal nerves are more susceptible to pressure than the adductor fibers. After a number of amendments this law is stated as " In the course of a gradually progressing organic lesion involving the recurrent laryngeal nerve three stages can be observed. In the first stage only the abductor fibers are damaged, the vocal folds approximate in the midline and adduction is still possible. In the second stage the additional contracture of adductors occur so that the vocal folds are immobilized in the median position. In the third stage the adductors become paralysed and the vocal folds assume a cadaveric position".

This theory is fraught with clinical and experimental inconsistencies. It was assumed that the nerve fibers supplying the abductors of the vocal folds lie in the periphery of the recurrent laryngeal nerve and any progressive lesion involves these fibers first before involving the deeper fibers that supply the adductors. It was even suggested that adductors being phylogentically older are more resistant to insults than the newer abductors. According to this theory in all progressive lesions involving the recurrent laryngeal nerve the abductors paralyze first followed by the adductors.

When recovery takes place the first muscle group to recover will be the adductors before the abductors could recover.

Differential innervation theory: This theory was based on the anatomic fact that the recurrent laryngeal nerve often branched outside the larynx. Injury to individual branches could cause paralysis of specific groups of muscles accounting for the varying positions assumed by the paralysed cord.

Changes in the cricoarytenoid joint and paralysed muscles: These changes have been proposed to explain the position of the cord in vocal fold paralysis. This theory of progressive fibrosis of muscles has no anatomical proof.

Interarytenoid muscle contraction: In this theory the paramedian position of a paralysed vocal cord is attributed to contraction of interarytenoid muscle which is supposed to receive bilateral innervation. In reality this is not true as the interarytenoid muscle just helps to close the posterior glottic chink.

Disturbance of autonomic supply: This theory has no experimental evidence. It suggests that the vocal cord position is determined by the laryngeal muscle tone due to autonomic innervation.
Wagner and Grossman theory: This is the most popular and widely accepted theory which could account for the varying positions assumed by a paralysed vocal cord. This theory was first proposed by Wagner and Grossman (1897). This theory states that in complete paralysis of recurrent laryngeal nerve the cord lies in the paramedian position because the intact cricothyroid muscle adducts the cord. (Because the superior laryngeal nerve is intact). If the superior laryngeal nerve is also paralysed the cord will assume an intermediate position because of the loss of adductive force. This theory has been confirmed by electro myological studies.
According to this theory, chest lesions should cause recurrent laryngeal nerve paralysis alone, but in many patients with lung cancer the cord assumes a intermediate position. This has been attributed to the phenomenon of retrograde atrophy of the vagus nerve up to the level of nucleus ambiguus.
Paralysed vocal cords may demonstrate some movement due to the action of interarytenoid muscle which gets bilaterally innervated.

Pathogenesis of vocal cord paralysis:

Vocal cord paralysis is a sign of disease and is not a diagnosis. It may be due to a lesion anywhere from the cerebral cortex to the neuromuscular junction. Because of the large size of the nucleus ambiguus, small lesions in it may produce isolated laryngeal and pharyngeal motor losses. Lesions involving the nucleus ambiguus may cause bilateral paralysis more often than unilateral palsy.
Peripheral damage to the laryngeal innervation may be of three types:

1. Damage to the vagus trunk above the nodose ganglion, the origin of superior laryngeal nerve

2. Damage to the vagus nerve below the level or to the recurrent laryngeal nerve

3. Damage to the superior laryngeal nerve alone.

Vocal cord paralysis may be congenital or acquired.

Congenital vocal cord palsy: Many infants with stridor may have congenital paralysis of vocal cords. This could occur with or without other associated abnormalities (i.e. neurologic, laryngeal and cardiac defects). The most commonly associated anomaly in these patients is the presence of hydrocephalus. The mechanism of vocal cord palsy in these children are still not clear. It could be due to stretching of the vagus nerve, due to complicated delivery etc.

Acquired causes of vocal cord palsy:

Malignant disease: One third of all vocal cord paralysis are caused by malignancies like lung cancer, oesophageal cancer and thyroid malignancies. Other rare causes could include temporal lobe malignancies, posterior fossa tumors, paragangliomas etc.

Surgical trauma: is the second commonest cause of vocal cord paralysis. Thyroid surgeries are the commonest. Mediastinal surgeries, oesophageal surgeries can also cause vocal cord palsy.

Nonsurgical trauma: Injuries to neck caused by automobile accidents and penetrating neck injuries can cause vocal cord palsy.

Inflammatory causes: By far the most common cause is tuberculosis. This could be due to apical scarring of the mediastinum or enlargement of hilar nodes. Other rare causes include jugular vein thrombophlebitis following csom, subacute thryoiditis, meningitis both viral and bacterial.

Neurologic causes: Include brain stem ischemia, multiple sclerosis and head injuries.

Miscellaneous causes: include hemolytic anemia, thrombosis of subclavian vein, syphilis, collagen disorders, lead and arsenic poisoning.

Idiopathic causes: A major chunk of the recurrent laryngeal nerve paralysis fall under this group where in no demonstrable abnormality could be attributed to recurrent larygneal nerve paralysis. Left vocal cord is commonly involved in these patients. Many of the idiopathic recurrent laryngeal nerve paralysis is caused by viral infections (subclinical). Recovery is common in these patients.

Clinical features:

Unilateral superior laryngeal nerve injury:

These patients have very slight voice change. Patients may even complain of hoarseness of voice. Singers find it difficult to maintain the pitch. Diplophonia is common in these patients (defect in the production of double vocal sounds). The pitch range is decreased in these patients. This is due to the fact that cricothryoid muscle is very important in maintenance of vocal cord tension and this muscle is supplied by the superior laryngeal nerve.

On indirect laryngoscopy examination the vocal folds appear normal during quiet respiration. There could be seen a deviation of the posterior commissure to the paralysed side. The posterior commissure points towards the side of the paralysis. At rest the paralysed vocal fold is slightly shortened and bowed and may lie at a lower level than the opposite cord.

There is also associated loss of sensation in the supraglottic area causing subtle symptoms like frequent throat clearing, paroxysmal coughing, voice fatigue and foreign body sensation in the throat.
Bilateral superior laryngeal nerve injury: Fortunately, this condition is very rare. It could result in fatal aspiration and pneumonia. This condition is in fact difficult to diagnose as there is no asymmetry between the vocal folds.

Unilateral recurrent laryngeal nerve injury: Is the most common situation encountered. Left cord is affected commonly than the right as the left vagus nerve takes a more tortuous course. To start with the voice is breathy, but the normal vocal cord starts to compensate soon. The air way is adequate and there is no stridor in these patients. On indirect laryngoscopic examination the affected cord could assume any of the 6 positions described above. The cord may appear not to move, while the opposite cord will compensate for the lack of mobility. When right vocal cord is paralysed then tuberculosis or bronchial malignancies should be considered to be a possibility. Left vocal cord is involved in oesophageal malignancies, and in viral infections.

Unilateral superior and recurrent laryngeal nerve injury: This occurs usually in high vagal or brain stem lesions. Vocal folds are in intermediate position and the patient tends to have a breathy voice. There is also a tendency to aspirate.

Bilateral recurrent laryngeal nerve palsy: In this condition both cords assume a paramedian position compromising the airway. This commonly occurs following total thyroidectomy or in thyroid malignancies. The patient will commonly manifest with stridor. The voice will be near normal.

Bilateral superior and recurrent laryngeal nerve injury: Bilateral vocal cords are intermediate, flaccid, and motionless. The patient experiences aphonia and is at high risk for aspiration.

Evaluation:

The standard diagnostic workup and evaluation of a patient with vocal cord paralysis of unknown etiology is as follows: CXR, cervical spine series, barium swallow, thyroid scan, CT or MRI of head,

neck, and possibly thorax, CBC, Thyroid function tests, ESR, Rheumatoid factor, Parathyroid hormone, calcium and glucose levels, PPD, VDRL, fungal titers, lyme titers, and possibly a lumbar puncture.
Another adjuvant diagnostic aid to be considered is laryngeal electromyography. Described by Miller et al in 1982, this method of evaluation of laryngeal muscle innervation is gradually gaining acceptance by otolaryngologists. It is an analysis of the electrical activity generated by a motor unit. It is performed percutaneously, under local anesthesia on the cricothyroid muscles and thyroarytenoid muscles to test both the superior laryngeal nerve and recurrent laryngeal nerve, respectively. Miller, et al claims that laryngeal EMG is the most accurate method of determining superior laryngeal nerve paralysis. It also appears to
be helpful in cases of mechanical fixation of the cords and predicting outcome of certain cases of paralysis.

Imaging has a very important role to play in the evaluation of causes for vocal cord palsy. CT scan and MRI imaging of neck and thorax to rule out lesions that could involve the recurrent laryngeal nerves in these areas must be performed.

Management:

The voice of the patient should be recorded, since the major complaint is going to be hoarseness of voice.

Indications of early intervention include:

1. Life threatening aspiration

2. A known etiology which leaves no chance of recovery

3. Psychological and professional factors (relevant in singers)

Ideally speaking a wait and watch approach is useful in patients with unilateral idiopathic paralysis of vocal cords. Spontaneous recovery is the order of the day in these patients. The wait period may last up to 6 months in some patients.

Treatment for unilateral paralysis of vocal cord include:

1. Speech therapy

2. Surgical medialisation of the paralyzed cord

3. Intracordal injections

4. Selective reinnervation

Speech therapy: Can be used alone or in conjunction with other surgical modalities of management.

Surgical medialization:

Is currently the accepted modality of management for all cases of refractory unilateral paralysis of vocal cords.
This is currently the procedure of choice for most cases of unrecovered or uncompensated unilateral vocal cord paralysis.
Laryngeal framework surgery was first introduced by Payr in 1915 with the development of a thyroid cartilage flap. This failed to provide enough medialization and further developments were not introduced until the 1950's. Several authors then introduced different modifications but the procedure did not become popular until the late 1970's when Isshiki introduced his thyroplasty technique. This involved displacing and stabilizing a rectangular, cartilaginous window at the level of the vocal cord, therefore pushing the soft tissue medially. This technique gained wider acceptance after Isshiki reported the successful use of Silastic as the implant material. Silastic works very well because it is easier to carve than cartilage and can be tailor made for each patient.

The technique is performed under local anesthesia to allow the patient to phonate during the procedure. Thus, the degree of medialization can be determined immediately, intraoperatively by the quality of the patient's voice. A horizontal skin incision is made overlying the mid-thyroid ala. A window is made in the thyroid cartilage on the involved side corresponding with the level of the true vocal cord. The Silastic implant is then carved (many different modifications) to approximate the defect. A subperichondrial window is made in the endolarynx, and the Silastic implant is inserted into the window. The implant is fashioned so that is it wedged in place, therefore no suturing is required. The quality of the patient's voice is checked and glottic closure can be assessed using flexible endoscopy. If the desired voice is not obtained, or the airway is impaired, the implant can easily be removed and another redesigned.

Complications:

1. Airway compromise
2. Wound infection
3. Hematoma
4. Extrusion of the implant
5. Laryngocutaneous fistula formation

Advantages of surgical medialisation procedure:

1. Reversible
2. Can be effective even with fixed cord
3. The patient has immediate benefit

Disadvantages:

1. Skin incision

2. Edema can distort glottic defect

3. Results variable

4. Posterior commissure closure is not adequate

An adjuvant procedure to surgical medialization, also described by Isshiki, is arytenoid adduction. This procedure can help close the posterior glottic chink that medialization alone often fails to do. This procedure can be performed alone, or in combination with medialization. This procedure can produce excellent results, especially in patients with combined superior and recurrent laryngeal nerve paralysis (hence, an intermediate cord), however it is irreversible, technically difficult, and has a relatively high rate of complications (33% in one study). It should be reserved for surgeons experienced in laryngoplastic phonosurgery.

Intracordal injections:

Intracordal injection of polytetrafluoroethylene (Teflon), popularized in the 1960's, is still performed by some in the treatment of uncompensated unilateral vocal cord paralysis. Gelfoam paste may be used instead if the paralysis is thought to be temporary. Collagen has also been introduced as a potential substitute for Teflon. The technique is best performed under local anesthesia, when possible, as this allows for intraoperative evaluation of the patient's voice. Voice quality improvement during the procedure is an important guide to the location and amount of paste injected.

First, the pharynx and larynx are anesthetized. An anterior commissure laryngoscope is then used to visualize the cords and, by rotating the tip toward the paralyzed cord, displace the false cord so that as much of the true cord as possible is exposed. A Brunings syringe in then used to inject the paste. The tip of the needle should be placed between the vocal process of the arytenoid and the posterior aspect of the thyroid ala. The needle should be inserted approximately 5 mm and enough paste injected until the cord approaches midline. The patient is asked to say "E". If further improvement is needed, another injection is made. It is usually necessary to repeat the process 2-3 times. Voice improvement can be dramatic, but can be variable due to edema.
Since Teflon cannot be removed easily, it is always better to inject too little than too much. Gelfoam paste is injected in the same manner, but will gradually absorb over 1-3 months.

Complications:

1. Airway edema

2. Granuloma formation

3. Results not predictable

Advantages:

1. No skin incision required

2. Out patient procedure

3. Results satisfactory in majority of cases

Selective reinnervation:

Nerve-Muscle Transfer:

Originally described by Tucker in 1977, this procedure uses a branch of the ansa hypoglossi attached to a small block of omohyoid muscle as a nerve-muscle pedicle to innervate the thyroarytenoid muscle on the involved side. The procedure is based on the strap muscles being accessory muscles of respiration. Prerequisite to reinnervation is a mobile cricothyroid joint, and that the cause of the paralysis has not left the ansa hypoglossi denervated as well.

The technique is performed under local or general anesthesia. A lateral neck-crease incision is made approximating the lower border of the thyroid cartilage. The ansa hypoglossi is identified as it lies on the jugular vein. It is traced to its point of entry into the anterior belly of the omohyoid muscle. A free block (approximately 2-3mm on a side) of muscle from the omohyoid is excised, including the point of entry of the nerve. A window is created in the thyroid ala exposing the thyroarytenoid muscle.
The nerve-muscle pedicle is then sutured to this muscle. The incision is closed after placement of a penrose drain.

The results of this procedure have been very good. Tucker reports an 80% success rate, and other authors (May and Beery) have reported similar results. Granted, there is a delay, usually 2-6 months before voice improvement begins.

This procedure can be combined with surgical medialization for immediate improvement of voice quality. The surgical exposure is similar to that necessary for thyroplasty. The combined procedure should be performed under local anesthesia.

Advantages:

1. The vocal fold is medialised without resorting to any implants

2. Better pitch control

3. Other methods can be attempted even if this fails

Disadvantages:

1. Skin incision

2. Prolonged wound healing

Management of bilateral vocal cord palsy:

The initial aim is to secure the airway as these patients will manifest withs stridor. A tracheostomy should be performed on an immediate basis.

Vocal cord lateralisation procedures:

This involves several techniques that surgically widen the glottic opening. While this improves the airway, the patient's voice quality suffers. The three most commonly utilized techniques are arytenoidectomy, arytenoidopexy, and cordectomy.

Arytenoidectomy:

Classic arytenoidectomy involves removal of some or all of the arytenoid cartilage. This procedure can be performed in a variety of ways, from endoscopically by microsurgical or laser technique to an external, lateral neck approach (Woodman). The Woodman procedure seems to be a popular choice. This involves a lateral neck incision, exposure of the arytenoid cartilage posteriorly with removal of the majority of the cartilage, sparing the vocal process. A suture is then placed into the remnant of vocal process and fixed to the lateral thyroid ala. This technique seems to cause less voice deficit than other approaches.

Arytenoidopexy:

Involves displacing the vocal fold and arytenoid without surgical removal of any tissue. It can be done endoscopically with a suture passed around the vocal process of the arytenoid and secured laterally. This procedure, however, has a relatively high failure rate.

Cordectomy:

Dennis and Kashima (1989) introduced the posterior partial cordectomy procedure using the carbon dioxide laser. This involves excising a C-shaped wedge from the posterior edge of one vocal cord. If this posterior opening is not adequate after 6-8 weeks,
the procedure can be repeated or a small cordectomy can be performed on the other vocal cord. They claim relief of airway
obstruction with preservation of voice quality.

Reinnervation:

Tucker proposed a nerve-muscle transfer to the posterior cricoarytenoid muscle for the treatment of bilateral vocal cord paralysis. The technique is similar to the one used for unilateral vocal cord paralysis. Prerequisites are that the cricothyroid
joint not be fixed and that the necessary nerve for the graft not have been affected by the process that caused the paralysis.
Tucker reports a high success rate.

39. Vocal cord paralysis-Theories on position

Various theories have been proposed to explain the various positions assumed by a paralysed vocal cord.

Semon's law: This theory proposed by Rosenbach and Semon in 1881, depends on the concept that abductor fibres in the recurrent laryngeal nerves are more susceptible to pressure than the adductor fibers. After a number of amendments this law is stated as " In the course of a gradually progressing organic lesion involving the recurrent laryngeal nerve three stages can be observed. In the first stage only the abductor fibers are damaged, the vocal folds approximate in the midline and adduction is still possible. In the second stage the additional contracture of adductors occur so that the vocal folds are immobilized in the median position. In the third stage the adductors become paralysed and the vocal folds assume a cadaveric position".

This theory is fraught with clinical and experimental inconsistencies. It was assumed that the nerve fibers supplying the abductors of the vocal folds lie in the periphery of the recurrent laryngeal nerve and any progressive lesion involves these fibers first before involving the deeper fibers that supply the adductors. It was even suggested that adductors being phylogentically older are more resistant to insults than the newer abductors. According to this theory in all progressive lesions involving the recurrent laryngeal nerve the abductors paralyze first followed by the adductors. When recovery takes place the first muscle group to recover will be the adductors before the abductors could recover.

Differential innervation theory: This theory was based on the anatomic fact that the recurrent laryngeal nerve often branched outside the larynx. Injury to individual branches could cause paralysis of specific groups of muscles accounting for the varying positions assumed by the paralysed cord.

Changes in the cricoarytenoid joint and paralysed muscles: These changes have been proposed to explain the position of the cord in vocal fold paralysis. This theory of progressive fibrosis of muscles has no anatomical proof.

Interarytenoid muscle contraction: In this theory the paramedian position of a paralysed vocal cord is attributed to contraction of interarytenoid muscle which is supposed to receive bilateral innervation. In reality this is not true as the interarytenoid muscle just helps to close the posterior glottic chink.

Disturbance of autonomic supply: This theory has no experimental evidence. It suggests that the vocal cord position is determined by the laryngeal muscle tone due to autonomic innervation.
Wagner and Grossman theory: This is the most popular and widely accepted theory which could account for the varying positions assumed by a paralysed vocal cord. This theory was first proposed by Wagner and Grossman (1897). This theory states that in complete paralysis of recurrent laryngeal nerve the cord lies in the paramedian position because the intact cricothyroid muscle adducts the cord. (Because the superior laryngeal nerve is intact). If the superior laryngeal nerve is also paralysed the cord will assume an intermediate position because of the loss of adductive force. This theory has been confirmed by electro myological studies.
According to this theory, chest lesions should cause recurrent laryngeal nerve paralysis alone, but in many patients with lung cancer the cord assumes a intermediate position. This has been attributed to the phenomenon of retrograde atrophy of the vagus nerve up to the level of nucleus ambiguus.
Paralysed vocal cords may demonstrate some movement due to the action of interarytenoid muscle which gets bilaterally innervated.

40. Bilateral abductor palsy

Bilateral vocal fold (vocal cord) immobility (BVFI) is a broad term that refers to all forms of reduced or absent movement of the vocal folds. Bilateral vocal fold (cord) paralysis (BVFP) refers to the neurologic causes of bilateral vocal fold immobility (BVFI) and specifically refers to the reduced or absent function of the vagus nerve or its distal branch, the recurrent laryngeal nerve (RLN). Vocal fold immobility may also result from mechanical derangement of the laryngeal structures, such as the cricoarytenoid (CA) joint. Fiberoptic laryngoscopy is the mainstay of clinical assessment. Management strategies include medical treatment of causative inflammatory conditions, with surgical procedures such as tracheostomy employed as necessary.

Although a small number of conditions account for most cases of vocal cord immobility, this article presents a comprehensive differential diagnosis, followed by the clinical presentations, diagnostic workup, and treatment options. The goal of the article is to provide the clinician with a basic understanding of the rare entity of bilateral vocal fold immobility (BVFI).

Fiberoptic laryngoscopy is the mainstay of clinical assessment. Management strategies include medical treatment of causative inflammatory conditions, with surgical procedures such as tracheostomy employed as necessary.

According to Benninger's findings in a series of 117 cases BVFI can be attributed to the following causes: surgical trauma (44%), malignancies (17%), endotracheal intubation (15%), neurologic disease (12%), and idiopathic causes (12%).

In adults, conditions that mimic vocal fold immobility include paradoxical vocal fold motion and functional disorder.

Causes of vocal fold fixation differ in adults and in children. In adults, these include mechanical causes, inflammatory processes (affecting the CA or larynx), malignancy, surgery, neurologic causes, radiation injury, metabolic causes, and toxins. Mechanical derangement of the posterior glottis may also be referred to as posterior glottic stenosis (PGS). Bogdasarian and Olson classified PGS into the following 4 grades:

Grade I - Interarytenoid scarring with normal posterior commissure

Grade 2 - Interarytenoid and posterior commissure scarring

Grade 3 - Posterior commissure scarring involving one cricoarytenoid joint

Grade 4 - Posterior glottic scarring involving both cricoarytenoid joints

Management:

Since these patients will be in stridor air way should be secured by tracheostomy.

Definitive treatment:

1. Medialization procedures
2. Cordectomy
3. Kashima’s surgery

41. Left vocal cord palsy

Already discussed

42. Tracheostomy

Already discussed

43. Pharyngeal pouch

Pharyngeal pouches are mainly acquired and rarely congenital diverticula in and around the pharynx and oesophagus. These diverticula are nothing but circumscribed pouches caused by a protrusion of mucosa through a preexisting weakness / opening in the muscle layers of pharynx / oesophagus.

This is in contrast to the congenital diverticula like Meckel's diverticula where in the diverticula is covered by all the muscle layers of the wall of the viscous.

These pharyngo oesophageal diverticula may be single of multiple. It could be sited laterally or posteriorly. Majority of them arise above the level of cricopharyngeus muscle, rarely some of them may arise at the level of oesophagus.

Inferior constrictor: is the most important muscle in the understanding the pathophysiology of pharyngeal diverticula. This muscle is described in two parts: Thyropharyngeus and cricopharyngeus respectively. The thyropharyngeus muscle arises from an oblique line on the thyroid ala, and a fibrous arch between the thryoid and cricoid cartilages. Its upper fibers overlaps the superior and middle constrictors, and the lower fibers lie edge to edge with the cricopharyngeus muscle. There is a potential space between the middle and inferior constrictors, lying over the pyriform fossa. This space is bounded anteriorly by the thyrohyoid muscle. This space is pierced by neurovascular bundle of the third arch.

Killian's dehiscence: Is the junction between thyropharyngeus and cricopharyngeus muscles. This is a potentially weak area not supported by other constrictor muscles. The cricopharyngeus muscle is thicker and bulkier than the thyropharyneal component of inferior constrictor. The pressure generated by the constriction of cricopharyngeus muscle is sufficient to cause prolapse of mucosal lining through this potentially weak area.

The following are the weak spots around pharynx and oesophagus:
Lateral:

1. Above the superior constrictor

2. Between the superior and middle constrictors

3. Between the middle and inferior constrictors

4. Below cricopharyngeus - Killian-Jamieson's area

Posterior:

1. Laimer-Hackermann's area

2. Killian's dehiscence

Classification of pharyngeal diverticula:
Lateral:

1. Congenital

2. Acquired:

a. Normal bulges

b. Traumatic

c. Raised intrapharyngeal pressures (Pharyngoceles)

Posterior:

1. congenital

2. Acquired:

a. Traumatic

b. Intrapharyngo-oesophageal pressure

c. Posterior pharyngeal pulsion diverticulum (Zenker's diverticulum)

History: The first pharyngeal pouch was described by Ludlow in 1769, when he performed autopsy on a patient who had dysphagia throughout his life. Wheeler performed the first documented surgical excision of pharyngeal pouch. Dohlman first performed endoscopic removal of pharyngeal pouch.

Etiopathology of pharyngeal diverticula: Is still not clear. A lot of it has been attributed to the cricopharyngeal muscular contractions. Wouters and Van Overbeek proposed that an anatomical predisposition to a large Killian's dehiscence, which lies between the propulsive oblique fibres of

the thyropharyngeus and the horizontal fibres of the cricopharyngeus, which have a sphincteric action, plays a prominent part in mucosal herniation.

Cook et al using videoradiography and manometry found intrabolus pressures were greater in patients with a diverticulum compared with an age matched healthy population and concluded that a disorder of diminished upper oesophageal opening was the cause.

Histologically it has been demonstrated that the cricopharyngeus muscle fibres are gradually replaced by fibro adipose tissue in these patients.

Congenital lateral pharyngeal diverticula: Is a very rare entity, with only a few cases reported. These diverticula have been attributed to remnants of branchial cleft. These diverticula communicates internally, and usually found to be associated with second cleft commonly.

These patients commonly present with swelling over left side of neck since the ultimobranchial body growth is diminished or absent on the right side. These patients presented within two decades of life. These patients present with tender fluctuant swelling in the anterior triangle of neck. The patient may also have mild fever, with dysphagia.

Plain radiographs of neck usually show air inside the diverticulum. These diverticula can be better demonstrated by the use of contrast swallow using high density barium. Surgery must be performed once the acute episode has been treated with a course of antibiotic.

Acquired lateral pharyngeal diverticula:

Normal bulges: Small lateral pharyngeal bulges can be seen either arising in the pyriform fossa, or more rarely from the tonsillar fossa. This could become evident while performing a modified valsalva manuver. These normal lateral bulges are more common in elderly, probably due to reduced muscle tone, and loss of elasticity of the tissues. These bulges are usually bilateral and asymptomatic, and hence are thought to be normal variants.

Radiologically contrast studies demonstrate these bulges as smooth hemispherical prominences arising from either the tonsillary fossa or pyriform fossa, hence they are termed as pharygneal ears. They enlarge in size during performance of modified valsalve manuver.

Traumatic: It has been reported by Atkins, that a certain group of criminals in central and north india had self inflicted diverticula where they hid stolen valuables. These diverticula have been produced by repetitive attempts to introduce a lead mass (size of a pigeons egg) in to the tonsillar fossa creating a diverticula. This diverticulum probably lies between the superior and middle constrictor muscles.

Raised intrapharyngeal pressure (Pharyngoceles): These large sometimes symptomatic diverticula arises from the precursor pharyngeal ears. This is due to repetitive increase in intrapharyngeal

pressure. Loss of muscular tone due to ageing could also play a role. These diverticulae protrude through areas of weakness in the lateral pharyngeal wall and develop into pouches known as pharyngoceles. Men are commonly affected than women.

Symptoms: Entrapment of food inside the diverticula causes dysphagia, which could be intermittent. Sometimes there may be regurgitation of entraped food from the diverticula casuing foul taste and bad odor. Patients may also have nocturnal coughing and choking. If the mass enlarges enough to affect the larynx, voice changes can occur. Patients may also manifest with chronic pulmonary problems. The mass may lie anterior to the sternomastoid muscle. This mass is compressible, and on compression may empty its contents with a gurgling sound. This is known as Boyce sign. Indirect laryngoscopy may not reveal much. Sometimes a small slit may be seen close to the pyriform fossa.

Plain radiographs of neck may reveal the diverticula as a translucency lateral to the pyriform fossa. Videofluroscopic techniques using high density barium really clinches the diagnosis. The high density barium effectively coats the diverticula and is retained for a longer duration to make diagnosis reliable.

Posterior pouches:

Posterior pharyngeal diverticula are more common than lateral diverticula. Among the various types of posterior pharyngeal diverticula, the posterior pulsion diverticulum (Zenker's diverticulm) is more commonly encountered.

Congenital Posterior diverticula: These are very rare. This diverticulum arises from above the cricopharyngeus, and is lined by normal pharyngeal mucosa. The whole diverticula is covered with muscle differentiating it from an acquired pulsion diverticula.

Acquired posterior diverticula:

Traumatic posterior pharyngeal diverticulum: This is a very rare condition. The etiological factor is hypopharyngeal trauma, either from damage caused by obstertitian's finger during breech delivery, or due to blind passage of suction / endotracheal tubes. Initially, there may be reactive spasm of cricopharyngeal muscle causing dysphagia and drooling.

Diverticulum due to raised intrapharyngo-oesophageal pressure: This rare variety of posterior diverticulum protrudes through the Laimer-Hackermann area. It commonly occurs in old individuals probably due to weakness of supporting musculature.

Posterior pharyngeal pulsion (Zenker's) diverticulum: Zenker called this pulsion diverticula. Earlier foreign body impaction was attributed as a cause of this diverticulum. Killian attributed spasmodic contraction of the circular fibers at the upper end of the esophagus.

Etiology:

Theories attributed to the formation of Zenker's diverticulum:

Negus theory: Tonic spasm of cricopharyngeus muscle

According to Negus Killian's dehiscence occur as a result of man assuming erect posture, causing the larynx and cricopharynx to move lower down into the neck resulting in other constrictors to lie obliquely. There is also a lack of posterolateral longitudinal muscles which are seen elsewhere in the alimentary tract. Criopharyngeal muscle spasm associated with the pharyngeal stippling peristalsis causes prolapse of the mucosa through the weak portion in the posterior portion of pharynx (Killian's dehiscence).

Dohlmann's theory: Lack of inhibitory stimuli to the cricopharyngeus muscle.

Dohlmann postulated that lack of inhibitory stimuli to the cricopharyngeus muscle causing increased intraluminal pressure in the pharynx causing prolapse of mucosa through killian's dehiscence.

Wilson's theory: Second swallow due to pharyngeal laxity.

In patients with posterior pharyngeal diverticulum, the pharynx was found to be large. A second swallow becomes necessary to completely empty the pharynx. This second swallow occurs against a closed circopharyngeal sphincter. The attendant increase in the intraluminal pressure causes the pharyngeal mucosa to prolapse through the killian's area.

Korkis theory: Neuromuscular inco-ordination and associated congenital weakness could cause this problem.

Stages of posterior pharyngeal diverticulum:

Stage I: Small mucosal protrusion (initial stage) Patients may have a sticky sensation in the throat.

Stage II: A definite pouch with oesophagus and hypopharynx still in line (intermediate stage) Regurgitation and gurgling sounds are common in these patients (Boyce sign).

Stage III: A large pouch with the hypopharynx in line with the neck of the diverticulum. The oesophagus inlet is pushed anteriorly. Patients in this stage have severe dysphagia.

Clinical features:

1. Difficulty in swallowing

2. Regurgitation of undigested food from the diverticulum

3. Weight loss

4. Halitosis

5. Hoarseness of voice

Radiography: Plain radiographs of neck may show a triangular lucency in the prevertebral tissues, with the apex at the level of the cricoid cartilage. This is due to the presence of air in the upper part of the pouch. Contrast videofluoroscopy clinches the diagnosis.

Treatment:

1. Dilatation of cricopharyngeus

2. Diverticulectomy

3. Inversion

4. Diverticulopexy

Endoscopic:

1. Dohlmanns electocoagulation

2. Dohlmanns laser treatment

3. Stapling

Endoscopic diathermy: Dohlmann's operation:

This procedure was first described by Mosher. The septum between the diverticulum and oesophagus is divided. Initially scissors was used to divide it. Since bleeding produced was enormous use of diathermy to divide the septum was resorted to. Major risk of this surgical procedure is the ever-present danger of mediastinitis. In contrast to external approach this procedure can also be performed in elderly individuals. For this procedure a double beaked oesophagoscope is used. The larger beak is introduced into the oesophagus while the smaller beak is inserted into the diverticulum. The intervening septum is divided.
External surgical approach is resorted to in patients with stage III diverticulum. The diverticulum is exposed through a transverse skin incision in the neck. After excising the diverticulum the mucosa should be carefully approximated / stapeled.

44. Investigations for dysphagia

Already discussed

45. Plummer winson syndrome.

Already discussed

46. Oesophagoscopy

Indications of oesophagoscopy:

1. Removal of foreign bodies

2. Examination and biopsy of lesions from oesophagus

3. Dilatation of oesophagal strictures (benign)

4. Treatment of pharyngeal pouch

Anesthesia used:

Local (topical) 4% xylocaine spray, and pyriform fossa block.

General anesthesia is reserved for removal of impacted foreign bodies

Premedication:

1. Injection Glycopyrrolate 0.2 mg intra muscular injection is given as premedication to prevent excessive secretions in the throat.

2. Injection fortwin 1 ampule intramuscular is given

Instruments used:

1. Rigid oesophagoscope - Two types are available. Negus type and Jackson's type. They differ from each other in the type of illumination.

Negus type: In this oesophagoscope the illumination is at the Proximal end of the scope. The biggest disadvantage is that the illuminating tip is present at the proximal end and is not very bright.

Jackson's type: This type of oesophagoscope has distal illumination. The illumination is brighter than that of Negus type since it is present at the distal end. The major disadvantage is that it could get soiled with blood and secretions.

2. Flexible oesophagoscope

Position of patient on the table: Boyce position.

In this position the patient lies supine with ring below the head. The neck is flexed and the head is extended at the atlanto-occipital joint.

Complications:

1. Oesophageal perforation

2. Injury to teeth, lips, gums, and cervical spine

3. Rupture of aortic aneurysm

47. Etiology of carcinoma esophagus

Squamous cell carcinoma	Adenocarcinoma
Cigarette smoking	Gastro-esophageal reflux disease
Alcohol drinking	Barrett's esophagus
ALDH2 deficiency	Reflux symptoms
Drinking very hot liquids	Obesity
Achalasia	Cigarette smoking
Caustic injury	Diet (high in processed meat, low in fruits, vegetables)
History of thoracic radiation	History of thoracic radiation
Tylosis	Anticholinergic agents
Human papilloma virus infection	Family history
N-nitrosamines	*Helicobacter pylori* infection (decreased risk)

48. Stricture oesophagus

Already discussed

49. Foreign bodies in ENT

Children are known to insert a myriad of foreign bodies into their body cavities. Ears, nose and throat are easily accessible to children. Mentally deranged adult individuals are also commonly known to insert foreign bodies into their ears nose and throat.

Children insert foreign bodies in to their ears /nose / throat due to ignorance and curiosity.
Mentally deranged individuals insert foreign bodies into their body cavities.
Accidental insertion of foreign bodies are common in adults
Type of foreign bodies inserted can be classified into:

Organic – This again may be subdivided into animate and inanimate foreign bodies. Inanimate foreign bodies include seeds. Animate foreign bodies include insects. Maggots are known to commonly involve nasal cavities with foul smelling discharge. Insects and worms can accidentally enter the ear. When seeds are inserted into ear or nose they have a tendency to swell on exposure to moisture. Aural syringing should not be resorted to as it will cause rapid swelling of the seed within the ear canal, making their removal difficult. Organic foreign bodies in nose if present for a prolonged duration may cause foul smelling unilateral nasal discharge. Animate foreign bodies like insects, maggots should be stifled with liquid paraffin before removal.
Inorganic foreign bodies: These include commonly available house hold items. Impacted in organic foreign bodies in the ear should always be removed under anesthesia. Unimpacted foreign bodies inside the external ear canal can be removed by aural syringing. Foreign bodies involving the throat get impacted just above the level of cricopharynx. It could also get impacted in any of the narrowed portions of oesophagus. Oesophagoscopy should be done to remove these foreign bodies.
Foreign bodies involving the airway are highly risky to remove. The air way should be shared between the anesthetist and the surgeon performing Bronchoscopy. This adds further to the risk. Foreign bodies involving the air way gets commonly stuck in the right main bronchus, since this bronchus is a direct continuation of trachea. Rigid Bronchoscopy should be performed under jet ventilation anesthesia to remove these foreign bodies.

Role of X-rays in the management of foreign bodies:

X-rays are useful in identifying the site of foreign bodies if they get lodged in the airway or food passages. Radio opaque foreign bodies like coins, bone pieces can be clearly visualized on an x-ray. Fish bones are commonly radiolucent and less readily visualized in an x-ray. X-ray chest will reveal either direct or indirect evidence of foreign body in the air passage. It also reveals the presence of collapse, emphysema or abscess of the involved lobe of the lung. These events are more common in long standing foreign bodies.

50. LASERs in ENT

Lasers used in ear surgeries include:
Argon - 514 nm
KTP -532 nm
Carbondioxide - 10,600 nm
Er:YAG - 2960 nm

In ear surgeries lasers are used to vaporize small glomus tumors, acoustic neuromas, small A-V malformation, granulation tissue or adhesions in the middle ear cavity. Lasers have also been used to perform myringotomy, perforation of foot plate during stapes surgery, and coagulation of membranous posterior canal in BPPV.

Operational parameters:
1. Wave length of laser beam : The exact properties of laser depends on its wavelength

2. Power: Is actually the output from the machine and is measured in watts. Higher the power, more is the energy delivered to the tissues

3. Exposure tine: It is measured in seconds

4. Spot size: This is actually the area exposed to the laser beam. Spot size is the minimum at focal length. Focussed beam is used for cutting and decofocussed beam is used for coagulation / ablation of tissues.

5. Power density: It is the power delivered per unit area of spot size and is measured in watts/square cm. This indicateste intensity of the beam.

6. Exposure to laser: This value is the power density multiplied by duration of exposure in seconds and is measured in joules/ cubic cm.

Laser delivery mode:
Continuous mode:
It provides constant stable energy as the active medium is continuously kept in a stimulated mode.

Pulsed mode:
This gives interrupted beam as the active medium is intermittently activated for a short time.

Q - switched mode:
This mode provides very short pulses in a controlled manner. Pulses range between 10 ns and 10 milliseconds.

Advantages:

Precision

Rapid ablation of tissues

Excellent hemostasis

Minimal post op pain

Minimal tissue oedema

Disadvantages:

Expensive

Expensive to maintain

Safety precautions need to be taken

Types of lasers used in otolaryngology:
Argon laser:
This lies in the visible spectrum. Does not eed pointing ray. It is absorbed by hemoglobin. Hence it is used to treate portwine stain, hemaingioma and telengiectasis. When focussed to a small point it can vaporize the target tissuee. This laser is used to create a hole in the foot plate of stapes. It needs a drop of blood to be placed over the foot plate for this effect to occur.

KTP laser:
This laser also lies in the visible spectrum. It has a wavelength of 532 nm. These waves are absorbed by hemoglobin and can be delivered via optical fibers. This laser is also used ins tapes surgery, endoscopic sinus surgery to remove polypi, inverted papilloma and vascular lesions.

Nd:YAG laser:
This laser has a wavelength of 1064 nm and lies in the infrared zone. It is in the invisible range and requries a separate aiming beam of visible light to focus. It can pass through clear fluids and is also absorbed by pigmented tissue as the case may in eye and urinary bladder. In otolaryngology it has been usedto debulk tracheobronchial and esophageal lesions for palliation.

Carbondioxide laser:
It has a wavelength of 10,400 nm in the invisible range. It requires an aiming beam of visible light to focus the laser beam. This laser does not pass through optical fibers. It needs special articulated arms and mirrors that reflect the laser beam to the spot of the lesion. This laser beam is absorbed by tissues high in water content and is not color dependent. Reflection and scatter through tissues is minimum. Its tissue effect is in depth and in adjacent tissues laterally. Clinically it is used in laryngeal surgeries to excise papillomas.

Diode laser:
This has a wave length of 600-1000 nm. It can be delvered via optical fibers and is moderately absorbed by melanin and hemoglobin. Diode lasers are used for turbinate reduction, laser assisted stapedectomy and tonsillar ablation.

Safety precautions:
Eye protection to surgeon and assistant by wearing goggles. Wavelenght specific glasses should be worn to prevent retinal damage. Patient's eye should be protected by double layer of saline soaked cotton eye pads. All exposed areas of face are covered by saline soaked pads.

Endotracheal tubes:
Wave specific tubes are available. Rubber tubes are better than PVC as they are more resistant to laser hits. PVC tubes when hit by laser can generate toxic fumes. These endotracheal tubes should be covered by reflective aluminium foils. The cuff of the endotracheal tube should be inflated with blue dye mixed saline and covered with wet cottonoids. In case of accidental hit by laser blue color effusion will warn the surgeon.

Anesthetic gases:
Non inflammable gases are used. Halothane / enflurare are preferred to nitrous oxide. Concentration of oxygen should not exceed 40%.

Smoke evacuation:
Constant suction should be used to suck out fumes released out of laserization of tissue.

51. Thyroglossal cyst

Embryology: The thyroid gland originates from the foramen cecum present in the floor of the pharyngeal gut on the 17th day of gestation. The gland then descends in front of the pharynx as a bilobed diverticulum which is initially patent. It reaches its final position in the neck by the 7th week of gestation. The duct usually disappears by the 10th week of gestation. Persistence of any portion of this duct could give rise to thyroglossal cyst.

Since the hyoid bone develops later and joins from lateral to medial, the thyroglossal duct may get trapped in the substance of the body of hyoid bone, resulting in the tract running inside the body of the bone. The hyoid bone rotates to reach its adult position dragging the duct posteriorly and superiorly at the inferior edge of its body.

No natural internal opening of thyroglossal duct has been demonstrated at the level of foramen cecum so far. This has been attributed to the fact that the tongue and foramen cecum forms after the complete descent of the thyroglossal duct. Rarely a tract could be found at the level of foramen

cecum. This tract has been attributed to the persistence of lingual duct, which represents the point of union between the anterior and posterior components of the tongue.

The lowermost portion of the thyroglossal duct could remain in some as the pyramidal lobe of the thyroid gland.

Theories of thyroglossal cyst:

Cystic degeneration: This theory suggests that recurrent throat infections could possibly stimulate the epithelial remnants of the tract causing it to undergo cystic degeneration.

Retention phenomenon: This theory suggests a block in the thyroglossal duct could cause the cyst to expand because of retained secretions. Foramen cecum has been postulated as the possible site of obstruction. The thyroglossal duct epithelium is supposed to contain mucinous and serous glands. The continued secretions from these glands could cause enlargement of the cyst.

Clinical features: Thyroglossal cyst is the second commonest benign neck lesion next only to lymphadenopathy. Commonly it manifests as a cystic midline neck mass before the age of 5. The mass moves with deglutition and on protrusion of the tongue. Theoretically speaking the cyst could lie anywhere within the thyroglossal tract. These cysts may also be located laterally as well.

Site of occurrence:

1. Hyoidal - 61%

2. Suprahyoidal - 24%

3. Infrahyoid - 13%

4. Intralingual - 2%

The masses are invariably painless. When these cysts are associated with pain then infection of the cyst should be considered. Enlargement of intralingual cysts may cause respiratory obstruction.

Differential diagnosis: These cysts should be differentiated from

1. Dermoid cyst - cheesy secretion

2. Infected lymph node - purulent secretion

3. Lipoma - slippery edges

4. Sebaceous cyst - doughy feel

5. Hypertrophic pyramidal lobe of thyroid

Commonly these cyst could contain thyroid tissue, hence I 131 study should be considered in all patients with suprahyoid and infrahyoid masses. Rarely this could be the only functioning thyroid gland tissue.

Treatment:

Is mainly surgery. The procedure commonly performed is Sistrunk operation which involves exposing the whole cyst along with its tract. The anterior portion of the body of the hyoid bone should be included in the dissection to prevent recurrence. It was Wenglowski who suggested that along with the body of hyoid bone a core of tissue between the hyoid bone and the tongue should also be removed to reduce the incidence of recurrence.

Causes of failure of Sistrunk operation:

1. Missing a dumb-bell cyst deep in the back of hyoid

2. A cyst which has already ruptured with formation of a number of pseudocysts

3. Leaving part of a cyst wall

4. Presence of multiple tracts

In the case of infected thyroglossal cyst a fistula may also form. If fistula occurs then it must be removed in toto. The mouth of the fistulous tract must be included in the incision.

52. Pleomorphic Adenoma

Pleomorphic adenoma, the most common salivary gland tumor, is also known as benign mixed tumors (BMT's), because of its dual origin from epithelial and myoepithelial elements.It is the commonest of all salivary gland tumors constituting up to two-thirds of all salivary gland tumors.

The etiology of pleomorphic adenoma is unknown, but the incidence of this tumor has been increasing in the last 15-20 years in relation to the exposure of radiation. One study suggests that oncogenic simian virus (SV40) may play a role in the onset or progression of pleomorphic adenoma. Prior head and neck irradiation is also a risk factor for the development of these tumors.

Microscopically pleomorphic adenoma has a highly variable appearance, hence the name pleomorphic. It is characterized by mixed proliferation of polygonal epithelial and spindle-shaped myoepithelial cells in a variable stroma matrix of mucoid, myxoid, cartilaginous or hyaline origin. Epithelial elements are usually of polygonal, spindle or stellate-shaped cells which may be arranged

to form duct-like structures, sheets, clumps, or interlacing strands. The ducts and tubules are seen usually exhibiting an outer lining in addition to an inner cuboidal epithelial cell layer. This is outer myoepithelial cell layer (or layers) which merges into the surrounding stroma which also contains dispersed or clumped myoepithelial element cells. Areas of squamous metaplasia and epithelial pearls can be found. The tumor lacks the true capsule and is surrounded by a fibrous pseudo capsule of variable thickness. The tumor extends through normal glandular parenchyma in the form of finger-like pseudopodia. These microscopic extensions account for the high risk of recurrence in cases treated with simple enucleation or surgical resections performed with inadequate surgical margins.

Histology will reveal proliferation of myoepithelial and epithelial cells of the ducts. There is also a marked increase in stromal components. The tumor is usually not well encapsulated.

Previously carried out enucleation procedure has been abandoned because of high associated rates of recurrence. Presently pleomorphic adenoma of the parotid gland is treated either with superficial (Patey's operation) or total parotidectomy with the latter being the more frequently performed procedure due to lower incidence of recurrence. Meticulous technique is required to preserve the facial nerve. The tumors of the submandibular glands are treated with simple excision procedure with preservation of adjacent nerve including the mandibular branch of the trigeminal nerve, the hypoglossal nerve, and the lingual nerve.

SHORT ANSWER QUESTIONS

EAR

1. Draw the diagram of medial wall of middle ear and name the parts

Already answered

2. Intratympanic Muscles

Tensor tympani- Originates from the cartiagenous and bony portions of the eustachean tube.

It gets inserted into the handle of malleus after going around the processus cochleariformis.

Contraction of this muscle pulls the malleus medially and anteriorly, at right angles to the normal direction of vibration. Contraction of this muscle can be seen as in drawing of the ear drum.

Stapedius - Arises from the pyramid present in the posterior wall of middle ear cavity.

It gets inserted to the neck of stapes.

Contraction of stapedius muscle causes fixation of stapes. It increases the stiffening effects of middle ear conduction mechanism. Contraction of this muscle can reduce transmission by up to 30 dB for frequencies less than 1-2 KHz.

Contraction of these two muscles serves to dampen unwanted resonances in the middle ear system causing spoken words to be heard with clarity.

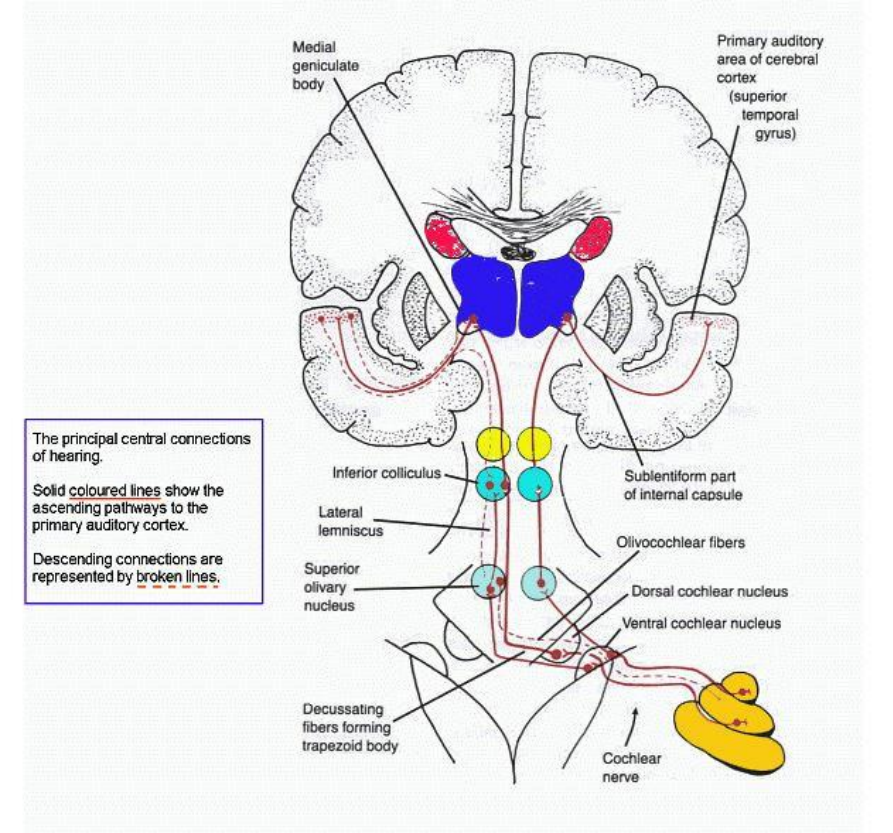
3. Stapes - The stapes consists of a head, neck, two crura and a base (footplate). The head of the stapes points laterally and has a small cartilage covered depression for articulation with the lenticular process of the incus. The tendon of the stapedius muscle attaches to the posterior part of the neck and the upper part of the posterior crura. The neck of the stapes gives rise to two crura, the anterior crura is thinner and less curved than the posterior crura. The two crura join the foot plate which closes the oval window during life. The average dimensions of the foot plate is 3mm x 1.4mm. The long axis of the foot plate is almost horizontal, with the posterior end being slightly lower than the anterior.

4. Mesotympanum – This is middle ear proper. The most prominent portion of this part of middle ear is the promontory which is raised by the basal turn of cochlea.

5. Nerve Supply of External Auditory Canal

Since it originates from branchial arch it is innervated by 5th, 7th, 9th and 10th cranial nerves. Auriculo temporal branch of the mandibular nerve innervates the anterior portion of the pinna, tragus, and the anterior wall of the external canal. The well of the concha and the posterior wall of the external canal receive innervation from the 7th, 9th, and 10th cranial nerves. This complex innervation of the external canal accounts for several clinical findings involving the external canal : i.e. vesicular eruption in the skin of the external canal with facial palsy is caused by herpetic infection of the geniculate ganglion is known as the Ramsay Hunt syndrome. Hypesthesia of the concha and external canal caused by facial nerve compression from cerebello pontine angle tumors is known as Hitselberger's sign. Instrumentation of the external canal can cause nausea or coughing through stimulation of the vagus nerve via the Arnold's nerve.

6. Auditory Pathway



7. Functions of eustachian tube

Functions of eustachean tube are:

1. Ventilation of middle ear

2. Drainage of middle ear secretions

3. Protection of middle ear from nasopharyngeal commensals and pathogens

For any middle ear surgical procedure to succeed a normal and functioning eustachean tube is a must.

It is always better to have a clear understanding of the functional status of ET before embarking on tympanoplasty / myringoplasty.

8. Valsalva maneuver

This maneuver is not popular now. It was first proposed by Antonio Maria Valsalva during 17th century. This maneuver is performed by exhaling forcibly exhaling against a closed airway. This is a very difficult maneuver to perform. To overcome this difficulty a modified valsalva Maneuver has been proposed. In this maneuver, the patient is made to expire against closed glottis.

9. Caloric Test

In this test attempt is made to stimulate labyrinth with cold / warm water irritation to induce nystagmus. The temperature of fluid used for irrigation is 7 degrees centigrade above or below normal body temperature. If cold water alone is used then it is known as Kobrack test while if both warm and cold water is used it is known as bithermal caloric test. Caloric testing is a clinically useful bed side tool to isolate peripheral vestibular system and to rule out central causes of vertigo. It is also used to test for brain stem functions in comatose patients.

Before attempting to perform caloric testing otoscopic examination should be performed to ensure that there is no obstruction in the external canal (cerumen impaction), there is no infection involving skin lining of the external auditory canal.

Position of the patient:

Patient is placed in supine position with head elevated to 30 degrees. This position ensures that the lateral canal is placed in a vertical plane thereby optimizing stimulation. Patient should clearly be explained about the procedure. A kidney tray or catch basin is placed under the ear which is to be irrigated. Water flowing out of the external canal will be caught in the kidney tray. An irrigation system is used to deliver 250 cc of either warm or cold water solution over a period of 25 - 30 seconds to the ear under test. The irrigated solution will freely dribble out of the external canal and can be collected in the basin. Nystagmus starts 30 seconds after the onset of water delivery and will build in intensity over the ensuing 30-45 seconds. After an interval of 5 minutes after cessation of nystagmus the other ear can be tested. The pneumonic COWS help in identifying the direction of nystagmus (cold opposite and warm same side).

Canal paresis - Caloric test indicates (based on the duration of nystagmus) after stimulation with water is less than that of the opposite side.

Directional preponderance - This takes into consideration the duration of the nystagmus to the left or right irrespective of the fact that it is elicited from the right or left labyrinth. If nystagmus is 30% more on one side then the other then it is known as directional preponderance to that side. Directional preponderance occurs towards the side of a central lesion and away from the side in a peripheral lesion. Canal paresis and directional preponderance can also be seen together in the same patient.

Modified Kobrak test:

This is a quick office procedure where cold water is used for the test. In this test the patient is seated with head tilted 60 degrees backwards to ensure that the horizontal canal is in vertical position. The ear is irrigated with cold water for 60 seconds. The temperature of the irrigated water should be 7 degrees centigrade below the normal body temperature. Initially 5 ml is used. If there is no response then the volume should be increased to 10, 20 and 40 ml if needed. In normal persons the nystagmus starts beating towards the opposite ear when 5 ml is irrigated. If higher volumes of water is needed to elicit nystagmus then the labyrinth is considered to by hypoactive. If there is no response even after irrigating 40 ml of water then it indicates dead labyrinth.

10. Fistula test

The fistula test is designed to elicit symptoms and signs of an abnormal connection (fistula) between the labyrinth and surrounding spaces. Fistulas may be acquired, most commonly as a result of cholesteatoma or, less commonly, as a dehiscence of bone overlying the superior semicircular canal. Iatrogenic causes include chronic ear surgery and stapes surgery.

The test involves the application of pressure to the patient's ear canal and observation of eye movements with Frenzel lenses in place. Occluding the ear canal with the patient's tragus or using a

Bruening otoscope can provide pressure to the ear canal. The direction of nystagmus depends on the site of the fistula, a topic that is beyond the scope of this article.

11. Tuning fork tests

Rinne's test: is a tuning fork test used to clinically test hearing deficiencies in patients. It is designed to compare air conduction with bone conduction thresholds. Under normal circumstances, air conduction is better than bone conduction.

Ideally 512 tuning fork is used. It should be struck against the elbow or knee of the patient to vibrate. While striking care must be taken that the strike is made at the junction of the upper 1/3 and lower 2/3 of the fork. This is the maximum vibratory area of the tuning fork. It should not be struck against metallic object because it can cause overtones. As soon as the fork starts to vibrate it is placed at the mastoid process of the patient. The patient is advised to signal when he stops hearing the sound. As soon as the patient signals that he is unable to hear the fork anymore the vibrating fork is transferred immediately just close to the external auditory canal and is held in such a way that the vibratory prongs vibrate parallel to the acoustic axis. In patients with normal hearing he should be able to hear the fork as soon as it is transferred to the front of the ear. This result is known as Positive Rinne test. (Air conduction is better than bone conduction). In case of conductive deafness the patient will not be able to hear the fork as soon as it is transferred to the front of the ear (Bone conduction is better than air conduction). This is known as negative Rinne. It occurs in conductive deafness. This test is performed in both the ears.

If the patient is suffering from profound unilateral deafness then the sound will still be heard through the opposite ear this condition leads to a false positive Rinne.

Use of Rinne test in quantifying conductive deafness:

Conductive deafness of more than 25 dB is indicated by negative Rinne with 512 Hz fork, while it is positive for 1024 Hz. If Rinne is negative for 256, 512 and 1024 Hz then conductive deafness should be greater than 40dB.

Weber's test:

Is a tuning fork test (quick) used to assess hearing levels in an individual. This can easily detect unilateral conductive and unilateral sensorineural hearing loss. This test is name after Ernst Heinrich Weber (1795 – 1878).

Procedure:

Tuning forks used - 256 Hz / 512 Hz

Commonly used frequency is 512 Hz.

A vibrating fork is placed over the forehead / vertex / chin of the patient. The patient should be instructed to indicate which ear hears the sound better. In normal ear and in bilateral equally deaf ears the sound will be heard in the mid line. This test is very sensitive in identifying unilateral deafness. It can pick out even a 5 dB difference between the ears.

Theory:

A patient with a unilateral (one-sided) conductive hearing loss would hear the tuning fork loudest in the affected ear. This is because the conduction problem masks the ambient noise of the room, whilst the well-functioning inner ear picks the sound up via the bones of the skull causing it to be perceived as a louder sound than in the unaffected ear.

Inadequacies:

This test is most useful in individuals with hearing that is different between the two ears. It cannot confirm normal hearing because it does not measure sound sensitivity in a quantitative manner. Hearing defects affecting both ears equally, as in Presbycusis will produce an apparently normal test result.

Absolute Bone conduction test:

This test is performed to identify sensorineural hearing loss. In this test the hearing level of the patient is compared to that of the examiner. The examiner's hearing is assumed to be normal. In this test the vibrating fork is placed over the mastoid process of the patient after occluding the external auditory canal. As soon as the patient indicates that he is unable to hear the sound anymore, the fork is transferred to the mastoid process of the examiner after occluding the external canal. In cases of normal hearing the examiner must not be able to hear the fork, but in cases of sensori neural hearing loss the examiner will be able to hear the sound, then the test is interpreted as ABC reduced. It is not reduced in cases with normal hearing.

12. Weber test

Weber's test:

Is a tuning fork test (quick) used to assess hearing levels in an individual. This can easily detect unilateral conductive and unilateral sensorineural hearing loss. This test is name after Ernst Heinrich Weber (1795 – 1878).

Procedure:

Tuning forks used - 256 Hz / 512 Hz

Commonly used frequency is 512 Hz.

A vibrating fork is placed over the forehead / vertex / chin of the patient. The patient should be instructed to indicate which ear hears the sound better. In normal ear and in bilateral equally deaf ears the sound will be heard in the mid line. This test is very sensitive in identifying unilateral deafness. It can pick out even a 5 dB difference between the ears.

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Inadequacies:

This test is most useful in individuals with hearing that is different between the two ears. It cannot confirm normal hearing because it does not measure sound sensitivity in a quantitative manner. Hearing defects affecting both ears equally, as in Presbycusis will produce an apparently normal test result.

13. Mention any two tuning fork tests

Rinne test

Weber test

14. Two causes for perichondritis of pinnae

Frost bite

Ear pricking

15. Otomycosis

Fungal infection of external auditory canal. White cotton like flakes indicate candida infection while black flakes indicate aspergillus. These patients have intense itching and blocking sensation in the ear.

16. Malignant otitis externa.

Involves bony portion of the external auditory canal. This condition is common in diabetics. Pseudomonas organism have been implicated. Since this infection involves bone it is very difficult to eradicate. It can even involve skull base.

Carbenicillin is the drug of choice.

17. Signs of malignant otitis externa

The patient gives history of trivial trauma to the ear often by ear buds, followed by pain and swelling involving the external auditory canal. Pain is often the common initial presentation. It is often severe, throbbing and worse during nights. It needs increasing doses of analgesics. On examination granulation tissue may be seen occupying the external canal. It often begins at the bony cartiagenous junction of the external canal. Discharge emanating from the external canal is scanty and foul smelling in nature. When the discharge is foul smelling it indicates the onset of osteomyelitis. Ironically the patient does not have fever or other constitutional symptoms.

Otoscopy: Reveals granulation tissue at the bony cartiagenous junction. The ear drum is usually normal. The external auditory canal skin is soggy and edematous.

18. Keratosis obturans

Already discussed.

19. Pseudocyst Pinna

This condition involves the Pinna and can frequently recur even after successful treatment. It goes by various names i.e. intercartilagenous cyst, endochondral Pseudocyst and idiopathic cystic chondromalacia. This condition was first described by Engel in 1966.

Clinical features:
1. Presents as painless, spontaneous dome shaped cystic swelling on the anterior surface of auricle.

2. This condition is predominantly seen in adult males

3. It is uncommon before 20 and after 60 years of age.

4. Majority of these cysts are found in the scaphoid and triangular fossae of the pinna

5. Majority of these cysts have been reported in Chinese. Chinese have attributed this problem due to the firm pillow they use to sleep. Studies have not demonstrated any racial differences.

6. Right ear is more commonly affected than the left. This has been attributed to the habit of majority of individuals to sleep on their right side.
Management:

1. Antibiotics 2. Anti-inflammatory drugs 3. Steroids (systemic / intralesional) ??

Wide bore needle aspiration – The fluid from pseudocyst can be aseptically aspirated using a wide bore needle. Compression dressing is applied in order to avoid recurrence due to fluid reaccumulation. POP can also be used as compression dressing.

Button surgery: Incision and drainage always was associated with a high recurrence rate. This procedure is performed under local infiltrative anesthesia. A helical incision is made, and the skin flap is elevated well beyond the anterior cartilage segment of the pseudocyst. The anterior wall of the cyst is excised along the margin. After the straw-colored fluid is drained completely the posterior wall is curetted. After obtaining hemostasis, similar sized buttons (sterile) is sutured on the anterior and posterior surfaces of the pinna providing compression at the operated area. This helps in preventing recurrence in these patients. No other dressing is essential. The patient is put on antibiotics and anti-inflammatory drugs for a week after which the suture is removed along with the shirt button.

20. Otalgia.

Unilateral pain in the ear can be classified into:

i. External ear causes

ii. Middle ear causes

iii. Pain referred from adjacent areas due to segmental innervation.

21. Four important causes of referred otalgia

    1. Dental pain
    2. Tonsillitis
    3. Post tonsillectomy
    4. TM joint dysfunction

22. Referred otalgia

Pain to the ear can be referred from disorders affecting other portions of head and neck. This is due to the same segmental innervation. These include:

1. Temporomandibular joint dysfunction

2. Dental pain

3. Quinsy

4. Tonsillitis

5. Post tonsillectomy pain always radiates to the ipsilateral ear.

Causes for referred otalgia should be diligently searched for in a patient with ear pain, with clinically normal ear.

23. Tinnitus-Classify

Nodar's classification: This classification was based on the importance of 6 factors related to tinnitus. These include:

1. Description, 2. presence, 3. continuous or pulsatile, 4. single or multiple, 5. level and 6. annoyance. These variables are more or less similar to that of Nodar's classification, but a little simpler and user friendly. This system of classification has also not found wide use.

Shulman's classification: This classification divides tinnitus into two main categories: otologic and neurotologic. Patients belonging to otologic category include those with disease of external or middle ear, cerumen impaction, abnormal mobility of ear drum or ossicular chain, and abnormal contractions of the middle ear musculature.

The neurotologic classification is based on a complete cochlear - vestibular evaluation. It includes history and physical examination, audiological testing, vestibular evaluation and radiologic studies. Cochlear evaluation includes: pure tone audiometry, auditory brain stem response, speech

audiometry, tone decay testing and tests for recruitment. Vestibular testing includes complete electronystagmography.

Modified Nodar's classification: In 1996 Nodar came out with a new version of his classification. He used the pneumonic ABC and C-CLAP. The term ABC denotes aurium (one ear), binaural (two ears), and cerebri (centered in the head). C-CLAP denotes cause, composition (patient's description), loudness, annoyance and pitch. This is the commonly used classification system in use these days.

24. Causes of red ear drum

1. Acute otitis media
2. Glomus jugulare

25. Mention the different types of perforations of Tympanic Membrane

Central perforation
Attic perforation
Marginal perforation

26. Signs of retracted tympanic membrane

1. Prominent handle of malleus
2. Apparent foreshortening of handle of malleus
3. Diminished ear drum mobility
4. Presence of retraction pockets

27. Glue ear./ Secretory otitis media
28.

Otitis media with effusion is defined as chronic accumulation of mucus within the middle ear, and rarely this could involve the mastoid air cell system. This accumulation causes conductive hearing loss.

A high index of suspicion is necessary to identify this condition. Every child with upper respiratory infection must be otoscopically examined.
Otoscopic findings: The tympanic membrane may be bulging, or retracted with a distorted cone of light. The ear drum may appear yellow, blue or simply clear white. Pneumatic otoscopy will reveal a ear drum which has a restricted mobility.

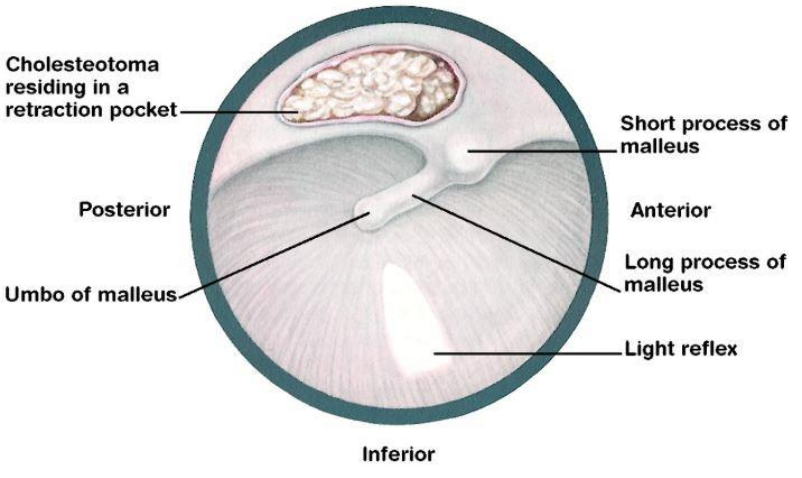
Grommet insertion is the ideal treatment modality.

29. Myringotomy

Myringotomy with grommet insertion was introduced by Poltizer of Vienna in 1868. He used this procedure to manage “Otitis media catarrhalis”. Soon it became the common surgical procedure performed in children.
Indications: Bluestone and Klein (2004) came out with revised indications for grommet insertion which took into consideration the prevailing antibiotic spectrum. Chronic otis media with effusion not responding to antibiotic medication and has persisted for more than 3 months when bilateral or 6 months when unilateral.
A myringotome is used to perform a radial incision in the antero inferior quadrant of the ear drum along the direction of radial fibers of the middle fibrous layer of ear drum.

30. Define Cholesteatoma and draw diagram

Cholesteatoma is defined as a cystic bag like structure lined by stratified squamous epithelium on a fibrous matrix. This sac contains desquamated squamous epithelium. This sac is present in the attic region. Cholesteatoma is also defined as 'skin in wrong place'. Cholesteatoma is known to contain all the layers of skin epithelium. The basal layer (germinating layer) is present on the outer surface of cholesteatoma sac in contact with the walls of the middle ear cleft.



31. Extra cranial complications of chronic suppurative otitis media.

Sub periosteal abscess
Bezold’s abscess
Facial nerve paralysis
Zygomatic abscess

32. Four intra-temporal complications of CSOM

Facial nerve paralysis
Lateral sinus thrombosis
Labyrinthitis
Gradenigo syndrome

33. Gradenigo syndrome/Petrositis.

Gradenigo classically described the following as the classic features of this syndrome:

Discharging ear

Retro orbital pain

Abducent nerve paralysis causing diplopia

Causative factors:

Uncontrolled mastoiditis

Epidural abscess following mastoiditis

34. Griesinger's sign

The Griesinger sign refers to edema of the postauricular soft tissues overlying the mastoid process as a result of thrombosis of the mastoid emissary vein. It is a complication of acute otomastoiditis and may be associated with dural sinus occlusive disease (DSOD). It is said to be a pathognomonic finding

35. Fistula sign

Already discussed

36. Otogenic brain abscess

Already discussed

37. Medical treatment of meniere's disease.

Already discussed

38. Name four complications of cortical mastoidectomy

1. Facial nerve paralysis
2. Injury to lateral semicircular canal
3. Injury to tegmen plate
4. Dislocation of incus

39. Indications for radical mastoidectomy

1. Malignant lesions of middle ear cavity
2. Promontory cochlear fistula
3. Unresectable cholesteatoma extending into the eustachean tube

40. Myringoplasty

Already discussed

41. Pure tone audiometry

Already discussed

42. Tympanoplasty

Already discussed

43. Masking

Masking means that one puts in some "noise" in the opposite ear while testing an ear. The reason to do this is to prevent sound from the side being tested from going over to the good side This is called the "cross-over problem". See the illustration below for an example of the cross-over problem.

When to do masking --

Bone conduction testing in persons with hearing loss should be done with masking to prevent sound from the stimulated side from going over to the good side.

Air conduction testing should be done when there is a 40 db or greater threshold.

44. Tymanogram

Already discussed

45. Impedance audiometry

Already discussed

46. Mention the any four causes of Sensory Neural Hearing loss

1. Presbyacusis
2. Ototoxicity
3. Noise induced hearing loss
4. Otosclerosis

47. Cochlear implant- indications.

1. Deaf mutism
2. Profound sensorineural hearing loss not responding to conventional hearing aids

48. Hearing aids

Already discussed

49. Otosclerosis

Already discussed

50. Otosclerosis – diagnosis, medical management

Pure tone audiometry showing conductive hearing loss / Cookie bite audiogram

Impedance audiometry – Showing As curve

The aim of medical management is to convert an active otosclerotic foci into an inactive or quiescent foci. Fluoride is the drug of choice.

Indications of fluoride therapy:

1. Patients with surgically confirmed otosclerosis who show progressive sensorineural deafness disproportionate to age.

2. Patients with pure sensorineural loss with family history, age of onset, audiometric pattern and good auditory discrimination indicate the possibility of cochlear otosclerosis.

3. Patients with radiological demonstration by CT scan of spongiotic changes in the cochlear capsule

4. Patients with positive Schwartz sign.

5. Post op treatment: If patients are found to have an active focus during surgery, fluride therapy is prescribed for 2 years.

51. CARHART'S notch.

Is classically found in bone conduction audiograms of patients with otosclerosis. This is actually a dip centered around 2000 Hz.
Some authors consider Carhart's notch to be an artifact. This notch is closely related to the Carhart's effect.

Carhart's effect: was initially described following successful stapes surgery. There was an over closure of air bone gap following successful surgery. Classically this effect leads to an improvement in hearing levels particularly at 2 KHz frequency levels.

How Carhart's effect is created?

When skull is vibrated by bone conduction, sound is transferred to cochlea via three routes. i.e.

1. By direct vibration of skull

2. By vibration of ossicular chain which is suspended within the skull

3. By transmission via external auditory canal (normal route)

In conductive hearing loss routes 2 and 3 are affected, but can be regained following successful stapes surgery. Hence bone conduction thresholds improve around 2 KHz frequency range.

52. Paracusis of Willisi

Otosclerotic patients may hear better in noisy environment because the speaker has a tendency to raise his voice because of excessive ambient noise. This phenomenon a feature of otosclerosis is known as Paracusis Willisii.

53. Bell's palsy
Already discussed

54. Crocodile tears
The term "crocodile tears" is derived from the ancient belief that crocodiles weep after killing their victims. "crocodile tears syndrome," also known as Bogorad syndrome, is the shedding of tears while eating or drinking in patients recovering from Bell's Palsy. It is also referred to as gustatory lacrimation.

55. Surgery for facial palsy

1. Facial nerve decompression
2. Facial nerve anastomosis
3. Sural nerve anastomosis

NOSE

1. Littles area

This area is located in the anterior part of the cartiagenous portion of the nasal septum. Here there is extensive submucous anastomosis of blood vessels both from the external and the internal carotid systems. Bleeding commonly occurs from this area since it is highly vascular and is also exposed to the exterior. Anastomosis occur between the septal branches of sphenopalatine artery, greater palatine artery, superior labial artery and the anterior ethmoidal artery. This plexus is also known as Keisselbach's plexus. Bleeding from this area is common because mucosal drying occurs commonly here and this area is easily accessible to nose picking. Among the vessels taking part in the anastomosis the anterior ethmoidal artery is from the internal carotid system while the other vessels are from the external carotid system. Bleeding from this area is clearly seen and easily accessible and flows through the anterior nasal cavity hence it is known as anterior bleed.

2. Mention the arteries involved in kiesselback's plexus

Anterior ethmoidal artery (branch of the ophthalmic artery)
Sphenopalatine artery (terminal branch of the maxillary artery)
Greater palatine artery (from the maxillary artery)
Septal branch of the superior labial artery (from the facial artery)

3. Osteomeatal complex

The ostiomeatal complex (OMC) or ostiomeatal unit (OMU), sometimes less correctly spelled as osteomeatal complex, is a common channel that links the frontal sinus, anterior ethmoid air cells and the maxillary sinus to the middle meatus, allowing airflow and mucociliary drainage.

The ostiomeatal complex is composed of five structures:

Maxillary ostium: drainage channel of the maxillary sinus
Infundibulum: common channel that drains the ostia of the maxillary antra and anterior ethmoid air cells to the hiatus semilunaris
Ethmoid bulla: usually a single air cell that projects over the hiatus semilunaris
Uncinate process: hook-like process that arises from the posteromedial aspect of the nasolacrimal duct and forms the anterior boundary of the hiatus semilunaris
Hiatus semilunaris: final drainage passage; a region between the ethmoid bulla superiorly and free-edge of the uncinate process

4. Haller cell.

These are also known as Infra orbital recess cells.

Introduction:

These are pneumatized ethmoid air cells that project along the medial roof of the maxillary sinus and the most inferior portion of the lamina papyracea.

This air cell lies below the ethmoid bulla and lateral to the uncinate process.

Commonly these cells arise from anterior ethmoid air cells and are closely related to the infundibulum. Rarely these cells can arise from posterior

ethmoidal cells in which case it does not compromise the infundibulum.

Infections involving these cells can compromise and narrow the infundibulum causing obstruction to the drainage of maxillary sinus ostium. It has been
suggested that infections involving these cells could be a factor in recurrent maxillary sinusitis.

5. Sphenoid sinus

Is located in the skull base at the junction of the anterior and middle cranial fossa. Pneumatization of sphenoid starts during the 4th year of childhood and gets completed by the 17th year. The sphenoid sinuses vary in size and may be asymmetric.

They drain through the superior meatus via a small ostium about 4mm in diameter located disadvantageously 20mm above the sinus floor.

This sinus is related to several important vital structures. They are:

1. Pituitary gland lies above the sphenoid sinus.

2. Optic nerve and internal carotid arteries traverse its lateral wall.

3. The nerve of pterygoid canal lie in the floor of the sinus.

Hence infections of sphenoid sinus may involve the optic nerve if the canal of the optic nerve is dehiscent.

6. Functions of Nose-5 functions

    1. Protection of lower airway
    2. Olfaction
    3. Humidification of inspired air
    4. Adds resonance to voice
    5. Respiration

7. Rhinomanometry

Rhinomanometry is used to assess the patency of the nose. There are two types of rhinomanometry, active and passive rhinomanometry. Active rhinomanometry involves the generation of nasal airflow and pressure with normal breathing. Passive rhinomanometry involves the generation of nasal airflow and pressure from an external source, such as fan or pump which drives air through the nose.

Active rhinomanometry: can be divided into anterior and posterior methods according to the siting of the sensor tube. In active anterior rhinomanometry, the pressure sensing tube is taped to one nasal passage. This method measures resistance of one nasal cavity at a time and must be repeated on the other side. The total air flow through the nose is measured with the help of the sensor tube. In active posterior rhinomanometry, the pressure sensing tube is held within the mouth and it detects the post nasal pressure. Air flow through each nose can be measured by taping the opposite nose.

8. Furuncle Nose

Nasal furunculosis is a localized infection of the hair-bearing nasal vestibule. It is usually caused by the bacteria S aureus. It can occur as a primary infection or secondary to chronic rhinorrhea, upper respiratory infections, and nose picking. Patients usually present with painful swelling in the vestibule. The skin over the nose becomes tense and red, and a boil may be visible in the nostril. If not treated properly, the patient can develop complications like facial cellulitis and cavernous sinus thrombosis, which is characterized by fever, headache, chemosis, proptosis, and cranial nerve III, IV, V, and VI palsies.

9. Potato nose

Already discussed

10. Dangerous area of face

The danger triangle of the face is a triangle with two corners at both corners of the mouth and one corner in the middle of the nose between the eyes. The way the blood flows to the human nose is special, so it is possible (but very unlikely) for infections to spread directly to the brain from a cut, scratch or a popped pimple.

Almost all people have valves in the veins of the face. But even with one-way valves, blood flow between the facial vein and cavernous sinus can spread infection from the face. It is the direction of blood flow that is important. Infection may possibly lead to cavernous sinus thrombosis, meningitis or brain abscess.

11. Cottles line and its significance

Perpendicular line drawn between the nasal crest and the nasal bone. Septal deviations anterior to this line can be managed by septoplasty while deviations posterior to this line can be managed by SMR.

12. Bleeding polypus septum

Hemangioma involving the nasal septum has been reported in children and pregnant mothers. Pyogenic granuloma of nasal septum has also been erroneously christened as bleeding polypus nasal septum. Enlarged retro columellar vein also resembles bleeding polypus. This condition can be managed by cauterizing the offending area.

13. Epistaxis digitorum

This is also known as epistaxis due to nose picking. This is common in children.

14. Sluder's Neuralgia

A pain syndrome originally described by Sluder as a symptom complex referable to the nasal ganglion. • The sphenopalatine ganglioneuralgia is a type of headache or neuralgia, which was once believed to be attributed to the irritation of the sphenopalatine ganglion.

15. Management of septal haematoma

Septal hematoma causes swelling of the nasal septum. This is caused due to accumulation of blood between the nasal septal cartilage and its overlying perichondrium. Stripping of perichondrium away from the cartilage will compromise its nutrition causing the cartilage to undergo liquefaction necrosis. Within 3 days following the development of septal hematoma, nasal septum gets destroyed causing collapse of the bridge of the nose.

This condition can be managed by evacuation of hematoma via a septoplasty incision situated on the side of the swelling.

16. Septoplasty

Already discussed

17. Complications of septal surgery

Complications of SMR:

1. Septal hematoma

2. Septal abscess

3. Septal perforation

4. Nasal deformities due to excessive removal of dorsal strut of the septum

5. Removal of the columella cartilage will cause pig snout deformity

18. Oroantral fistula

This is a fistulous communication between the floor of the maxillary sinus to the oral cavity. This commonly occurs following dental extraction of infected upper molar and premolar tooth.

The upper lateral teeth when removed has a tendency to form blood clots. Fibrosis sets in within the clot material aiding the healing process. Fibrosis inside the clot is the most critical stage in the healing process. During this process of healing the air pocket within the maxillary sinus could keep constantly extruding hampering the healing process. This eventually leads to the formation of oroantral fistula. In order to prevent this fistula formation, the mucosal flaps after extraction of upper lateral teeth should be sutured.

Management:

1. Wait and watch approach: A significant amount of these fistulas tend to heal spontaneously. This is more so if the size of the fistula is 2 mm or less. If the size is 3 mm or more then spontaneous healing is hampered because of sinus infection in the periodontal area.

2. Caldwell Luc procedure: This surgery aims at creating a more permanent drainage via the antrostomy performed through the inferior meatus. This helps in spontaneous healing of the fistula.

3. Direct closure of the fistula can be attempted using palatal flaps.

19. Indications for Caldwell-luc operation

Indications:

1. Mycotic maxillary sinusitis

2. Multiseptate maxillary sinus mucocele

3. A/C polyp (Recurrent)

4. Oroantral fistula

5. Revision procedures

6. Access for transantral sphenoidectomy, orbital decompression, orbital floor repair, exploration of pterygoplatine fossa

7. Excision of tumors involving the antrum (inverted papilloma)
8. Visualization of orbital floor during orbital floor decompression surgeries
9. Removal of foreign bodies from maxillary antrum

20. Mention two important causes of Sinusitis

Anatomical obstruction like grossly deviated nasal septum

Infections hampering the Mucociliary clearance mechanism

21. Two surgical approaches to maxillary sinus

Inferior meatal antrostomy

Caldwell Luc surgery

22. Name major complications of FESS.

Bleeding
Injury to orbital contents
Injury to optic nerve
CSF rhinorrhea

23. Enumerate four complications of FESS (Functional Endoscopic Sinus Surgery)

Bleeding
Injury to orbital contents
Injury to optic nerve
CSF rhinorrhea

24. Any two clinical findings in nasal cavity of a patient suffering from allergic rhinitis

Watery rhinorrhea

Enlarged pale inferior turbinate

25. Ethmoidal polyposis

Already discussed

26. Antrochoanal polyp

Already discussed

27. Surgical management of atrophic Rhinitis

Already discussed

28. Rhinitis sicca

Rhinitis sicca or generally speaking dry nose is a rather frequent problem involving many people. The term “dry nose” has not yet been uniformly defined. Otolaryngologists often use the terms “rhinitis sicca” or “dry rhinitis,” although no clear definition exists. Many symptoms during dry nose could be encountered ranging from subjective sensation of the dry nose and itching up to mild burning, nasal obstruction, crusting associated with unpleasant smell, epistaxis, and diminished sense of smell. Rhinitis sicca anterior means a chronic inflammation in the region of the anterior part of the nose, affecting the anterior and caudal septum and/or the corresponding lateral nasal vestibule. Mechanical as well as environmental irritations lead to crust formation. In rare cases, patients suffer from a slight stench due to bacterial colonization of the crust formations.

The treatment of rhinitis sicca involves mainly elimination of promoting factors, moistening, sufficient daily drinking amount, cleansing of the crusts, care of the mucosa and inhibition of possible infections, or in rare cases the elimination of overlarge endonasal space.

Saline drops have been used with great effect to manage these patients.

29. Rhinitis Medicamentosa

Rhinitis medicamentosa is a condition characterised by nasal congestion without rhinorrohea or sneezing. This condition is caused by the use of topical nasal decongestants for a prolonged period of time. Use of these topical decongestants for more than a week is sufficient to cause this problem. This condition should be differentiated from rhinitis caused by use of drugs like oral contraceptives, antihypertensives and psychotrophic drugs.

Possible mechanisms of rhinitis medicamentosa include:

Secondary decrease in the production of endogenous norepinephrine through a negative feed back mechanism
Sympathomimetic amines used as topical decongestants have effects on both alpha and beta receptors. Their alpha effects predominate over beta effects causing nasal decongestion. This beneficial alpha effect is short lived while beta effect is more prolonged. After cessation of alpha stimulation the sympathomimetic amines still keep stimulating beta receptors causing rebound nasal congestion.
Rebound increase in parasympathetic activity causing increased nasal secretion and nasal mucosal congestion.

Management:

Nasal douching

Intranasal steroids

Oral prednisolone

30. Rhinoscleroma.

Already discussed

31. Two important clinical features in fungal sinusitis

Polypoidal tissue seen inside the nasal cavity

Orbital enlargement is seen in these patients

32. Mucormycosis.

Fungal infections involving the nasal cavity. White cheesy material can be seen within the nasal cavity

33. Two common sites of CSF leak in Nose
Cribriform plate
Roof of ethmoid

34. Rhinolith

Already discussed

35. Nasal myiasis

Already discussed

36. Inverted papilloma/ Ringertz Tumor

Already discussed

37. Maxillectomy - types and indications

Total maxillectomy

Partial maxillectomy

Maxillectomy with orbital exenteration

38. Orbital Blow out Fracture

Blow out fracture is defined as a clinical syndrome in which there is fracture of orbital walls with intact rim. When rim is not involved it is known as pure orbital blow out fracture. If orbital walls and rims are also involved in the fracture then it is known as impure orbital blow out fracture.

39. Lignocaine in ENT

2% xylocaine is used in infiltration anesthesia

4% xylocaine is used for topical anesthesia

THROAT

1. Boundaries of parapharyngeal space

This space is situated lateral to the fascia covering the constrictor muscles of the pharynx (buccopharyngeal fascia). Lateral to this space lie the pterygoid muscle, mandible and carotid sheath.

Superiorly it extends up to the skull base while inferiorly it ends at the level of hyoid bone because of the attachment of the submandibular gland sheath to the sheaths of the stylohyoid muscle and the posterior belly of digastric muscle.

The carotid sheath lies close to the posterolateral wall of this space.

Postero medially this space communicates with the retropharyngeal space.

Anteriorly and inferiorly this space communicates with the spaces associated with the floor of the mouth.

This space is most commonly involved in neck space infections. Infections from this space can easily spread to the carotid and retropharyngeal spaces.

2. Parts of hypopharynx.

Pyriform fossae

Posterior pharyngeal wall

Post cricoid growth

3. Gerlach's Tonsil

Also known as the tubal tonsil. This is an accumulation of lymphoid tissue around the pharyngeal end of eustachean tube. This forms one of the components of inner waldayer's ring. Inflammation of this structure is known to cause eustachean tubal block and its attendant ear problems like secretory otitis media.

4. Components of waldeyers ring

    1. Palatine tonsils
    2. Lingual tonsils
    3. Adenoid
    4. Tubal tonsils
    5. Lymphoid accumulation in the mucosal lining of posterior pharyngeal wall

5. Blood Supply of Tonsil

The main artery of the tonsil is the tonsillar branch of the facial artery which enters the tonsil near its lower pole by piercing the superior constrictor just above the styloglossus muscle. Other arteries supplying the tonsil are lingual artery through its dorsal lingual branches, ascending palatine branch of facial artery, and ascending pharyngeal vessels.

Venous drainage occurs through the para tonsillar vein, and the vessels also pass through to the pharyngeal plexus or facial vein after piercing the superior constrictor.

6. Parapharyngeal space

Already discussed

7. Reinke’s space

Reinke's space is a potential space between the vocal ligament and the overlying mucosa. It is not an empty space, but contains cells, special fibers and extracellular matrix. It plays an important role in the vibration of the vocal cords. Edema of this space is called Reinke's edema.

8. Rima Glottidis

The rima glottidis is the opening between the true vocal cords and the arytenoid cartilages of the larynx.

It is normally subdivided into two parts: that between the arytenoid cartilages is called the intercartilaginous part (or the intercartilaginous glottis, respiratory glottis, or interarytenoid space), and that between the vocal folds the intermembranous part (3/5th) or glottis vocalis.

9. Unpaired cartilages of larynx.

Epiglottis

Thyroid cartilage

Cricoid cartilage

10. Nasopharyngeal tonsil

Also known as adenoid. Adenoid is a collection of lymphoid tissue in the mucous membrane overlying the basisphenoid area. It has an oblong shape, similar to that of a truncated pyramid. It in fact virtually hangs from the roof of the naso pharynx. Its anterior edge of this tissue is vertical and lie in the same plane as the post nasal aperture. Its posterior edge gradually merges into the posterior pharyngeal wall. and its lateral edges incline towards midline. It is lined by ciliated columnar epithelium. The surface of adenoid has furrows. It feels like a bag of worm to touch. Laterally adenoid is continuous with lymphoid tissue around the pharyngeal end of eustachean tube.

11. Phonation

The phonatory process, or voicing, occurs when air is expelled from the lungs through the glottis, creating a pressure drop across the larynx. When this drop becomes sufficiently large, the vocal folds start to oscillate. The minimum pressure drop required to achieve phonation is called the phonation threshold pressure (PTP),and for humans with normal vocal folds, it is approximately 2–3 cm H2O. The motion of the vocal folds during oscillation is mostly lateral, though there is also some superior component as well. However, there is almost no motion along the length of the vocal folds. The oscillation of the vocal folds serves to modulate the pressure and flow of the air through the larynx, and this modulated airflow is the main component of the sound of most voiced phones.

12. Aphthous ulcers

They are also called aphthae, aphthosis, aphthous stomatitis and canker sores. An aphthous ulcer is typically a recurrent round or oval sore or ulcer inside the mouth on an area where the skin is not tightly bound to the underlying bone, such as on the inside of the lips and cheeks or underneath the tongue.

13. Oral submucous fibrosis

Oral Submucosal fibrosis is a chronic debilitating potentially malignant condition of the oral cavity associated with betel nut chewing. It is characterized by fibrosis of the oral soft tissue, resulting in marked rigidity and inability to open the mouth. The inability to open the mouth is slowly progressive in nature.

Buccal mucosa was the most commonly involved site, but no part of the oral cavity is immune to this condition. Almost all the patients were pan chewers. Pathophysiology of this disease is not well established. A number of factors trigger the disease process by causing a juxtaepithelial inflammatory reaction in the oral mucosa. Factors such as areca nut chewing, ingestion of chilies, genetic and immunologic processes, nutritional deficiencies can lead to this condition.

Betel nut chewing:

The areca nut component used along with betel leaf has been implicated. A recent study has clearly demonstrated the dose and frequency relationship of areca nut chewing in the pathogenesis of this disorder. Arecoline the active ingredient of areca nut is known to stimulate fibroblasts to increase its production of collagen by 150%. Flavinoids, catechin and tannin present in betel nuts cause collagen to cross link making them less susceptible to collagenase enzyme degradation. The disease process of Submucosal fibrosis is active even after cessation of betel nut chewing suggesting that arecoline not only affects the fibroblasts it also affects gene expression in fibroblasts causing them to produce increased amount of collagen with intense cross linkages.

Clinical features:

1. Progressive inability to open the mouth due to fibrosis and scarring

2. Oral pain and burning sensation when the patient consumes spicy food

3. Increased salivation

4. Change in taste

5. Secretary otitis media due to stenosis of the pharyngeal end of Eustachian tube

6. Dryness of mouth

7. Dysphagia when consuming solids if esophagus is involved

Treatment:
Medical: Weekly Submucosal intralesional injections of steroids may help in prevention of progression of the disease. Cessation of betel nut chewing is a must. Placental extracts can be injected intra lesionally to reduce the inflammatory effects of the disease. Use of topical hyaluronidase in doses of 150 units in association with steroids has proved beneficial.

Intralesional injection of IFN-gamma has a role due to the immuno regulatory effect of the molecule.

Surgical management is reserved only for advanced cases with severe trismus. This include excision of fibrous bands, with split thickness skin grafting.

14. Mention two premalignant lesions of oral cavity

Leukoplakia

Erythroplakia

15. Keratosis tonsil

Keratosis Pharyngis is a medical condition where keratin grows on the surface of the pharynx, that is the part of the throat at the back of the mouth. Keratin is a protein that normally occurs as the main constituent of hair and nails.

16. Reactionary Haemorrhage

Hemorrhage is one of the most important complication following tonsillectomy. Reactionary hemorrhage occurs within the first 24 hours after the surgical procedure.

Causes of reactionary hemorrhage is as follows:

1. During surgery the blood pressure of the patient is diminished due to the effects of anesthetic medications. After the effects of the medications wear off, the blood pressure returns to normal causing bleeding from the operated site. This commonly occurs within the first 24 hours after surgery.

2. Slippage of ligatures. During surgical procedure bleeding vessels are ligated. These ligatures can slip due to the repeated swallowing efforts of the patient causing bleeding from the operated site.

Management:

1. Blood grouping and cross matching should be performed.

2. If blood loss is more than 200ml then blood transfusion should be initiated

3. Patient should be shifted to the theater, anesthetized and the bleeding vessels are ligated again.

17. Adenoid facies

The characteristic adenoid facial appearance consists of:

Underdeveloped thin nostrils
Short upper lip
Prominent upper teeth
Crowded teeth
Narrow upper alveolus
High-arched palate
Hypoplastic maxilla

18. Eagle's Syndrome

Elongated styloid process causes pain in the throat. This is known as Eagle's syndrome. This condition can be managed by excision of the elongated styloid process.

19. Tonsillolith

Tonsillolith is a rare dystrophic calcification occurring in the tonsil as a result of chronic inflammation. Most commonly tonsilloliths are intratonsillar and are asymptomatic. They have been identified incidentally. Commonly patients with tonsillolith complain of foul breath and throat pain. Throat pain is usually very intense during acts of swallowing. Also known as tonsil concretions or tonsillar stones.

Deposition of calcium salts (Calcium phosphate) occurs normally in the skeleton. When calcification occurs in soft tissues in an unorganized fashion it is known as heterotopic calcification. Calcium salts (Calcium phosphate) occurs normally in the skeleton. When calcification occurs in soft tissues in an unorganized fashion it is known as heterotopic calcification. This heterotopic calcification can be further subdivided into three categories:

Metastatic calcification: This calcification occurs in normal tissues due to deposition of calcium. This is the result of higher than normal levels of serum calcium as in the case of hyperparathyroidism or higher levels of serum phosphate as in patients with chronic renal failure. Metastatic calcification usually occurs bilaterally and symmetrically.

Idiopathic calcification: This condition refers to deposition of calcium in normal tissue despite normal serum levels of calcium and phosphate. Examples include chondrocalcinosis and phleboliths.

20. White patch on Tonsil-3 conditions

Infectious mononucleosis
Diphtheria
Vincent's angina

21. Two absolute indications for tonsillectomy

Obstructive sleep apnoea

Group A beta hemolytic streptococci infections involving tonsil

22. Name four complications of tonsillectomy

Hemorrhage
Injury to lips
Injury to teeth
Nasal regurgitation of swallowed liquids

23. Causes and treatment of secondary haemorrhage

Secondary hemorrhage following tonsillectomy occurs after the first 7 days following surgery. It is due to infection. This condition can be managed by using systemic antibiotics.

24. Four modalities for tonsillectomy

Cold steel tonsillectomy
Laser tonsillectomy
Coblation tonsillectomy
Bipolar cautery tonsillectomy

25. Causes of Reactionary hemorrhage after tonsillectomy

Already explained

26. Quinsy.

Already explained

27. Retro pharyngeal abscess

Already explained

28. Four causes for unilateral tonsillar enlargement

Lymphomas

Squamous cell carcinoma

Quinsy

FB tonsil

29. Ludwig's angina

Already e xplained

30. Rhinolalia Aperta-Mention any 3 causes

Cleft palate
Velopharyngeal incompetence
Velopharyngeal mislearning

31. Rhinolalia Clausa

Enlarged adenoid
Enlarged lingual tonsil
Oropharyngeal growth

32. Four causes of Rhinolalia clausa

Already discussed

33. Laryngomalacia

Already discussed

34. Laryngocele.

Already discussed

35. Microbiology of acute epiglottitis

H influenza

36. Steeple sign

This is a classic feature seen in children with croup. Their X-ray chest picture shows pencil like narrowing resembling the steeple of the church.

37. Features of Laryngeal Tuberculosis

Hoarseness
Odynophagia
Loss of weight

38. Turban epiglottis

Is a feature of tuberculous laryngitis. This is due to lymphatic oedema of the epiglottis. These patients manifest with intense pain while swallowing (odynophagia).

39. Vocal nodule.

Already discussed

40. Reinke's Edema

Reinke's edema is the swelling of the vocal cords due to fluid (edema) collected within the Reinke's space. First identified by the German anatomist Friedrich B. Reinke in 1895, the Reinke's space is a gelatinous layer of the vocal cord located underneath the outer cells of the vocal cord. When a person speaks, the Reinke's space vibrates to allow for sound to be produced (phonation). The Reinke's space is sometimes referred to as the superficial lamina propria. Reinke's edema is characterized by the "sac-like" appearance of the fluid-filled vocal cords

41. Functional Aphonia

Functional aphonia is defined as loss of speech suffered by the patient without any attributable organic cause. Patient afflicted by this condition suddenly loses voice / sometimes voice gets reduced to a whisper. These patients don't suffer from hoarseness of voice.

Aetiology:

This condition is caused by sudden psychological trauma / emotional shock.

Affected patients are mostly more than 15 years of age.

Females are commonly affected.

Symptoms:

Aphonia: Patient suddenly loses voice / sometimes voice gets reduced to a whisper. There is no hoarseness of voice. Importantly these patients are able to cough normally.

These patients normally have associated psychological problems. These patients may have pain in the neck.

42. Juvenile papilloma of larynx

Already discussed

43. Laryngeal papillomatosis

Already discussed

44. Premalignant lesions of larynx

Leukoplakia of vocal folds
Papilloma of vocal folds

45. Name four causes for left recurrent laryngeal nerve palsy

Carcinoma lung
Malignancy thyroid
Malignant disorders of oesophagus
Mediastinal adenopathy

46. Semon's Law

This theory proposed by Rosenbach and Semon in 1881, depends on the concept that abductor fibres in the recurrent laryngeal nerves are more susceptible to pressure than the adductor fibers. After a number of amendments this law is stated as " In the course of a gradually progressing organic lesion involving the recurrent laryngeal nerve three stages can be observed. In the first stage only the abductor fibers are damaged, the vocal folds approximate in the midline and adduction is still possible. In the second stage the additional contracture of adductors occur so that the vocal folds are immobilized in the median position. In the third stage the adductors become paralysed and the vocal folds assume a cadaveric position".

47. Bilateral abductor paralysis

Already discussed.

48. Causes of Right Recurrent Laryngeal Nerve Palsy-any 3 causes

Pulmonary tuberculosis
Thyroid malignancy
Oesophageal malignancy

49. Cricothyroidotomy

Cricothyroidotomy: Is not performed commonly nowadays because of high incidence of postoperative subglottic stenosis. This procedure is indicated only under extremely emergency conditions.

Cricothyroid membrane is incised through vertical incision and tracheostomy tube is inserted through it. Ideally these patients must be converted into a regular tracheostomy within 48 hours.

50. Laryngeal crepitus

Laryngeal crepitus is felt by the examiner when the larynx is moved from side to side with a slight posterior pressure. When absent, it is a clinical sign of a mass in the retrolaryngeal space or hypopharynx, probably due to a laryngeal trauma.

51. Two important indications for total laryngectomy

Malignant growth involving both vocal folds
Post cricoid malignancy (total laryngectomy oesophagectomy with gastric pull up)

52. Any four speech rehabilitation methods after laryngectomy

Oesophageal speech
TEP
Artificial larynx

53. Tracheostomy

Already discussed

54. Name four complications of tracheostomy

Sudden apnoea
Damage to apical pleura
Damage to subclavian vein
Injury to thyroid

55. Immediate complications of tracheostomy

Bleeding
Damage to apical pleura
Sudden apnoea

56. Prevertebral shadow widening

Caused in retropharyngeal abscess
FB cricopharynx

57. PV syndrome (Plummer Vinson syndrome

Already discussed

58. Investigations in a case of Dysphagia.

Barium swallow
UGI endoscopy
Oesophagus manometry
Barium swallow screening

59. Dysphagia Lusoria

Dysphagia lusoria (or Bayford-Autenrieth dysphagia) is an abnormal condition characterized by difficulty in swallowing caused by an aberrant right subclavian artery. It was discovered by David Bayford in 1761 and first reported in a paper by the same in 1787.

60. Pharyngeal pouch

Already discussed

61. Strictures of oesophagus

Already discussed

62. Barret's Oesaphagus

Barrett's esophagus is a condition in which there is an abnormal (metaplastic) change in the mucosal cells lining the lower portion of the esophagus, from normal stratified squamous epithelium to simple columnar epithelium with interspersed goblet cells that are normally present only in the small intestine, and large intestine. This change is considered to be a premalignant condition because it is associated with a high incidence of further transition to esophageal adenocarcinoma, an often-deadly cancer.

63. Oesophageal diverticulum

There are different ways to categorize esophageal diverticulum. Esophageal diverticula can be divided into true and false diverticula. True diverticula are outpouchings that include all layers of the esophageal wall while false diverticula only include the mucosa or submucosa.

An esophageal diverticulum can also be characterized by how it is formed: pulsion or traction. Pulsion diverticula are created when there is increased intraluminal pressure causing herniation of the esophageal wall in an area of weakness and usually occur in the setting of dysmotility of the esophagus. Traction diverticula occur when there is an external force on the esophageal wall such as mediastinal inflammation that adheres and pulls on the esophageal wall creating a defect or diverticulum.

An esophageal diverticulum can also be categorized based on location as pharyngeal (Zenker) diverticula, mid-esophageal diverticula, and epiphrenic diverticula. Pharyngeal diverticula are considered false diverticula. They usually occur in the hypopharynx where there is a weakness in the area known as Killian's triangle. Killian's triangle is an area bound by the cricopharyngeus muscles and inferior pharyngeal constrictor muscles. These are usually formed by pulsion. A mid-esophageal diverticulum is usually true diverticulum and normally caused by traction from mediastinal inflammation. Epiphrenic diverticula are usually false diverticula located in the distal 10 cm of the esophagus. Similar to pharyngeal diverticula, they are also usually caused by pulsion from motility disorders that cause an increase in lower esophageal sphincter pressure such as achalasia.

64. Heimlich maneuver

This is used to dislodge FB from air passages.
In performing the maneuver, the rescuer stands behind the choking victim and wraps his arms around his upper abdomen, joining his two hands just below the rib cage and pressing his balled left hand into the victim’s belly. Grasping one fist in the other, the rescuer then makes four sharp upward squeezes or thrusts into the victim’s abdomen, thus forcing out of his lungs the air that will expel the foreign object from the throat. An unconscious victim is laid on his back and the thrusts administered from above. The abdominal thrusts are repeated until the foreign object is expelled.

65. Obstructive Sleep Apnoea

Snoring is defined as a rough rattling noise made on inspiration during sleep by vibration of the soft palate and the uvula. On inspiration, air on its way to the lungs travels by the tongue, soft palate, the uvula and the tonsils. In awake persons the muscles at the back of the throat tightens to hold these structures in place and prevents them from collapsing and vibrating in the airway. During sleep, the soft palate and uvula may vibrate causing the sounds of snoring. Snoring occurs nearly in a third of adults.

Sleep disordered breathing an overview:

This condition encompasses a spectrum of disorders with implications in many fields of medicine. The following are the spectrum in the order of increasing significance:

Primary snoring

Upper airway resistance syndrome

Obstructive sleep apnea hypopnea syndrome

Apnea:

This is defined as a cessation of breathing for 10 seconds. In obstructive sleep apnea, the apneais accompanied by observed ventilatory effort (rise and fall of chest).

Sleep disordered breathing is represented by snoring. Rarely snoring could be totally benign, occurring as a consequence of a removable cause like nasal congestion, excessive fatigue, abnormal sleep position, CNS depressants etc.

It should be stressed that snoring indicates an underlying pathological change that is indeed more significant than the auditory annoyance of one's bed partner.

66. CPAP

Continuous positive airway pressure (CPAP) is a form of positive airway pressure ventilator, which applies mild air pressure on a continuous basis. It keeps the airways continuously open in people who are able to breathe spontaneously on their own, but need help keeping their airway unobstructed. It is an alternative to positive end-expiratory pressure (PEEP). Both modalities stent the lungs' alveoli open and thus recruit more of the lung's surface area for ventilation, but, while PEEP refers to devices that impose positive pressure only at the end of the exhalation, CPAP devices apply continuous positive airway pressure throughout the breathing cycle. Thus, the ventilator itself does not cycle during CPAP, no additional pressure above the level of CPAP is provided, and patients must initiate all of their breaths.

67. Four uses of laser in ENT

Papilloma larynx excision
Vascular tumor removal from larynx
Laser tonsillectomy
Stapedotomy

68. Wegner Grossmann theory

This is the most popular and widely accepted theory which could account for the varying positions assumed by a paralysed vocal cord. This theory was first proposed by Wagner and Grossman (1897). This theory states that in complete paralysis of recurrent laryngeal nerve the cord lies in the paramedian position because the intact cricothyroid muscle adducts the cord. (Because the superior laryngeal nerve is intact). If the superior laryngeal nerve is also paralysed the cord will assume an intermediate position because of the loss of adductive force. This theory has been confirmed by electro myological studies. According to this theory, chest lesions should cause recurrent laryngeal nerve paralysis alone, but in many patients with lung cancer the cord assumes a intermediate position. This has been attributed to the phenomenon of retrograde atrophy of the vagus nerve up to the level of nucleus ambiguus. Paralysed vocal cords may demonstrate some movement due to the action of interarytenoid muscle which gets bilaterally innervated.

69. Trotter’s Triad

Trotter's syndrome is a cluster of symptoms associated with certain types of advanced nasopharyngeal carcinoma. The cause of pain is the mandibular nerve of the foramen ovale, through which the tumor enters the calvarium. Symptoms include the following:

Unilateral conductive deafness due to middle ear effusion
Trigeminal neuralgia due to perineural spread
Soft palate immobility
Difficulty opening mouth

70. Levels of neck lymph nodes