**Online learning module**

Welcome to the asynchronous formal learning for Year 4 ORL/ENT teaching.

You have been allocated 4 hours to complete this online module. *Some questions in your exam will be sourced from this material.*

The purpose of this online module is to give you a good overview of ORL conditions that you are likely to encounter in your early clinical practice.

Otorhinolaryngology has four sub-specialties:

1. Head and Neck
2. Laryngology
3. Otology
4. Rhinology

On the left hand side you can navigate through the teaching materials under each of these clinical topics.

**Summary of objectives:**

1. Spend a *minimum of four hours* learning from this module

2. Become comfortable with the principles in the basic sciences, diagnosis and management of common ORL diseases

3. Enjoy the module!

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# Head and Neck

## Sore Throat

**Differential diagnosis for a sore throat:**

Tonsillitis

Peritonsillar abscess (Quinsy)

Supraglottitis / Epiglottitis

Deep neck space infections

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**And the following that will be covered elsewhere in the curriculum:**

Pharyngitis, common cold, foreign body, dental infections

### Tonsillitis

Tonsillitis is inflammation of the pharyngeal tonsils. The inflammation usually extends to the lingual tonsils and adenoids therefore pharyngotonsillitis and adenotonsillitis are synonymous with tonsillitis

**Aetiology**

Most commonly viral - adenovirus, rhinovirus, RSV, EBV

15 - 30% are bacterial - mostly group A beta-hemolytic *Streptococcus pyogenes* (GABHS).

**Epidemiology**

* Tonsillitis most often occurs in children; however, the condition rarely occurs in children younger than 2 years.
* Tonsillitis caused by *Streptococcus* species typically occurs in children aged 5-15 years

**History**

* Fever
* Sore throat
* Halitosis (foul breath)
* Dysphagia (difficulty swallowing)
* Odynophagia (painful swallowing)
* Mild airway obstruction e.g. mouth breathing

**Examination**

1. Degree of respiratory distress
2. Full examination of oral cavity including the oral mucosa, dentition, salivary ducts. *In particular look for: tonsillar erythema, oedema +/- exudate (see pictures above)*
3. Tender cervical lymphadenopathy
4. Flexible nasoendoscopy - especially if severe to assess for degree of airway obstruction

**Possible complications of *Streptococcal* pharyngotonsillitis**

**Non-suppurative complications**Scarlet fever
Rheumatic fever
Post-streptococcal glomerulonephritis

**Suppurative complications**Peritonsillar abscess (Quinsy)
Deep neck space infections
Cervical lymphadenitis

**Management**

1. ABC's

* Airway secured
* Fluid resuscitation

2. Antibiotics

* for acute inpatients - Penicillin IV 2MU Q6h
* for outpatients - Penicillin PO for 10 days

3. Steroids

* Reserved for inpatients - usually Dexamethasone either 8mg stat or 8mg TDS for one day

4. Supportive therapy

* Anti-emetics
* Analgesia and antipyretics

### EBV Tonsillitis

*Consider infectious mononucleosis particularly when tonsillitis is accompanied by:*

*- tender lymph nodes
- splenomegaly
- severe lethargy
- white/gray membrane may cover tonsils that are infected with EBV (see image)*

However, EBV diagnosis can only be confirmed via blood tests. The importance of this is that EBV tonsillitis takes longer to resolve and patients should avoid contact sport

### Peritonsillar Abscess (Quinsy)

Each tonsil is surrounded by a capsule.It is in the potential space **(peritonsillar space)** between the tonsil and capsule that abscesses can form - a peritonsillar abscess, or in layman's terms, quinsy

*IMPORTANT: the peritonsillar space is contiguous with several deeper spaces and infections can involve the parapharyngeal and retropharyngeal spaces (see deep neck space infections)*

*Compare these pictures above with the pictures shown in the tonsillitis section. What are the differences?*

*Note carefully the following:*

* *Uvula deviation to contralateral side*
* *Inferior and medial tonsil displacement*
* *Localised fluctuance (easier to appreciate when examining a patient than in pictures)*
* *Swelling of supratonsillar fold/soft palate rather than tonsil itself*

**Aetiology**

Tonsillitis can progress to cellulitis and then via tissue necrosis and pus formation to a peritonsillar abscess
*or* starts via an infection of minor salivary glands

**Microbiology:**

* A polymicrobial flora is isolated from peritonsillar abscesses
* Predominant organisms are anaerobes
* Aerobic organisms present are commonly *Strep*, *S aureus* and *H influenzae*

**Epidemiology**

Peritonsillar abscess (PTA) usually occurs in teens or young adults but may present earlier and occur in later adulthood

**History**

Symptoms are the same as for tonsillitis with a few other symptoms that are red flags for PTA and deep neck space infections:

* Neck pain
* Throat pain, more severe on the affected side +/- referred ear pain
* Trismus (lock-jaw) - due to inflammation of chewing muscles
* Voice change - in PTA pharyngeal edema and trismus can cause a *"hot-potato" voice* - as if the patient is struggling with a mouthful of hot food

*Remember: nasty dental infections can mimic PTA therefore examine the oral cavity carefully*

**Examination**

As for tonsillitis.
Apart from the visual findings as noted above:

* Drooling
* Trismus
* More severe dehydration

Patients with quinsy should have a flexible nasoendoscopy/laryngoscopy performed to rule out epiglottitis

**Investigations for tonsillitis and PTA**

General:
1. FBC, U+E (especially if dehydrated)
2. Monospot test/heterophile antibody test - to rule out *infectious mononucleosis*

With PTA or if you suspect deep neck space infections:
1. Lateral neck X-ray
If clinically concerned or X-ray suggestive then proceed to CT scan

**Management**

*Always first think ABC's:* In severe circumstances endotracheal intubation or a surgical airway may be required.

The cornerstone of PTA treatment is: **incision and drainage**

In addition the treatment regime includes the same medication and considerations as for *tonsillitis.*

Patients with PTA tend to be more toxic - therefore require more IV fluid resus (and greater care)

### Supraglottitis/Epiglottitis

Epiglottitis and supraglottitis are interchangeable terms meaning:

*inflammation of structures above the insertion of the glottis in the oropharynx including the epiglottis, vallecula, arytenoids and aryepiglottic folds (see picture below for nasoendoscopic view of the larynx)*

**Aetiology**

*Haemophilus influenzae* type b (Hib) used to be responsible for >90% of epiglottitis cases
Due to vaccination the incidence has dropped markedly

Now other bacterial pathogens are responsible for the majority of cases including *Strep, Staph* and a number of gram negative bacilli

*A Danish study demonstrated an incidence of paediatric epiglottitis of 4.9 cases per 100,000 per year in the decade before Hib vaccination.*

*With the introduction of widespread Hib vaccination - 1996 to 2005 - an incidence of only 0.02 cases per 100,000 per year was seen. (1)*

**History**

* Sore throat
* Odynophagia/dysphagia
* Muffled voice - "Hot potato voice"
* Adults may have had a preceding upper respiratory tract infection (URTI)

Always ask about vaccination if you suspect supraglottitis

**Examination**

* "Toxic" appearance of patient
* Sitting or leaning forward. Extreme = **"Tripod position"** - Sitting up on hands, with the tongue out and the head forward
* Drooling/inability to handle secretions
* Irritability
* Stridor: A late finding indicating advanced airway obstruction

+ Muffled voice, cervical adenopathy, fever, respiratory distress, mild cough

*Flexible laryngoscopy is required for diagnosis + assessment of airway (if patient will tolerate it)*

*Remember: The progression of supraglottitis can be rapid (i.e. over hours!)*

**Investigation**

1. Lateral Neck XR - may be useful (see image below)
Most adults are stable and may safely undergo imaging. When clarking patients with possible epiglottitis, lateral neck XR can be a useful screening tool.

2. As mentioned above - judicious use of flexible nasoendoscopy

3. Blood cultures

This lateral neck X-ray demonstrates a classic radiographic finding in epiglottitis - the **"thumb sign"**
- This is due to swelling of the epiglottis
- The swelling of the epiglottis on the X-ray is shown by the blue dotted line - the left side is normal and the right has the abnormality

**Management**

1. Airway is the priority - ensure early ENT review. You should know where the surgical airway (cricothyrotomy) kit is.
Unstable patients/severe respiratory distress may need immediate intubation or surgical airway management.

If patient is stable they may still need monitoring in ICU.

Minimise distress - particularly important in paediatric patients.

2. Antibiotic - 1st choice is ceftriaxone

3. Supportive therapy
Analgesia, anti-emetics, IV fluid

**Deep Neck Space Infections**

The anatomy of the neck is complex. To understand the origin and spread of deep neck space infections (DNSI) a basic understanding of the anatomy is required.

**Summary of anatomical concepts:**

* Spatial compartments in the neck are created by fascial planes
* These spatial compartments can communicate with each other -> spread of infection
* Anterior to the prevertebral fascia is the "**danger space**" that extends from the skull base to the diaphragm - making **mediastinitis** a possible complication of abscesses that spread into this space
* There are a number of important structures in this area including blood vessels, nerves & bone that can become affected

**Types of DNSI:**1. PTA/quinsy (see previous page)
2. Retropharyngeal
3. Parapharyngeal
4. Prevertebral
5. Submandibular (relatively superficial)

**Challenge to diagnosis:** DNSI may be covered by layers of unaffected tissues & therefore is hard to visualise and palpate

**Etiology**

DNSI are most commonly complications of pharyngitis or dental infection
Other causes include: sialadenitis, IVDU, malignancy

*Note: DNSI is often a complication of an inadequately treated pharyngitis, dental abscess or tonsillitis*

Important possible sequela of DNSI:

1. Internal jugular vein thrombophlebitis (Lemierre Syndrome) - causes septic emboli and sepsis
2. Mediastinitis - signalled by chest pain or widened mediastinum on CXR

Carotid artery rupture, meningitis and cavernous sinus thrombosis are rare.

**History**

* Sore throat, dysphagia, odynophagia, trismus
* Neck pain and pain on neck movement
* Can have painful neck mass

**Examination**

* Posterior pharynx erythema + swelling in *retropharyngeal abscess*
* Medial displacement of tonsil and lateral pharyngeal wall in *parapharyngeal abscess*
* Torticollis (holds neck in rotated position)
* Tender lymphadenopathy

Findings suggesting complications:
- Neurological deficit - *cranial nerves* (eg. hoarseness from vocal cord paralysis with carotid sheath and X/recurrent laryngeal nerve involvement)
- *Horner syndrome* from involvement of the cervical sympathetic chain

**Investigations**

1. Lateral neck X-rays +/- CT scan

2. FBC, U+E

3. Blood cultures

**Management**

1. ABC's As with PTA/Quinsy the airway is of paramount importance.
Followed by IV fluid resus for the "C"

2. Antibiotics

3. Surgery - incision and drainage

## Neck Lump

The etiology of neck lumps are numerous and present a good opportunity to cover the **"surgical sieve"**.

The surgical sieve allows you to answer a "what are the causes of..." question (whether on ward rounds or exams) in a systematic way. Using the VITAMIN CD acronym, for neck lumps it can be constructed like this:

**Vascular:** AV malformation, aneurysm

**Inflammatory:**

**Traumatic:** Ranula, haematoma

**Autoimmune/allergic:** Thyroiditis

**Metabolic:** Goitre

**Infective:** Lymphadenitis, reactive lymphadenopathy

**Neoplastic**Benign: Carotid body tumour/chemodectoma
Malignant: Squamous cell carcinoma, thyroid cancer, lymphoma, malignant lymphadenopathy

**Congenital:** Branchial cleft cyst, thyroglossal duct cyst, dermoid cyst

**Degenerative**

**-----------------------------------**

**Branchial cleft cyst**

- These are congenital and present as an inflamed mass at the anterior border of the sternocleidomastoid
- They occur due to the failure of branchial cleft obliteration during fetal development
- Like the thyroglossal duct cyst these remain asymptomatic until they become infected - usually during an URTI

**Goitre**

- This is enlargement of the thyroid gland
- It may be diffuse or nodular and the patient may be euthyroid, hyper- and hypo- thyroid
- The treatment varies with the type  *Note: goitre is commonly more subtle than the one in this picture*

**Thyroglossal duct cyst**- They are congenital and are located in the midline of the anterior neck
- These cysts remain asymptomatic until they become infected - usually in the setting of an URTI
- On examination they move with swallowing (like the thyroid) - but unlike the thyroid, these cysts also move superiorly with protrusion of the tongue
- Treatment is with excision

**History**

Important points to cover when taking a neck lump history:

- Pain: chronic oral pain is suspicious of malignancy and ***referred unilateral otalgia*** can be associated with tumours at the base of the tongue, larynx and hypopharynx (due to CN IX and X innervating both the pharynx and the ear)

- Dysphagia: range of occasional "catching" to inability of swallowing solids. Tumours generally cause gradual decline in ability to swallow food and weight loss. Nasal regurgitation or aspiration suggests neurological cause.

- Stridor: inspiratory sounds - caused by airflow blockage **at or above the vocal cords** i.e. is a symptom of upper respiratory obstruction.

- Hoarseness: suggests laryngeal disease - needs referral to ORL

- Constitutional symptoms: weight loss, night sweats, anorexia, chills/fevers - suggestive of malignancy

- Social factors: smoking and alcohol - highly associated with head and neck cancers.

**Examination**

1. A thorough neck examination includes the thyroid, lymph nodes, parotid & submandibular glands (including CN VII) - *Note: the neck is examined from behind the patient after initial inspection*

Lumps should be assessed for:

Position
Size
Contour - smooth, craggy
Texture - soft, firm, hard or fluctuant
Mobility
Tenderness

2. Look for signs of hypo- or hyper- thyroidism

3. In Australia and New Zealand adults neck lumps are commonly metastatic SCC therefore check the skin and oral cavity for a possible primary tumour

**Investigations**

1. Imaging: Ultrasound, CT or MRI

2. Cytology/histology: Fine needle aspiration (FNA) or biopsy

3. Blood tests
FBC - useful if haematological condition suspected
Thyroid function tests - useful in thyroid disease

# Rhinology

## Sinusitis

The terms rhinosinusitis and sinusitis are synonymous as inflammation of the sinuses is always accompanied by inflammation of the nasal cavity

Acute and chronic rhinosinusitis are independent clinical entities with different underlying pathophysiological mechanisms

**Examination** (this is identical for all types of sinusitis)

1. Anterior rhinoscopy - can be performed with otoscope
- look for mucopurulence, oedema, erythema and polyps
2. Flexible fibreoptic endoscopy

### Acute Rhinosinusitis

Up to 4 weeks of purulent nasal discharge accompanied by nasal obstruction, facial pain, facial pressure or fullness

**Aetiology and History**

Both viral and bacterial RS are preceded by an URTI

Viral:
Symptoms of acute rhinosinusitis are present less than 10 days
Symptoms do not worsen

Bacterial:
10 days+ beyond onset of URTI symptoms
*Symptoms worsen within 10 days after initial improvement*Common bacteria that cause sinusitis are the same as for otitis media:

* *Streptococcus pneumoniae*
* *Haemophilus influenzae*
* *Moraxella catarrhalis*

Purulent rhinorrhea, facial pain/pressure and nasal obstruction strongly suggest a bacterial cause

**Management**

*Antibiotics - amoxicillin + clavulanate for 7 days*

Further investigation and surgery may be indicated for recurrent or persistent sinusitis and complications

### Chronic Rhinosinusitis

**Aetiology**

The pathophysiology of CRS is multifactorial and forms a large part of the research that the University of Auckland ORL department performs
Factors include:

1. Immunology - intramucosal B and T cells
2. Microbiology - intramucosal *S. aureus*
3. Anatomy - sinus ostia obstruction (outlet of sinus)

**History and Diagnosis**

*12 weeks or longer of 2+ of the following:*
Mucopurulent discharge (anterior, posterior or both)
Nasal obstruction (congestion)
Facial pain/pressure/fullness
Decreased sense of smell

*and inflammation as seen by:*
Purulent mucus or oedema in the middle meatus or ethmoid region
Polyps
Imaging - CT

**Management**

1. Antibiotics
- ideally culture-directed; duration 3-4 weeks

2. Anti-inflammatory
- intranasal or oral steroids
- allergy management (if previous allergy known)

3. Mechanical
- nasal saline irrigation

4. Surgery - performed if symptoms remain despite maximal medical therapy for 4 - 6 weeks
- Functonal endoscopic sinus surgery (FESS)

### Complications

*Complications of sinusitis are rare in the antibiotic era*

**Orbital infection**This represents a progression of the infection"

1. Periorbital oedema
2. Orbital cellulitis
3. Subperiosteal abscess
4. Orbital abscess
5. Cavernous sinus thrombosis

**Intracranial complications**
Meningitis
Epidural abscess
Pott Puffy tumour (osteomyelitis of frontal bone with subperiosteal abscess)

### Allergic Rhinosinusitis

This is one of the most common ENT diseases and a differential diagnosis for sinusitis

It is the inflammation of the nasal mucous membranes caused by an IgE-mediated reaction to one or more allergens

**History**

Patients may be atopic and complain of:

- clear and watery nasal discharge
- itching of nose, eyes and throat
- nasal congestion

Seasonal symptoms may include exposure to pollen or cats

**Management**

Mainstay:
1. Antihistamines
2. Intranasal corticosteroids

Others:
3. Systemic corticosteroids - if intractable symptoms
4. Decongestants - be aware of rebound effects

### Epistaxis

Epistaxis is bleeding from the nose

**Aetiology**

Bleeding occurs when the mucosa erodes and the vessels become exposed. Can be due to the following:

- infections e.g. cold

- trauma e.g. nose picking, foreign body, inhalation of dry air (on an aeroplane for example)

- medications e.g. anticoagulants, topical nasal meds, illicit drugs

- *rare:* systemic conditions e.g. coagulopathy, sarcoidosis, Wegener's granulomatosis + tumours

but often idiopathic

>95% of bleeds are from Little's area (Kiesselbach's plexus) in the anterior septum

Very small number are posterior nose bleeds (*NB these are more difficult to manage & there is a larger chance of airway compromise*)

**Epidemiology**

Very common - only small percentage of patients present for medical assessment

*Epistaxis in children is often due to nose picking or URTI*

**History**

*- Did bleeding being unilaterally or bilaterally?* -> anterior bleeds start unilaterally and become bilateral whereas posterior bleeds are bilateral from the beginning

- *Foreign body?* -> very important to ascertain in young children

- *General medical history* -> particularly systemic conditions, medications (anticoagulants!) and smoking

- *History of easy bruising, prolonged bleeding or recurrent epistaxis* -> consider systemic conditions

- *Family history of bleeding disorders*

**Examination**

*Before starting make sure you have all equipment prepared including cautery, packing and topical meds*

First you should spray local anaesthetic into both sides
If bleeding is obviously from one side - soak a small cotton ball with a mixture of transexamic acid and local anaesthetic with adrenaline *then examine 15 minutes later - this will provide anaesthesia and may stop bleeding allowing better examination*

Use the following:
- headlight: allows two free hands
- nasal speculum
- may need gentle suctioning of blood or clot to allow better view

Inspect both sides - if packing does not stop bleeding then a posterior bleed is possible and **flexible naso-endoscopy** needs to be performed

*Batteries can cause an alkali burn that can cause tissue necrosis*

**Investigations**

1. FBC - to assess severity of haemorrhage via the Hb
2. Coagulation screen - especially if on warfarin
3. X-rays if FB suspected (paediatric population) - see below

# Laryngology

## Airway

### Stridor

Stridor is a harsh noise produced by turbulent airflow through a partially obstructed upper airway

It is commonly **inspiratory** indicating an obstruction at the larynx
*Note: expiratory and biphasic stridor are rarer and imply obstruction at the level of the trachea and subglottis*

Stridor is a symptom more commonly encountered in children and therefore we will focus on paediatric presentations of stridor

**Aetiology**

In a child the differential diagnosis is (using a surgical sieve):

*Traumatic***:** Foreign body

*Autoimmune/allergic:*Anaphylaxis

*Infective:* Laryngotracheobronchitis (croup), tracheitis, supraglottitis/epiglottitis, DNSI (see head and neck section)

*Neoplastic*
- Benign: respiratory papillomatosis/vocal fold papilloma, vocal fold cysts & nodules (see hoarseness section)

*Congenital:*Laryngomalacia, laryngeal web, vocal cord paralysis, subglottic stenosis (can be secondary to prolonged intubation)

*Stridor should be treated as a medical emergency*

In adults the following are the most common causes of stridor:

* Anaphylaxis
* Foreign body
* Trauma e.g. laryngeal fracture
* DNSI
* Malignancy: laryngeal or mediastinal
* Benign external compression e.g. goitre

*Chronic stridor in an adult should raise concern and malignancy needs to be excluded*

**Laryngomalacia**

* Stridor occurs as a result of prolapse of the supraglottic structures into the laryngeal inlet on inspiration
* Most common cause of stridor in infants and most common congenital laryngeal abnormality
* Majority of infants can be managed conservatively. ~10% require surgery

**Picture of flexible nasolaryngoscopic examination of a patient with laryngomalacia**- The epiglottis is omega-shaped and folded upon itself to that the lateral margins lie close to one another
- The aryepiglottic folds are foreshortened and thin
- The arytenoids are large with redundant mucosa
- Mucosal oedema from vibratory trauma to the supraglottic area exacerbates the symptoms

**Laryngeal web**

* Congenital condition that presents with an abnormal cry and stridor
* Arises from the failure of the recanalization of the larynx in the embryo
* Treatment is with incision for thinner webs; thicker webs may require stenting

**Subglottic stenosis**

* This is the partial or complete narrowing of the subglottic area - which is the narrowest portion of the airway in children
* *Congenital* - present with stridor at birth or intermittent stridor if mild stenosis e.g. only with URTI or
*Acquired* - commonly secondary to prolonged endotracheal intubation (~90% of cases)
* Lateral neck X-ray can show stenosis
* Treatment is dictated by age of patient, grade and type of stenosis
Procedures range from observation for mild cases to laryngotracheal reconstruction for severe cases

### Tracheostomy

This is an operative technique that creates a surgical airway in the cervical trachea - it is formed by anastomosing the proximal tracheal stump to the skin surface

Indications - can be elective or acute:
1. Relieve upper airway obstruction - e.g. congenital deformity, trauma, infection, neoplasm
2. Prophylactically in head and neck surgery (temporary airway)

### Laryngeal fracture

* Caused by trauma
* Symptoms: hoarseness, pain, crepitus, loss of normal midline neck landmarks
* Investigations: CT and fibreoptic laryngoscopy for diagnosis
* Management:
*- If airway stable* and minimal displacement then treatment is conservative with antibiotics if mucosal tears present +/- steroids for the oedema
*- If airway compromised* or large amount of displacement then surgery is advised

## Voice

Hoarseness or Voice Change

Voice change or dysphonia (including hoarseness) is a symptom of primary laryngeal disease although rarely it can be due to other diseases e.g. lung cancer or hypothyroidism

The function of the larynx is primarily phonation and airway protection

**Aetiology**

*Every patient that presents with dysphonia needs to have malignancy excluded*

The common causes of dysphonia are (remember the surgical sieve):

**Trauma -** phonotrauma - voice abuse & misuse leads to trauma of the vocal cords. Repetitive phonotrauma leads to a local inflammatory response which can progress to nodules and polyps

**Neoplasm**
Benign lesions: Vocal fold nodule, vocal fold polyp, vocal fold papilloma, laryngeal cysts
Malignant neoplasms: Laryngeal SCC (>90% of laryngeal cancer)

Multiple underlying pathological mechanisms: vocal cord paralysis or recurrent laryngeal nerve paralysis

### Recurrent respiratory papillomatosis

Bimodal distribution:
Juvenile onset - ages 2 to 4 due to infection with HPV during pregnancy or birth
Adult onset - ages 30 to 50 due to HPV infection in adulthood

Treatment is with CO2 laser resection or cold steel resection. Recurrence is very common

### Vocal cord polyp

Associated with smoking and voice abuse

Treatment is excision to exclude malignancy and provide resolution

### Vocal fold cyst

Formed by mucous glands that are found throughout the larynx

Treatment depends on location

### Vocal fold nodule

Commonly affects professional voice user and are the most common cause of persistent dysphona in children (they shout a lot)

Usually bilateral & mainstay of treatment is speech therapy

**History**

* The onset, duration and progression of any voice change
* Preceding upper respiratory tract infections, direct or vocal trauma and endotracheal intubation
* Social factors: particularly *smoking* and alcohol
Occupation - professional voice users have higher rates of benign laryngeal disease as they have higher rates of voice misuse and abuse - therefore a careful history of voice use is needed
* Systemic enquiry should include symptoms of hypothyroidism and gastro-oesophageal reflux disease (GORD)

Age is very important:
- Adults have a greater incidence of malignant disease
- In children the main differential diagnosis is vocal cord nodules and laryngeal papillomatosis

*Persistent and progressive dysphonia in a person with a smoking history is a red flag for laryngeal malignancy - especially if associated with dysphagia or odynophagia*

**Examination**

1. A full ear, nose and throat exam

2. A neck exam - particularly looking for lymphadenopathy

3. Flexible nasoendoscopy with particular focus on laryngoscopy

4. Video stroboscopy - allows visualisation of the mucosal wave and has an appearance of a "slow motion" film. It is useful in diagnosis, monitoring rehabilitation and speech therapy

**Treatment of Laryngeal Cancer**

Treatment strategy depends on histology type, grade, tumour stage and overall health of the patient

Like most major cancers, this requires an MDT approach involving:

* Surgeon
* Speech language therapist
* Specialist nurses e.g. tracheostomy nurse
* Dietician
* Radiologist
* Radiation oncologist
* Medical oncologist
* Pathologist

The mainstay of treatment involves:

1. Surgery - partial or total laryngectomy (the latter requiring a tracheostomy)
+/- reconstruction using surgical flaps
2. Radiotherapy - primary or adjuvant
3. Chemotherapy in some cases

## Swallow

Dysphagia is difficulty eating due to disruption of the swallowing process

**Differential diagnosis:**

**Vascular -** Stroke
**Inflammatory** - GORD
**Trauma
Autoimmune/allergy -** Eosinophilic oesophagitis, scleroderma
**Metabolic
Infective
Neoplastic -** Barret's oesophagus, oesopheageal cancer, brainstem tumours
**Congenital
Degenerative -** Achalasia, Zenker diverticulum

### Chronic Cough

Cough that lasts more than 8 weeks

**Aetiology**

3 conditions account for ~95% of chronic cough:

1. Postnasal drip syndrome/upper airway cough syndrome (UACS)
2. Asthma
3. GORD

*Be aware that a number of other conditions make up the remainder e.g. ACE inhibitor therapy, smoking, CHF, non-asthmatic eosinophilic bronchitis etc.*

**UACS** - most common cause - secretions containing inflammatory mediators stimulate pharyngeal and laryngeal sites -> cough

**GORD** - either via:
1. Distal oesophageal acid exposure that stimulates an oesophageal-tracheobronchial cough reflex via CN X
2. Microaspiration of acid to the larynx/trachea (laryngopharyngeal reflux) *this is most often silent i.e. patients do not get heartburn*

**Investigation and Management**

*For this presentation history has very little yield for diagnosis*

**UACS**Treat any underlying sinusitis, avoid allergens
Hallmark of this syndrome is the lack of pathognomonic findings - diagnosis made by response to:
1. Antihistamines
2. Decongestants
NB: response may take weeks to months

**Non-asthmatic eosinophilic bronchitis**
Diagnosis made by induced sputum showing increased eosinophils
Highly responsive to inhaled corticosteroids

**GORD**
Treated with PPI

Therefore investigation should proceed with:

1. Stop smoking and other factors e.g. ACE inhibitor therapy (response may take one month)
2. Chest XR - to rule out pulmonary lesions
3. Trial of UACS treatment
4. Induced sputum
5. 24 hour pH monitoring
6. If the above show no yield then more extensive investigation required: sputum for TB, HRCT, bronchoscopy

### Globus

Globus is a persistent or intermittent painless sensation of a lump or foreign body in the throat

**Aetiology**

1. GORD - accounts for ~25 to 50% of patients
.
2. Non-specific oesophageal motility disorder
.
3. *Malignancy
.*
4. Psychological/stress
.
5. Others: retroverted epiglottis, thyroid disease, TMJ dysfunction etc

**Investigation and Management**

Like with chronic cough, the history is low yield but should be used to evaluate for malignancy, reflux & psychological factors

1. Full ENT exam including laryngoscopy - to exclude malignancy + other causes
2. 3 month trial of PPI therapy (e.g. omeprazole)
3. If unresponsive to PPI then further Ix: 24 hour pH monitoring, endoscopy and barium swallow
4. If above negative consider: psychiatry input

*If suspicious findings on history, exam or laryngoscopy then more aggressive investigation is needed*

### Pharyngoesophageal diverticulum

The herniation of the posterior pharyngeal/oesophageal mucosa and submucosa due to increased intraluminal pressure. Either due to lack of coordination of musculature or a hypertensive upper oesophageal sphincter

**History**Dysphagia
Regurgitation of undigested food from the pouch - with risk of aspiration
Halitosis

**Investigation**

Barium swallow
Fibreoptic endoscopic evaluation of swallow
+/- pH monitoring to assess for associated GORD

**Management**

The management of this condition is increasingly being handled by otorhinolaryngologists (c.f. general surgeons previously)

Transoral endoscopic treatment with CO2 laser or electrocautery is becoming standard

# Otology

## Ear infections

**Types of ear infections:**

* Otitis externa
* Acute otitis media (AOM)
* Otitis media with effusion (OME) or glue ear

*A large study performed in South Auckland found that over 25% of Pacific Island children have OME (2)*

*NZ Maori and Pacific Island children suffer from a high burden of OME*

### Otitis externa

Otitis externa is an inflammatory and infectious process of the external auditory canal (EAC) +/- auricle

Predisposing factors include:

* Heat
* Humiditiy
* Trauma (e.g. cotton bud)
* Exposure to water (e.g. swimming)
-> swimmers are particularly prone to it because repetitive swimming results in removal of cerumen & drying up of the EAC

**Aetiology**

Most common pathogens: *Pseudomonas aeruginosa, S. aureus*

Less commonly: *S. epidermidis, Proteus* spp., *E. coli,* diphteroids

**History**

* Otalgia (*ear pain*)
* Otorrhoea (*ear discharge*)
* Aural fullness
* Pruritis *(itchiness)*
* Tenderness
* Hearing loss *(due to oedema and debris obstructing EAC)*
* *If advanced there may be oedema and erythema of the auricle/pinna*

**Examination**

Using an otoscope: EAC erythema, oedema + otorrhoea
+/- Pain on distraction of the pinna
+/- Advanced: periauricular and cervical lymphadenopathy

**Management**

1. Antibiotic drops - Sofradex (framycetin sulphate/gramicidin/dexamethasone) *or* Ciproxin (ciprofloxacin)

2. Earwick insertion to stent open the EAC if there is occlusion (occlusion shown in right-most picture above)
- this is important in order to allow the antibiotic to reach the infected tissues

3. Aural suctioning (atraumatic debridement) with the use of a microscope - if experienced enough

4. Analgesia

5. If exostoses (surfer's ear) present: may need surgical management to stop recurrence of otitis externa

*Note: the steroid component in the ear drops helps with decreasing ear canal oedema*

### Acute otitis media

AOM is the inflammation and infection of the middle ear

**Aetiology**

Normal function of the pharyngotympanic tube (PT tube) is:

1. Middle ear aeration to allow pressure equilibration between the atmosphere and the middle ear
2. Mucociliary clearance of the middle ear space
3. While doing above it prevents aspiration from nasopharynx to the middle ear

*Underlying pathogenesis is pharyngotympanic (Eustachian) tube dysfunction - leading to pathogens passing from the nasopharynx into the middle ear*

AOM is usually preceded by a viral URTI - causes PT tube inflammation & therefore dysfunction
The most common pathogens are:

* *Streptococcus pneumoniae*
* *Haemophilus influenzae*
* *Moraxella catarrhalis*

**Epidemiology**

Peak incidence is is in *children aged 3 to 18 months* with incidence tapering as a child approaches adolescence

**History**

* Otalgia
* Fever
* Hearing loss
* Otorrhoea (if ear drum perforation)
* Can have decreased appetite and a concurrent URTI

*Note: AOM in children can present with fussiness and irritability and therefore an otoscopic examination should be part of a general paediatric assessment*

**Examination**

On otoscopy a bulging tympanic membrane (TM) with erythema can be seen

**Management**

1. Reassurance with analgesia and watchful waiting is appropriate for the majority of children as 80% will have spontaneous resolution within 2 to 14 days. In certain cases antibiotics are appropriate 1st line treatment e.g. severe illness, <6 months of age and not improved within 48 hours of watchful waiting

2. Oral antibiotics:
*1st line:* Amoxicillin
*2nd line:* Erythromycin or co-trimoxazole

*In paediatrics:
Drug doses are often given in ranges (e.g. 50 - 100 mg/kg/dose) - always give maximum dosage for their weight
Underdosing, by using lower end of range dosing, is one of the primary causes of failure of treatment*

3. Analgesia:
Paracetamol is 1st line
Ibuprofen if no contraindications

### Otitis media with effusion

**Aetiology**

* Like AOM the pathogenesis includes *PT tube dysfunction*
* Usually *follows AOM*

A number of hypotheses exist to explain the above findings but the following two are most often quoted:
1. PT tube dysfunction leads to loss of pressure equilibration of the middle ear with the astmosphere
- nitrogen is absorbed by the middle ear mucosa leading to the middle ear having a relatively negative pressure
- this elicits a transudate secretion by the middle ear mucosa and increased passage of pathogens into the middle ear
- this leads to chronic inflammation and effusion

2. The initial trigger is inflammation of the middle ear (via AOM and/or ongoing reflux from the nasopharynx into the middle ear via PT tube dysfunction)
- the inflammation induces a mucin-rich transudate

In both of these hypotheses the transudate also acts as a further culture medium for bacteria

The following risk factors are thought to worsen PT tube dysfunction:

* Parental smoking
* Absence of breast feeding
* Adenoid hypertrophy
* Day care attendance (increased exposure to pathogens)

**Epidemiology**

Most common in children up to the age of 15

Prevalence much higher in Maori and Pacific Island children compared to European (see blue box above)

**History**

* Often asymptomatic
* Commonest complaint = decreased hearing (usually noticed by parent)
* Recent AOM or URTI common
* Parents notice poor sleep in child (likely due to sensation of pressure)

**Examination**

*Otoscopy* (include pneumatic otoscopy):
Dull grey or yellowish immobile TM
If the TM is clear air-fluid levels can occasionally be seen

In adults flexible nasoendoscopy should be performed to exclude a nasopharyngeal tumour

**Investigation**

*Tympanometry* - allows testing of TM mobility and middle ear function (abnormal in OME)

*Audiology -* conductive hearing loss in OME

**Management**

Firstly patients are categorised into two groups:

1. **High risk group**: *children at risk for speech, language or learning problems*
This includes suspected hearing loss, language delay, autism, developmental delay or uncorrectable visual impairment

2. **Low risk group**: children with no suspicion of above problems

The low risk group can be managedvia *"watchful waiting":*

*-* Review 3 months after diagnosis. If still has OME -> audiology testing
- If hearing normal review again in 3 months time -> if OME persists (i.e. 6 months) needs ORL review
- A 10 day course of oral amoxicillin can be trialled but evidence base is small

The high risk group needs ORL review

**ORL review**The surgeon will consider placement of tympanostomy tubes (grommets) - see pictures below
Adenoidectomy will be considered in children with repeat need for grommets

## Cholesteatoma

**A destructive lesion of the skull base and middle ear formed by trapped squamous epithelium**

**Aetiology**

The trapped squamous epithelium forms a sac with keratin debris
Chronic inflammation and infection ensue leading to:
- growth and migration of the squamous epithelium
- osteoclastic activity
- causes PT tube dysfunction and oedema; providing a culture medium for bacteria

Can be *congenital or acquired*
- secondary to retraction of TM *(primary acquired)*
- squamous epithelium migration during surgery e.g. grommet placement *(secondary acquired)*

**History**

* persistent or recurrent purulent otorrhoea
* hearing loss
* tinnitus
* rarely: vertigo, ataxia, facial nerve paresis (from invasion)

*Hallmark symptom is painless otorrhoea*

**Examination**

*If due to retractions (primary acquired):*

* retraction of parts of the TM
* purulent otorrhoea, polyps and granulation tissue
* ossicular erosion

*If due to TM perforation (secondary acquired):*

* debris visible through the perforation

*If perforation has healed or occured secondary to surgery (also secondary acquired)*

* TM can appear normal
* once cholesteatoma is large - it may be able to be seen through the TM

*Cranial nerve function especially facial nerve should be checked in all patients*

**Investigations**

CT scan
Audiometry
MRI if facial nerve, labyrinth or intracranial involvement suspected

**Management**

*The mainstay of treatment is surgical - mastoidectomy and the extent of the operation will depend on the location of the cholesteatoma*

Medical adjunctive therapy:

* microscopic removal of debris from EAC
* keep all water out of ears - prevent contamination
* topical antibiotics

**Complications**

* bone erosion - including ossicular chain
* sensorineural hearing loss, dizziness
* facial nerve injury
* infection including: *mastoiditis, intracranial abscess, sigmoid sinus thrombosis, meningitis*

## Hearing Loss

**Aetiology**

Hearing loss can be divided into two types:

Conductive hearing loss (CHL) - *anything that decreases the transmission of sound to the cochlea*

1. cerumen impaction
2. middle ear effusion *including OME*
3. otosclerosis
4. cholesteatoma

Sensorineural hearing loss (SNHL) -*anything that stops transmission from the cochlea onwards*

1. congenital
- syndromic e.g. *Usher syndrome*
- non-syndromic - *80% is autosomal recessive*
2. perinatal causes *e.g. low birth weight or sepsis*
3. infections  *-* prenatal *e.g. CMV, rubella, toxoplasmosis, varicella*
- postnatal *e.g. OM, mumps, meningitis or HIV*
4. trauma
5. ototoxic drugs *e.g. aminoglycoside*
6. presbycusis/age-associated
7. neoplasms - *acoustic neuroma, cerebellopontine angle tumours*

**History**

- *include*: duration, nature of onset, progression and which side(s) affected

- *presence or absence of*: tinnitus, vertigo, imbalance, otorrhoea, headache, facial nerve dysfunction

- *and:* head trauma, ototoxic exposure, occupational or recreational noise exposure and a family history of hearing impairment

**Examination**

- Otoscopy - assess TM for retraction & with insufflation for TM mobility + compliance

- Nose, nasopharynx and oral exam +/- flexible nasoendoscopy to assess for neoplasm

- Cranial nerve examination with special focus on V, VII and VIII

- Rinne and Weber tuning fork tests

**Investigations**

1. Audiometry

2. Imaging

- CT if conductive (look for ossicular and other bone abnormality)
- MRI if asymmetric or unilateral SNHL (look for neural lesions)

**Management**

1. Environment
- reduce back ground noise
- ensure good lighting on speaker's face etc.

2. Amplification
- hearing aids
- bone anchored hearing aid (BAHA)

3. Cochlear implants

### Otosclerosis

Otosclerosis is a disease of the temporal bone - nearly exclusively of the otic capsule. It is characterised by abnormal removal of mature bone and replacement with thicker woven bone

This disease process has a predilection for the bone of the oval window and stapes footplate

*In 80 - 90% of patients the disease is limited to the oval window and stapes footplate*

**Aetiology**

Normal bone turnover that happens in other bones of the body does not occur in the healthy otic capsule after initial development
In otosclerosis increased osteo-clastic and -blastic activity leads to fixation of the stapes (*causing CHL)* and rarely extend into the inner ear *(causing SNHL)*

Thought to be due to genetic and environmental factors:
- often there is a family history (autosomal dominant with incomplete penetrance)
- associated with Measles virus (*? activates responsible gene)
-* fluoridation of water thought to be contributing to decreased incidence now

**Epidemiology**

~10% Caucasian population affected (clinical disease only ~0.5%)

Asians and Africans have much lower rate

**History**

* Slowly progressive hearing loss - usually bilateral and asymmetric (~30% unilateral)
* Tinnitus common
* Usually present between the age of 20 and 35

**Examination**

1. Otoscopy - usually normal but done to help exclude other causes of CHL e.g. cholesteatoma, OME etc
2. Rinne and Weber tests - CHL findings

Sometimes patients will talk quietly as they perceive their voices to be loud due bone conduction

**Investigations**

*Given normal examination findings, investigations are important*

1. Imaging - **CT diagnostic**
2. Audiometry - demonstrates CHL
3. Tympanometry - middle ear not affected therefore normal in early and mid stages. Abnormal once stapes fixation severe

**Management**

Management strategy depends on severity of disease and symptoms

Options are:

1. Observation with yearly follow up
2. Medication - occasionally sodium fluoride and bisphosphonates
3. Hearing aid
4. Surgery - stapedectomy (total or partial) with prosthesis placement

### Presbycusis

Presbycusis is otherwise unexplained SNHL in the elderly - usually bilateral & symmetrical

**Aetiology**

1. Genetic predisposition
2. Noise trauma
3. Diet, nutrition, ototoxin exposure
4. Multifactorial age-associated changes:
- Central pathology: decreased cell population in auditory cortex with increased synaptic and information processing time
- Cochlear pathology: loss of hair cells and supporting cells
- CNVIII fibre loss

**History**

Progressive hearing loss - particularly worse with ambient noise

Ask about:
- occupation especially military, industry (high noise jobs)
- family history

*Examination usually normal but performed to exclude other diagnoses*

**Investigations**

Audiometry is diagnostic
Consider other tests e.g. imaging if suspect other diagnosis (e.g. unilateral hearing loss)

**Management**

Options:

1. Hearing aid
2. Assistive devices e.g. telephone amplifier, headsets for TV
3. Cochlear implant (reserved for profound hearing loss)

### Investigations

**Audiometry**

Pure-tone audiometry is the most common test of hearing
The patient is asked to respond to sounds of decreasing intensity across the frequency range
Both air conduction and bone conduction can be tested

**Tympanometry**

A tympanometer presents a low frequency sound to the ear and measures the sound energy reflected from the eardrum

The eardrum is most floppy when the pressure on both sides of it is equal. If fluid is present in the middle ear, the eardrum is unresponsive to changes in pressure of the external ear canal and a flat tracing is observed

Tympanometry does not depend on major patient co-operation, so the test is quite useful in testing children for "glue ear" (otitis media with effusion)

## Vertigo and Tinnitus

Tinnitus is the perception of sound in the head or the ears.

Vertigo is defined as perception of movement in the absence of movement. It is caused by asymmetry in the baseline input into the vetibular centres, which causes the vertigo but also nystagmus and vomiting.

*It is very important to differentiate this symptom from other symptoms that patients may complain of* Typically patients will describe "dizzines", "light-headedness" etc. These are not specific and must be explored further by the doctor and differentiated into:
- pre-syncope
- syncope
- vertigo
- weakness
- unsteadiness or imbalance

**Differential Diagnosis**

Central

1. Ischaemia: TIA, stroke, vertebrobasilar insufficiency + migraine
2. Neoplastic: Acoustic neuroma (usually presents with unilateral progressive hearing loss)
3. MS

Peripheral (the first 4 diagnoses are in order of incidence)

1. Benign paroxysmal positional vertigo (BPPV)
2. Meniere disease
3. Vestibular neuronitis
4. Labyrinthitis
5. Others: otitis media, sinusitis

*Note: vertigo that doesn't start to improve within 48 hours may indicate a central cause*

**History**

- *As above, ensure you understand exactly which symptomatic entity the patient is describing i.e. is the patient truly describing vertigo?*- Timing: how long does it last?
- Onset: is it sudden?
- Vertigo association: do certain postures bring it about?
- Presence of tinnitus, hearing loss, otalgia, otorrhoea, aural fullness
- Preceding URTI
- Smoking and other medications or herbal remedies

- Review of systems:
- associated gait problems or ataxia (suggest a neurological cause)
- head trauma
- other medical problems
- other ENT diseases

**Examination**

**-** Vital signs including orthostatic BP
- Full ENT exam (particularly for signs of infection, hearing loss etc.)
*- Thorough neurological exam is mandatory with specific focus on cranial nerves, gait and cerebellar function*

Special tests:
- Dix Hallpike test
- Romberg and tandem gait
- Head thrust
- Caloric testing

**Investigations**

A range of specialist vestibular reflex testing exist

Imaging with MRI is indicated where there is asymmetric hearing loss an (acoustic neuroma may be suspected)

Baseline bloods should be performed e.g. FBC, U+E, Glucose

### BPPV

**Aetiology**

BPPV is defined as vertigo that is elicted by certain head positions. The positions trigger nystagmus.
The cause of this is unknown but is postulated to be due to 2 pathological mechanisms:

1. *Canalithiasis*: This is due to otoliths that have become detached from the otolithic organs and are free-floating; exerting a force on the cupula mechanism within the semicircular canals
(*analogy is: pebbles in a tyre - when the tyre turns the pebbles tumble down due to gravity; this tumbling triggers the nerves inappropriately*)

2. *Cupulolithiasis:* This is due to deposits of otoliths on the cupula themselves; rendering the cupula more sensitive to gravitational force with certain head positions
(*analogy is: heavy object placed on a skinny pole; extra weight makes pole unstable and harder to keep in upright position - pole easily clunks from one side to another and tends to keep tilted position*)

**History**

Onset of vertigo is sudden and severe - lasting ~30 seconds
Typically occur when moving the head in certain positions e.g. from lying to sitting
May be associated with nausea and vomiting

**Examination**

Diagnosis is made by Dix-Hallpike test
Remainder of examination should be normal

**Management**

1. *Canalith repositioning via Modified Epley Maneuver*

2. In the acute setting vestibulosuppresants can be tried for symptomatic relief:
- Promethazine
- Benzodiazepines e.g. lorazepam
- Scopolamine

Surgery reserved for severe intractable cases

### Meniere Disease

**Aetiology**

Also known as *endolymphatic hydrops* (hydrops is another term for oedema)

The cause is thought to be due to impaired reabsorption of the endolymphatic fluid
The precipitent factors is still unknown but postulated to be infectious, immunologic or allergic

**History**

Meniere disease occurs as attacks lasting for hours. The four symptoms are:

1. *Unilateral fluctuating SNHL*2. *Vertigo* lasting minutes to hours
3. Constant or intermittent *tinnitus* - typically increasing in intensity during vertiginous attack
4. *Aural fullness*

The attack is associated with nausea and vomiting - followed by feeling lethargic for a few days

**Investigation**

Meniere disease is a clinical diagnosis - based on history and normal examination
Audiometry is used to confirm SNHL

The following are performed to help rule out differential diagnoses:
- *Syphilis as this can perfectly mimic Meniere disease*- As noted before if hearing loss is asymmetric then MRI needs to be performed

**Management**

Acutely:
1. Vestibular suppresants - promethazine, benzodiazepines, scopolamine
2. Betahistine

Long term suppression
1. Dietary restriction of salt
2. Thiazide diuretic
3. Betahistine
4. Aminoglycoside injection into middle ear
5. Surgery reserved for severe and intractable cases

### Vestibular neuronitis

**Aetiology**

This is an acute, sustained dysfunction of the peripheral vestibular system causing vertigo, nausea and vomiting

*The most likely cause is reactivation of HSV in the vestibular ganglion and nerve* - other viruses e.g. adenovirus are also potential pathogens (note: do not confuse this with Herpes Zoster Oticus - which is a Varicella Zoster virus mediated disease)

**Epidemiology**

Commonly in 4th to 6th decades

**History**

Commonly have preceding URTI
Sudden and acute vertigo, nausea and vomiting *without hearing loss*Lasts for days -> cannot work or do usual activities

**Examination**

*Should have normal hearing and neurological exam (otherwise suspect something more sinister)*

Nystagmus will be present with slow phase towards injured ear

**Management**

1. Vestibular suppressants
2. Corticosteroids for 3 weeks (decreases chances of long term vestibular function loss)

### Labyrinthitis

**Aetiology**

This is inflammation of the labyrinthine structures including the vestibular and cochlear components

This may be caused by viral or bacterial pathogens

Viral
*Rubella and CMV* - can cause prenatal SNHL
*Mumps and measles -* can cause postnatal SNHL
Other viruses e.g. adenovirus, parainfluenza virus etc.
VZV can cause a special kind of labyrinthitis - Herpes Zoster Oticus - and if the facial nerve is involved it is called Ramsay Hunt Syndrome

Bacterial
Occurs secondary to otitis media or meningitis

Rarely, labyrinthitis can also be autoimmune

**History**

*The key difference between labyrinthitis and vestibular neuronitis is that you get SNHL in labyrinthitis*

Viral
Sudden loss of unilateral loss of vestibular function and hearing
Bedridden for days to weeks - some unsteadiness can last for months
Preceding URTI common

In HZV oticus Sx are deep, burning auricular pain followed by eruption of vesicular rash in EAC and concha

Bacterial
Can have symptoms of AOM, meningitis and cholesteatoma in addition to the above

**Examination**

A full examination as outlined in the "Overview" bar
In addition consider looking for signs of meningitis

**Investigation**

Baseline blood tests
Audiometry
Consider CSF in suspected meningitis

**Management**

Viral
As per vestibular neuronitis

Bacterial
IV antibiotics

If OM -> myringotomy with effusion evacuation
Mastoiditis and cholesteatoma will need surgery
Neurosurgery if intracranial abscess

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