ENT Lectures

Introduction to Otolaryngology

- **Pre-slides**
    - Everything in *Lecture Notes: ENT* is required in the exam + a book called *Key Topics: Foreign Bodies* chapter.
- ENT is an acronym for Ear, Nose, and Throat. This is the layman's term. The medical term is otorhinolaryngology.
    - Oto: ears
    - Rhino: nose
    - Laryngo: larynx
    - HNS: Head and neck surgery, maxillofacial, plastic surgery is an overlap area.
    - Base of skull surgery - Roof of the nose is the base of the anterior cranial fossa, etc.
- A patient comes through to our clinic with epistaxis (bleeding from the nose); physical examination will start before history passively. History is not always necessarily preceding the physical examination.
- History
    - Ear symptoms: Hearing Loss
        - hearing loss, ak.a. hearing impairment or deafness. When we say a person is deaf this doesn't mean that he doesn't hear me, this can be partial or complete deafness. other symptoms are ear pain (otalgia), discharge from the ear (otorrhea), tinnitus (hearing abnormal noise), vertigo (dizziness), ear itching, aural fullness (heaviness in the ear).
        - We have to ask the patient if its sudden or gradual, partial or complete, stable or progressive, constant or episodic, and unilateral or bilateral. We also have to tailor our treatment based on what the patient wants, what sort of occupation they have, etc. THe most important is bilateralism of the symptoms
    - Ear symptoms: Otalgia
        - We have to ask the normal SOCRATES associated with pain. Primary otalgia is a pathology in the ear, secondary is pathology outside of the ear (referred otalgia). Referred pain - two organs with same innervation getting the same symptoms. This means that we have referred otalgia.
            - REFERRED OTALGIA IS MORE COMMON THAN PRIMARY OTALGIA.
            - THE MOST COMMON CAUSE OF OTALGIA IS REFERRED OTALGIA.
        - Thus we have to know the sensory supply for the ear and the organs that they supply to allow us to know where to look for secondary pathologies. We will be able to do that in the physical examination. Upon finding an abnormal ear, this

is primary otalgia. If the ear is normal in examination, this means that there is secondary otalgia.

- Sensory innervation of the ear:
    - Vagus nerve (X) gives a branch to the external ear → Arnold's branch of the vagus nerve for pain sensation.
    - Trigeminal (V)
        - V1 Opthalmic
        - V2 Maxillary
        - V3 Mandibular: this is the one that supplies the ear.
    - Glossopharyngeal (IX) gives the pharynx and might be a cause of referred pain in the ear.
    - C2-C3 in the cervical plexus giving us two branches, greater auricular nerve and lesser occipital nerve supplying the external ear.
    - Facial nerve (VII) is mainly a motor nerve (⅔ of the fibers); the rest are sensory. They give special taste sensation through the chordi tympani nerve.
        - V3: Trigemenal for sensation, pain, touch and temperature.
        - Posterior one third is glossopharyngeal (IX) for taste, pain touch and temperature
    - Which structures might have pathologies and may result in referred otalgia?
        - Oral cavity through trigeminal
        - Pharynx through glossopharyngeal
        - Nasal cavity through trigeminal
        - Dental through mandibular branch of the trigeminal
            - DENTAL IS THE MOST COMMON SITE OF ORIGIN OF REFERRED OTALGIA
        - TMJ through trigeminal
        - Larynx through vagus nerve (2 branches - recurrent laryngeal and superior laryngeal nerve)
        - Cervical disc prolapse/occipitus skin lesion through C2-C3
- Ear Symptoms: Otorrhea
    - Colour - blood-stained or not. If it's yellowish, bacterial infection. Greenish → pseudomonas aeroginosa (gram-negative bacilli) is one of the worst bacterias to have an infection from because it's multi-drug resistant.
        - History of trauma: clear, serous fluid due to CSF leak or otorrhea.
    - Character - serous fluid from CSF, purulent → bacterial infection, mucoid → tympanic membrane perforation or rupture.
    - Tympanic membrane layers: Middle fibrous layer, outer layer is stratified squamous, inner layer → anything but stratified squamous. ADD THE LAYERS OF THE EAR AND THEIR COVERING
- Ear Symptoms: Tinnitus

- Objective and subjective tinnitus
    - Objective: examiner may even hear the tinnitus " TMJ problems "
    - 99.9% of the tinnitus is subjective; only the patient hears the tinnitus.
- Very common symptoms, 10% of the general population have tinnitus. Presbycusis (presby - age related changes in hearing) due to nerves resulting in hearing loss. A lot of the older population like to stick the radio to their ears. This is so they can overcome or ignore the abnormal tinnital noise with a different kind of noise of their choice. Some of the 10% have tinnitus so severe that it is affecting their everyday function and environment. It will usually affect the patient more in night because it is a quiet environment.
- Pulsatile/nonpulsatile: one of the best descriptions of tinnitus. The patient might hear their heartbeat, which is pulsatile tinnitus. Anything can cause either. With pulsatile tinnitus, we have to think of a few things to rule out. Anything with hearing rule may cause tinnitus.
    - Causes of pulsatile tinnitus:
        - Carotid atherosclerosis
            - In the middle ear, we have the carotid artery running from below, curving anterior and inferior to the middle ear, going inside and then curving again and then anastamoses with the circle of willis. This kink at the very beginning with atherosclerosis at that area will cause the change of blood flow from normal to turbulent flow. This change will be right beside the ear.
        - AVM, carotid aneurysm
        - Glomus Jugulare Tumour
            - The venous drainage from the brain is the superior and inferior sagittal sinus, to transverse, lateral, sigmoid sinus. At the sigmoid sinus, we have the jugular bulb, which is right below the middle ear. Some cells in the jugular bulb form a tumour resulting in venous hum and pulsatile tinnitus.
        - Hypertension
            - Uncontrolled HTN patients will have bilateral tinnitus because the blood tension will increase on the blood vessels.
        - Hyperdynamic circulations
            - the blood will keep being turbulent → anemia polycythemia vera, thyrotoxicosis, pregnancy, exercise, fever, thiamine deficiency, Paget's disease of the bone (if it hits the temporal bone → new vascularization → turbulent blood flow)
            -

- Ear symptoms: Dizziness/Vertigo
    - What is the difference between dizziness and vertigo?
        - Vertigo is a true rotatory movement of the patient, the things around him, or anything. Dizziness is a general term than encompasses vertigo as well. Anything may cause dizziness and/or vertigo. the inner ear comprises only 20% of cases of dizziness; ear is not the only cause behind dizziness. Hyper/hypotensive/glycemia, electrolyte imbalance are major causes of dizziness. tumour, MI, CVA, hemorrhage, cervical disc prolapse are other minor causes of dizziness. Hypo/hyperthyroidism, vitamin D deficiency are other causes of dizziness. NOT EVERY DIZZINESS → EAR CAUSE
            - Syncope, light-headedness, vertigo are kinds of dizziness.
    - A patient with dizziness or vertigo, we suspect the problem is in the ear. We ask the patient about the duration (excluding non-otological causes) and they respond with
        - Seconds and minutes especially related with changes of position → Benign Paroxysmal Positional Vertigo BPPV
        - Hours - 2 days → Menierre's disease
        - Days - weeks → Labyrinthitis or vestibular neuritis
    - we have to ask about the impact on daily life → is it getting better or worse? This will determine how aggressive we are in the management or treatment of the patient.
- Ear symptoms: Ear itching
    - Any inflammatory process in the external ear may cause itching.
- Ear symptoms: Aural fullness
    - heaviness in the ear
    - eustachian tube: connects the nasopharynx and the middle ear
        - Functions (3)
            - Ventilation of the middle ear
                - Equalization of the pressure around the tympanic membrane
            - drainage of the middle ear secretions
                - The middle ear is lined by mucosa. This mucosa goes through the eustachian tube down to the nasopharynx.
            - Prevents reflux of nasopharyngeal contents
                - By closing. The levator vili pallatini and tensor vili pallatini muscles will constrict. Salpingo
                - Tensor villi is supplied by the mandibular nerve from the trigiminal but levator villi and sulpingopharengeous are supplied by the pharyngeal plexus mainly from the pharyngeal branch of the vagus

        - With eustachian tube dysfunction, the ventilation in the middle ear will decrease, the pressure will thereby decrease as well through the resorption of air. Thus, it will have a relative negative pressure resulting a retracted tympanic membrane. the patient who gets that will feel aural fullness. When we go down in the plane, this will reverse and the ear will 'pop'.
- Nasal symptoms: General
    - We have to stress asking about the unilateral/bilateral causes. Bilateral → function problems such as inflammation. Unilateral might be mass (polyp), fracture, foreign body.
    - We have to ask the patient if it's continuous or intermittent. If continuous, this might mean it's an anatomical problem.
- Nasal symptoms: Sleep apnea, snoring, mouth breathing, etc.
    - sleep apnea - cessation of breathing for at least 10 seconds
        - We can divide it according to
            - Obstructive
                - At the nasal cavity, nasopharynx, oral cavity, oropharynx
            - Central
                - Respiratory center in the brainstem, especially in prolonged congested patients
            - Mixed
    - Snoring - Stertor is snoring when awake
        - Partial obstruction. The level of obstruction will be <u>supralaryngeal</u>. if it was complete obstruction, we would get apnea and no air would come out.
        - A partial obstruction at larynx or trachea, the patient will get stridor
    - Rhinorrhea - Nasal discharge
        - Colour, uni/bilateral, unilateral serous (CSF rhinorrhea and leak), character (serous → viral rhinosinusitis or allergy), purulent (bacterial), mucoid (allergy)
        - Sneezing probably due to any inflammatory condition
        - Lining epithelium for the mucosa → ciliated pseudostratified columnar epithelium. This mucous through ciliary movement is pushed into the oropharynx. The amount of mucous production daily is 500 - 1500 ml.
        - Sometimes the nerve endings in the pharynx (GERD reflux) will cause increased sensitivity to the nerve endings that the patient will get annoyed with even the normal production of mucous.
    - Headache
        - The patient will feel pain right where the sinuses are affected. sphenoid → parietal or occipital pain. facial → facial headache. second premolar

and first two molars' roots go into the floor of the maxillary sinus → with a dental pathology, this will cause sinusitis.

- Epistaxis - bleeding from the nose
    - Unilateral vs. bilateral
    - have to ask about the symptoms of anemia
    - bleeding from other orifices
    - drugs: anticoagulant, antiplatelets.
    - history of trauma
- Nasal deformity
    - Too big or small nose
- Change in smell sensation
    - Temporal lobe of the brain is responsible for smell sensation. The pathology might thus be central, like temporal lobe seizures, CVA, tumours, hemorrhages all in the area of the temporal lobe.
    - Olfactory nerve (I) might have a pathology.
        - hyposmia is the reduction in the ability to smell in the patient.
        - anosmia - total loss of smell sensation in the patient.
        - dysosmia - abnormal smell sensation

1) parosmia: smell an orange like a banana. Smell something like another

2) cacosmia: Smell something that is bad when there's a normal smell. "caca"smia (I died a little inside putting this in the notes). Chronic rhinosinusitis might result in cacosmia.

§**Lectures**

§**Physical Examination**

§Ear examination

○Review anatomy

§External auditory canal is around 1 inch. The outer one third is cartilaginous. The inner two thirds are bone. The outer one third has hair follicles, inner two thirds with no hair. The outer one third has seromenous (check) glands (ear wax) and the inner two thirds with no glands.

§The middle ear cavity has three ossicles: malleus, incus and stapes. A part of the malleus is stuck to the tympanic membrane and it is called the handle of the malleus. The smallest bone in the body is the stapes. The stapes is also the most complete ring. The largest and most lateral is the malleus. The stapes is stuck to the middle ear, called Foot plate of the stapes. This plate articulates with the oval window.

§The Eustachian tube connects the middle ear to the nasopharynx.

§Temporal, zygomatic, buccal, marginal mandibular, cervical are the branches of the facial nerve.

§Chorda tympani, a branch from the facial nerve, innervates the taste in the anterior two-thirds of the tongue.

§Tensor tympani muscle and stapedius muscle with innervation from trigeminal and facial respectively.

§Tegmen tympani splits the roof of the middle ear.

§Cochlea and vestibular apparatus for hearing and balance respectively. The cochlea is two and a half turns. The cochlear or auditory nerve comes out of the cochlea. The vestibular apparatus is divided into vestibule and semicircular canals. The vestibular has urticle and saccule. Out of these canals, we get the vestibular nerve which joins the cochlear. The vestibulocochlear nerve passes the CP angle and goes to the pons.

§The inner ear has two types of fluid: perilymph, which is high in sodium low in potassium like ECF. The endolymph is like ICF, which is high in potassium low in sodium.

○We have to clean our hands and wear the headlight with every single examination, especially for the OSCE. We have to ask the patient about which ear the patient has pathology. This will allow us to know the normal background anatomy of the patient. This will also allow me to start with the ear that has no pain and establish rapport with the patient.

○*Inspection*: We look for ear deformities. The helix, anti-helix, tragus, anti-tragus, lobule and cocha.

§We have protruding or bat ears, which is a cosmetic deformity. We can correct that through otoplasty.

§Microtia: small ear. Anotia: the pinna would not be present.

§Aural atresia: the external auditory meatus would be closed off.

§Sinus/pit: preauricular or postauricular sinus or pit. The difference between sinus and fistula – abnormal communication between two epithelial surfaces. The sinus – communication between an epithelial surface and a blind cavity.

§Accessory auricles(?) – any skin tag or cartilage that is "extra".

§Cauliflower ear (also called Wrestler's Ear) – occurs after trauma followed by auricular hematoma.

·Cartilage is an avascular structure. It gets its perfusion from perichondrium. With trauma, there will be spacing between them, ischemia, and necrosis. Thus we have to do incision and evacuation of the hematoma.

·The patient will come with a swelling and we will feel it tense. We will enter with a needle aspiration, if blood, it's positive. To prevent recollection, we have to cover with a gauze and compress with a bandage to prevent further damage.

·The hematoma might become an abscess as well and create further problems.

§We look for signs of inflammation, such as redness, swelling, or discharge, ulceration, hypo or hyperpigmentation.

§Scars: post-auricular scar from surgery. End-aural scar will be present obliquely in the preauricular surface. With tympanic membrane perforation, we have to repair that through tympanoplasty or myringoplasty (myringo = tympanic membrane). We use fascia, periosteum, veins, etc. But we mostly use facia and/or cartilage (most popular two types of treatment).

·Tragal scar – evidence of using the tragus for previous repair.

○Palpation:

§We move the pinna and see if we have auricular tenderness, press on the tragus and see if we have tragal tenderness. This might be a sign of otitis externa.

§We also palpate the mastoid bone. If tendernesss, sign of mastoiditis.

§Fistula Test (perilympathic fistula): we press on the tragus, close the canal for 10 seconds, then sudden release. Positive test is if the patient had nystagmus, or the patient told you they felt dizzy afterwards.

·Nystagmus: rapid, oscillatory, involuntary movement of the eye

○Percussion:

§Percuss the mastoid for mastoid tenderness.
○Otoscope:
§We always have to choose the right ear piece for the right patient. It isn't universal for everyone. We use the biggest but most appropriate ear piece for each patient. An adult = big, pediatric = small. Adult with otitis media = small.
§Pneumatic otoscope: checks the mobility of the tympanic membrane as well as doing the otoscopy. When we press, the tympanic membrane should go medially.
§We always have to examine the patient with the right ear pathology with our right hand.
§We have to grab the otoscope like a pen.
§We should pull the pinna upwards, backward, outwards, to straighten the canal. This is for adults.
§Pediatrics -> just backwards.
§Neonates -> backwards and inwards.
§Is the external auditory canal adequately patent or is it narrow? We have mention any signs of inflammation, discharge (with its own classifications as well), skin lesions, masses, foreign bodies, tumours, neoplasms.
§We look at the tympanic membrane, comment on its normal or abnormal structure.
·Normal: semi-transparent, grey-pearly colour, intact, normal position (not retracted nor bulging), presence of cone of light, presence of handle of malleus, mobile tympanic membrane through Valsalva maneuver
○Cone of light: anteroinferior quadrant.
○Handle of malleus: backwards and downwards
○Valsalva: have the patient close their nose and mouth and try to "breathe"
○Tympanic membrane is made up of pars flaccida, e.g. attic (upper small part with two layers, missing the fibrous layer), and pars tensa (lower larger part). With Eustachian tube dysfunction, pars attica moves easier. When it retracts, it's called retraction pocket. Cholesteatoma occurs with the keratinization of pars attica when it gets retracted; presence of squamous epithelia in the middle ear (Google this and add to notes).
§Impacted wax – wax closes off the view of the meatus completely.
§Tympanic membrane perforation
·Site, size (if less than ¼ of tympanic is small sized, if ¼ - ½ medium, more than ½ large), wet/dry (depending on presence of secretions), marginal or central
§Myringotomy – hole in the tympanic membrane, we put a device called ventilation tool for ear effusions.
○Tuning fork tests: At least 256 forks, even better is the 512
§Rinne Test: We grab the tuning fork with the stem. We hit the olecranon process or patella. We go in lateral to the ear by one inch, at the mastoid process. Air conduction louder -> positive (? Check). We have to do it at three positions behind the ear, at the pinna, then at the tragus.
§Weber Test: Any midline ear prominence then we ask which side is louder. Again, with the stem to the bone.If both are heard equally, centralized. If one side is more, then it is lateralized to [that side]. If the patient can't hear any side, it's called indifferent.

§These examinations are not called normal or abnormal, they're called positive or negative and centralized/lateralized. Don't use any other terminology.

○Free field hearing test: talk to the patient and see what the intensity of their hearing is.

○Facial nerve: Ask the patient to elevate their eyebrows, and have them look at the examiner's finger from up to down. Then close your eyes tightly and don't let me open them. Show me your teeth (look at angles of the mouth for drooping), whistle, and blow out your cheeks. Ask the patient to grimace for symmetry, then ask the patient for taste.

○Nasopharynx examination: (check)

§Hearing loss:

○Conductive hearing loss: pathology in the external/middle ear.

○Sensory hearing loss: pathology inner ear, nerve, brainstem.

§Nose Examination:

○Anatomy

§Nasolacrimal duct connects the inferior meatus to nasolacrimal sac. It is the only structure that connects to the inferior meatus.

§Maxillary sinus, frontal sinus and anterior ethmoidal air cells connect to the middle meatus.

§Superior meatus – posterior ethmoids

§Sphenoethmoidal recess – sphenoid sinus

§Osteomeatal complex (OMC) is the part of the middle meatus that connects the sinuses. This is important.

§Ciliated pseudostratified columnar is the lining epithelium.

§Base of the tongue is the posterior 1/3 of the tongue. Floor of the mouth is inferior to the tongue.

§The distance is 7-8 cm between the anterior aspect of the nose to the posterior.

○Inspection:

§Front, lateral, and behind the patient for any nasal deformities. Comment if the patient is a mouth-breather. Comment if the patient has epiphora (hyperlacrimation). Comment if the patient has allergic salute (transverse skin crease superior to the nose holes). Comment if the patient has allergic shiners (black spots around the eye). Comment on scars (lateral rhinotomy scars for example). Lift the tip of the nose and look at the transverse columellar scar and anterior dislocation of the nasal septum.

○Palpation:

§palpate the nose and look for localized tenderness or crepitus, emphysema, any fracture lines, etc.

§palpate the sinuses

○Percussion: tenderness on the sinuses.

○Anterior rhinoscopy:

§Look at the nose from anteriorly using a nasal speculum. Here, we use the Thudichum's speculum. We examine both sites using our left hand. We go in the nose closed, we go out of the nose open.

§Five things:

·Nasal septum: is it straight or deviated? If deviated, to which side? Which is wider cavity as well? Intact or perforated?

·Turbinates: mesotrophic (normal size), hypertrophic or atrophic?

·Mucosa: Normally pink in colour. Pale mucosa -> allergic rhinosinusitis.Red -> erythematous inflammatory mucosa.

·Masses: Polyps, neoplasm, foreign body, clot, rhinolith. We do suction to differentiate the polyp from mucous. The polyp has yellow-greyish colour. The polyp is shiny, the turbinate isn't. The polyp is soft to the touch, turbinate is hard. The polyp has no nerve endings -> insensate. The polyp has a stalk while the turbinate doesn't. The polyp most commonly isn't bloody, if so, we have to determine if there's a neoplasm.

·Discharge: Colour, where? Character, amount, etc.

○Nasal patency test:

§Metallic testing with vapourization upon breathing. If on the right side there's more condensation, this means the right is more open.

§We can also bring little cotton pieces that move upon exhaling out.

○Posterior rhinoscopy:

§Looking at the nasopharynx through a mirror. We put a long mirror through the oral cavity. We have to do warming of the mirror and depress the tongue.

○Nasal endoscopy: There's rigid and flexible nasoscopy.

○Lamina papyracea divides the ethmoid and the orbit. It's oblique to the orbit.

§***Throat Exam:***

○Lips

§Cleft lip, ulcers, masses, lesions

○Teeth

§Cavitations, dental carries, teeth cap, filling, braces, dentures for upper and lower teeth

○Gingiva

§Masses, ulcers, lesions

○Buccal mucosa

§Parotid duct (Stinson) opposite to the upper second molar tooth.

○Tongue

§Macroglossia, fasciculations, motility of the tongue, geographic, glossitis

○Floor of mouth

§Submandibular ducts (Warthon's duct – lateral to the lingual frenulum), frenulum

○Palate

§Cleft palate, ulcers, masses, lesions

○Uvula

§Bifid or not. 30% of bifid uvula have submucous cleft palate -> Eustachian tube dysfunction.

○Tonsils

§Anterior pillar and posterior (palapharyngeal and palatoglossal pillars). Ulcers, masses, lesions. The tonsil if they are large or small. Presence of crypts and they are preserved -> no tonsillitis. Pus, follicles, lesions.

○Posterior pharyngeal wall

§Ulcers, masses, lesions, postnasal drip or discharge.

○Larynx

§Indirect laryngoscopy

·Warm the mirror and go through the oral cavity. Pull the tongue forward and point the mirror down. Ask the patient to make a sound to see the vibration of the vocal cords. Vocal cords with phonation go through adduction. At inspiration, they go through abduction. With adduction, there is turbulent air flow. Articulation of speech is through the tongue, buccal, mouth, lips, etc.

·Hyponasal speech: when a person has a flu or sneezing. This is because the nose or the sinuses is less than usual, if more than usual -> hypernasal speech. This is similar in the cleft palate patients.

·False vocal cords are at a higher level than true vocal cords.

§Neck exam

○Scars, masses, striae, lesions, hyperpigmentation

○Palpation

§Mass, I have to test it. I also have to test thyroid and lymph node groups.

○Percussion

§Mass

○Auscultation

§Mass or goiter

○Consistency, tenderness, fixed or mobile, attached to underlying structures, pulsations, transilluminations, fluctuance.

○Lymph nodes

§Submental -> submandibular -> upper anterocervical, middle anterocervical, lower anterocervical -> posterior border of cervicals -> pre-auricular -> post-auricular -> suboccipital

§Cranial nerve examination

○Add this

External Ear Diseases

§Complications of auricular hematoma -> necrosis, abscess, and cauliflower ear.

§Wax becomes darker and darker with time. Starting with white-yellowish, to yellow-brown, to dark brown after long periods of oxidation. It can be soft or hard. Wax presence is usually asymptomatic. When symptomatic, it's usually total obstruction.

○soft/hard, colour, impaction

○Hygroscopic feature: if it comes in contact with water, it will expand.

§Ear syringing: position has to be posterosuperior. 3% of people who get ear syringing get tympanic membrane perforation. Solution used is normal saline, tap water; it has to be warm (37 +/- 4). This is done to prevent caloric reaction -> patient will get vertigo or nystagmus if outside of that range. If the wax is too hard, we have to give the patient waxolytic, such as olive oil. The patient can use dewax, or Waxol as well if he prefers to take drugs.

○Complications: Tympanic membrane perforation, caloric reaction, impaction, injury of the canal wall, otitis externa. Otitis externa patients need to keep their ear dry.

§Otitis externa

○Bacterial

§Can be localized or diffuse. The most common type is the diffuse. The localized usually occurs in the outer one third in the hair follicles. This is called furunculosis. This is infected hair follicles. The furunculosis occurs in the outer one third because it is the only part that has hair follicles. If you see one that extends into the middle ear, you have to suspect a neoplasm. The most common to do diffuse is pseudomonas aeruginosa (g – bacillus) followed by staph aureus.
§Otalgia, otorrhea (purulent), hearing loss (conductive), usually no fever
§Signs: redness in the meatus, discharge, swelling
§Palpation: tenderness on the auricular and tragal aspects.
§Percussion: no tenderness on mastoid.
§Otoscopic examination: Stenosed canal with decreased patency, discharge and debris.
§Management: aural toilet (suction discharge) and keeping the ear dry, topical antibiotics (neomycin/gentamycin/fluoroquinolones (ciprofloxacin)/chloramphenicol) + systemic (if there's cellulitis/furunculosis)
Fusidic acid also
○Otomycosis
§Aspergillis niger, candida albicans respectively are the most common causative agents for otitis externa.
·Ear itching is more in fungal infections
§Conductive hearing loss
§Palpation: aural pain (auricular and tragal)
§Management: aural toilet and keeping the ear dry, with topical anti-fungal (clotrimazole, miconazole, nystatin)
○Viral
§Also called herpes zoster oticus, e.g. Ramsay–Hunt syndrome
§Management: Aural toilet and keeping the ear dry. Oral steroids (this will decrease the chance of residual problems such as facial nerve) and acyclovir
And you should protect the eye because of the facial palsy
○Malignant otitis externa
§It behaves like a malignant disease but it isn't one. It is so severe that it affects the bone. The ear is present in the temporal bone. This disease is also called necrotizing otitis externa, another name is base-of-skull inflammation (base of the skull Osteomyelitis). Most commonly caused by pseudomona aeruginosa, most affects immunocompromised patients (chronic steroid users/abusers, elderly, diabetic, AIDS, etc. have a higher chance of getting this infection).
§Aural toilet and keeping the ear dry, inpatient, topical and system, debridement of necrotic tissue, control the underlying immunocompromised status.
§On xray scan, the temporal bone is "hot". The bone is eaten on CT scan.
§Antibiotics for at least 6 weeks, IV antibiotics. Take a swab culture as well. Pipracillin (tazocin), third-generation (ceftazidime, cefotaxime), fourth-general (Cefepime ?), fluoroquinolones, carbapenems, impinem (Check this)
§Possible complications: Brain abscess, meningitis, encephalitis, any cranial nerve from 3-12 can be affected by this disease.
§**Nasal and Paranasal Diseases**

§Started with a review of basic nasal anatomy. Discussed in the aforementioned notes.
§We can have obstructive causes and neural causes of anosmia/hyposmia.
○Obstructive: congestion
○Neural: trauma and fracture that tears the nerve fibers (anosmia that is permanent if transverse cut of the fibers). URTI is the most common cause of permanent anosmia, a serotype virus would have destroyed the nerve.
§**Rhinosinusitis**
○They are combined together because they are common cavities that are opening into each other. More than 90% of the cases come as both rhinitis and sinusitis. They also have the same epithelia: ciliated pseudostratified columnar epithelia. It's divided into allergic and non-allergic.
○Allergic (seasonal and perennial)
○Non-allergic (infective and non-infective). The non-infective are rhinitis medicamentosa and vasomotor and others (hormonal drugs and so on). Infective is acute and chronic.
§Alpha-2 agonist drugs are the ones that cause rhinitis medica. When we stimulate the alpha-2 receptor drugs, the congestion goes down. This is for the sympathetic. The parasympathetic will increase congestion. There is usually a balance between these.
§In vasomotor RS, they have parasympathetic overflow → ~20% of the patients have it. This can be seen with patients who need a constant supply of napkins while eating for example. The rhinorrhea is usually serous → mucoid. Bilateral usually.
·H-1 Blockers: 1st gen. Diphenhydramine and chlorphenramine
·H-1 Blockers: 2nd gen. Citrazine, Loratidine, Fixofinadine
·H-1 Blockers: 3rd gen. Levocitrazine, desloratidine
·We can give antihistamines to these patients, or topical nasal steroids to decrease the inflammatory process. Mometasone, declomethasone as topical nasal steroids.
§The patient will get rebound phenomena or tachyphylaxis <CHECK SPELLING> if we give alpha-2 agonists. This usually occurs after 3-5 days of starting the treatment. This is why we tell patients not to take these drugs more than 3-5 days.
§Alpha-2 agonists' examples: ephedrine, xylometazoline, naphzoline, dimethindene.
§Allergic rhinosinusitis
○Allergic RS is IgE mediated, i.e. Type I Hypersensitivity reaction.
○Symptoms:
§Rhinorrhea: serous in acute, mucoid in chronic.
§Sneezing/itching
§Epiphora
·Due to congested mucosa closing the lacrimal duct
·Or due to allergic conjunctivitis " in atopy syndrome"
§Postnasal drip
§Cough/Sore throat
§Eustachian tube dysfunction symptoms
·Aural fullness
·Hearing impairment (conductive)

§Olfactory: Hyposmia/anosmia due to congestive mucosa
§Headache: the OMC closed, collecting the secretions and pushing against à headache
○Signs:
§Epiphora, rhinorrhea, mouth breather, allergic salute and shiners
○Investigations:
§Hypertrophied turbinates, postnasal drip
○Diagnosis:
§Skin-prick test: Intradermal or subdermal injection of most common 25-30 allergens. Wait 15 minutes. The area will become elevated (more than 3 mm) and red = allergy from that allergen.
·Risk of anaphylaxis
§IgE levels: we can do a blood test for the total Ig. It's non-specific
§Radio Allergo Sorban Test (RAST): Testing IgE levels against specific allergens, up to 300-400 allergens.
○Management (4-line therapy):
§The only cure in all of these is avoidance.
§Medical: topical nasosteroids and antihistamines. If the patients' main symptoms are nasal itching, sneezing and rhinorrhea, then antihistamine is better. If the main symptoms are nasal obstruction, postnasal drip, etc., steroids are better.
§Joke: ليش الناس بتخاف من الحسد؟ لانه ملك الغابة. #مطارنة #نفر_فورغيت
·Nasal steroids usually start working after a week. The dose is usually for a month then there's a check-up. We can give the patient oral/IV steroids, but they will have these side effects.
·Antileukotrienes, such as monteleukast and zafirlukast
·Omalizomab <Check this>: anti-Ig antibody.
§Surgery: We do surgery so the patient gets better with medical treatment. It's not curative in any way. It includes getting rid of nasal polyps and such.
§Immunotherapy or desensitization: if the patient is allergic to pollen for example, we give them the same allergen and then increase the number of pollen gradually. There are subcutaneous (called allergic shot à subcutaneous allergic therapy) and sublingual drops. This will allow us to get immunomodulation changing it from IgE pathway à IgG pathway.
·Its problem is that the patient might be allergic to something else as well. After treatment:
○1/3 have a permanent cure
○1/3 temporary cure
○1/3 No change
§Septal deviation
○When we have an opacified maxillary sinus, we think of a dental problem. There will usually be a problem in the second premolar and first two molars.
§Acute Rhinitis
○Acute vs chronic
§< 12 weeks: acute ; >12 weeks: chronic
○More than 85-90% of the cases are viral. Most common virus is *rhinovirus*, but it may be caused by any other virus
○Coryza is another name for acute infective viral RS.

○10-15% are bacterial. Most common cause is Streptecoccus Pneumonia , H. Influenzae , Moraxella Cataralis <CHECK SPELLING>
§H. Influenza is type B in RS, nontypable in otitis media.
○Symptoms:
§Sneezing, itching, etc.
○Signs:
§Mouth breather, red nose, Epiphora, etc.
○Investigation:
§Rhinoscopy: hypertrophied turbinates, congested mucosa, postnasal drip
○Management:
§Usually self-limited. Bed rest, antipyretic, analgesic, supportive treatment, decongestant (topical not more than 5 days).
§7-10 days: I start giving antibiotics for at least 10-14 days. Amoxicillin + clavulanic acid is first line. Second-general cephalosporin is second line. I can give the patient macrolides as well.
§Surgery:
·FESS: Functional Endoscopic Sinus Surgery
§Complications of RS
○Intracranial
§Epidural abscess
§Subdural abscess
§Cerebral (brain) abscess
§Meningitis
§Encephalitis
§Cavernous sinus thrombosis
○Extracranial
§Orbital
·Orbital cellulitis
·Subperiosteal abscess
·Retro-orbital abscess
·Cavernous sinus thrombosis
§Non-orbital
·Otitis media
·Pharyngitis
·Laryngitis
·Tracheaitis
·Bronchitis
·Pneumonia
·Gastroenteritis
·pott puffy tumour: Not a true neoplasm. Osteomyelitis in the anterior table of the frontal sinus. It is a sequestrum. Managed with incision, drainage and debridement and then give the patient antibiotics.

○The medial aspect of the forehead to the eyes and a bit of the nose is a dangerous zone because it shares the venous drainage with the cavernous sinus.
§What passes through the cavernous sinus?
·3,4, first two 5, 6, and internal carotid artery
§Presentation?
·Ill, sick, febrile, proptosis, headache, cranial neuropathy, pain or hypoesthesia in V1 and V2, CVA
§Management
·Broad-spectrum IV antibiotics
§Investigation
·Magnetic Resonance Venogram MRV to see the sinus
§Nasal polyps
○The polyp is edematous mucosa. They are divided according to the cause to three:
§Inflammatory (most common), neoplastic, and *antrochoanalpolyp*
○The nasal polyps are common in adults, uncommon in pediatrics. I will have to check children for cystic fibrosis (exam question), immotile cilia syndrome (Kartegener Disease) <CHECK SPELLING>,
§Never do biopsy for the polyp in children; it might be meningoencephalocele.
○Choana is the communication between nasal cavity and nasopharynx. Antrochoanal polyp is an uncommon unilateral pathology that mostly affects adults. It is the benign proliferation of mucosa. Management of it is surgery.
○*Neoplastic*: We have to take a biopsy, and determine its malignancy and its nature. Then I have to do staging and the management is according to type and stage. If small, we do total excision. If not, we take a biopsy.
○Inflammatory
§Caused by allergic RS or infectious RS. Most commonly bilateral.
§Symptoms:
·Nasal obstruction, rhinorrhea, headache, anosmia, hyposmia, postnasal drip, Eustachian tube dysfunction symptoms, Epiphora
§Investigations:
·Discharge, postnasal drip, etc.
§Management
·All of these patients will require surgery and all of them will have a recurrence. After surgery, I will start medical therapy. I will give them anti-allergic medications. Antihistamines, nasal steroids, antibiotics, monteleukast. Once the medical therapy doesn't work, I will do FESS. Doing this will slow the recurrence and/or prolong it. Then get another medical treatment. The polyps will still probably come back again.
§SAMTER Triad:
·Triad of nasal polyposis, bronchial asthma, and aspirin intolerance. The patients with the triad are the most difficult patient to control the nasal polyps and bronchial asthma.
§**Vertigo**
§Vertigo is true rotatory movement; part of the umbrella term of "dizziness".
§What are the inputs of balance in the person?

○Inner ear (NOT MIDDLE EAR), cerebellum, ocular, joints and tendons, C-spine, proprioception. All of these go to the cortex which coordinates them and controls the body position.
§Most important question: What is the duration of the attack?
○Seconds: Benign paroxysmal positional vertigo
○Minutes – hours : Meniere's disease
○Hours – days: Labyrinthine failure
§Cerebellar exam: Finger-nose test, rapid alternating hand-movement (dysdiatokinesia), heel-toe test, heel-shin test <CHECK SPELLING>, nystagmus (if currently vertigo), Romberg's test (heels together with supinated extended arms) (positive is if the patient falls or loses balance).
§*Benign Paroxysmal Postural Vertigo***
○The macula is the organ of proprioception in the vestibule. They get stimulation for the body change acceleration, position of the head, etc. A part of the macule is the otolith. They are calcium crystals. Sometimes, they disjoin from the macule and go to the lateral semicircular canal (most commonly) to the cupula (organ of proprioception in the semicircular canals).
§No hearing loss, no otalgia, no otorrhea, nothing but vertigo
○Dick's Hallpike Test
§Ask the patient if they have a problem with the c-spine. The head will be over the end of the bed and the patient will be looking to the right, ask them to look at a single point for 30 seconds, then have them sit and do the other side.
·Ask the patient to look at a single point throughout the movements (i.e. look at the examiner's nose).
·Ask the patient to keep their eyes open no matter what (a positive test is nystagmus. Thus they have to keep their eyes open).
§If nystagmus on one side, they will have positive BPPV on the ipsilateral side. If they have BPPV, the examiner will do Epley Maneuver.
·Epley Maneuver: <ADD THIS>
§When they sleep, they have to sleep with 60-70 degrees.
§Success rate for the Epley Maneuver: 70-80%
§Meniere's Disease
○There is perilymph and endolymph. Meniere's disease is Endolymphatic Hydrops (increase in endolymph)
§Four symptoms (any 2 is Meniere's):
·Hearing loss (sensory)
·Vertigo
·Aural fullness
·Tinnitus
§These are positive provided you excluded the neurological causes
○75% unilateral
○When patients get the vertigo attacks, it lasts hours up to 2 days. They get the symptoms throughout this period.
○As the attacks repeat, the sensory-hearing loss gets worse. At first there's no effect but repeated attacks à problems.

§Chief complaint in first years: vertigo, later years: hearing loss and tinnitus become more severe.
○Management:
§Medical Therapy:
·Low-salt diet will decrease the repetition of the attacks
·Low-dose of loop diuretics such as hydrochlorothiazide
·Vestibular sedative for patients who *just* got an attack. This is best with a benzodiazepine (diazepam, metazolam, etc)
·Patient with nausea and vomiting à anti-emetic and IV fluids.
·Beta-histine (betacirc) is a partial H1-agonist and antagonist (agonist in places antagonist in others). It decreases secretion of endolymph and <SOMETHING ELSE>
§Surgical Therapy:
·Labyrinthectomy for the middle ear
·Vestibulocochlear nerve cutting (losing sensation from that side of the ear)
·Medical labyrinthectomy –injection in the middle ear in the tympanic membrane putting in a grommet and injection of aminoglycoside which diffuse through the round ear that does ototoxicity à labyrintehectomy
·Endolymphatic sac decompression
§Labyrinthitis
○Usually viral, preceded by URTI, self-limiting, symptoms are vertigo and sensorineural hearing loss and tinnitus
○Vertigo days to weeks
○Management: bed rest, anti-emetic, vestibular sedative, supportive treatment.
§Vestibular neuritis
○Same as labyrinthitis
○Virus goes to vestibular nerve, vertigo without hearing loss and tinnitus
§Perilymphatic fistula
○Opening between inner ear and middle ear, symptoms are vertigo, sensorineural hearing loss and tinnitus
§Management: ear exploration and closing up the hole
§Acoustic neuroma:
○Shwann-cell tumour in the vestibular branch but may be in the acoustic nerve. It's also called vestibular shwannoma. More than 2/3 of the cases in the vestibular nerve.
○They come with unilateral (95%), bilateral (5%) in NF-II
§Sensorineural hearing loss and tinnitus
○Investigation: MRI for the CP angle (most common place for tumours)
○Management based on site, size, and symptoms.
§Radiation, surgery, etc.
§Nystagmus
○Can the nystagmus help us know if the cause is peripheral (inner ear) or central (brain etc)
§Nystagmus is horizontal in peripheral
§Nystagmus can be horizontal, vertical, or pendular in central

§Thus, unidirectional nystagmus is more peripheral. Multidirectional nystagmus is more central.
§Peripheral nystagmus has latency (a couple of seconds); central has no latency
§Peripheral vertigo's nystagmus is fatigable (nystagmus goes away after 30 seconds); central isn't
§Peripheral nystagmus improves with visual fixation (look at my finger); central doesn't

Adenotonsillar diseases

-Drugs can be a cause of adenotonsillar diseases. There will be deposition of the inhaled material on the throat. Anything that results in obstruction can result in sore throat. Smoking and alcohol are chronic irritants of the throat à sore throat. GERD is another.
-Systemic causes: drugs such as methimazole, metronidazole. Aplastic anemias, systemic infections such as HIV, infectious mononucleosis (CMV less commonly).
-Head and neck areas' surgeries will cause sore throat because they will cause damage to the salivary glands à minimal saliva à dryness and irritation to the throat.
-*Diphtheria*: gram-positive bacillus, part of the immunization program. Pseudomembrane or membrane on the tonsils and pharynx. Sometimes, they extend downwards. They might come with dyspnea, stridor, etc. easily bleeding from the membrane. There are two problems with this bacillus: the symptoms might result in airway compromise. It may produce cardiotoxins and neurotoxins. I will take a swab for culture and sensitivity but I will start with penicillins and macrolides, such as azithromycin etc. as empirical treatment. However, we have to give the patient antitoxin serum as well to counteract the effect of toxins from the diphtheria.
○Infectious mononucleosis (glandular fever/kissing disease) is a diffuse lymphadenopathy disease. Patients come with fever, sore throat, dyspnea, etc. These patients may have the disease up to 6 weeks or two months. If you think it's infectious mononucleosis, we have to do monospot test or Paul-Bunnel Test.
§Management: supportive therapy, encourage oral hydration, antipyretic for fever, analgesic for pain. It's a self-limited disease so we shouldn't give antibacterial/antiviral.
-Waldeyer's Ring
○We have a lymphoid collection like a ring, called adenoids/nasopharyngeal tonsils at the midcircle are the tubal tonsils, further down are palatine tonsil, and at the base of the circle is the lingual tonsil. Check the picture in the slides for a better representation. The palatine tonsil are the largest collection of them.
§What's the difference in anatomy between adenoids and tonsils?
·Adenoids are posterosuperiorly, palatine tonsils are in the tonsillar fossa in the oropharynx on both sides. The adenoids -> pseudostratified ciliated columnar, tonsils à stratified squamous epithelium. Adenoids are usually a collection of one while tonsils are 2. Palatine tonsils have crypts (8-16); adenoids have furrows on their surface (wrinkling on their surface) (2-4 furrows). Adenoids are unencapsulated. Tonsils are encapsulated. Adenoids have afferent and efferent lyphatics. The tonsil only has efferent lymphatics.
-Adenoids

○Adenoids are a pediatric disease, they grow until 6-7 years then they get smaller until 12-14. Check the slides for Fig. 1: Adenoidal size in relation to age.
○Nasal polyps on the other hand are an adult disease but it can occur in pediatrics uncommonly.
○Symptoms of adenoids:
§Mouth breathing, sleep apnea, snoring.
·The most common cause of ??is adenotonsillar hypertrophy
§Hyponasality, Eustachian tube dysfunction. Patients will come with impaired hearing.
§Mouth-breathing children might also have teeth crowding. This occurs because there is no contact between the jaws due to the mouth breathing; the teeth would all grow as they wish rather than how they're supposed to.
§Bilateral rhinorrhea: the nose and paranasal sinuses produce mucus and we usually swallow it. If the patient has adenoids, they will instead expel the mucous through the nose.
○Signs of adenoids:
§Rhinorrhea, mouth breather
§Typical age with typical symptoms
§Rhinoscopy going through the nose and looking at the adenoids. We can also do lateral neck soft tissue xray (as seen in the slides). We look at the airway column that got constricted in the area of the nasopharynx. This is probably due to a soft tissue mass. The true diagnosis is intra-operative.
○Indication for surgery:
§Symptomatic
§Surgery is called adenoidectomy using the curette.
○Complications of surgery:
§Complication from the mouth gag, lacerating the tongue, hard palate, tongue, etc.
§TMJ dislocation while opening the mouth
§C-spine injury while extending the neck
§Bleeding and infection at the site of surgery. Infection from the Eustachian tubes giving us otitis media.
§Regrowth of adenoid tissue. More common in younger patients.Seen in 10% of the patients.
§Rhinolalia apperta
·Velum is another name for the soft palate. When we eat, the velum would go up and the posterior wall to move to create the velopharyngeal sphincter. Patients with cleft palate will have velopharyngeal incompetence. With the adenoid present, there would be no nasal regurgitation of food. With getting rid of the adenoids, the patients will become hypernasal, nasal regurgitation of fluids and food. This condition post-surgery is called rhinolalia apperta.

§10% of Down Syndrome's patient have atlantoaxial subluxation. Thus, we can't extend the head of the patient. Before doing any adenoidectomy for them, we have to make sure they don't have the subluxation. If they are, I have to know what angle they have as well.
-Tonsils
○Grading system of tonsils
§The numbers go from 1-4, draw a line through the midline and anterior pillar and see how many "quarters" of the boundary between these lines the tonsils take up. +4 tonsils are sometimes called kissing tonsils.
○Acute tonsillitis
§Tonsillitis usually extends to the pharynx so it's somewhat synonymous to discuss both at the same time under the term tonsillopharyngitis.
§85% of the cases of acute tonsillopharyngitis are viral. 15% are bacterial. Adenovirus, and other viruses.
§Most common bacteria are group A beta hemolytic bacteria; strep pyogenes. This lateral group may be part of the normal flora in 30% of the population as well. Since it is most commonly viral, it's usually self-limiting. The symptoms get worse for 4 days and then get better.
§Symptoms:
·Odynophagia, sore throat, fever, otalgia (referred through glossopharyngeal (9) nerve), cough, etc. Halitosis (bad odour)
§Signs:
·Enlarged tonsils, absence of crypts, pus,hyperemia, tonsillar lymph nodes, upper anterocervical lymph nodes may be enlarged.
§Management:
·Supportive; encourage oral intake, bed rest, analgesic, antipyretic when needed.
·We give antibiotic upon seeing pus on the tonsils, it stayed for more than a week-10 days, or when the patient has precious time.
○Amoxicillin + clavulanic acid
§Clavulanic acid gets rid of the pencillinase from the bacteria, allowing for bacterial resistance to go down.
§Complications:
·Septic: peritonsillar abscess (quinsy), parapharyngeal abscess, retropharyngeal abscess, otitis media, laryngitis, tracheitis, bronchitis, pneumonia, or go down to the GI tract to complicate gastroenteritis
○Quinsy: there's a capsule on the lateral aspect of the tonsil. Pus will collect between the capsule and lateral aspect and create an abscess. It will push the tonsil medially. When you examine the tonsils, you will find that one of the tonsils is pushed medially. You will also find edema on the soft palate. To test for peritonsillar abscess, we go to the upper pole of the

anterior pillar, laterally from the upper pole at 1 cm, and push in a needle up to 1 cm and aspirate. Pus is positive for Quinsy. Management is incision and drainage. DEPTH IS NO MORE THAN 1 CENTIMETER. There's a 30% chance of first recurrence. 90% chance of second recurrence. First recurrence is usually indication for tonsillectomy.

·Aseptic: Rheumatic fever, glomerulonephritis, scarlet fever, and febrile convulsions

○Rheumatic fever criteria John's criteria

§Major (any 2 majors, or 1 major and 1 minor)

·Sydenham's Chorea

·Erythema marginatum

·Rheumatic heart disease

·Subcutaneous nodules

○Rheumatic fever cause: antibodies against strep pyogenes. There's antigen mimicry. The antigens on the heart are attacked by the antibodies due to this mimicry.

○Glomerulonephritis: immune complex deposition in the glomeruli (post-streptococcus GNP – crescentic).

○Scarlet fever: maculopapular rash from the neck down. They will get circumoral pallor. The tongue may be red; strawberry tongue. Scarlet fever is pathognomonic for strep pyogenes.

○Febrile convulsions: 6 months – 6 years, children get convulsions due to raise in temperature but not a neurological cause.

§Indications of tonsillectomy:

·Recurrent acute tonsillitis

○An attack came, completely healed, then another attack, completely healed, etc. Recurrent acute tonsillitis = at least 7 attacks/year or 5 attacks/2 consecutive years (10), or 3 attacks/3 consecutive years (9).

·Obstructive symptoms from large tonsils

○Mouth breathing, sleep apnea, snoring, etc.

○Dysphagia is another type of obstructive symptoms. The children will have failure to thrive, post-tonsillectomy à their weight increases.

·Complicated tonsillitis: i.e. febrile convulsions

○Quinsy

○Rheumatic heart disease: to slow the progression of RHD.

○Glomerulonephritis

·Asymmetric tonsils (one is +1, another is +4)

·Suspicion of malignancy: a tonsil may have a chronically ulcerated surface unilaterally. This may be a cancer.

Otitis Media

- The pathophysiology usually starts with Eustachian tube dysfunction. Otitis media is commoner in pediatrics rather than adults, because the ET is wider, shorter and more horizontal in the pediatric age group. Pediatric disease is in the nasopharynx (adenoids), which may result in ET dysfunction. Thus, they have a higher chance of that.
- Borders of middle ear?
    - Lateral: Tympanic Membrane
    - Medially: Inner ear
    - Superior: Tegmen tympani
    - Inferior: Jugular bulb
    - Posteriorly: Additus ad antrum
- Reasons behind ET dysfunction:
    - Anatomical: nasopharyngeal masses (adenoid), nasopharyngeal cancer, cleft palate, Down Syndrome patients
        - Tensor levi palatine, salpingopharyngeus, levator palatine muscle. There will be abnormal orientation of the muscle fibers in cleft palate and thus the ET will not open and close properly.
        - Down syndrome patients have a patulous ET.
    - Functional: Mucociliary abnormality,
    - Risk factors: Not being breastfed, daycare attendants
- Its Classifications
    - < 12 weeks: Acute
    - > 12 weeks: Chronic
- Acute Otitis Media (AOM), Chronic Suppurative Otitis Media (CSOM), and Otitis Media with Effusion (OME)
    - CSOM's characteristics are TM perforation that lasted more than 3 months.
        - Any perforation that lasts more than 3 months is CSOM; even if it's a dry perforation.
    - OME's characteristics is an intact TM and collection of fluid behind it.
- Recurrent Acute Otitis Media (RAOM)
    - 3 attacks in 6 months or 4 attacks in a year. A child with RAOM needs to be tested for hypogammaglobinemia. This child will take a prolonged course of antibiotics.

AOM

- This is usually preceded by an URTI. ET dysfunction, affecting mucociliary movement, leading to stagnation and otitis media. Usually bacterial but 25% of the cases are viral.
- "its most common to be viral (RSV) but there will be super added bacterial infection in 50% of cases " not sure about this
- Once diagnosed, we have to give the patient antibiotics regardless.

- Streptococcus pneumonia, H. influenza non-typable, Moraxella catarrhalis
- Symptoms
    - Conductive hearing loss, tinnitus, otalgia, fever (especially in pediatrics), 10% mucopurulent otorrhea (when there's perforation of the TM),
        - Pediatrics patients will show up with ear tugging and inconsolable crying as a sign of otalgia.
- Signs
    - Discharge in 10% of the patients
- Palpation
    - Mastoid tenderness if accompanied with mastoiditis
- Percussion
    - Mastoid tenderness
- Otoscopy
    - Discharge in 10%; early-stage OM – hyperemic TM, late-stage OM – bulging, and/or perforated TM, fluids and pus behind the TM
    - Aural toilet and keep the ear dry if perforated TM
- Management
    - Oral antibiotic (systemic)
    - Augmentin (Amoxicillin + Clavulanic acid) for two weeks
    - We don't give fluoroquinolones to children, because they cause early growth of the epiphyseal growth plate.
    - Traumatic tympanic perforation healing rate is 94% at 3-months.
    - Ragged/serrated edges with blood clots at the edges are a sign of traumatic tympanic membrane perforation.
- Perforation healing process
    - Squamous epithelium, mucosa with the middle fibrous layer
    - Epithelium, mucosa without the middle fibrous layer – slight scar
    - Epithelium, mucosa, with sclerosis – myringosclerosis
- Perforation description
    - Site, size (small (< 25%), medium (25-50%), large (<50%)), discharge, central/marginal

OME

- Usually bilateral ear effusion in the middle ear cavity that has been there for at least 3 months with an intact TM. Before 3 months, it's called simply middle ear effusion.
- Also called secretory otitis media, serous otitis media, and glue ears. Usually these names are added intra-operatively.
- Management

        - Surgery through myringotomy with ventilation tube insertion.
            - Short-acting ventilation tube: Grommet tube (97% success rate in 3-months with the appropriate treatment), if I assume the patient's ET dysfunction will get better within a year
            - Long-acting ventilation tube: T-tube (73%), if I assume the patient's ET dysfunction will need more than a year to get better.
            - Possible complications to this procedure: otorrhea (due to infection) → aural toilet and keeping the ear dry and ear drops. If the patient doesn't get better, this would be a foreign body reaction and remove it. Other complications are dislodgement of the tube, residual tympanic membrane perforation, myringosclerosis, and tympanosclerosis.
            - the myringotomy may not heal and the case will be converted into CSOM
        - During the waiting period of 3 months, we give the patient antibiotics, topical steroids, etc. " we treat him as acute otitis media"
        - We can also do adenoidectomy
- Symptoms
    - Ear discomfort (no discomfort), aural fullness, hearing loss (conductive), tinnitus, sits close to TV
    - However, most of these patients are pediatric and thus are giving you wrong symptoms.
    - MOST COMMON CAUSE OF CONDUCTIVE HEARING LOSS IN PEDIATRICS IS OME.
- Signs
    - Otoscopy: Intact, dull tympanic membrane. Retracted "may be bulged also" , bluish-hue or yellow amber TM, air bubbles behind TM

CSOM

- Classification
    - Active (wet)
        - Most common bacteria causing this is pseudomonas aeruginosa
    - Inactive (dry); or
    - Safe
        - This means that it is a dry pars tensa perforation
        - Safe or unsafe according to chance of acquiring an associated cholesteatoma
    - Unsafe
        - Chronically discharging pars tensa perforation
        - Pars flaccida perforation
    - Atticoantral
        - Pars flaccida perforation (addicus ad antrum perforation)

        - Tubotympanic
            - Pars tensa perforation
- Symptoms
    - Otorrhea, hearing impairment (conductive), tinnitus, vertigo (if it reaches the inner ear)
- Physical examination
    - Inspection: otorrhea if active CSOM
    - Palpation: mastoiditis if associated
    - Percussion: mastoiditis if present
    - Otoscopy: perforated TM
    - Postnasal space: normal

Questions

- TM perforation
    - Central: remnants all the way around
    - Dry
    - Medium-to-large (taking up more than 50%)
    - Myringosclerosis

there is a sheet for the otitis media lecture was done by the previous batches

https://www.facebook.com/groups/142839389386457/permalink/187089651628097/

Epistaxis

- Blood supply for the nose
    - Internal carotid system from the anterior ethmoid and posterior ethmoid.
    - Superior labial artery, sphenopalatine, and greater labial artery from the external carotid
    - Thus it is rich in blood supply. They anastomose anteriorly on the nasal septum on the Little Area. This plexus is called Kisselbach's plexus. They all anastomose except for the posterior ethmoidal artery. Thus, anterior epistaxis is more common (90% of case). This is because of the Kisselbach's area that is more prone to trauma. Posterior epistaxis is thus less common.
    - What are the causes of epistaxis?
        - Localized

            - Most common cause is spontaneous/idiopathic bleeding. This excludes children, whose most common cause of epistaxis is nasal picking, also called nasal digitorum.
            - Trauma, foreign body, sinonasal tumours, septal perforation, septal deviation (the submucosal blood vessels become exposes and easily torn from the area that the septum is going to where the outside air would hit that area and dry it out making it easier to get vascular injury then.
        - Systemic
            - Hemophilia, thrombocytopenia, warfarin/aspirin drugs, leukemia, lymphomas, hypertension, Hereditary hemorrhagic telangiectasia (Ossler-Weber-Rendu syndrome – autosomal dominant disease with dilated blood vessels usually in the nasal cavity, oral cavity, tracheobronchial tree, GI tract)
            - Hypertension
                - A patient with 160/100 who has epistaxis. Stress and anxiety from the bleeding → epinephrine and norepinephrine secreted which increase the HR and BP.
                - A patient with 200/130 who has epistaxis. Stress and anxiety from the bleeding are not enough to describe this case and thus this patient probably has underlying hypertension.
                - Hypertensive patients get two problems:
                    - First: Epistaxis is at a higher risk for posterior ethmoidal than anterior ethmoidal (10 – 30%). The former is more different to control.
                    - Second: They get arteriosclerosis. Even with the sclerosis, it wouldn't close. The sclerosis would be in tunica media.
    - How long have you been bleeding? Which area? Unilateral or bilateral? Scanty or plenty? Symptoms of anemia? Bleeding tendency? Trauma? Drugs for bleeding? Nasal symptoms? Has it ever happened before? Any underlying medical problems?
- Outline of management of epistaxis:
    - Manual digital pressure: External compression on the alar cartilage in a slightly flexed position. This clot will create a tamponade effect. Bleeding time is usually 2-8 minutes, thus we press continuously for 10 minutes.
    - Cautery in its two kinds: Chemical/electrical

        - Chemical cautery with silver nitrate sticks. Just touch a dilated blood vessel or a mild ooze, it will turn into white and then black. If the ooze is severe, there will be washout of the silver nitrate solution. Thus don't use it for severe cases.
    - Anterior pack
        - We leave it 1-3 days. We give this patient prophylactic antibiotic adjuvantly.
        - Merocel: a cotton about 6-7 cm, which you put lidocaine on, and then just put it inside the nares of a patient. It's easier to put than the anterior pack in its classic form.
        - Balloon catheter: 7-9 cm balloon catheter
    - Posterior pack
        - We remove the anterior pack, then administer the Foley's catheter through the nose. When we see it in the mouth, I will pull it slightly until I can't see it, where it will be in the nasopharynx. I will put 10 cc and clamp it. We leave it 3-5 days according to the patient's position.
    - Angiogram with embolization
    - Surgery: ligation of the area of bleeding

Foreign Bodies

- Pediatric age group, especially toddlers, 1-4 years are the most common age group to ingest foreign bodies. Mentally retarded patients are another group. Up to 25% of people with a foreign body might have another.

Ear foreign bodies

- Symptoms: bleeding per ear, hearing loss, tinnitus
- Signs: part of foreign body, swelling, redness, wax
- Otoscopy: Foreign body, inflammation signs, TM perforation
- Complications: Ossicular complications
- Management: Depends on symptoms, size, and nature of foreign body.
- If insect, kill it first

Nose foreign bodies

- Nostril excoriation (abrasion) is a sign of a foreign body closing off that area. "especially when it is unilateral"
- Symptoms: Epistaxis, leakage, postnasal drip, headache, rhinosinusitis, Epiphora
- Signs: Part of foreign body, rhinorrhea, CSF
- Rhinoscopy: Site, size, laceration, swelling
- Complications: rhinosinusitis, epistaxis, rhinolith, foreign body inhalation and aspiration.

- Management: Nature of foreign body, your skills, and patient's condition. You can put topical local anesthesia, and local decongestant.

Pharynx

- Symptoms: Bleeding per mouth, sore throat
- Most common sites: Vallecula, base of the tongue, tonsils
- Management: take it out

Larynx

- Symptoms: Choking, cough, wheezing, stridor, SOB, hemoptysis, sore throat, otalgia (referred pain)
- Management: direct laryngoscopy and take it out.
- Foreign body might actually go back and deposit in the retropharyngeal space and becomes a retropharyngeal abscess. You can go in with a needle and aspirate that area to make sure of the nature of the swelling.

** there is more notes about foreign bodies in the esophagus in the sheet :)

Upper Airway Obstruction

- Signs and symptoms
    - Cough, shortness of breath, apnea, stridor (awake or asleep; partial obstruction at the larynx or trachea), sturtor (snoring while awake – partial obstruction supralaryngeal), use of accessory muscles (rectus abdominus, intercostal, subcostal), cyanosis, nasal flaring
- Stridor
    - Divided into inspiratory, biphasic and expiratory.
    - Inspiratory: upon breathing in; supraglottic or glottis site of stenosis
    - Biphasic: subglottic stenosis or extrathoracic trachea (cervical trachea)
    - Expiratory: Thoracic trachea

Choanal atresia

- Choana is the communication between the nasal cavity and the nasopharynx (also called posterior nares).
- Can be unilateral or bilateral
- Unilateral:
    - Patient gets unilateral rhinorrhea. It starts from a mucoid discharge then it becomes a mucopurulent discharge due to the overriding infection.
    - Age of presentation is months to years.

- Bilateral:
    - Presentation at birth. This is because children are obligate nasal breathers until 6-9 months. They can't be mouth breathers.
- It can be membranous, bony or mixed. It's usually mixed.

Croup

- Also called laryngotracheobronchitis.
- This is an inflammatory condition usually caused by viral. Most commonly parainfluenza virus. It is self-limited. Usually stays a week. Peaks at 3rd day. Any other virus can cause it, but the most common virus is parainfluenza virus. Usually, it is preceded by upper respiratory tract infection. When a person as a sneeze and gets this. The cause of symptoms is inflammation and edema.
- Symptoms: Dyspnea, shortness of breath, stridor, cough, hoarseness, low=grade fever. Some patients get barking cough. Patients usually come to the ER only at maximum edema (usually on the 3rd day of presentation as well).
- Investigations:
    - Chest x-ray: tapering airway column, also called Steeple Sign or Pencil Sign.
- Management: Racemic nebulizer (3 nebulizers, 30 minutes between each) + steroids (takes 1.5 hours to work). Then the patient goes home with oral steroids. (for 2-3 days)
- Sometimes, these patients come back to the ER worse than they already were. Thus, we have to give them endotracheal intubation.

Laryngomalacia

- Most common cause of stridor in infants. The laryngeal cartilages are usually pliable, and easily collapsible upon birth. The muscles and neural reflexes are not completely developed and thus it is easier for them to collapse upon each other. Sometimes, these patients have such a severe case that it would suck the air in and cause obstruction. The patient will then get sleep apnea, failure to thrive due to eating dysfunction, and 10% get cor pulmonale. At 2-3 years of age, the patient will outgrow the disease. Severe cases, however, will need surgery because sometimes the patient won't make it.
- The breathing of the patient will be louder in different positions, prone or supine.
- Signs: Omega sign (omega-shaped glottis)
- Management: Observation for mild, surgery for severe.

Management outline of upper airway obstruction

- Oxygenation: I have to ask myself if the oxygen is enough to bypass the problem.
- Medication: Do I need to add B-2 agonists, cholinergic, and steroids?
- Endotracheal intubation

- Cricothyroidotomy
- Tracheostomy

Cricothyroidotomy

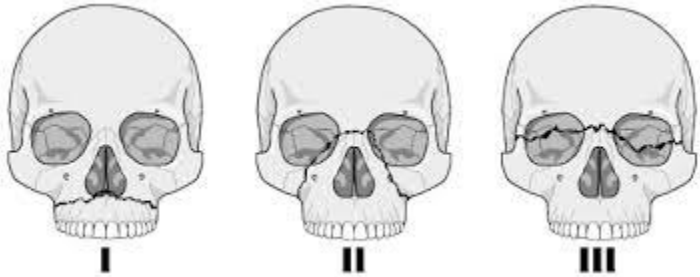
- Contraindicated in pediatrics younger than 12 years of age.

Tracheostomy

- Incision (vertical or horizontal) usually above the suprasternal notch (one or two fingerbreadths). We have to retract the strap muscles laterally, until we get to the thyroid gland, after that we see the pretracheal fascia. We make an incision between the 2-3 or 3-4. Not 1-2 rings because it increases the chance of subglottic stenosis. Not 4-5 because that's too low.
- Indications
    - Most common indication is prolonged intubation; a patient that needs prolonged ventilation for at least two weeks.
    - Upper airway obstruction (laryngeal tumour, oropharyngeal cancer, etc.)
    - Part of another procedure
    - Pulmonary toilet
    - Prevent aspiration
    - Sleep apnea
- Complications
    - Injury to blood vessels, nerves, esophagus, C-spine
    - Pneumothorax
    - Decanulation and false passage
        - After five days, we get a well-formed tract between the tracheostomy and the trachea. The first five days are dangerous. Decanulation might occur and it gives a false passage. The patient will get subcutaneous emphysema, pneumothorax, etc.
    - Tracheoinnominate fistula
        - Fistula will occur between the tube and the underlying innominate artery
- Benefit: Easier nursing care; easier to suction, decreases dead space

Facial Trauma

- We should always ask the patient about the mechanism of trauma. It may be soft tissue injury or skeletal injury. It may be blunt trauma or penetrating injury, or it might even be a slap to the face. We should ask about the other symptoms; CSF leak, constitutional symptoms, etc.
    - We ask about other problems, such as nasal deformities, otalgia, otorrhea, blood per ear, decreased hearing, tinnitus, vertigo? Pain anywhere else in the mouth/face?
- Physical examination

    - Localized pain anywhere
    - Nasal deformities, lacerations, ulcers, lesions, epistaxis, CSF leak, feel for crepitus, anterior rhinoscopy, blood clots, lacerations in the mucosa, septal hematoma, foreign body in the nose
    - Ears: avulsion, lesions, canal exam, perforation, CSF leak, etc.
    - Eyes: Raccoon eyes, swelling, bruises, extraocular motility (limitation to eye movement), visual acuity
    - Mouth: lacerations, missing teeth, dentures, ulcers
    - Neck: subcutaneous emphysema, hoarseness of the voice, mobility to the vocal cords, ecchymosis in the larynx, hematoma, avulsion of the vocal cord
- Management
    - Depends on mechanism of injury to the patient.
    - Tetanus for dirty wounds.
- Le Fort Fractures
    - Just know which type of fracture they are
    - 

- Blow-out fractures
    - What passes through the floor of the orbit? The infraorbital nerve, which is the continuation of VII. There might be compression and impaction of the nerve leading to paresthesia of that area. The orbital contents may herniate through the orbital sinus. The patient would then get enophthalmos. There might be impaction of the inferior rectus muscle, leading to limited upward gaze.
- The nose is more prominent and thus is more at risk for trauma. It also has different structures which may react to different forms of injury. For this patient, if the patient gets injury to one side, it will go to the other side. If anterior injury, they will get depressed nose.
- Complications of nasal trauma
    - Septal hematoma: blood pooling in the nasal septum, there will be separation between the cartilage and perichondrium. If we didn't diagnose this properly, there might be necrosis of the cartilage. Hematoma is also a good medium for bacterial overgrowth leading to septal abscess. The septum in septal hematoma is widened. If we touch it or try to probe it, it will be soft to the touch. In this case, to make sure, we will go in with a needle and do aspiration. Management is incision and evacuation of the septal

hematoma. We might even put splints or silastics and suture for 24-48 hours. This will prevent the recollection of the hematoma. We will give them prophylactic antibiotics as well.

- CSF leak
- Cribriform plate fracture
- Septal deviation
- Septal perforation
- Nasal deformity
- Epistaxis
- Rhinolith

Septal perforation

- Causes can be divided to traumatic and non-traumatic
    - Traumatic (more common)
        - Iatrogenic (more common in adults)
            - This is due to medical practice. An example of this is septoplasty. We usually make an incision from inside the nose, lift the mucosa and perichondrium, and cut the deviated parts. If while lifting, we get trauma on either side, the cartilage inside will die leading to septal perforation.
            - Another reason is cautery. If we cauterize an epistaxis patient on both sides, it might lead to perforation. Proper procedure entails that we cauterize one area only and switch every two weeks and three weeks. We only cauterize one at a time. Don't forget that.
            - Another cause is nasal cannula. Some patients take oxygen. If the tip of the nasal cannula keeps harming the patient, it might lead to perforation.
            - Chronic use of topical decongestants. Due to the severe vasoconstriction, it will lead to death of blood supply and death of cartilage
        - Non-iatrogenic (more common in children)
            - Foreign body, nasal piercing, nasal digitorum, cocaine abuse (potent vasoconstrictor and traumatic mechanism of abrasion)
    - Non-traumatic
        - Sinonasal tumour (malignancy leading to septal perforation due to erosion of the septum by the tumour), chronic inhalation, granulomatous diseases of the nose (TB and Syphilis),
        - Hereford's syndrome: sarcoidosis

- Symptoms
    - Mostly asymptomatic
    - Divided according to perforation diameters
        - Small
            - Less than 1 cm
        - Medium
            - 1-2 cm
        - Large
            - > 2 cm
    - If small perforation, 70-80% cure
    - If medium, 40-50%
    - If large, < 10%
    - Patient might come with nasal dryness, crusting of the nose (might supersede an infection), nasal obstruction (due to open laminar flow), whistling (from the nose with small perforations),
- Surgical
    - Graft from the ear
    - Mucosal rotation
    - In large perforations, there's something called septal button (a grooved plastic piece) and try to put it on the septum.

Hearing Loss

- Round window's function is to release the pressure as a protective mechanism.
- The cochlea is two and a half turns of the vestibular apparatus.
- A cross-section of the cochlea will give us three compartments <ADD>
- Tonotopic arrangement: Every part of the cochlea is used for a specific frequency. The higher we go (to the apex), the lower the frequency.
- Characteristics of CHL
    - Rinne: negative; Weber: Toward the affected side; PTA: ABG > 10 and upper line > 30
- Preserved speech discrimination: this is unlike sensorineural hearing loss, which has poor speech discrimination.
- Paracusis: Patient can hear in a noisy environment better than a quiet environment.
- Recruitment phenomenon: Found in SNHL not CHL. The nerve fibers are weak, to do action potential they need to reach the threshold. Sometimes, you are unable to reach the threshold, when you keep making your voice louder, it almost reaches the threshold. When it reaches the threshold, the patient hears everything very loudly due to the amount of nerve fibers stimulated.

- Presbycusis: As age increases, the ability to hear decreases. Associated with SNHL
- Congenital hearing loss
    - Congenital vs. hereditary: Congenital is present at birth.
- Hereditary hearing loss:
    - Usher syndrome: Combined hearing loss and retinitis in the mucosa.
    - Waardenburg syndrome: hearing loss, hypopigmentation in the hair (white forelock), heterochromia iridis, dystopia canthorum
    - Pendred syndrome: hearing loss and goiter
    - Alport's syndrome: glomerulopathy and hearing loss.
    - Treacher Collins syndrome: maxillomandibular hypoplasia (flat face), cleft lip/palate, aural atresia

Otosclerosis*

- Occurs in the footplate of the stapes. It sometimes can go into the inner ear and cause mixed hearing loss (less common). Cochlear otosclerosis starts in the cochlea as SNHL then becomes CHL.
- In active form of disease, there will be otospongeotic (active stage) followed by otosclerosis upon calcification.
- Autosomal disease with variable penetrance, two thirds of cases are bilateral.
- It is the second most common cause of conductive hearing loss in 15-50. The most common cause of conductive hearing loss is impacted wax.
- Clinical vs. histopathological
    - Histopathological: if 100 M and 100 F died and we studied their ears, we will find otosclerotic changes. 1:1 F:M
    - Clinical: symptomatic with 2:1 F:M ratio. This is usually due to hormonal changes in females.
- Findings:
    - Inspection, palpation, percussion – normal
    - Otoscopy: Normal, but 10% of cases will have Schwartz Sign – Redness behind the tympanic membrane posterosuperiorly. This will give us an indication of an active disease.
    - Weber: Lateralized to the affected.
    - Postnasal: normal
    - Paracusis Welsi: Hearing in noisy environment better than quiet
    - Tympanometry: As changes
    - PTA: ABG > 10, > 30
    - Carhart notch: two lines with ABG > 10, upper line > 30. There will be a notch around 2000 Hz.

- Management:
    - Observation if asymptomatic
    - Hearing aids (intracanal, retroauricular)
    - Surgery: Stapedectomy or stapedotomy
        - Take out the defective stapes and put a prosthetic in place.
        - 10% of patients who get this surgery get vertigo
        - 2% of patients will get permanent sensorineural hearing loss.
    - Sodium fluoride: used in active otosclerosis (in otospongeosis) to force it into otosclerosis. Not used anymore.

Ototoxicity

- Changes in inner ear due to medications. Aminoglycosides, loop diuretic, antimalarial, salicylates, cisplatin, vincristine.
- Findings:
    - All negative
    - Rinne: +
    - Weber: lateralized to normal ear

Weber loves to lateralize to the conductive hearing loss and hates the SNHL

    - PTA: ABG < 10; below 30
- Management:
    - Stop the offending medication
    - Patients might not get better. We ask them to avoid loud exposure.
    - Hearing aid if the patient has a severe problem

Presbycusis

- Age-related hearing loss. It's noise-induced hearing loss; might be high intensity noise or low intensity continuous noise.
- Bilateral, symmetrical, progressive, high-frequency SNHL. We can't guess who has it or will get it. It might start with one era and then go to the other ear.
- Symptoms: hearing loss, tinnitus
- Inspection, palpation, percussion, otoscopy normal
- Rinne positive on both ears with Presbycusis. Weber lateralized to contralateral side.
- Dip in PTA 3000-6000 Hz.
- Management: Hearing aid, avoid further noise exposure
- In PTA – 30-50: mild; 50-70: moderate, 70-90: severe, > 90: profound

BAHA

- Born Anchored Hearing Aids

- Patient who is born with atresia might have this. You would put a screw inside the skull, it becomes osteointegrated. You would then put a device on it that is a receiver and transmitter of audio waves, etc.

Cochlear implants

- Problems: there are channels, up to 120 frequency only. Patients who hear normally 125 Hz to 12000 Hz have infinite channels though.
- Done for bilateral severe hearing loss.

Neural plasticity

- Pre-lingual hearing loss with no stimulus until 11-13 years, the temporal lobe would atrophy. If you give the patient a cochlear implant, it'd be useless. Thus, we have to give this before that age. The earlier the better.