

The logo features a horizontal, oval-shaped graphic with a dark background. Inside the oval, there are stylized, glowing elements in shades of red, orange, and yellow, resembling a cross-section of an ear or a medical instrument. The text "ENT SCHOLAR" is overlaid in white, uppercase letters across the center of the graphic.

ENT SCHOLAR

# Clinical Examination Techniques in Otology

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# Introduction

Clinical reasoning and bed side examination was part of clinical medicine was practised by ancient Indian, Egyptian, Babylonian and Chinese physicians.

“He who studies medicine without books sails an uncharted sea, but he who studies medicine without patients does not go to sea at all.” Osler.

This in a nutshell is the importance of patient in the learning process of a medical graduate. New technologies have pushed the art of clinical examination to the back seat. Still clinically examining the patient is a must before ordering investigations<sup>1</sup>. The current technology tempts the physician to order the tests before examining the patient actually. This is amplified by the fact that a comatose patient invariably undergoes a CT scan brain before being examined by a physician only to be discovered later that the altered consciousness was due to metabolic abnormalities / drug overdose. Current study<sup>2</sup> involving medical students during their clerkship period revealed that according to these students the relative contributions of history taking, clinical examinations, investigations towards the diagnosis were as follows:

1. History taking 62%
2. Physical examination 17%
3. Diagnostic tests 21%

## History Taking:

Before proceeding with clinical examination perse a good history taking is a must. Without proper history taking it is not possible to come to a reasonably correct diagnosis by clinical examination alone. The importance of accurate history taking and documentation should be stressed and restressed at this juncture. This is rather important in evaluating neuro-otological disorders. If a patient presents with vertigo as the primary complaint, clinical examination of the ear could turn out to be unremarkable, and one has to resort to accurate history alone to come to a conclusion<sup>3</sup>. A questionnaire completed by the patient will go a long way in improving the accuracy of clinical examination.

## Ideal questionnaire:

This should include:

1. Various aspects of history including history of present ailment
2. Previous evaluations if any
3. Previous medical / surgical therapy
4. Social history
5. History of trauma

## 6. Details regarding onset and exacerbation of symptoms

History should include:

History of:

1. Previous ear surgery - This is rather important. This helps in deciding whether the ailment is caused by a recurrence of the disease (recurrent mastoiditis), or iatrogenic (caused as a complication of the surgical procedure). For example injury to torus tubaris during adenotonsillectomy could lead to acute otitis media due to blocked eustachean tube orifice. This could also explain a scar seen in the post aural region.

2. Previous head injury<sup>4 5</sup> - This can cause giddiness. Fractures involving petrous portion of temporal bone can cause giddiness, sensorineural hearing loss (commonly high frequency type), and facial palsy. BPPV has been documented to occur most commonly following head injury. Studies reveal that nearly a quarter of these patients have history of head injury<sup>6</sup>. Other probable causes of auditory manifestations following head injury include:

a. Brain stem concussion

b. Injury to 8<sup>th</sup> nerve complex

c. Post traumatic endolymphatic hydrops (secondary Meniere's disease)<sup>7</sup>

d. Perilymph fistula

e. Labyrinthine concussion – This is actually an enigma. If post traumatic vertigo resolves spontaneously over time then this diagnosis is considered. This diagnosis is infact made retrospectively.

f. Cervical vertigo – This is again a poorly understood condition. Current theories suggest that cervical vertigo occurs due to compression of blood vessels in the neck and alteration of sensory input to the vestibular system.

3. Systemic diseases like diabetes<sup>8</sup>/ hypertension – Diabetes mellitus is known to cause sensorineural hearing loss.

4. Use of ototoxic drugs – This history is a must. Drugs used to other ailments like Streptomycin can prove to be toxic to the inner ear<sup>9</sup>. This could cause tinnitus, hearing loss and vestibular symptoms. Ototoxicity is usually associated with bilateral high frequency sensorineural hearing loss. It should be stressed that currently there is not therapy available to reverse drug induced sensorineural hearing loss.

5. Exposure to noise during work – Continuous exposure to loud noise during work is one of the common cause of sensorineural hearing loss<sup>10</sup>. It is of two types. Temporary threshold shift where the sensorineural hearing loss is reversible if the patient is removed from the offending environment. Continuous exposure to noise would lead to permanent threshold shift when the deafness become irreversible.

6. Family h/o deafness – Familial history of hearing loss is of commonly sensorineural type. If

conductive hearing loss is reported then otosclerosis should be considered. There is clearly no consensus at present regarding the molecular basis of familial sensorineural hearing loss<sup>11</sup>.

7. Atopy / allergy – can lead to infections of nasal and sinus mucosa causing secretory otitis media.

The classic symptoms of ear disease are as follows:

1. Deafness
2. Discharge
3. Tinnitus
4. Pain
5. Vertigo

Deafness:

The patient must be asked whether deafness was sudden in onset, or gradual in onset. If deafness is sudden in onset the triggering event if any must be sought for. For example, deafness following head injury may be caused by a fracture of petrous portion of temporal bone. If the damage occurs to the auditory nerve the patient may have sensori neural hearing loss. Damage to 8th nerve is common following transverse fractures of temporal bone. Sometimes acute trauma may lead to dislocation of the ossicles causing conductive hearing loss. Of the 3 ossicles incus is the most commonly dislocated bone following trauma.

Deafness could be of 3 types:

1. Conductive deafness – Caused by pathology in the conducting system of external ear and middle ear cavities. Rarely inner ear causes that spare the hair cells of the cochlea but affecting scala vestibuli, helicotrema, scala tympani can cause conductive deafness. This concept of inner ear conductive hearing loss was proposed by Gloris & Davis during 1960<sup>13</sup>. They attributed conductive deafness to the stiffness of cochlear partition in these patients.
2. Sensorineural hearing loss – This is caused by inner ear / auditory nerve causes
3. Mixed deafness – This type has varying degrees of both the above mentioned types of deafness

Clinically sensorineural hearing loss can be suspected if the patient volunteers history of inability to comprehend speech. These patients classically say that they can perceive some sound but unable to comprehend the spoken word. This could be due to lack of speech discrimination which is commonly seen in sensorineural hearing loss. Associated complaints of tinnitus also point towards sensorineural hearing loss.

Conductive deafness can be differentiated from sensori neural deafness in a conscious patient easily by doing a tuning fork test. Commonly used tuning fork tests are 1. Rinne, 2. Weber, and 3. Absolute bone conduction test.

Transient deafness after head injury may be commonly caused by a haematoma in the middle ear cavity. Following head injury the other common triggering event for deafness is viral infections. Common among them are mumps, measles etc. Deafness following viral infections are purely sensorineural in nature. The presence of wax is sufficient to cause fluctuating hearing loss which is conductive in nature.

Causes of fluctuating hearing loss are:

1. Presence of wax (conductive deafness) - Patient will c/o severe itching in the affected ear . These patients also have tinnitus because the masking effect of the external ear to endogenous noise produced by human body is lost / reduced.
2. Meniere's disease (sensorineural deafness)
3. Peri lymph fistula (sensorineural deafness) <sup>12</sup> could be due to the ability of the fistula to seal on its own.

In patients with deafness associated with ear discharge the presence of perforation in the ear drum could be the cause.

In all patients with c/o deafness a proper drug history is a must. Ototoxic drugs like streptomycin, gentamycin and aspirin may cause irreversible damage to the hair cells of the cochlea causing sensori neural hearing loss. These drugs also sensitises the hair cells of the cochlea to damage due to noise exposure, hence occupational history of these patients is a must. H/O exposure to loud noise must be sought.

# causes of conductive deafness

**Congenital causes**

**Acquired causes**

**Ext ear**

Atresia

Exostosis

Impacted wax

**Ext ear**

Perforated Ear drum

Tympanosclerosis

**Middle ear**

**Middle ear**

Congenital malleus fixation

Congenital stapes fixation

S.O.M.

Otosclerosis

A.O.M.

Ossicular disruption

Trauma

Haemotympanum

## Discharge:

Ear discharge is one of the common problems that brings the patient to the doctor. Before examining the patient a detailed history regarding

1. Duration of the discharge
  2. Quantity of discharge
  3. Quality of discharge
  4. Aggravating & relieving factors
- must be sought for.

Causes of ear discharge include:

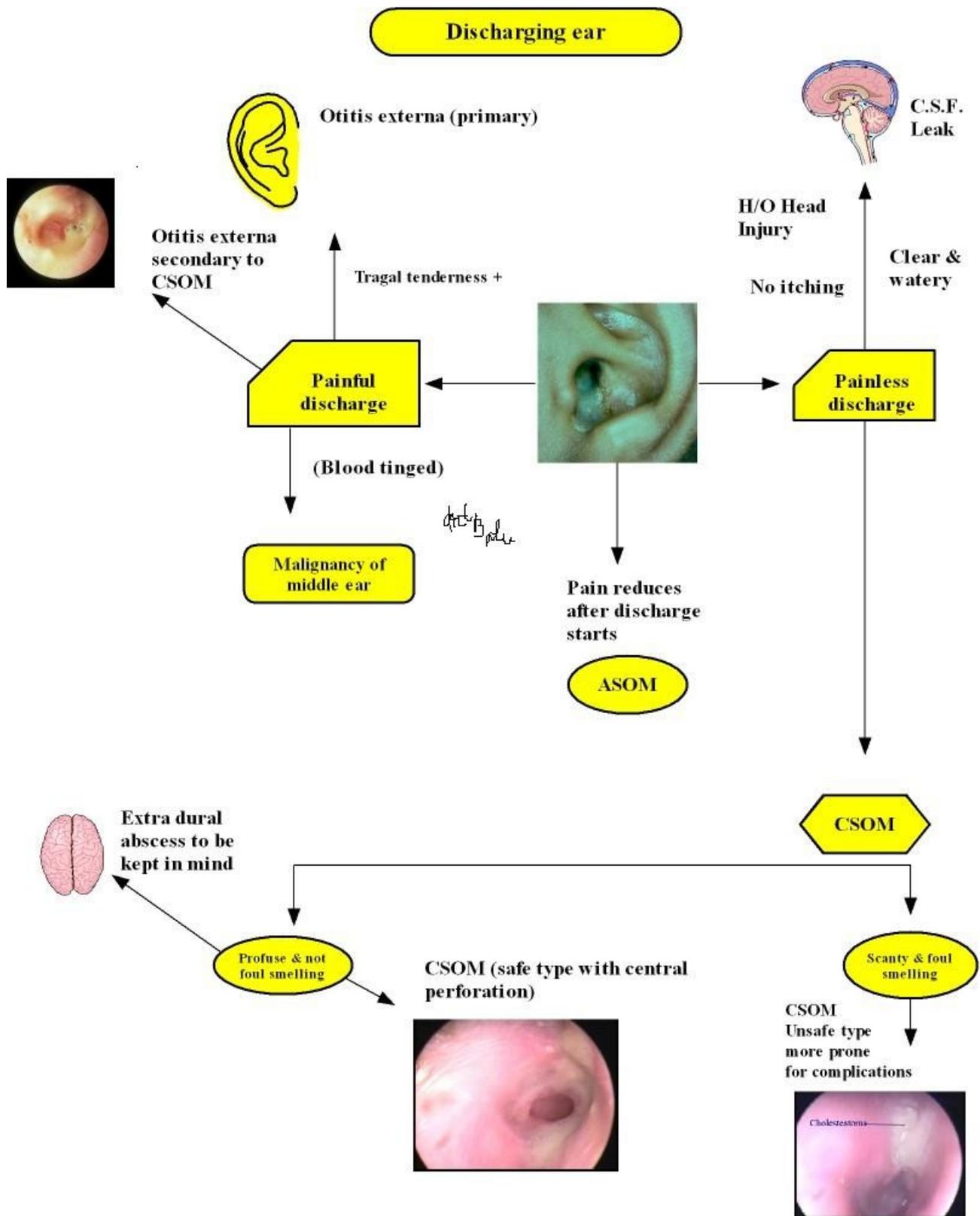
1. ASOM
2. CSOM (with / without cholesteatoma)
3. CSF Otorrhoea
4. External / Middle ear neoplasm
5. Granulomatous diseases like tuberculosis, atypical mycobacteria, Wegner's granulomatosis
6. Parotid gland fistula presenting as saliva discharging out of the ear. These patients invariably have persistent Foramen tympanicum (Foramen of Huschke) through which discharging saliva finds its way into the external auditory canal. These patients characteristically complain of ear discharge during mastication.
7. Otitis external (due to bacterial / fungal infections)
8. Foreign body in the external canal. Vegetative foreign bodies are more prone to cause discharge from the external auditory canal. Usually this discharge is foul smelling.

If the duration of discharge is short then acute conditions must be borne in mind. Common acute conditions which can lead to ear discharge are

1. A.S.O.M. - here the discharge is Serosanguinous in nature (blood tinged), preceded by an episode of severe ear pain, pain subsides as soon as discharge starts, preceding episode of upper respiratory infection.
2. Otomycosis - common fungi affecting the external canal are candida and aspergillus fumigatus. Candida gives a curdy appearance in the external ear canal. In a dried up state it could appear like a cotton wool. Aspergillus fumigatus appears as a black color patches in the external auditory canal. These patients have ear discharge mostly just wetness, not profuse in nature, associated with intense itching.
3. C.S.F. Otorrhoea - The discharge is watery in nature, there is absolutely no mucoid elements in the discharge. This clear discharge starts when the affected ear assumes a dependent position. Biochemical analysis of this discharge will show that it contains glucose which is normally absent in purulent ear discharges.

The color of ear discharge could be a pointer to the cause of otorrhoea. Purulent discharge indicates presence of infection, where as a bloody discharge could be caused by trauma or presence of granulation tissue associated with infection.

Mucoid discharge	Common in CSOM
Mucopurulent	CSOM associated with mastoiditis
Serous	ASOM
Serosanguinous	ASOM and Otitis externa, trauma
Watery	CSF otorrhoea
Saliva	Parotid fistula



Ear discharge associated with itching is commonly due to otomycosis.

Presence of mucoid discharge indicates perforation of ear drum because the external auditory canal does not contain mucous glands. Hence mucoid discharge will have to come from the middle ear cavity where there are lots of mucinous glands.

Foul smelling ear discharge is almost always associated with cholesteatoma or neoplasm. Cholesteatoma characteristically causes a musty odor.

If discharge is associated with pain then it is probably due to otitis externa since acute otitis media tends to become painless as soon as the discharge starts. Discharge with persistent pain indicate otitis externa.

Otitis externa is characterised by thin scanty discharge associated with ear itching.

Tests to identify CSF Otorrhoea:

Bedside test - One useful bedside test for CSF otorrhoea is Handkerchief test. If the secretion is mopped with a handkerchief and allowed to dry, there will be stiffening of the handkerchief if the discharge is from the middle ear because of the presence of mucous, if the discharge is csf there is no stiffening seen.

Most sensitive diagnostic test is estimation of Beta 2 transferrin in the secretions. Beta 2 transferrin is seen only in the CSF and is absent in other types of discharges.

Another important factor in the history taking is asking for the quantity of discharge. If the discharge is profuse the following conditions must be borne in mind :

1. Chronic mastoiditis
2. Mastoid reservoir
3. Extra dural abscess.

Of these three in extra dural abscess the discharge is so profuse the external canal fills up with pus immediately after mopping. The presence of mastoiditis or mastoid reservoir can be ruled out by looking out for tenderness in the mastoid tip area. In children with well pneumatized mastoids the pus may cause erosion of the outer cortex and present as a collection just under the mastoid periosteum. This condition is known as sub periosteal abscess. If the ear discharge is scanty and foul smelling osteitic reaction due to infection must be suspected. This is frequently caused by the presence of cholesteatoma in the middle ear cavity associated with bone erosion.

### Otalgia:

This is defined as ear pain. There are two distinct types of otalgia encountered during clinical examination of a patient.

1. Primary otalgia
2. Referred otalgia

Causes of Primary otalgia include:

1. Otitis externa
2. Otitis media
3. Mastoiditis

Referred otalgia is caused by pathologies involving areas other than the ear. This is due to the common sensory innervation of those areas with that of the ear.

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.

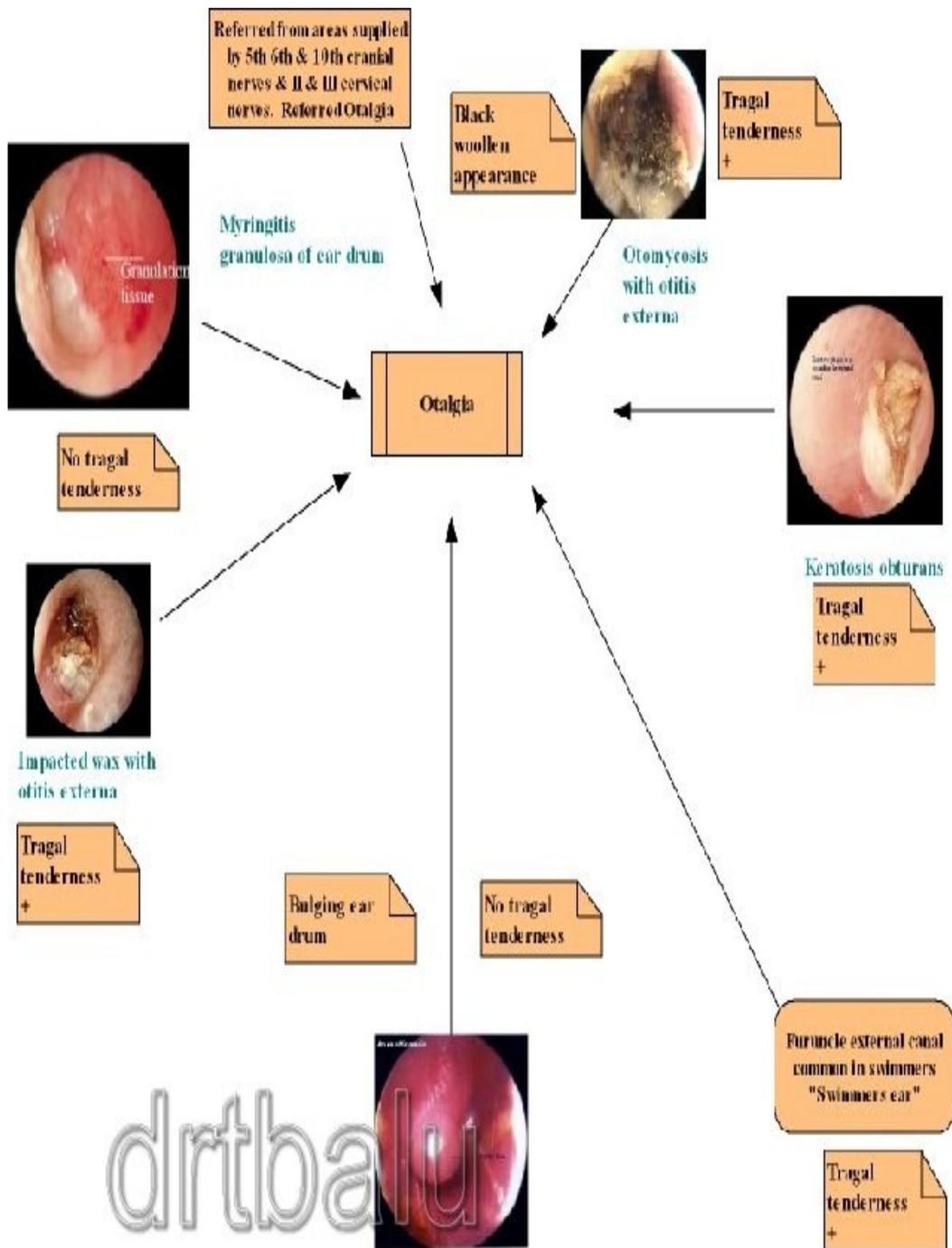
Causes for referred otalgia should be diligently searched for in a patient with ear pain, with clinically normal ear.

Innervation of the pinna and external auditory canal is from the following sources:

1. Auriculo temporal branch of V cranial nerve
2. C1 and C2 nerves
3. Jacobson's branch of glossopharyngeal nerve
4. Arnold's branch of Vagus nerve
5. Ramsay Hunt branch of facial nerve

Hence any of the noxious stimulations conveyed by these nerves will be interpreted as otalgia by brain.

Pain due to inflammation in the external ear is intense and is associated with swelling of the external auditory canal. This can be differentiated from pain arising from middle ear inflammation by the presence of tenderness on pressing the tragus. This sign is known as the tragal sign. Tragal sign is negative in otalgia due to middle ear causes. Pain due to mastoiditis (inflammation of mastoid air cells) can be differentiated from pain due to otitis externa by the presence of three point tenderness. Three point tenderness is elicited by using the middle finger to apply pressure over the well of the concha, index finger is applied over the mastoid process, and the thumb is used over the mastoid tip. The pressure over the well of the concha indicates tenderness over the antral area, tenderness over the mastoid process indicates the presence of mastoiditis, and tenderness over the tip of the mastoid process indicate inflammation and thrombosis of mastoid emissary vein.



drtbalu

(Acute Otitis Media)

### Innervation of Middle ear:

The middle ear receives sensory afferents primarily from the glossopharyngeal component of tympanic plexus. These sensory afferents are largely formed by the Jacobson's branch of glossopharyngeal nerve. The facial nerve receives sensation from the medial portion of pars flaccida and pars tensa by way of its chorda tympani branch.

### Innervation of Inner ear:

The inner ear is supplied by vestibulocochlear nerve. This nerve doesn't have pain fibers. Hence significant inner ear pathology can develop without pain. These nerves are sensitive to stretch hence in patients with endolymphatic hydrops there may be fullness of the membranous portion of inner ear which is perceived as pain by the patient.

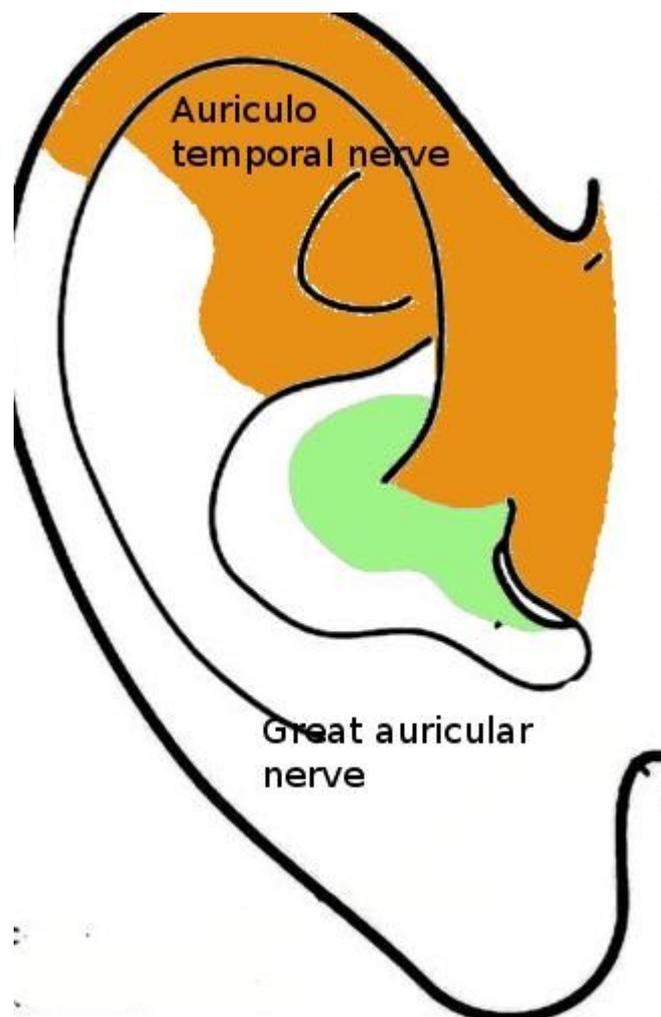


Figure showing innervation of Pinna

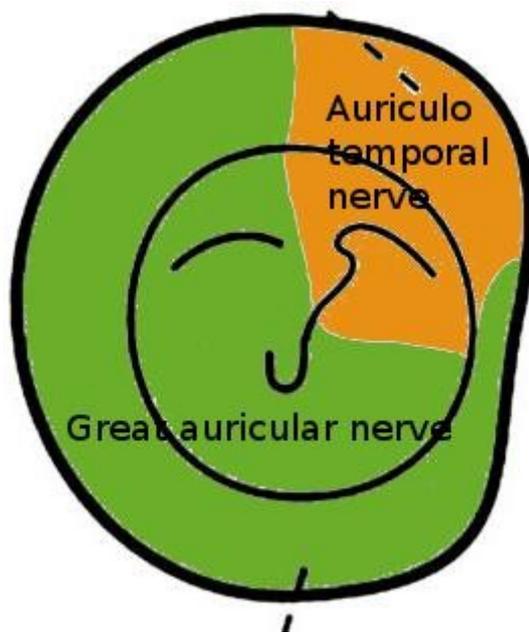


Figure showing innervation of external auditory canal

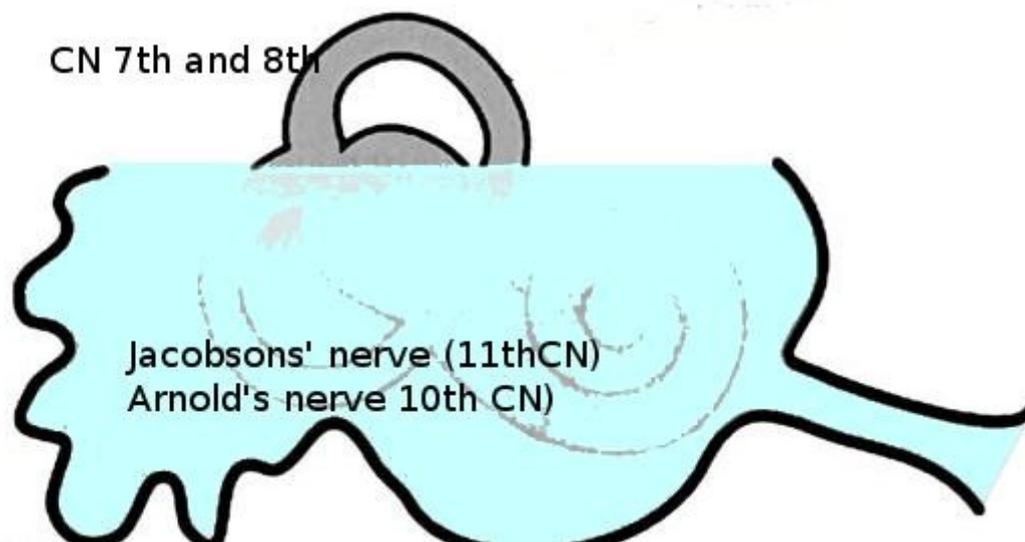


Figure showing innervation of middle ear

External ear causes of otalgia:

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 externa 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 of 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 traumatises 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 colonisation. 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 mastigation
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 characterised 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: *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.

Acute localised 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

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 mucopurulent 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.

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 characterised by intense itching, infact 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.

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 Fungal debris (black in case of aspergillus and white in the case of candida).

Invariably the infection is mixed type.



Otomycosis Aspergillous Niger



Otomycosis *Candida albicans*



Figure showing furuncle involving external auditory canal

## 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 cartilagenous portion of the external canal. This condition can cause complications like facial nerve involvement, and spread to the intracranial structures.

## Chronic Myringitis:

Is defined as loss of tympanic membrane epithelium for more than a month in the absence of middle ear pathology. The etiology of this condition remains largely unknown. It may follow acute infections, trauma or previous ear surgery. It is often seen without any of these so called predisposing conditions.

The common presenting symptom is otalgia with associated otorrhoea, hearing loss, tinnitus, aural fullness and pruritis.

Otalgia in these patients are mediated by sensory afferents from the external auditory canal and lateral portion of ear drum. Granulation tissue may be seen in the external auditory canal and lateral surface of ear drum. Identification of this condtion is vital as it would avoid unnecessary tympanomastoid surgery.



Figure showing chronic myringitis

### Bullous myringitis:

This condition is characterised by acute infection of ear drum / external auditory canal producing intraepithelial fluid collection. These patients commonly present with bullae in the lateral surface of ear drum (Bullous myringitis) or over the external auditory canal (Bullous otitis externa). These patients classically present with severe otalgia associated with serous / bloody discharge. If otalgia is intense decompression of the bullae can be performed. These patients usually respond well to topical antibiotic drops, oral antibiotics, steroids and analgesics.



Figure showing Bullous myringitis

### Middle ear causes of otalgia:

#### Acute otitis media:

Acute otitis media is one of the common middle ear causes of otalgia. This condition is common in children. It is caused by eustachian 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 eustachian tube. In patients with intractable pain then myringotomy will have to be resorted to. Majority of them will respond well to oral antibiotics, analgesics and nasal decongestants.

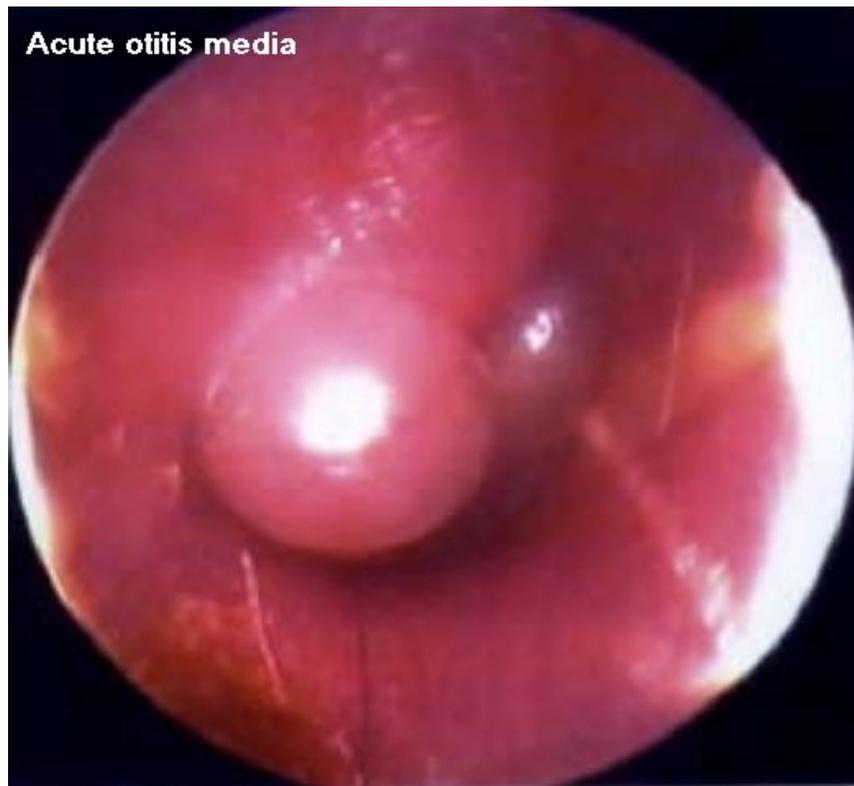


Figure showing acute otitis media

Eustachean tube dysfunction:

This is one of the common middle ear causes of otalgia. Patients with eustachean tube dysfunction may have otalgia even in the absence of otitis media. This occurs because of the inability of middle ear to equalize with atmospheric pressure resulting in distortion to the middle ear mucosa and ear drum. Patients with retracted drum may experience acute ear pain when middle ear equalization manages to peel the retracted drum away from the promontory.

Otitis barotrauma:

This is caused due to descent in an aircraft which has not been pressurized / in air travellers with pre existing eustachean tube dysfunction. Since there is failure of equalisation due to inability of eustachean tube to open normally the superficial blood vessels of middle ear mucosa ruptures leading on to hematoma formation. This is an acute painful condition. In some patients secretory otitis media may also be caused.

In these patients the middle ear fluid is believed to be transudate in nature. Where as in secretory otitis media due to infection the middle ear fluid resembles exudate. These patients should be treated with antibiotics (systemic), analgesics, and nasal decongestants. They are also encouraged to perform repeated valsalva manuever in order to open up their eustachean tubes.



Figure showing otitis barotrauma

Ramsay Hunt syndrome:

This syndrome is characterised by otalgia, lower motor neuron type of facial palsy and the characteristic rash seen in the pinna / external auditory canal. Otolgia in these patients are caused by irritation / inflammation of the affected cranial nerve (7th nerve in this case). This condition is caused by Herpes infection.

Miscellaneous causes of otalgia:

1. Trauma
2. Hematoma
3. Seroma
4. Frost bite
5. Burns and thermal injuries

Otolgia associated with indurated oedema of pinna should arouse suspicion of cellulitis.

### Chondrodermatitis nodularis chronica helicus:

This is an inflammatory nodule seen over helix of pinna with otalgia. This is commonly seen in males. Cases have also been reported on women<sup>14</sup>. It is a benign appearing tender lesion which can be treated with local excision and steroid infiltrations. The exact cause for this condition is not clearly known. Some authors believe it could be caused by prolonged and excessive pressure. This is possible because pinna has very little subcutaneous tissue for insulation and padding and is hence prone for injury due to trivium. This condition has also been associated with autoimmune disorders.

### Secondary / Referred otalgia:

This is due to complex innervation of the ear. Noxious stimuli from remote areas of head and neck can manifest with otalgia due to the shared anatomic innervation of the ear. The causes for secondary otalgia are extensive and numerous. The three logical sites for initial attention in a case of referred otalgia are temporomandibular joint, entire neck and teeth. Among other probable causes head and neck malignancy should be excluded.

### Temporomandibular joint syndrome:

In this condition the patient has difficulty / pain during mastication. These patients also manifest with aural fullness, tinnitus and vertigo. This syndrome goes by the name Costen syndrome. It was Costen who described this condition in precise detail in 1934. The symptoms associated with this syndrome are supposed to arise from posterior displacement of condyle of mandible, which leads to compression of auricular temporal branch of trigeminal nerve of the chorda tympani branch of facial nerve.

Temporomandibular joint should be examined in all patients with otalgia. Focus should be on joint and muscles of mastication. It should also be seen whether careful palpation of joint structures reproduces otalgia.

Pterygoid muscles should be palpated intraorally in order to rule out spasm of the muscle. Mandible should be distracted laterally to assess pain during various movements.

Presence of click / crepitus should be sought while performing this movement test.

Oral cavity of these patients should be examined with emphasis on identification of malocclusion which could cause excessive strain on the temporomandibular joint and its muscles.

### Infections causing otalgia:

Infections that cause referred otalgia include:

1. Tonsillar infections (i.e. Quinsy, tonsillitis) via the glossopharyngeal nerve
2. Mumps parotitis – due to stretching of the sensitive parotid fascia via trigeminal nerve
3. Rarely sinus infections also can cause otalgia
4. Dental infections like tooth decay may cause referred otalgia

### Eagle syndrome:

This is another potential cause of referred otalgia. This syndrome is defined as otalgia, facial pain, throat pain secondary to elongated styloid process / ossified

stylohyoid ligament. Pain in Eagle's syndrome could be due to:

1. Direct compression / irritation of trigeminal, facial, glossopharyngeal and vagus nerves.
2. Direct compression / irritation of carotid vessels (Carotidynia)
3. Inflammation of tendinous part of stylohyoid ligament
4. Infection of styloid process and adjacent tissue (styloiditis)

Gastro oesophageal reflux disease:

A large number of disorders may be caused by gastro oesophageal reflux. It can cause otalgia by irritating the upper aerodigestive tract in the sensory distribution of glossopharyngeal and vagus nerves. Sometimes reflux of acid contents from the stomach may irritate the pharyngeal end of eustachean tube causing acute otitis media. This GERD induced otitis media is common in children and toddlers.

Neuralgias: The extensive innervation of the ear predisposes it to neuralgias. Neuralgic pain is usually brief, lancinating and episodic in nature. This type of pain is self limiting and needs only reassurance. Neuralgias involving trigeminal, geniculate, glossopharyngeal and sphenopalatine ganglia manifest as otalgia.

Neoplasms:

Tumors involving various sites of head and neck can present with otalgia.

Tumors involving the anterior portion of the tongue may cause otalgia via the involvement of chorda tympani branch of facial nerve.

Nasal and paranasal sinus malignancies may cause otalgia secondary to eustachean tube dysfunction or direct neural involvement. Direct neural involvement will cause otalgia through afferents from posterior lateral nasal nerves by way of sphenopalatine ganglion associated with the second division of trigeminal nerve.

Hypopharyngeal tumors especially those involving the pyriform fossa can cause otalgia via the vagus nerves.

Tonsillar malignancies may cause otalgia via the glossopharyngeal nerve.

The cervical plexus covers a large surface area of skin extending from the posterior aspect of the auricle on the mastoid to the lateral neck and thyroid. This explains referred ear pain in inflammations / malignancies of thyroid.

Lesions arising from infratemporal fossa can cause referred otalgia due to involvement of Arnold's nerve or Jacobson's nerve.

Cervical arthritis as a cause of referred otalgia:

Otalgia can be caused due to occipital nerve root irritation from cervical osteoarthritis. Spasm involving the cervical muscles can cause significant bilateral otalgia.

Otalgia can also be caused due to intracranial lesions due to stretching of Dura.

Vertigo: is defined as a sensation of unsteadiness / rotation. The commonest peripheral causes for vertigo are the diseases affecting the inner ear. It is always associated with tinnitus/ blocking sensation in the ear. Peripheral vertigo can be differentiated by central vertigo by its fatigability. In peripheral vertigo the vertigo tends to diminish with time because the higher center learns to adjust with the problem. It is always positional. The patient will have to assume the provoking position for vertigo to manifest. Vertigo due to Meniere's disease is self limiting and short lived. It never lasts for more than a day after which the patient gradually improves. Peripheral vertigo is always

associated with horizontal nystagmus, which is again fatiguing, whereas central nystagmus due to cerebellar pathology manifests with rotatory / vertical nystagmus. They also show other positive cerebellar signs like past pointing, dysdiadokokinesis etc.

Symptoms of vertigo should not be dismissed as due to aging. If diligently sought specific etiology is identifiable in almost all cases <sup>15</sup>.

Detailed history is invaluable in diagnosing the cause for unsteadiness / vertigo.

The history should focus on:

1. Duration of the symptom
2. Severity of the symptom (The patient should be quizzed specifically whether the quality of life is affected)
3. Progression of the symptom (Improving / worsening)
4. Onset – Gradual / sudden
5. Whether giddiness is episodic (duration of each episode) / constant
6. Exacerbation and remitting factors
7. Relation of giddiness to head position
8. Associated hearing loss / tinnitus / nausea / vomiting
9. H/O trauma / head injury / barotrauma
10. H/O chronic ear disease
11. How much is the patient hampered by this symptom?

### Inspection:

The external ear is inspected with the following in mind:

1. Size & shape of the pinna
2. Presence of tags / preauricular sinuses / pits
3. Evidence of trauma to pinna
4. Skin condition of pinna & external auditory canal
5. Evidence of previous surgery / presence of scars in the post aural / end aural region
6. Discharge from the external canal
7. Neoplastic lesions of pinna

Movement of auricle may be painful in patients with cellulitis, dermatitis and otitis externa.

Examination of external auditory canal:

The external auditory canal is inspected for:

1. Stenosis
2. Furuncle
3. Cysts
4. Oedema
5. Dermatitis
6. Exposed bone
7. Neoplastic change

Presence of granulation tissue, purulent discharge, mucoid discharge, squamous debris is assessed.

Bony osteomas and exostosis can be confirmed by means of gentle palpation.

Aural polypi if present should be manipulated and palpated for ascertain its site of attachment. It is not prudent to remove aural polyp in the out patient clinic in order to visualize the ear drum because it could be attached to vital structures like middle ear ossicles, facial nerve etc.

### Otoscopy:

The ear drum can be examined using an otoscope. The pinna should be grasped between the index finger and thumb and is pulled postero superiorly. This maneuver straightens the external canal bringing the ear drum into full view. This maneuver should be done only in adults. In infants the pinna must be pulled posteriorly and downwards in an effort to straighten the external canal. This is because of the fact the bony portion of the external canal is not fully developed in infants.



Image showing the procedure used to straightening the external auditory canal

Otoscopy is a procedure which is used to examine the external auditory canal and tympanic membrane. This is a very crucial part of clinical examination in otology. The external auditory canal is not a straight tube. It shows two crucial angulations:

1. Outer third of external auditory canal which is formed of cartilage is directed postero superiorly.
2. Inner two thirds of external auditory canal is bony and is directed antero inferiorly.

As a whole the external auditory canal measures about 2.5 cms.

The external auditory canal is not in a straight line. The aural speculum when introduced straightens the external canal thereby helps in better visualization of the ear drum. Appropriate sized aural speculum should be chosen in order to minimize patient's discomfort. Disposable aural speculum is preferable.

The following points should be noted while performing otoscopy:

1. Status of external auditory canal skin. (Normal / thickened / weepy)
2. Presence of flakes in the external canal (Could mean desquamated epithelium / fungal flakes)
3. Presence of narrowing (stenosis) of external canal
4. Presence of cerumen / discharge / blood / foreign body in the external canal
5. Presence of masses arising from bony portion of external canal - exostosis / osteoma. If present

whether single / multiple.

6. Whether the ear drum is visible in its entirety?

7. Is the ear drum intact?

8. What is the color of the drum? Normal drum is pearly white in color.

9. Is cone of light visible in the antero inferior quadrant of the pars tensa? The cone of light is actually a reflection of light due to the angulation of the ear drum with that of external auditory canal. This cone of light is triangular in shape, with its apex pointing towards the umbo and the base towards the antero inferior quadrant of the ear drum.

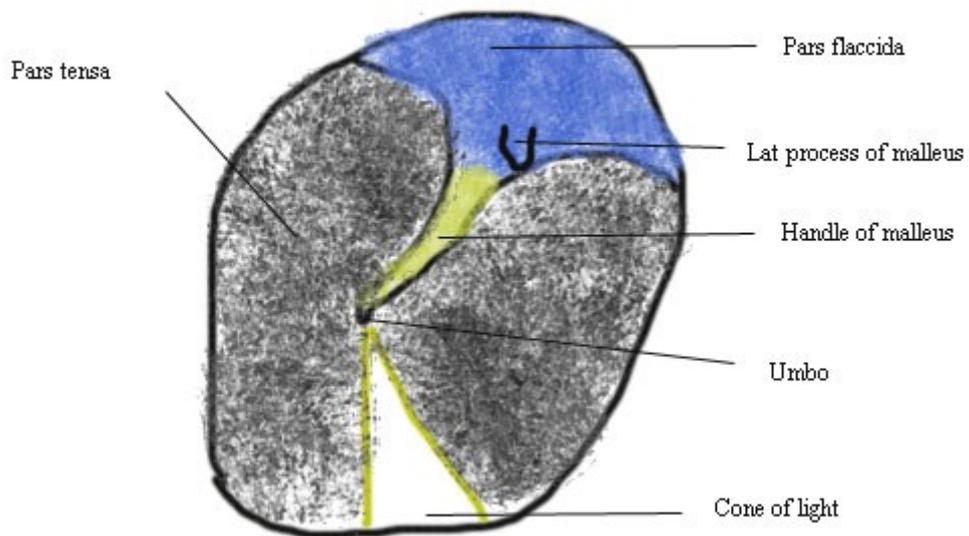


Figure showing ear drum and its various parts

Cone of light is distorted in :

1. Acute otitis media because of the bulging ear drum
2. Adhesive otitis media because the ear drum is retracted
3. Secretory otitis media
4. When there is perforation the quadrants involved by the perforation should be mentioned. It should not be described in numerics.



Image showing retracted ear drum

Retracted drum can be identified by the following features:

1. Distortion of cone of light
2. Prominent handle of malleus
3. Impaired /loss of mobility on siegelization
4. Presence of retraction pockets

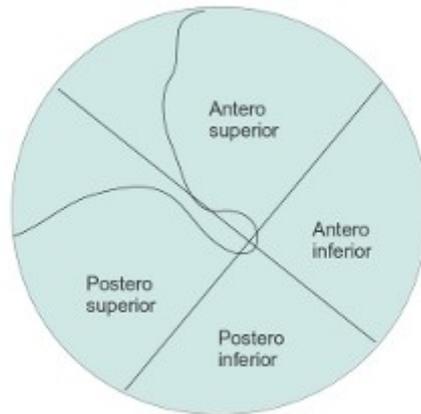


Figure showing quadrants of the ear drum

A monomeric area of ear drum should be differentiated from true perforation. This can be done by performing powder insufflation test. In this test gentamycin powder is infiltrated into the aural cavity using a powder insufflator. In patients with perforated ear drum the powder tends to be stuck to the wet middle ear mucosa.

Use of Gruber's aural speculum itself is sufficient to straighten the external auditory canal.

The ear drum is roughly oval in shape and about 1 cm in diameter. Normal ear drum is pearly white in color. The following structures of ear drum are visualised:

1. Attic area
2. Pars tensa
3. Cone of light
4. Handle / lateral process of malleus

Rarely the following structures also can be seen:

Long process of incus

Head of stapes

Promontory

Eustachean tube orifice

Perforations any must be identified, its position clearly documented. Through the perforation the status of the middle ear mucosa must be observed and documented. Presence of tympanosclerotic plaque (chalky mass over the ear drum) is an indicator of previous ear disease.

The cone of light must be observed for any distortion. Cone of light is absent in perforated ear drums, is distorted in retracted ear drums. It is also distorted when the ear drum is bulging as in the case of Acute otitis media.

The color of the ear drum must also be noted:

Red drum - is seen in acute otitis media, glomus jugulare

Blue drum - is seen in haemotympanum, secretory otitis media

Flamingo drum - is seen in otospongiosis

Mobility of the ear drum must be tested using a pneumatic otoscope, or a siegele's speculum. The mobility of the ear drum is restricted in adhesive otitis media.



Image showing a red drum (Glomus tympanicum)



Image showing multiple retraction pockets in the ear drum

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.



Image showing siegles pneumatic speculum

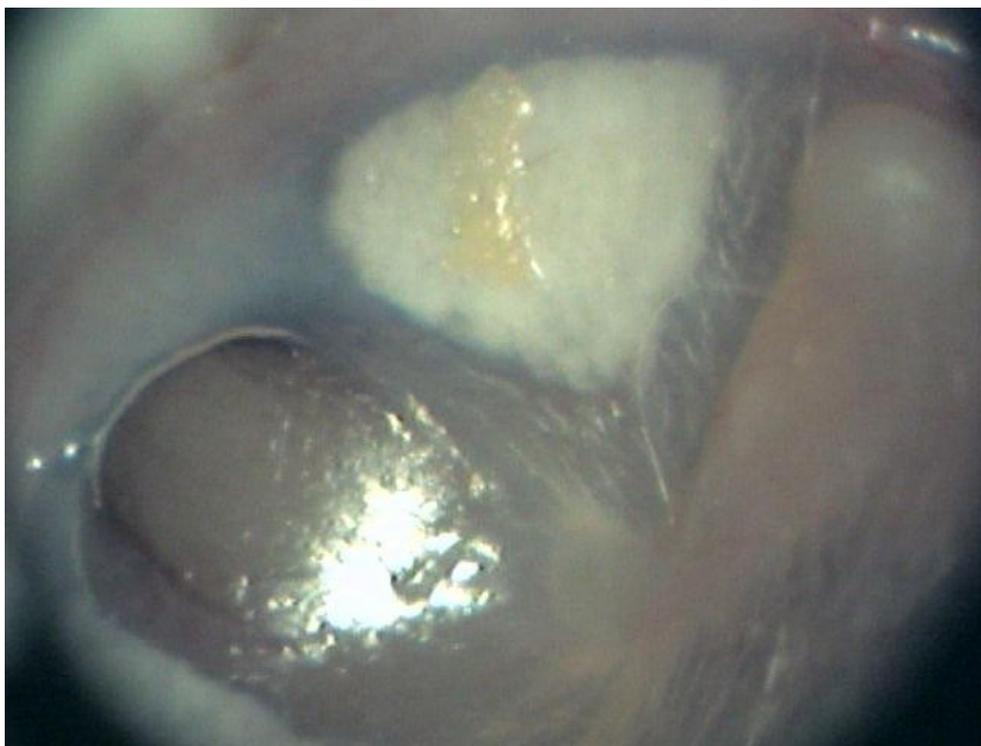


Image showing tympanosclerotic plaque

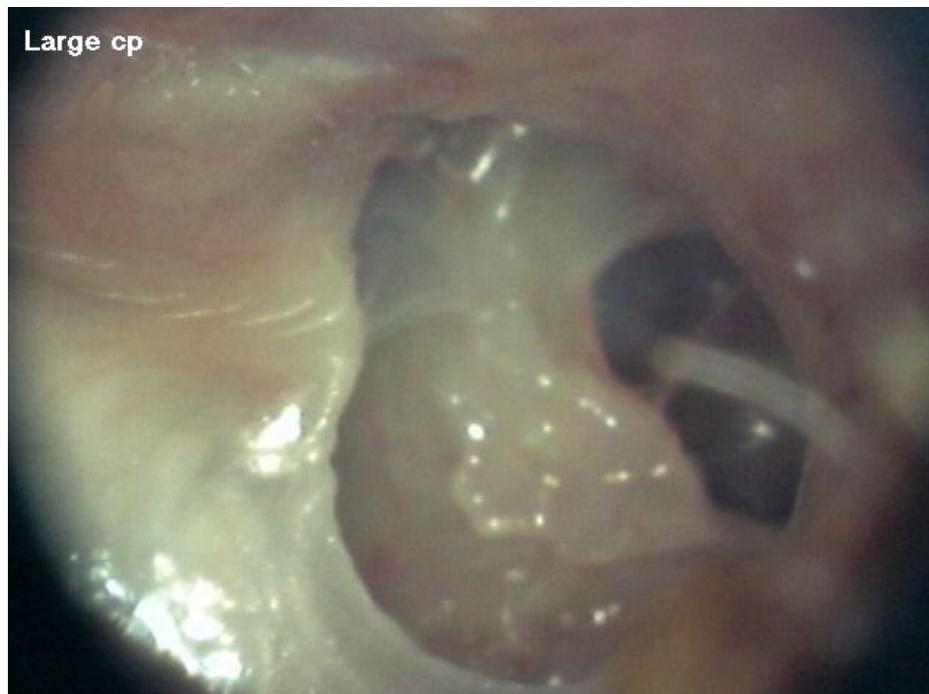


Image showing large central perforation



Image showing attic perforation

### Tests for hearing:

This is one of the important functions of ear which must be tested. This can be performed in clinical setting by using tuning forks.

Advantages of tuning fork tests:

1. Easy to perform
2. Can even be performed at bed side
3. Will give a rough estimate of the patient s hearing acuity

The following tests can be performed using a tuning fork:

1. Rinne test
2. Weber test
3. ABC test

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:

1. It should be made of a good alloy.
2. It should vibrate for one full minute.
3. It should not produce any over tones.

Rough estimation of hearing loss would be:

quite whisper - normal

Loud whisper - 20 - 30 dB

Quite voice - 30 - 45 dB

Loud voice - 45 - 60 dB

Shout - 60 - 80 dB

Rinnes test:

Ideally 512 tuning fork is used. This frequency is preferred because it falls within the range of normal speech frequency. A fork vibrating at this frequency is better heard than felt. It also produces fewer overtones. 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.



Figure showing Rinne test being performed (air conduction)

#### 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.



Image showing weber's test being performed

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 sensorineural 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.

Other tuning fork tests that could be performed include:

Bing test:

This is actually a modification of Weber's test. The vibrating fork is placed over the mastoid process and when it ceases to be heard the examiner's finger is used to occlude the external auditory canal. In normal individuals the sound will be heard again. This is because by occluding the external auditory canal the examiner is preventing sound from escaping via the external canal. The external auditory canal acts as a resonating chamber. If the vibrating fork is not heard again after the external canal is occluded then it is construed that the middle ear conduction is the cause for deafness. In patients with pronounced deafness if the vibrating fork is heard after occlusion of external canal then deafness is construed to be due to labyrinthine causes.

Politzer test:

In this test the vibrating fork is held in front of open mouth and the patient is asked to swallow. If the Eustachian tubes are patulous then sound will be intensified during swallowing. If only one tube is patulous then sound will be accentuated only in that ear. Sometimes normal persons too may not hear the vibrating fork.

Bing Entotic test:

Hypothetically this test is supposed to differentiate between deafness due to ankylosis of foot plate of stapes from that of conditions interfering with mobility of other ossicles. This test is actually of historic value only. Eustachian catheter is passed and to one of its ends is attached a speaking tube. If the patient is able to hear the fork better via this tube than that from the external auditory canal then middle ear ossicles other than foot plate of stapes is supposed to be at fault.

Stenger's test:

This test is performed to identify feigned hearing loss and malingering. This test is based on the auditory phenomenon known as "Stenger's principle". This principle states that when two similar sounds are presented to both ears only the louder of the two would be heard. Patients usually are not aware of this phenomenon. When two similar tuning forks of same frequencies are made to vibrate and held simultaneously in the acoustic axis of both ears only the louder fork will be heard.

Loudness of vibrating fork can be adjusted by adjusting the distance of the fork from the external canal. Usually the vibrating fork is held closer to the allegedly deaf ear of the patient. The patient will not acknowledge hearing in that ear. According to Stenger's principle he should be able to hear the louder fork. If the hearing loss in worse ear is genuine, patient will respond to the signal presented to the better ear. This is known as negative Stenger's test. Feigning patient will not acknowledge hearing when louder sound is presented to the worse ear. This is known as positive Stenger's test.

Gelle test:

In this test, the air pressure in the external canal is varied using a Siegle's speculum. The vibrating fork is held in contact with the mastoid process. In normal individuals and in those with sensorineural hearing loss, increased pressure in the external meatus causes a decrease in the loudness of the bone conducted sound. In stapes fixation no alteration in the hearing threshold is evident.

Chimani Moos test:

This is actually a modification of Weber test. When the vibrating fork is placed on the vertex, the patient indicates that he hears it in the good ear and not in the deaf ear. The meatus of the good ear is then occluded. A genuine deaf patient will still be able to lateralize the sound to the good ear, whereas a malingerer will deny hearing the sound at all.

### Tests for evaluating vestibular functions:

Nystagmus should be looked for. This is actually to and fro movement of the eyes. This involuntary rapid eye movement can occur as a result of vestibular, optokinetic or pursuit system dysfunction. Disturbance involving any of the three systems can cause drift of the eyes while optical fixation is attempted. This drift is known as slow phase of the nystagmus, which is followed by a rapid corrective phase which resets the drift. The direction of nystagmus is decided by the direction of the fast phase corrective nystagmus.

Classification of nystagmus:

Clinically nystagmus is classified either as spontaneous or evoked. Spontaneous nystagmus occurs without any provoking stimuli, whereas evoked nystagmus usually occurs in response to stimulation.

After clinical examination nystagmus is described using the following terms:

1. In terms of direction of fast phase – i.e. Right beating, left beating, geotropic, ageotropic.
2. Plane – horizontal, rotatory or vertical.
3. Intensity – I degree, II degree and III degree.

I degree nystagmus :

This is the least intense nystagmus which occurs when the gaze is towards the fast component of the nystagmus.

II degree nystagmus:

In this type of nystagmus, it occurs when the gaze is towards the fast component as well as in the midline.

III degree nystagmus:

This is the most severe type of nystagmus occurring in all directions of gaze.

Examination of nystagmus will be made easier if the patient is made to wear Frenzel's glasses. It is a + 20 diopter lens. When the patient wears it, the eye movements can be visualized in a magnified manner by the observer. Another advantage being, it removes optic fixation by the patient making the nystagmus easy to observe.



Image showing Frenzel glasses

Fistula test:

Otological examination is incomplete if this test is not performed. This test is usually performed by applying alternating positive and negative pressure to the ear drum using siegel's pneumatic speculum or tragal pressure. Pneumatic otoscope can also be used for this purpose. Nystagmus and vertigo in these patients is considered to be positive. Hennebert's sign is positive fistula test in a patient with intact ear drum and without evidence of ear disease.

Conditions causing positive fistula test:

1. Labyrinthine fistula
2. Labyrinthitis
3. Syphilis
4. Superior semicircular canal dehiscence syndrome

Dix-Hallpike Maneuver:

This test is routinely performed in all patient who complain of vertigo. It is mandatory when patient complains of vertigo when assuming certain provoking positions.

The Dix–Hallpike test is performed with the patient sitting upright with the legs extended. The patient's head is then rotated by approximately 45 degrees. The clinician helps the patient to lie down backwards quickly with the head held in approximately 20 degrees of extension. This extension may either be achieved by having the clinician supporting the head as it hangs off the table or by placing a pillow under their upper back. The patient's eyes are then observed for about 45 seconds as there is a characteristic 5–10 second period of latency prior to the onset of nystagmus. If rotational nystagmus occurs then the test is considered positive for benign positional vertigo. During a positive test, the fast phase of the rotatory nystagmus is toward the affected ear, which is the ear closest to the ground. The direction of the fast phase is defined by the rotation of the top of the eye, either clockwise or counter-clockwise.

Positive test:

There are several key characteristics of a positive test:

Latency of onset (usually 5–10 seconds)

Torsional (rotational) nystagmus. If no torsional nystagmus occurs but there is upbeating or downbeating nystagmus, a central nervous system (CNS) dysfunction is indicated.

Upbeating or downbeating nystagmus. Upbeating nystagmus indicates that the vertigo is present in the posterior semicircular canal of the tested side. Downbeating nystagmus indicates that the vertigo is in the anterior semicircular canal of the tested side.

Fatigable nystagmus. Multiple repetition of the test will result in less and less nystagmus.

Reversal. Upon sitting after a positive maneuver the direction of nystagmus should reverse for a brief period of time.

To complete the test, the patient is brought back to the seated position, and the eyes are examined again to see if reversal occurs. The nystagmus may come in paroxysms and may be delayed by several seconds after the maneuver is performed.

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