OTORHINOLARYNGOLOGY

OTORHINOLARYNGOLOGY

Solved Question Papers

Third Edition

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JAYPEE

Contents

MBBS PHASE III EXAMINATION

DECEMBER 2008

(Revised Scheme 2)

LONG ESSAYS

1. Discuss the physiological basis and mechanism of deglutition.

- Deglutition or swallowing is a process by which chewed food is emptied from mouth into stomach
- It is initiated voluntarily but ends reflexly.

Stages

Oral stage	*Pharyngeal stage*	*Esophageal stage*
Voluntary stage pushing food from mouth into pharynx	Involuntary stage pushing food from pharynx into esophagus	Involuntary stage transporting food into stomach through series of peristaltic waves in esophagus

a. Oral stage or first stage
   - Voluntary stage where food enters from mouth into pharynx.

   Mechanism
   - Once bolus of food is formed, it is projected on to back of the tongue
   - Tongue is elevated and pressed against hard palate and moved backwards
   - Soft palate is elevated and bolus is propelled into pharynx.

b. Pharyngeal stage or second stage
   - Involuntary stage pushing bolus of food from pharynx into esophagus
   - Also called swallowing reflex
   - Receptors of this reflex are present in vicinity of anterior and posterior pillars of fauces and tonsils
   - Impulse is sent to swallowing center in medullary reticular formation via trigeminal and glossopharyngeal nerves
   - Vth, VIth, IX, X and XII nerves carry motor impulse from swallowing center
   - This reflex also inhibits respiration, sneezing, coughing and vomiting during this stage.

   Mechanism
   - Once bolus enters pharynx, it can enter 4 ways which are:

◆ Back into mouth	◆ Upward into nasopharynx	◆ Forward into larynx	◆ Downward into esophagus

   - Various movements are coordinated, so that bolus enters only into esophagus
   - Entry of bolus in other ways is prevented as follows:

Back into mouth prevented by	*Upward into nasopharynx prevented by*	*Forward into larynx prevented by*
◆ Position of tongue against roof of mouth ◆ High intraoral pressure developed by movements of tongue	◆ Elevation of soft palate	◆ Approximation of vocal cords ◆ Forward and upward movements of larynx ◆ Backward movement of epiglottis to close larynx ◆ Deglutition apnea which is temporary unrest of breathing occurring in this stage

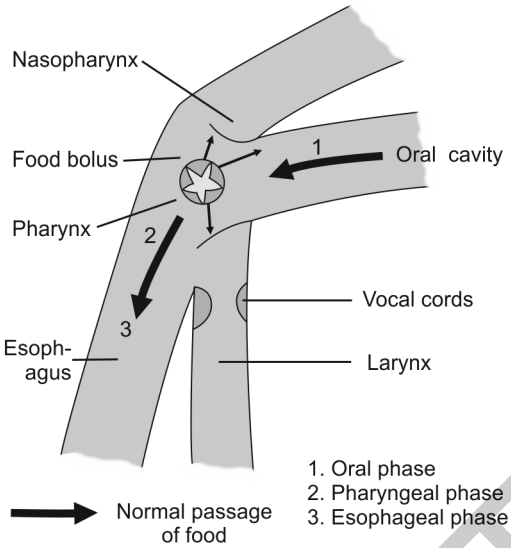

Figure 1: Phase of deglutition

- Entry of bolus into the esophagus is favored by:
  - Closure of rest 3 outlets
  - Stretched opening of larynx by upward movement of larynx
  - Relaxation of the upper esophageal sphincter
  - Peristaltic movements of pharynx
  - Elevation of the larynx lifts the glottis away from the food passage
- Above all movements occur simultaneously in a coordinated fashion to facilitate entry of bolus into esophagus in this 2nd stage of deglutition which lasts only for 1–2 seconds.

c. Esophageal stage or third stage
- Involuntary stage where bolus of food is transported to stomach by a series of peristaltic waves in esophagus.

Mechanism
- Two types of peristaltic waves occurring in esophagus are primary and secondary peristaltic contractions.

Primary peristaltic contractions	*Secondary peristaltic contractions*
◆ These start when bolus reaches upper part of esophagus ◆ These contractions pass downward and propel bolus towards stomach by developing a pressure	◆ Arise locally in esophagus due to distention of upper esophagus by bolus ◆ Travel downward producing a positive pressure (similar to primary peristalsis) ◆ Waves appear even in absence of primary peristaltic waves and push bolus into stomach

Significance

- Deglutition is first and an important process in gastrointestinal system helping food to move from mouth into stomach.

2. Discuss the etiopathology, clinical features and management of Meniere's disease.

- Meniere's disease is a disorder of inner ear characterized by vertigo, sensorineural hearing loss, tinnitus and aural fullness
- Also called as endolymphatic hydrops, as it causes distension of endolymphatic system
- Named after French physician Meniere who first described it in 1861.

Etiopathogenesis

a. Defective absorption by endolymphatic sac
   - Ischemia of endolymphatic sac → poor vascularity → defective absorption of endolymph → increased volume of endolymph → distension of endolymphatic system → rupture of Reissner's membrane → mixing of perilymph and endolymph → vertigo.

b. Vasomotor disturbances
   - Sympathetic overactivity → spasm of internal auditory artery and its branches → interference with functioning of cochlear or vestibular sensory neuroepithelium → deafness and vertigo
   - Anoxia of stria vascularis capillaries → increased permeability → increased transudation → increased production of endolymph → distension of endolymphatic system.

c. Allergy
   - Allergen (food or inhalant) → inner ear (shock organ) → increased production of endolymph.

d. Sodium and water retention
   - Retention of excessive amount of fluid → endolymphatic hydrops.

e. Hypothyroidism.

f. Autoimmune and viral etiologies.

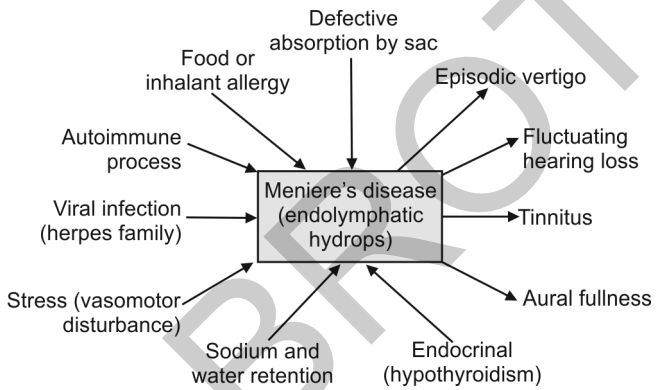

Figure 2: Meniere's disease—etiology and clinical features

Pathology

- Distension of endolymphatic system mainly affecting cochlear duct and saccule and to a lesser extent utricle and semicircular canals
- Complete filling of scala vestibule → marked bulging of Reissner's membrane → herniation of Reissner's membrane through helicotrema into apical part of scala tympani
- Distended saccule lie against stapes footplate
- Utricle and saccule show outpouching into semicircular canals.

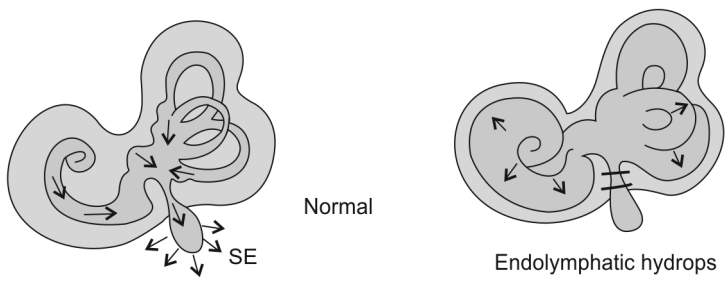

Figure 3: Meniere's disease—pathology

Clinical Features

- Commonly affects males in 35–60 years age group
- Unilateral (50%), but affects other ear in few years.

Symptoms

a. Giddiness (Vertigo)	◆ Episodic attacks in clusters with periods of spontaneous remissions lasting for weeks, months or years ◆ Sudden in onset of varying intensity, duration and interval ◆ Feeling of rotation or "to and fro" or "up and down" movements ◆ Accompanied by: – Nausea, vomiting, ataxia and nystagmus (mild attacks) – Abdominal cramps, diarrhea, cold sweats, pallor and bradycardia (severe attacks) ◆ Sometimes, vertigo preceded by sense of fullness in ear, change in character of tinnitus or discomfort in ear ◆ Sometimes, loud sounds or noise induce vertigo (Tullio phenomenon) due to distended saccule lying against footplate of stapes
b. Sensorineural hearing loss	◆ Fluctuating ◆ Accompanies or precedes vertigo ◆ Improves after attack and normal during remissions ◆ With recurrent attacks, incomplete improvement after attacks leading to slow, progressive and permanent deterioration of hearing ◆ Distortion of sound (tone of sound heard normally in one ear but with higher pitch in other ear) ◆ Intolerance to loud sound (due to recruitment phenomenon)
c. Tinnitus	◆ Low pitched roaring type (sometimes hissing character) ◆ Aggravated during acute attacks ◆ Persists during periods of remission ◆ Acute attack may be preceded by change in intensity and pitch of tinnitus
d. Sense of fullness or pressure	◆ Accompanies or precedes an attack
e. Other features	◆ Emotionally upset due to apprehension of repetitive attacks

Signs

Nystagmus	During acute attack Quick component towards unaffected ear

Investigations

a. Tuning fork tests (reveals sensorineural hearing loss)	i. Rinnes test – Positive (AC > BC) ii. Weber test – Lateralized to better ear iii. Absolute bone conduction test – Reduced in affected ear

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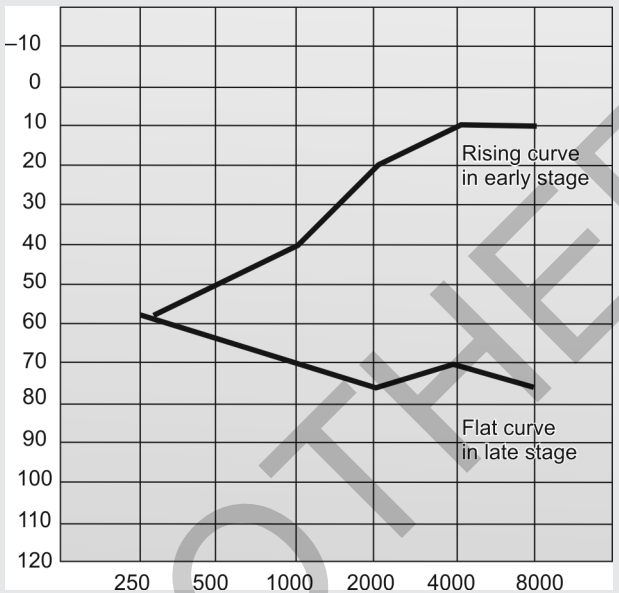
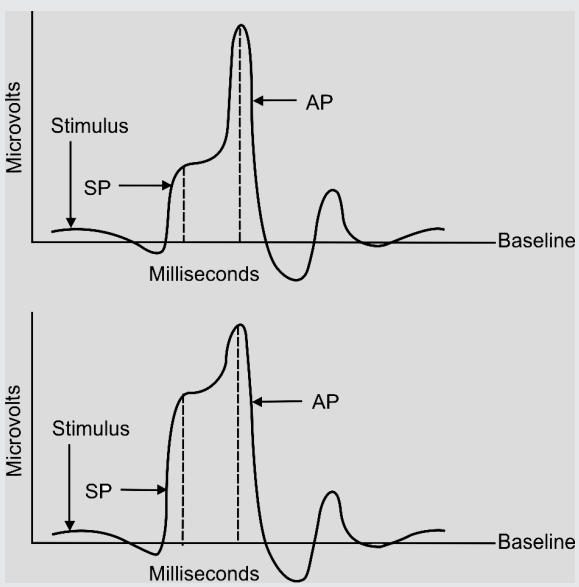
b. Pure tone audiometry	◆ Rising curve at early stage when lower frequencies are affected ◆ Flat or falling curve with involvement of higher frequencies  **Figure 4:** Meniere's disease—audiogram
c. Speech audiometry	◆ Discrimination score of 55–85% in remission phase but greatly impaired during and immediately after attack
d. Special audiometry tests (to indicate cochlear lesion and differentiate from retrocochlear lesions)	i. Recruitment test – Positive ii. Short increment sensitivity index (SISI) test – >70% iii. Threshold tone decay test – <25 dB
e. Electrocochleography	◆ Ratio of summating potential to action potential >30% (normal: 30%)  **Figure 5:** Meniere's disease—electrocochleograph

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f. Calorie test	◆ Reduced response on affected side (75%) ◆ Reveals canal paresis on affected side
g. Glycerol test	*Principle* ◆ Glycerol is a dehydrating agent which reduces endolymph pressure and causes improvement in hearing *Procedure* ◆ Record audiogram and speech discrimination score ◆ Give 1.5 mL/kg of 95% glycerol with equal amount of water and little flavouring agent ◆ Record audiogram and speech discrimination score at hourly interval for 2–3 hours *Interpretation* ◆ Positive results means improvement of 10 dB in two or more adjacent octaves or gain of 10% discrimination score ◆ There is also improvement in tinnitus and sense of fullness in ear *Significance* ◆ Test has diagnostic and prognostic value

Differential Diagnosis

Vestibular neuronitis	*Benign paroxysmal positional vertigo*	*Acoustic neuroma*
◆ Acute onset vertigo ◆ Recurrent vertigo not usual ◆ No sensorineural deafness ◆ No tinnitus ◆ No recruitment phenomenon	◆ Recurrent, momentary, positional vertigo ◆ Not associated with nausea and vomiting ◆ No sensorineural deafness ◆ No tinnitus ◆ No recruitment phenomenon	◆ Chronic unsteadiness ◆ Vertigo, neither marked nor paroxysmal ◆ Progressive unilateral sensorineural deafness ◆ No recruitment ◆ Associated cranial nerve palsies

Treatment

Conservative

A. Acute attack

Supportive	*Specific*
◆ Reassurance and psychological support to allay worry and anxiety ◆ Bed rest with head supported on pillow to avoid excessive movements	a. Vestibular sedatives (to relive vertigo) – 15–75 mg prochlorperazine daily orally or IM – 25 mg chlorpromazine TID – 5–10 mg diazepam IV (tranquillizing effect and suppresses activity of medial vestibular nucleus) – 0.4 mg atropine SC b. Vasodilators i. Carbogen inhalation (5% CO_2 and 95% O_2) - Good cerebral vasodilator and improves labyrinthine circulation ii. Histamine drip - 2.75 mg histamine diphosphate dissolved in 500 mL glucose by IV drip

B. Chronic phase

Supportive	*Specific*
◆ Reassurance, particularly important during acute attack to anxious patient ◆ Cessation of smoking to prevent vasospasm by nicotine ◆ Low salt diet (limit salt intake to 1.5–2 g/day) ◆ Avoid excessive consumption of water ◆ Avoid tea, coffee and alcohol	a. Vestibular sedatives – 10 mg prochlorperazine TID for 2 months, then reduced to 5 mg TID for 1month b. Vasodilators – 50 mg nicotinic acid TID just before meals – 8–16 mg betahistine TID c. Diuretics – 40 mg furosemide on alternate day along with potassium supplement (if not controlled by vestibular sedatives or vasodilators)

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◆ Avoid stress and prefer relaxation exercises like yoga, meditation ◆ Avoid activities requiring good body balance like flying, etc.	d. Propantheline bromide – 15 mg TID alone or in combination with vasodilator e. Elimination of allergen f. Hormone replacement g. Chemical labryinthectomy – Intratympanic injection of gentamicin daily or biweekly into middle ear to cause destruction of vestibular labyrinth

Operative

Indications

- Failure of conservative treatment.

Conservative procedures	***Destructive procedures***
Indications ◆ Hearing still useful with disabling vertigo *Techniques* a. Decompression of endolymphatic sac b. Endolymphatic shunt operation – Involves insertion of a tube connecting endolymphatic sac with subarachnoid space to drain excess endolymph c. Sacculotomy (Fick's operation) – Involves puncturing saccule with a needle through footplate of stapes d. Cody's tack procedure – Involves placement of a stainless steel tack through footplate of stapes to cause periodic decompression of saccule when it gets distended e. Cochleosacculotomy (otic-periotic shunt) – Involves puncturing cochlear duct by a curve needle passed through round window to drain it into perilymph f. Section of vestibular nerve – Selective sectioning of vestibular nerve by exposing it through retrosigmoid or middle cranial fossa approach g. Ultrasonic destruction of vestibular labyrinth h. Cervical sympathectomy (to correct microcirculatory fault in labyrinth)	*Indications* ◆ Unserviceable cochlear functions *Techniques* ◆ Labyrinthectomy – Involves complete destruction of membranous labyrinth by opening through lateral semicircular canal by transmastoid route or through oval window by transcanal approach

Recent Advances

- Intermittent low-pressure pulse therapy (Meniett device therapy)
  - Delivery of intermittent positive-pressure waves produced by an instrument called Meniett device (placed in external ear) to round window membrane through a ventilation tube inserted after myringotomy
  - Pressure waves produced pass through perilymph and reduce endolymph pressure by redistributing it through various communication channels.

Advantages

- Nondestructive
- Self administrable.

SHORT ESSAYS

3. Conductive deafness.

- Conductive deafness or hearing loss is due to defect in sound conducting mechanism anywhere between external auditory canal and footplate of stapes.

Causes

Congenital	*Acquired*				
	External ear	*Tympanic membrane*	*Middle ear*	*Eustachian tube*	*Systemic causes*
◆ Meatal atresia ◆ Fixation of footplate of stapes/ head of malleus ◆ Ossicular discontinuity ◆ Congenital cholesteatoma ◆ Congenital absence of oval window	◆ Wax (MC) ◆ Otomycosis ◆ Otitis externa ◆ Foreign bodies ◆ Polyps ◆ Traumatic stenosis ◆ Keratosis obturans ◆ Tumors (osteoma, exostosis)	◆ Bullous myringitis ◆ Traumatic rupture ◆ Perforation due to middle ear infection	a. Traumatic – Barotrauma – Hemotympanum – Ossicular discontinuity – Fracture base of skull b. Inflammatory – ASOM – CSOM – Serous OM – Adhesive OM c. Chronic infections – Tuberculous OM – Syphilitic OM d. Neoplasms (rare) – Glomus jugulare – Squamous cell carcinoma e. Miscellaneous – Otosclerosis	◆ Eustachian catarrh (Very common) ◆ Eustachian tube dysfunction (due to enlarged adenoids, nasopharyngeal growth, etc.) ◆ Barotrauma	◆ Wegener's granulomatosis ◆ Relapsing polychondritis ◆ Fibrous dysplasia ◆ Eosinophilic granuloma ◆ Sarcoidosis

Clinical Features

Symptoms	*Signs*
◆ Deafness ◆ Aural fullness ◆ Pain ◆ Associated with tinnitus, vertigo	◆ Otoscopy may reveal pathology in external ear or tympanic membrane ◆ Bone conduction better than air conduction ◆ Good speech discrimination

Degree of Deafness (Social Classification)

◆ Normal hearing	0–20 dB
◆ Mild deafness	20–40 dB
◆ Moderate deafness	40–60 dB
◆ Severe deafness	60–80 dB
◆ Profound deafness	>80 dB

Investigations

Tuning fork tests			*Audiometry*	*Radiography*
Rinne's test	Weber's test	Absolute bone conduction test		
Negative (BC > AC)	Lateralized to poorer ear	Normal	BC > AC with air-bone gap Loss not more than 60 dB	Schuller's view (X-ray)

Treatment

	Indications	*Techniques*
Conservative		
Hearing aid	◆ Unfit for surgery ◆ Refusing surgery ◆ Failure of surgery	

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	Indications	*Techniques*
Operative (principles)		
a. Removal of canal obstruction	◆ Impacted wax ◆ Foreign body ◆ Osteoma or exostosis ◆ Keratosis obturans ◆ Meatal atresia	
b. Removal of fluid	◆ Acute otitis media ◆ Serous otitis media ◆ Hemotympanum	i. Myringotomy with or without grommet insertion
c. Removal of mass from middle ear	◆ Middle ear tumors ◆ Cholesteatoma behind intact TM	i. Tympanotomy followed by removal of mass
d. Repairing TM perforation	◆ Traumatic rupture of TM ◆ Pathological rupture of TM	i. Myringoplasty ii. Tympanoplasty
e. Restoring ossicular continuity	◆ Otosclerotic fixation of footplate of stapes or head of malleus ◆ Traumatic disruption of ossicular continuity	i. Stapedectomy ii. Tympanoplasty iii. Ossicular reconstruction

4. Extratemporal complications of chronic suppurative otitis media.

- Extension of infection middle ear (suppurative otitis media) to adjacent structures is common if not properly treated.

Extratemporal (Cervical) Complications

- Subperiosteal abscess
- Zygomatic abscess
- Bezold's abscess
- Luc's abscess
- Citelli's abscess
- Parapharyngeal or retropharyngeal abscess
- Thrombophlebitis of jugular or subclavian vein.

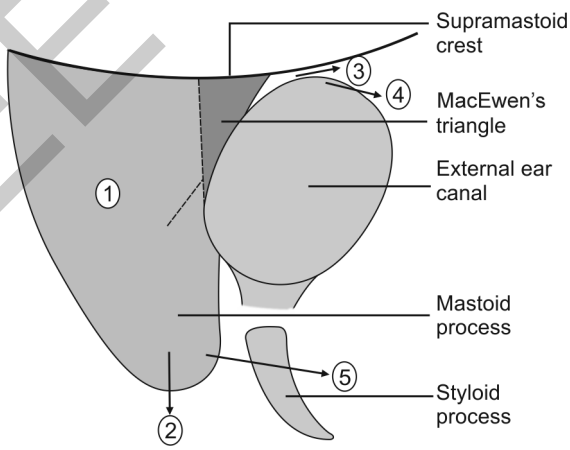

Figure 6: Showing directions of mastiod abscesses (1) Subperiosteal, (2) Bezold's, (3) Zygomatic, (4) Luc's and (5) Citelli's

a. Postauricular abscess (subperiosteal abscess)
   – Commonest abscess forming over mastoid

Location	*Clinical features*
◆ Over MacEwen's triangle	◆ Increased intensity of pain over mastoid antrum ◆ Pinna displaced forward, outward and downward (erection of pinna)

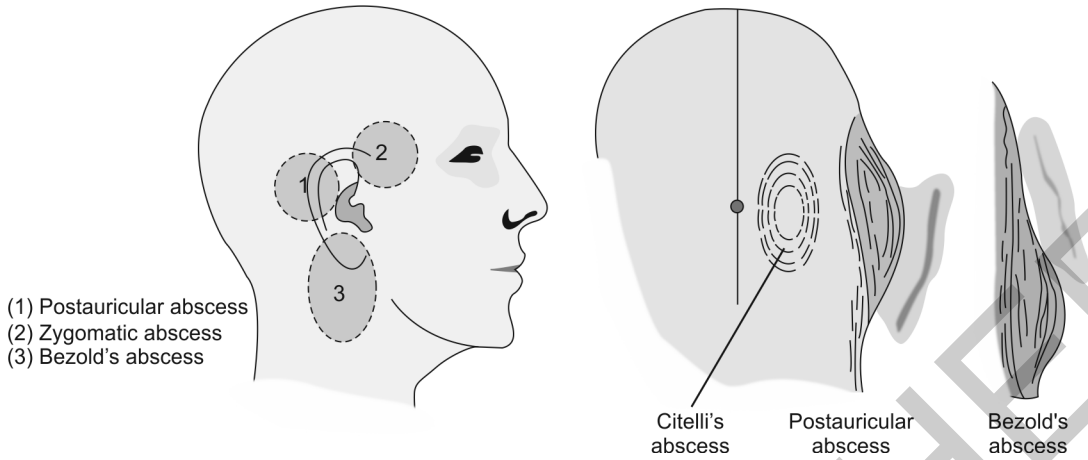

Figure 7: Extratemporal complications

b. Zygomatic abscess

Etiology	*Location*	*Features*
◆ Infection of zygomatic air cells situated at posterior root of zygoma	◆ Front of and above pinna, either superficial or deep to temporalis muscle	◆ Associated with edema of upper eyelids

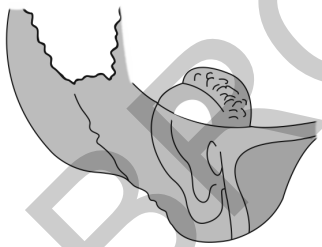

Figure 8: Zygomatic subperiosteal abscess

c. Bezold's abscess
   – Bezold's abscess is a subperiosteal abscess formed as a complication of acute coalescent mastoiditis.

Etiology	*Location*
◆ Perforation and necrosis of medial side of tip of mastoid	◆ Deep to anterior border of sternomastoid, pushing muscle outwards ◆ Between tip of mastoid and angle of jaw by followed posterior belly of digastric ◆ Upper part of posterior triangle ◆ Lower down in neck along carotid vessels

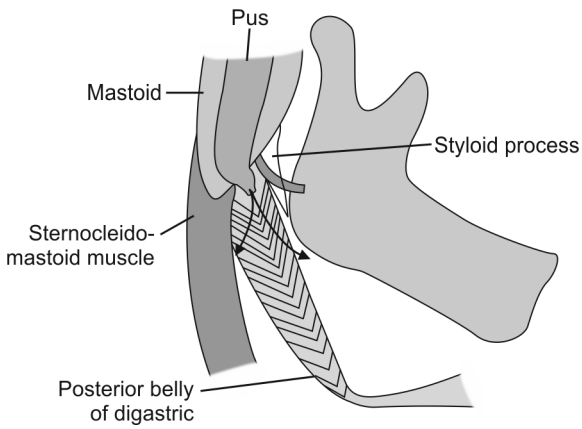

Figure 9: Bezold's abscess

Clinical features

– Sudden onset.

Symptoms	Signs
◆ Pain at back of ear or neck ◆ Fever ◆ H/O purulent ear discharge	◆ Tender brawny swelling in upper part of neck ◆ Torticollis ◆ Abscess may extend downward within sheath of muscle (sinking abscess)

Investigations	Differential diagnosis
◆ CT scan of mastoid and swelling	◆ Acute upper jugular lymphadenitis ◆ Abscess or mass in lower part of parotid gland ◆ Infected branchial cyst ◆ Parapharyngeal abscess ◆ Jugular vein thrombosis

Treatment

Operative

– Incision and drainage
  - Neck abscess is incised at dependent part and drained using drain
– Treatment of underlying cause
  - Cortical mastoidectomy for coalescent mastoiditis with careful exploration for fistulous opening
  - Intravenous antibiotics based on culture sensitivity at time of I and D.

d. Meatal abscess (Luc's abscess)

Location	Features
◆ Deep part of bony meatus	◆ Pus brakes through bony wall between antrum and external osseus meatus ◆ May burst into meatus

e. Behind mastoid (Citelli's abscess)

Location

– Behind mastoid, more towards occipital bone.

f. Parapharyngeal or retropharyngeal abscess
  – Results from infection of peritubal cells due to acute coalescent mastoiditis.

5. Ototoxicity.

- Ototoxicity is capacity of a drug or chemical to cause functional impairment and cellular degeneration of tissues of inner ear especially end organs and neurons of cochlear and vestibular divisions of VIII nerve.

Etiology (Ototoxic Drugs)

a. Aminoglycoside antibiotics	◆ Cochleotoxic – Neomycin – Kanamycin – Framycetin – Tobramycin
	◆ Vestibulotoxic – Streptomycin – Gentamicin
b. Diuretics	◆ Ethacrynic acid ◆ Furosemide ◆ Bumetanide

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c. Cytotoxic drugs	◆ Cisplatin ◆ Nitrogen mustard ◆ Carboplatin
d. Analgesics	◆ Salicylates ◆ Indomethacin ◆ Phenylbutazone ◆ Ibuprofen
e. Antimalarials	◆ Quinine ◆ Chloroquinine
f. Macrolide antibiotics	◆ Erythromycin
g. Glycopeptides antibiotics	◆ Vancomycin
h. Chemicals	◆ Alcohol ◆ Tobacco ◆ Marijuana ◆ Carbon monoxide
i. Miscellaneous	◆ Ampicillin ◆ Propranolol ◆ Propylthiouracil ◆ Deferoxamine ◆ Imipramine ◆ 5-hydroxytryptamine ◆ Carbamazepine

Clinical Features

- Same for all drugs but varies in severity, timing and duration.

Symptoms	*Signs*
i. Tinnitus – First warning symptom – Usually high pitched and continuous – May be unilateral or bilateral ii. Hearing loss – Sensorineural deafness affecting high frequencies iii. Vertigo (with vestibulotoxic drugs) – Sensation of continuous rotation after turning head or turning over in bed iv. Disequilibrium – Severely unsteady and bed ridden in severe cases	i. Bilateral loss of labyrinthine function (bobbing oscillopsia) – On caloric and rotational tests

Investigations

- Estimation of drug concentration in body
- Pure tone audiometry (high tone loss)
- Caloric test
- Electrocochleography
- Otoacoustic emission
- BERA.

Treatment	*Prophylaxis*
Conservative ◆ Mild hypnotics or tinnitus maskers for tinnitus ◆ Reassurance and regular physiotherapy for disequilibrium ◆ Hearing aids or cochlear implants for severe hearing loss	◆ Early recognition and discontinuation of ototoxic drug ◆ Regular monitoring of serum drug concentration ◆ Avoiding prescription of ototoxic drugs as much as possible ◆ Prescription of drug within maximum recommended doses

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