Primary Care Otolaryngology

- Major nerves running through ear: vestibulocochlear, facial
- Can tell which ear you’re looking at by direction of cone of light (always points forward)
- Tympanostomy tube is placed anteriorly because it creates less hearing loss

- **Auricular Hematoma**: occurs when physical trauma to the auricle causes tissue shearing and swelling
  - Treatment: I&D with dental roll bolstering
    - must be done stat because a coagulated hematoma will not break down but will eventually sclerose surrounding cartilage → cauliflower ear

- **Cerumen Impaction**

- **Otitis Externa**: infection or inflammation of the external auditory canal
  - Presentation: pain, hearing loss, otorrhea, fullness, itching
  - Investigation
    - differential diagnosis: bacterial and fungal infections can look very similar
      a.) bacterial source accounts for 90% of all infections
        - most commonly Pseudomonas, also Strep and Staph
        - pain with manipulation of tragus and auricle
      b.) fungal source is most commonly Aspergillus, also Actinomyces and Candida
        - lots of itchiness
      c.) chronic cause is due to underlying skin condition such as eczema
      d.) malignant: osteomyelitis of the temporal bone (not cancerous) as a result of a chronic infection that is misdiagnosed or untreated in diabetics or the immunocompromised
        - causes auditory canal to swell shut
        - bony breakdown can progress through to cranial cavity
        - diagnose with gallium uptake scan
  - Treatment
    a.) bacterial:
      - suction out purulent debris if qualified (not typically done in primary care)
      - insertwick for antibiotic drops if canal is narrowed
      - topical antibiotic drops:
        - neo/poly/HC is ototoxic and allergenic → only use if TM is intact and only when not using a wick
        - fluoroquinolones such as ciprofloxacin and ofloxacin
    b.) fungal:
      - remove debris
      - use topical acetic acid/hydrocortisone drops, or clotrimazole drops, or CASH powder (good for fungal or bacterial treatment, contains chloramphenicol, amphotericin B, sulfamethoxazole, hydrocortisone), or violet dye
    c.) chronic
      - first treat the eczema (steroid cream)
      - then use water/vinegar washes and avoid Q-tips
    d.) malignant is usually caused by *Pseudomonas* but is an emergency and requires referral to ENT
      - mild or moderate cases may heal with a non-antibiotic topical agent with ear washes
      - if patient is diabetic, immunodeficient, has history of radiation to the ear, has a swollen shut ear canal, or has severe disease, consider systemic antibiotic therapy
  - Follow-up: culture drainage for fungus if antibiotics fail

- **Myringosclerosis**: scarring of the tympanic membrane
  - Could be a result of tube placement or frequent infection
  - Usually benign but can cause a conductive hearing loss if severe
-Treatment: if not symptomatic, don’t do anything about it!

**Ear Drum Perforation**
-Presentation: perforation is usually posterior due to curvature of ear canal, with symptoms of hearing loss, tinnitus, otorrhea, bleeding
  a.) acute perforation: edges are jagged with lots of redness
    -ear pain is felt
  b.) chronic perforation: no redness, edges are smooth with invagination of the membrane → appearance of a rim
-Treatment: watch and wait, treat if infected with topical drops, possibly tympanoplasty with paper patch (clinic) or graft (OR)

**Eustachian Tube Dysfunction**
-Occurs with blockage of the eustachian tube that allows air to exit middle ear but not come back in → creation of negative pressure atmosphere in middle ear
  -can lead to tympanic membrane retraction (visualized as depression above malleus)
  -causes: nasal allergy, URI, nasopharyngeal mass, abnormal anatomy
-Presentation: ear pain, hearing loss, ear fullness
-Treatment:
  -if acute → should be self-limiting and heals with time
    -treat allergy with nasal steroid spray
    -oral or topical decongestants
  -if chronic + hearing loss → tube placement bilaterally
    -can surgically patch but it won’t treat underlying problem
-Prognosis: risk of cholesteatoma if unhealed/untreated

**Cholesteatoma:** noncancerous skin cyst arising from retracted piece of TM or from skin cells seeding the middle ear after a perforation event
-Eventually the growth eats away at the bone and causes permanent conductive hearing loss
-Presentation: textbook is a pearly white mass behind the eardrum, but it typically just appears as a bulging mass with granulations inside retraction pocket
-Treatment: surgical excision to prevent from destroying tegmen and reaching cranial cavity

**Otitis Media**
A.) **Chronic suppurative otitis media:** middle ear infection with otorrhea coming out through hole
  -occurs with TM perforation or tube
  -treatment: 10 days of topical antibiotic drops (quinolones only d/t toxicity issues) such as ciprohydrocortisone drops or ofloxacin
    -possibly surgery
    -consider chronic antibiotic suppression such as daily amoxicillin during winter and spring with monthly follow-up
B.) **Otitis media with effusion (serous otitis media):** when there is fluid behind the TM without presence of infection
  -caused by chronic eustachian tube dysfunction, aftermath of acute otitis media, or barotrauma
  -presentation: hearing loss, ear fullness, tinnitus
    -may have air-fluid line behind TM, bubbles, or retraction pocket
  -investigation: need to rule out mass
  -treatment: watch for 3-4 months, nasal steroids
    -tube placement if not better in 3-4 months
  -prognosis: hearing loss can last for months
C.) **Acute otitis media:** inflammation of the middle ear due to anatomic or physiologic dysfunction of the eustachian tube allowing secretions to accumulate in middle ear
  -offending agent is usually viral, bacterial (*Strep pneumo, H. flu, or Moraxella*), or fungal
  -frequently follows viral URI
  -prevention: vaccines may offer some protection
  -presentation: ear pain, hearing loss (hallmark! no loss = no infection), tinnitus, ear fullness
    -sharp pain with otorrhea if perforation
- bulging red ear drum with whiteness behind
- usually can’t distinguish viral from bacterial
- treatment: 10 days of oral antibiotics (DOC high dose amoxicillin), or observation with nonsevere illness
  - if pt had antibiotics in the last month → absolutely high dose amoxicillin +/- clavulanate, or
  - azithromycin is NOT used due to high levels of community Strep pneumo resistance
- if PCN allergic:
  - immediate (type I) hypersensitivities → clarithromycin, clinda
  - other hypersensitivities → cephalosporins
- no antihistamines or decongestants for kids
- consider analgesics: acetaminophen, antipyrine/benzocaine if > 2 years, ibuprofen
- follow up:
  - fluid will persist in middle ear for months after infection has resolved
  - amoxicillin failure due to β-lactamase producing bacterial strains
    → switch to amox + clavulanate, macrolides, cephalosporins
    - no quinolones in kids d/t risk of tendon rupture, abnormal bone development
      → if failure with recent antibiotic use, switch to clinda,ceftriaxone, or consider tympanocentesis
- prognosis: complications include labyrinthitis (inflammation of inner ear), meningitis, intracranial abscess, TM perforation, hearing loss, tympanosclerosis, facial nerve paralysis
  • mastoiditis: spread of infection to mastoid air cells → fever, otalgia, postauricular erythema, swollen/tender/protruded auricle
    - requires IV antibiotics, ENT consult, hospital admission, and frequently a mastoidectomy as it is close to critical structures

- Tympanic Membrane Abnormalities
  A.) Bullous myringitis: blistering and inflammation of the TM
    - usually caused by Mycoplasma, H. flu, or Strep pneumo
    - presentation: excruciating pain especially with coughing or sneezing
    - treatment: oral antibiotics (macrolide) + topical antibiotic if vesicular rupture present, short term pain management with opioids

- Ear Pain
  - Must distinguish true ear pain from referred pain
    - otologic pain from: otitis externa or media, myringitis, eustachian tube dysfunction, ear canal abscess, ENT tumor, shingles flare or prodrome
      - referred pain from: TMJ dysfunction, oral pain (pharyngitis, dental work, abscess), sinusitis, musculoskeletal neck pain, carotidynia, neck lymphadenopathy, parotitis, or trigeminal neuralgia

- Hearing Loss
  - Presentations:
    • sudden sensorineural hearing loss: sound gets in but you can’t process it; occurs suddenly within last 72 hours, usually without warning
      - sensory = problem with cochlea
      - neural = problem with brain
      - may be due to viral labyrinthitis, autoimmune issue, or vascular compromise
      - an otologic emergency! treatment must occur within 4 weeks of onset → refer to ENT without delay
      - treatment is steroids with treatment of underlying cause
    • conductive hearing loss: sound is blocked from getting in, could be one of a multitude of problems
      - problems with external auditory canal: cerumen impaction, foreign body, mass, exostosis, edema, otorrhea, congenital stenosis
      - problems with TM: sclerosis, perforation, retraction
      - problems with middle ear: OM + effusion, hemotympanum, acute OM, cholesteatoma

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-problems with the ossicles: discontinuity, sclerosis, malformation, fixation
  → treatment depends on identification and treatment of cause

-Investigation
  -take a careful HPI: acute/gradual loss, fluctuating/progressive, uni/bilateral, time course, preceding illness,
  other associated symptoms
  -medical history: use of ototoxic meds, ear surgery, trauma, TM perforation, or noise exposure
  -FH of hearing loss?
  -differential diagnosis: VINDICATE
    -vascular causes: HTN, CAD, DM, stroke, sickle cell
    -infectious cause: Lyme, syphilis, HIV, viral labyrinthitis, bacterial toxins, HSV, meningitis
    -neoplasm: acoustic neuroma, cancer mets to temporal bone
    -drugs: ototoxicity, general anesthesia
      -includes the aminoglycosides, vanco, erythromycin, chemotherapies (cisplatin, nitrogen
        mustard), furosemide, salicylates, quinine
    -idiopathic cause
    -congenital cause: absent CN8, intrauterine infection, teratogens, hypoxia, premature birth, low
      birth weight, hyperbilirubinemia
    -autoimmune cause: MS, autoimmune hearing loss, SLE, giant cell arteritis
    -trauma: noise, temporal bone fx, radiation
    -endocrine/metabolic cause: hypothyroidism, Meniere’s, presbycusis, cochlear otosclerosis
  -narrow down differential by ruling out conductive loss
  -then further narrow down by unilateral or bilateral sensorineural loss
    -if unilateral  → sudden SNHL, acute labyrinthitis, acoustic neuroma, Meniere’s disease,
      intracranial cause, noise induced trauma
    -if bilateral  → HTN, DM, CAD, ototoxicity, hypothyroidism, presbycusis, Lyme, HIV, syphilis,
      autoimmune, noise induced trauma
  -then narrow down based on when hearing loss occurred

-Treatment
  -identify and treat cause: medical therapy, office procedures, surgical correction, amplification (hearing
    aids)

-When to refer to ENT
  -tinnitus and hearing loss +/- vertigo
  -sudden hearing loss
  -chronic infection that is unresponsive to treatment
  -shingles of the ear
  -ear masses

☐ Audiometry: Measuring Hearing Loss
A.) When to do?
  -all newborns are screened with:
    • auditory brainstem response test: a brainwave recording of the infant’s reaction to sound in 8
      waves
      -mnemonic “ECOLI”
        1 = E = 8th CN
        2 = C = cochlear nucleus
        3 = O = olivary nucleus
        4 = L = lateral lemniscus
        5 = I = inferior colliculus
        6/7 test the medial geniculate nucleus
    • otoacoustic emission test: measurement of the cochlear response to sounds using a microphone

-neonates with risk factors such as FH, in-utero infection, abnormal facial features, low birth weight, severe
  hyperbilirubinemia, ototoxic med exposure, bacterial meningitis, low Apgar scores, respiratory failure
  requiring > 5 days ventilation, known syndrome associated with hearing loss
-children with hearing/speech/language/developmental delay, infections associated with sensorineural loss,
  head trauma with loss of consciousness or skull fracture, known syndrome associated with hearing loss, use
  of ototoxic meds, serous otitis media for 3+ months
-adults with self-perceived hearing loss, physical exam abnormality, exposure to ototoxic meds or loud
  nose, severe head trauma, infection associated with hearing loss, FH of hearing loss, hypoxia, respiratory
  failure, tinnitus

B.) Primary care techniques
- basic audiometer tells you whether or not a person can hear a 25 dB tone
- Weber tuning fork test is good for testing unilateral hearing loss
  - midline hearing is either normal or there is an equal deficit in both ears
  - when sound is best heard in affected ear, it means there is conductive hearing loss in that ear
    - may be because the conduction problem of the incus/malleus/stapes/eustachian tube
      masks the ambient noise of the room, while the well-functioning cochlea picks the sound
      up via bone, causing it to be perceived as a louder sound than in the unaffected/normal
      ear
    - or may be because lower frequency sounds that are transferred through the bone to the
      ear canal escape from the canal, but if an occlusion is present, the sound cannot escape
      and appears louder on the ear with the conductive hearing loss
  - when sound is best heard in unaffected ear, it means there is a sensorineural hearing loss in the
    affected ear
    - this situation is because the affected ear is less effective at picking up sound even if it is
      transmitted directly by conduction into the inner ear
- Rinne tuning fork test compares air conduction to bone conduction
  - here, a positive Rinne test indicates air conduction > bone conduction
    - could be normal or could be sensorineural loss in ear being tested
  - a negative Rinne means the tone is heard louder on the mastoid
    - indicates conductive hearing loss

C.) Pure tone audiometry: simultaneously tests air and bone conduction over a range of normal voice frequencies
- hearing level response is measured in dB (logarithmic)
  - normal hearing response @ 0-25 dB
  - mild hearing loss @ 25-45 dB for the tested frequency
  - moderate loss @ 45-65 dB
  - severe loss @ 65-85 dB
  - profound loss @ 85+ dB
  - deaf if you can’t hear at 120 dB
- measurement of air conduction
  - right ear response is marked by O’s, left ear response marked by X’s
  - can prevent cross-hearing (better ear compensating for ear with loss) by using a masking noise in
    the good ear
    - masked right ear now represented by Δ’s
    - masked left ear now represented by □’s
- measurement of bone conduction (headphones include a mastoid bone vibrator)
  ** bone conduction lines are usually deleted by the tech for simplicity if they are = to air
  conduction!
  - right ear response is marked by <’s
  - left ear response is marked by >’s
  - can mask to prevent cross-hearing
    - masked right ear now represented by [‘s
    - masked left ear now represented by ]’s

→ how to read the audiogram:
  1.) look at threshold of 25 dB
  2.) is hearing loss bilateral or asymmetric (R or L only)?
    - symmetric = lines for R and L ear match up fairly closely
    - asymmetric = hearing is different between ears
  3.) type of hearing loss: sensorineural, conductive, or mixed
    - if the air conduction thresholds show a hearing loss but the bone conduction thresholds
      are normal, then we call it a conductive hearing loss
    - if both the air conduction thresholds and the bone conduction thresholds show the same
      amount of hearing loss, we call it a sensorineural hearing loss
- A mixed hearing loss is when the bone conducted thresholds show a hearing loss and the air conducted thresholds show an even greater hearing loss

4.) severity
5.) slope
- flat when hearing loss is the same at all frequencies
  - common in diabetes
- “ski slope” or descending when loss is greater at higher frequencies
  - usually due to presbycusis or chemotherapy if sensorineural
- ascending slope when high frequencies are heard better than low frequencies
  - rare, this would be Meniere’s disease if sensorineural
- even slope with a notch in it indicates loss at a specific frequency
  - usually due to loud noise exposure if sensorineural
- U-shaped slope is rare and there is usually a genetic hearing loss

D.) Tympanometry: tests mobility of eardrum
- normally the middle ear and the rest of the world should have equal pressure
  \( \Rightarrow \) corresponds to a tympanogram peak @ 0 = zero difference = “Type A”
  - small area under curve means normal pressure but a more rigid TM
  - large area under curve means normal pressure but a more floppy TM
- when there is a middle ear effusion or a hole in the TM, the ear drum does not respond to pressure changes
  = “Type B”
  \( \Rightarrow \) corresponds to a flat tympanogram with no true peak
  - when there is eustachian tube dysfunction the middle ear is under negative pressure = “Type C”
  \( \Rightarrow \) corresponds to a negative tympanogram peak

E.) Ear canal volume
- too small (> 0.5) indicates obstruction or canal stenosis
- normal is 0.5-2.5
- too large (> 2.5) indicates perforated TM

F.) Speech audiometry: measures how well a patient recognizes speech
  - speech threshold recognition: how quiet of speech a person can recognize, as measured in dB
  - speech discrimination: how well a patient understands speech, normally > 88% of the words given

Otologic Surgeries
A.) Stapedectomy: performed when otosclerosis of the stapes \( \Rightarrow \) fixation and loss of vibration
  - prosthetic stapes put in
B.) Cochlear implant: indicated in profound bilateral deafness
  - implanted electrode wraps around cochlea, while external magnet transmits signal to electrode
C.) Bone anchored hearing aid (BAHA): metal stud implanted in skull transmits vibrations via bone to bypass the ossicles
  - helpful in someone who had to have their ossicles removed \( \Rightarrow \) conductive hearing loss
  - can also be used for unilateral sensorineural hearing loss, as the vibration will transmit through the skull to be received by the good ear
D.) Soundbridge: combination of cochlear implant and BAHA, where a wire carries external vibration to a floating mass transducer implanted in the middle ear
  - for individuals that can’t use a traditional external hearing aid due to having a small canal, etc.

Tinnitus: abnormal perception of sound in the middle ear in the absence of a corresponding sound in the external environment
- Different kinds
  - subjective tinnitus: a sound only the patient can hear due to aberrant neurological signalling in the brain
    - often a neurological response to hearing loss
    - high freq loss \( \Rightarrow \) hi freq tinnitus
    - roaring/low freq loss \( \Rightarrow \) low freq tinnitus
    - can also be caused by meds like aspirin
  - objective tinnitus: when a clinician can perceive the abnormal sound emanating from the patient’s ear
    - clicking with pharyngeal muscle spasm
    - breathy with patulous (abnormally open) eustachian tube
- Pulsatile/bruit with referred vascular sounds or tumor
- Can be high or low frequency
- Usually worse in quiet environments
- Investigation
  - Hearing test
- Treatment
  - Stop offending meds
  - Avoid caffeine and nicotine
  - If due to sensorineural loss, there is no known surgical or pharmacological intervention
    - Use of background noise
    - Tinnitus retraining therapy
  - If due to conductive hearing loss, resolving the underlying problem will resolve the tinnitus

- Vertigo: A specific kind of dizziness that results in a false impression of movement
- Contrast to the catch-all term dizziness that refers to having no impression of movement, but imbalance or lightheadedness/presyncope
- Different classes:
  - **Peripheral (otologic) vertigo**: Caused by problems with the inner ear
    - **Benign paroxysmal positional vertigo**: Occurs when otoliths dislodge into the semicircular canals
      - Intermittent vertigo lasting < 1 minute
      - Worse with head movements R/L when lying down
      - Better with head held still
      - Diagnose with Hallpike maneuver: Positive if rotational nystagmus seen
      - Treat with Epley maneuver: Repositioning the head over time to get the otoliths back in place
    - **Meniere’s disease**: A disorder of increased endolymphatic fluid → episodic, sudden unilateral sensorineural hearing loss, roaring tinnitus, and vertigo for hours
      - Treatment: Diuretics, low salt diet to lower ear pressure, anti-vertigo medication
      - Possible surgery for endolymphatic sac decompression, gentamycin injection, labyrinthectomy, or selective vestibular nerve resection
    - **Acute labyrinthitis/vestibular neuritis**: Infection or inflammation of the inner ear, often due to latent viral infection → Severe, disabling vertigo for 24-48 hours followed by weeks of imbalance
      - Treatment: Steroids and physical therapy
  - **Perilymphatic fistula**
  - **Superior semicircular canal dehiscence**
  - **Central (neurologic) vertigo**: A problem with the balance centers of the brain
    - Multiple sclerosis
    - Migraines
    - Benign intracranial hypertension
- Investigation
  - Vast differential diagnosis → Take a thorough history to narrow down
    - Cardiologic reasons: Orthostatic HTN, arrhythmia, CAD
    - Neurologic causes: Acoustic neuroma, TIA, stroke, Parkinson’s, neuropathy, migraine
    - Anemia
    - Psychologic causes (rarely): Anxiety, panic
    - Metabolic: Hyperthyroidism, menopause
    - Orthopedic: Cervical disc disease, lower extremity arthritis
    - Geriatric: Proprioception, off center of balance
    - Pharmacologic: Polypharmacy or side effects
  - PE: CN testing, Romberg, gait, nystagmus, ear exam, Dix-Hallpike test
  - Special tests ordered if you’re still stumped
    - Electro/videonystagmogram: Measure reaction of nervous system to certain stimuli/stressors
    - Rotary chair
    - Fistula test
    - High-res CT of the temporal bone
    - MRI of the internal auditory canal
- **Acoustic Neuroma:** a slow-growing noncancerous tumor of the Schwann cells surrounding CN 7/8
  - Early presentation: asymmetric hearing loss, tinnitus, imbalance but not vertigo
  - Late presentation due to brainstem compression
  - Investigation: MRI with contrast of the internal auditory canals
  - Treatment: observation or stereotactic radiation or surgery

- **Rhinology Background**
  - Types of URI include colds, influenza, acute bronchitis, acute exacerbation of chronic bronchitis, croup, bronchiolitis, otitis media, acute pharyngitis, sinusitis, epiglottitis
  - Having a tonsillectomy does not decrease incidence of colds
  - Smokers have more severe colds with prolonged course
  - No OTC cough and cold products available for children under 4
    - especially avoid ephedrine, pseudoephedrine, phenylephrine, diphenhydramine, brompheniramine, chlorpheniramine
  - Functions of the nose include nasal reflexes (may be linked to lower respiratory and vascular reflexes), and endocrine pheromone detection

- **Common Nose Issues**
  - Septal deviation: when septum is displaced from midline
    - becomes a problem when crookedness is severe enough to impact breathing through nose
  - Septal perforation: creates a hole going from one nostril to the other
    - creates disrupted air flow/breathing, nasal crusting, and bleeding
  - Nasal mucositis: irritation and infection of the nasal mucosa
    - treatment: topical or oral antibiotic
      - clindamycin especially good at penetrating cartilage
      - need more rigorous course if infection spreads past vestibule into cartilage
  - Epistaxis: nosebleed
    - anterior bleed occurs in Kiesselbach’s plexus (where you pick your nose)
    - posterior bleed occurs in Woodruff’s plexus
    - can be caused by picking, septal deviation, inflammation, cold or dry air, or a foreign body
      - systemic causes: clotting disorder, HTN, leukemia liver disease, anticoagulant therapy, thrombocytopenia
    - treatment: manual compression (hold soft part of nose and lean forward), oxymetazoline (Afrin; acts as vasoconstrictor to decrease blood flow), cauterization (one side at a time to avoid necrosis of septum), anterior or posterior packing (rarely done because it is painful), arterial ligation, surgical embolization (for patients with high BP and low platelets), “rapid rhino” inflation

- **Common Cold vs Influenza**
  - Colds
    - slow, insidious onset
    - fever only in kids
    - usually no headache or chills
    - sore throat, stuffy nose, sneezing
    - mild aches or weakness
  - Influenza
    - rapid onset with symptoms worsening over 3-6 hours
    - fever > 100 for 3-4 days
    - usually no sore throat, stuffy nose, or sneezing
    - headache, chills, severe aches and weakness

- **Allergic Rhinitis:** IgE-mediated reaction causing mast cells and basophils to release histamine, leukotrienes, serotonin, and prostaglandins → inflammation of the nasal mucosa
  - Presentation: nasal congestion, rhinorrhea, sneezing, itching, watery eyes, allergic shiner (dilation of veins under eyes causes dark under eye circles), blue/hypertrophied turbinates (due to venous dilation), allergic salute (crease across top of nose from constantly rubbing)
-may also have nasal polyposis: excess tissue created as a result of inflammation that destroys normal nasal tissue and can disintegrate nasal septum and orbital wall without treatment
  -results in being more prone to infections
  -remove surgically if obstructing air flow

- Samter’s triad: syndrome of aspirin sensitivity, nasal polyposis, and asthma that is often seen with allergic rhinitis, frequently leading to severe pansinusitis

-Treatment: avoidance of allergens, nasal saline lavage, nasal steroid spray, antihistamines (oral, nasal, eye), leukotriene inhibitor, allergy shots

-2nd generation antihistamines indicated for allergic rhinitis:
  a.) loratadine: for > 2 years old
  b.) cetirizine: for > 6 months old
  c.) fexofenadine: for > 6 years old
  d.) desloratadine: for > 12 years old
  e.) azelastine: nasal spray for > 12 years old
  f.) olopatadine: nasal spray for > 12 years old

-intranasal glucocorticoids
  -generic available:
    a.) fluticasone:
    b.) flunisolide:
  -no generic:
    a.) mometasone:
    b.) budesonide:
    c.) triamcinolone:
    d.) beclomethasone:
    e.) fluticasone furoate:
    f.) ciclesonide:

-consider anticholinergic nasal sprays only if all other therapies fail

☐ **Vasomotor Rhinitis:** non-allergy mediated inflammation of the nasal mucosa

- Causes: temperature, exercise, foreign body, fumes, food, medication
- Treatment: steroid or antihistamine sprays

☐ **Rhinitis Medicamentosa:** rhinitis induced by overuse of topical decongestants → rebound congestion

- Treatment: stop using spray
  - if needed, use nasal steroid taper, antihistamine spray, or Afrin taper

☐ **Viral Rhinitis:** URI caused by adenovirus, parainfluenza, coronavirus, rhinovirus, etc.

- Presentation: symptoms should last < 7 days
  - sore throat, nasal congestion, clear rhinorrhea, fever, cough +/- phlegm, malaise, fatigue, sneezing, itching
- Treatment: supportive with OTC antihistamines, decongestants, mucolytics, fluids, ibuprofen/Tylenol, antitussives (codeine), expectorants, rest
  - best for runny nose: anticholinergics like ipratropium spray, cromolyn sodium spray (mast cell stabilizer)
  - best for postnasal drip: corticosteroids
  - best for nasal congestion: decongestants (pseudoephedrine >> than phenylephrine), corticosteroids
    - pseudoephedrine can increase BP
  - best for sneezing: antihistamine, corticosteroids
  - best for chronic, nonproductive cough: dextromethorphan
  - nonopioid antitussive for recovering addicts: benzonatate
  - cocktail for acute cough associated with common cold: 1st gen antihistamine + decongestant like pseudoephedrine
    - newer 2nd gen antihistamines are ineffective in this situation
  - vitamin C and Zn supplements controversial
  - Zicam warning for permanent loss of smell associated with use

- Complications: acute otitis media, chronic middle ear effusions, asthma, dental problems, sinusitis, nasal polyps
Sinus Imaging

-X-ray
- good for showing air/fluid levels in the maxillary (Waters view) and frontal sinuses (Caldwell view)
- bad for looking at mucosal thickening or soft tissue abnormalities
- bones are often obscured and difficult to read

-MRI
- not the preferred modality for sinus imaging but you can get some information from it
- doesn’t detail bones well
- can falsely give the impression of inflamed mucosa
- good for evaluating neoplasms, mucoceles, and encephaloceles

-CT
- study of choice for evaluating nasal and sinus structures
- best to do after maximal treatment to reduce inflammation
- coronal CT without contrast is done in the plane of surgical approach and shows the osteomeatal complex best
- evaluation: be sure to look at whole CT, not just the sinuses
  - orbits, orbital wall, maxilla, nasal septum, turbinates, then sinuses anterior to posterior
  - mucus and polyps will be of water density (gray)
  - mucosal thickening indicates chronic sinusitis

Sinusitis: purulent infection of the sinus
- can’t tell whether it is viral or bacterial based on appearance!
- pathogens: Strept pneumo, H. flu, Moraxella, Staph aureus
- complications: facial or periorbital cellulitis, orbital abscess, meningitis, cavernous sinus thrombosis, intracranial abscess

A.) Acute sinusitis: when symptoms do not clear in 7-10 days, due to inflammation from viral URI trapping fluid in sinuses that incubates growth
- bacterial causes make up 90% of cases
- vaccine for influenza may help prevent
- presentation: “double sickening” where pt had viral symptoms, got better, then symptoms returned a week later, localized facial pain, unilateral sinus tenderness, upper tooth pain, purulent foul nasal discharge, fever, cough, fatigue
  - malodorous breath in young children with painless morning periorbital swelling
  - older children have tooth pain, headache, and low-grade fever
- treatment:
  - 10-14 day course of antibiotics
    - try amoxicillin first (Septra if allergic)
    - if severe or with recent antibiotic use ➔ use broad spectrum first (unless a child)
  - nasal saline lavage to help move infected mucus out, nasal steroid spray
  - antihistamine, decongestant, mucolytics
  - oxymetazoline (< 4 days) to help pt feel better before antibiotics kick in
  - severe frontal sinusitis needs referral to ENT
  - 63% of cases will be cured without any treatment ➔ can just observe mild cases for 7 days for improvement
- follow-up: if antibiotic failure, consider broad-spectrum
  - for failure with max treatment, surgical sinus aspiration
- complications: subperiosteal abscess of the orbit, intracranial abscess, exacerbation of COPD or asthma

B.) Subacute sinusitis: when symptoms last 4-12 weeks
C.) Chronic sinusitis: when symptoms are > 3 months, could be infectious or noninfectious cause
- could be allergic inflammation, cystic fibrosis, immunodeficiencies, ciliary dyskinesia, or anatomic abnormalities
- also consider Klebsiella, Pseudomonas, Proteus, Enterobacter, MRSA, anaerobes, fungus
- investigation: do a culture and sensitivity
- treatment
  - non-antimicrobial therapies that may help with clearance: decongestants, topical vasoconstrictors, nasal saline sprays, topical steroids, NSAIDs, cough suppressants
**Pharyngitis**
- differential diagnosis: post-nasal drip, virus, group A strep (most common cause), tonsillitis, mono, peritonsillar abscess
  - more rarely, gonorrhea, HSV, HIV, or cancer

A.) Viral pharyngitis
- often co-occurs with viral rhinitis
- agents: adenovirus, coronavirus, rhinovirus, influenza, parainfluenza, Coxsackie virus
- presentation: erythema, edema, dysphagia, pain, fever, lymphadenopathy, URI symptoms
  - soft palate is symmetrical, red but not to the extreme
- treatment: supportive meds
- prognosis: self-limiting in 3-7 days

B.) Strep pharyngitis
- presentation: sore throat, dysphagia, odynophagia, erythema, airway obstruction, tender lymphadenopathy
- investigation: must distinguish from viral pharyngitis by rapid *Strep* test +/- culture
- treatment: penicillin VK or amoxicillin or pen G injection for noncompliant patient, erythromycin if PCN allergic
  - but be aware of increasing macrolide resistance

C.) Acute tonsillitis: viral or bacterial
- commonly *Strep pyogenes*
- presentation: swollen tonsils with white plaques
- treatment: usually antibiotics
  - caution: if it is due to mono, certain antibiotics will cause a rash

D.) Peritonsillar abscess: a collection of mucopurulent material in the peritonsillar space
- often follows tonsillitis
- presentation: bulging, asymmetrical soft palate, “hot potato voice”, severe throat pain, dysphagia, trismus (inability to open jaw), deviated uvula, salivation/drooling, fever, severe malaise
- treatment: I&D by ENT, antibiotics with anaerobic coverage

E.) Mononucleosis: viral disease caused by EBV or CMV
- presentation: fatigue, malaise, sore throat with tonsillar edema/erythema/exudate, lymphadenopathy, hepatosplenomegaly
- investigation: Monospot rapid test (not reliable early in disease), CBC with atypical lymphocytes
- treatment: OTC pain control, possible steroids, splenomegaly precautions

F.) Ludwig’s angina: cellulitis of the floor of the mouth in the sublingual or submaxillary spaces
  - *angina* = Greek for strangling → an emergency as the airway can become blocked!
  - presentation: swollen neck, protruding tongue
  - treatment: hospitalization with airway management, IV antibiotics, surgical draining

**Laryngoscopy**
- Two kinds:
  - **direct laryngoscopy**: straight visualization of the larynx (no reflected images)
    - best image quality
    - can palpate vocal cords to distinguish paralysis vs fixation
    - can do injections and biopsies
    - done prior to laryngeal intubation
  - **indirect laryngoscopy**: the use of a mirror, angulated scope, or flexible scope to visualize an image or reflection of the larynx
    - mirror not invasive with no anesthetic but hard to do because of gag reflex and can’t see entire larynx
    - flexible scope goes through decongested/anesthetized nose
    - no gag reflex with better visualization of the upper airway
    - can be done in clinic
    - anesthetic tastes bad
    - can’t do biopsy

- When to do?
-complete head and neck exam, hoarseness, mass, foreign body, chronic sinusitis, chronic cough, recurrent otitis media, halitosis, obstructive sleep apnea, referred pain, SOB, hemoptysis, history of neck or cardiac surgery (to evaluate cord mobility)

-Reading scope images
-orientation: vocal cords always point to the front/anteriorly!

**Hoarseness**

- Investigation
  - take a good history
    - vocal strain?
    - recent surgery/intubation?
    - thoracic surgery → vocal cord paralysis?
  - differential diagnosis
    - if acute → postnasal drip, viral laryngitis, hypothyroidism, vocal fold paralysis, recent intubation, vocal hemorrhage (singers and performers)
      - paralysis a result of viral infection of a nerve or injury
      - bilateral paralysis is an emergency while unilateral paralysis could cause pneumonia
    - if chronic → smoking (Reinke’s edema), vocal strain, GERD, cancer, vocal nodules or polyps
      - nodules are a result of vocal misuse and allow gaps in vocal cords for air to escape
      - polyps are a result of acid reflux
      - could be squamous cell carcinoma
  - laryngoscopy
    - normal larynx should have sharp vocal cord folds with symmetric opening and closing
    - acute laryngitis will cause pink, puffy vocal cords with increased vasculature

- Treatment
  - acute laryngitis is usually self-limiting, treat with rest, fluids, and smoking cessation
  - DO NOT use steroids or antihistamines because they may cover up the injury and cause further damage to the vocal cords with permanent injury
  - vocal cord nodules can be surgically excised or overcome with voice therapy
  - vocal cord polyps heal with treatment of the underlying acid reflux but can be excised if they are large

**Head and Neck Tumors**

- how to evaluate?
  - rule out serious causes first, then think about the benign
  - weight loss, chronic ailment, age > 45, previous radiation to head/neck
  - rule out infectious cause (most likely) like bartonellosis or TB, congenital abnormalities, and metabolic causes
  - look for lymphadenopathy
    - postauricular → nasopharynx mets
    - submandibular → oral mets
    - submental → lip cancer
    - superficial cervical → oral/pharyngeal/laryngeal mets
    - deep cervical → nasopharyngeal/scalp/ear mets
    - supraclavicular → thyroid or upper esophageal mets
  - complete skin exam

A.) Congenital anomalies

  - **thyroglossal duct cyst:** occurs when duct persists after fetal development
    - a problem because they can cause dysphagia or become infected
    - investigation: have pt tilt head back, then provider takes hold of cyst and has pt stick tongue out
      - if action raises the cyst, it is a thyroglossal duct cyst
  
  - **branchial cleft cyst:** occurs with abnormal persistence of connections of the URT to the ear canal, upper neck, or lower neck
    - a problem because they can become infected and may need to be excised

B.) Malignant ear tumors

  - squamous cell carcinoma: refer to plastic surgery
- Ear canal tumors: very rare, but ear must be completely excised because they are highly metastatic.
  Diagnosis of all ear cancers require biopsy of lesions, surgical excision, radiation, and lymph node dissection if there is metastasis.

C.) Benign ear tumors
  - Glomus tympanicum: tumor of the tympanic membrane that appears as a bright red mass behind the membrane.
  - Causes hearing loss with pulsatile tinnitus.
  - Investigation: CT with contrast.
  - Treatment: surgical excision.
  - Glomus jugular: tumor occurs in the jugular foramen.

D.) Nasal tumors
  - Nasal osteoma: benign tumor of the skull base, usually found incidentally on CT scan.
    - Leave it alone if pt is asymptomatic.
  - Squamous papilloma: benign tumor caused by HPV but can transform into malignancy or cause obstruction and bleeding so they are typically excised.
  - Inverted papilloma: premalignant mass that is often confused for polyps, high chance of cancer conversion.
  - Juvenile angiofibroma: benign mass that occurs in adolescent males with chronic unilateral nosebleeds.
  - Squamous cell carcinoma: consider in smokers with history of nosebleeds and nasal pain.

E.) Nasopharyngeal masses
  - Adenoid hypertrophy: typically occurs in kids as adenoids usually regress by adulthood.
    - Can cause snoring.
  - Tornwald cyst: congenital cyst of the nasopharynx, leave it alone.
  - Mucocle: cyst caused by buildup of mucus from blocked gland.
    - Removed only if causing nasal obstruction.
  - Lymphoma.
  - Squamous cell carcinoma.

**Ophthalmology**

**Acute Conjunctivitis**
- Bacterial (Staph aureus, Strep pneumo, H. flu, Moraxella), viral, or allergic.
- Presentation: often hard to differentiate bacterial from viral.
  - Bacterial (Staph aureus, Strep pneumo, H. flu, Moraxella): red conjunctiva with yellow/white/green discharge bilaterally that is consistently purulent.
  - Viral (usually adenovirus): discharge may be more clear, watery, and stringy, gritty or burning feeling in eye.
    - Usually one eye affected first with second eye in 24-48 hours.
  - Allergic: bilateral redness, watery discharge, itching, injected conjunctiva with follicular appearance, may have morning crusting.
- Treatment
  - Bacterial → erythromycin ointment or sulfacetamide drops.
    - Fluoroquinolone drops preferred in contact wearers due to risk of *Pseudomonas*
  - Viral → symptomatic relief only with OTC antihistamine drops (Ocuhist, Naphcon-A, Visine AC).
    - Compresses, naphazoline, pheniramine.
  - Allergic → antihistamine/decongestant drops, mast cell destabilizer drops (olopatadine HCl), NSAID ophthalmic drops.
    - If severe: lodoxamide drops or *cromolyn sodium* drops.
- Prognosis: viral conjunctivitis takes 2-3 weeks to heal.
Glaucoma: refers to a group of diseases characterized by damage to the ocular nerve (cupping), with most cases also involving elevated intraocular pressure
- Causes progressive loss of retinal ganglion nerve axons → loss of visual field with eventual blindness
- Risk increases with age
- Several kinds:
  a.) secondary glaucoma: caused by injury or infection
  b.) congenital glaucoma: present at birth, with symptoms manifesting in first few years of life
  c.) closed (narrow) angle glaucoma: when contact between a malformed iris and trabecular network obstructs outflow of aqueous humor from the eye
    - means angle between iris and cornea is wide = normal
    - most prevalent in Asians
    - presentation is acute with redness and eye pain, headache
    - diagnostic criteria
      - must have 2 of these signs: ocular pain, nausea/vomiting, history of intermittent blurring of vision with halos
      - must have at least 3 of these signs: IOP > 21 mm Hg, injected conjunctiva, corneal epithelial edema, mid-dilated and nonreactive pupil
    - immediate treatment:
      - reduce IOP with carbonic anhydrase inhibitors (CO2 ↔ H2O)
        - acetazolamide or methazolamide
        - don’t give to liver/renal pts, adrenocortical insufficiency, or severe pulmonary obstruction
        - side effects: may increase blood sugar in diabetics, transient myopia, nausea, diarrhea, loss of appetite, loss of taste, paresthesias, lack of energy, renal stones, hematological problems
      - reduce IOP with topical beta blocker to decrease aqueous humor production
        - β-1 selective: betaxolol:
          - caution: may exacerbate or precipitate heart block, asthma, COPD, or mental changes
        - nonselective: carteolol, levobunolol, metipranolol, timolol
          - fewer cardiac and lipid effects
      - add alpha agonist if necessary to decrease aqueous humor production
        - apraclonidine or brimonidine (interaction with MAOIs, CNS effects, respiratory arrest in young children)
      - suppress inflammation → topical steroid like prednisolone
      - analgesics for pain
      - antiemetics for nausea/vomiting
      - place pt in supine
    - treatment one hour later:
      - administer miotics (pull iris away from trabecular meshwork to open up flow) such as pilocarpine (may cause headaches)
      - then osmotic agents if unsuccessful in lowering IOP → glycerin or isosorbide if diabetic, IV mannitol
        - but can’t use with pulmonary edema or anuria
    - treatment 1-2 days later is focused on creating a new opening in the iris for aqueous humor to drain out of: laser peripheral iridotomy, argon laser peripheral iridoplasty, anterior chamber paracentesis
    - meds to avoid in these patients because they will precipitate another attack: pseudoephedrine, phenylephrine, neo-synephrine, chlorpheniramine, diphenhydramine, Detrol, benzos, pupil dilation, tricyclics, hydralazine, citalopram, haloperidol, lithium, paroxetine, topimax
  d.) wide or open angle glaucoma (primary open angle glaucoma): clogged physiologic drain → increased intraocular pressure → results in sequential damage to the optic nerve → progressive loss of visual field
    - means angle between iris and cornea is closed = abnormal
    - most prevalent in blacks
-causes: idiopathic, steroids, pigment dispersion
-treatment: want to increase aqueous outflow while decreasing aqueous production to lower the IOP
  -suppress production:
    -beta-blockers
    -alpha agonists
    -alpha + beta agonists (vasoconstriction): dipivefrin (don’t give in angle closure glaucoma), epinephrine
  -topical carbonic anhydrase inhibitors: dorzolamide or brinzolamide
  -can increase glucose in diabetics
-increase outflow:
  -prostaglandin analogs: latanoprost, travoprost, bimatoprost, unoprostone
  -alpha agonists
  -cholinergic agonists:
    -act as cholinesterase inhibitors (choline ↔ HAc): echothiophate, demecarium, carbachol, physostigmine
  -combination agents available: dorzolamide + timolol, brimonidine + timolol
  \[ initial\text{ therapy}\text{ is}\text{ a}\text{ topical beta-blocker (unless cardiac/pulm contraindications) + topica}\text{ prostaglandins} \]
  -alternative are alpha/beta agonists, carbonic anhydrase inhibitors, and cholinergic agonists

- Refractive Errors
  A.) Hyperopia (farsightedness): when the eye does not bend light enough (low optical power) \(\rightarrow\) image is in focus behind the retina
    -correct with convex (+) lenses
  B.) Myopia (nearsightedness): when the eye bends light too much (high optical power) \(\rightarrow\) image is in focus too far in front of the retina
    -correct with concave (-) lenses
  C.) Astigmatism: abnormal curvature of the cornea causes vision to be out of focus
    -correct with a cylindrical lens
  D.) Presbyopia: symptomatic loss of normal accommodation with age \(\rightarrow\) loss of ability to focus on near objects
    -natural aging process
    -correct with reading glasses or bifocals

- Amblyopia: a reduction in vision of one or both eyes that is the result of eye/brain pathways not connecting well at birth and cannot be resolved by the use of corrective lenses
  -Causes:
    -sight deprivation such as cataracts or ptosis does not allow for development of correct pathways
    -strabismus
    -refractive errors such as anisometropia (unequal refractive power in eyes) or astigmatism
  -Not a result of a lesion in the visual pathway but a developmental problem
  -Treatment: close the good eye and force the bad eye to do extra work
    -occlusion via pharmaceutical or physical means

- Strabismus (Tropia): when eyes are not aligned with each other
  -Congenital or develops in early adulthood
  -May cause amblyopia
  -Eye deviations seen:
    - esotropia: cross-eyes
    - exotropia: wall-eyed
    - hypertropia: upward deviation
    - hypotropia: downward deviation
  -Presentation: no weakness in extraocular eye movements or nerve palsies, no diplopia
Disorders of the Lids and Lacrimal System

A.) Dacrocystitis: nasolacrimal duct inflammation
-usually due to nasolacrimal duct obstruction or infection (Staph aureus or Strep pneumo)
-presentation: pain, redness, swelling of the inner lower eyelid, constant tearing, recurrent refractory conjunctivitis, abscess in adults
-treatment:
  -infants: massage and observe unless large abscess forms
  -adults: oral antibiotics, warm compress, abscess usually requires surgery

B.) Ectropion: eyelid pulls down and away from eyeball
-caused by congenital abnormality, scarring, facial nerve palsy, aging
-presentation: sagging lid with a dull light reflex, irritation

C.) Entropion: when the eyelid turns in on itself and lashes rub against the eye
-caused by congenital abnormality, aging, scarring, spasm

D.) Chalazion: painless lipogranuloma of the meibomian glands due to trapped oil
-more of a bother than a problem
-treatment: warm compress, lid scrubs

E.) Hordeolum (stye): localized infection of the eyelid involving the hair follicles of exterior or meibomian glands if interior

F.) Blepharitis: inflammation of the eyelash follicles due to Staph if anterior and rosacea if posterior
-can progress to chalazion
-presentation: red, itchy eyelids with scales along eyelash bases
-treatment: warm compress, baby shampoo scrubs, topical erythromycin

G.) Lid (pre-septal) cellulitis:
-from insect bite, laceration, fracture into sinus
-presentation: redness, induration of lid, tenderness, pain
  -eyeball itself looks normal
  -no proptosis or limitation of eye movement
-treatment: antibiotics, warm compresses

H.) Orbital (post-septal) cellulitis:
-infection usually originates in infected sinus
-presentation: pain, swelling, proptosis of the eyeball, limited motion of eye, swollen conjunctiva, +/- vision loss
-investigation: visual acuity test, afferent pupillary defect, CT scan
  -consider mucormycosis in a diabetic patient or opportunists in an immunocompromised patient
-treatment: IV antibiotics, surgical draining of abscess

Cataracts: opacification of the eye lens
-Adult causes: age, steroids, diabetes, electrocution, congenital abnormality, trauma
-Childhood causes: metabolic disorder, infection, hereditary, trauma
-Presentation: gradual loss of vision, blurred or smoky vision, glares, decreased vision in bright light or at night
-Treatment: surgical removal of adult cataract when it interferes with ADLs, with replacement by an artificial lens
  -in kids the surgery must be done early to prevent amblyopia

Retinal Detachment: when retinal peels away from underlying support tissue
-Different kinds:
  • rhegmatogenous retinal detachment: occurs due to a tear in the retina that allows fluid to pass from the vitreous space into the subretinal space between the sensory retina and the retinal pigment epithelium ➔ progressive unzipping of the retinal
  • non-rhegmatogenous retinal detachment: when inflammation gets underneath the retina and causes the detachment
    -may be from fluid buildup (exudative) or from scarring created by the inflammation (tractional)
      -tractional is more common in diabetics
-Higher risk with cataract surgery, high myopia, aphakia, and peripheral lattice degeneration
-Presentation: usually acute, with floaters, vision loss, flashing lights, cut or line in vision
-Treatment: scleral buckle, endolaser, cryotherapy, vitrectomy + endolaser, treatment of underlying cause (diabetes, inflammation, etc)
Age-Related Macular Degeneration: damage to retina causes loss of central vision
- Two forms:
  - non-exudative (dry): when drusen (cellular debris) accumulate between the retina and the choroid → loss of cones
    - accounts for 90% of cases
  - exudative (wet): blood vessels grow up from the choroid behind the retina
    - less common but more severe
- Risk factors: age, genetics, smoking, HTN, micronutrient levels, serum lipids?
- Treatment: vitamins and supplementation with zinc, beta carotene, copper, lasering or surgical extraction for neovascularization, macular translocation, antiangiogenesis therapy

Ophthalmic Manifestations of Carotid Atherosclerosis
- Amaurosis fugax: 5-10 minutes of unilateral vision loss
- Hollenhorst plaque: cholesterol embolus in retinal arteriole
- Central retinal artery occlusion: severe painless loss of vision due to loss of blood supply to the retina from embolus
  - associated with atrial fibrillation, endocarditis, coagulopathies, CAD, hypercoagulable stages
  - presentation: temporal arteritis (afferent papillary defect with pale or swollen optic nerve with splinter hemorrhages), cherry-red spot and ground-glass retina, boxcar segmentation of vessels with severe obstruction
  - investigation: cardiovascular exam for murmurs or bruits, examination for temporal arteritis
  - treatment:
    - immediate lowering of IOP with acetazolamide
    - carbogen therapy
    - hyperbaric chamber
- Central retinal vein occlusion: when occluded veins back up and blood fills retina → causes painless loss of vision
  - causes: HTN, mechanical compression, glaucoma, inflammation of nerve, orbital disease, hyperviscosity disorders
  - investigation: determine whether it is ischemic or non-ischemic
  - treatment: treat underlying medical condition, aspirin therapy, laser ischemic retina, treat macular edema, treat associated glaucoma
- Diabetic retinopathy: occurs when hyperglycemia damages basement membrane of retinal capillaries → loss of pericytes and microaneurysm formation → leakage of capillaries, macular edema
  - proliferation of weak blood vessels
  - can cause neovascular glaucoma if they grow in the anterior chamber
  - risk with DMI and II → frequent eye exams
  - no known increased risk with gestational diabetes
  - presentation:
    - funduscopic exam: serum leakage will form hard exudates while red blood cell leakage will cause hemorrhage, capillary closures, cotton wool spots from retinal ischemia
  - treatment: laser ischemic areas
  - prognosis: diabetics also at increased risk for chronic open angle glaucoma, CN III/IV/VI palsies, early cataracts, orbital mucormycosis
- Hypertensive retinopathy: damage to vessels caused by high blood pressure
  - graded based on extent
    - grade I → mild arteriolar narrowing
    - grade II → moderate narrowing with AV crossing defects
    - grade III → severe narrowing with hemorrhage or exudates
    - grade IV → all of the above + optic edema from the hemorrhaging

Disorders of the Cornea
- Pterygium: when sun exposure results in growth of degenerate tissue that digs into the cornea, pulling on cornea as it grows out → astigmatism
  - treatment: excision with needle + antifibrotics
- Corneal abrasions:
-investigation: stain to view abrasion
-treatment: topical ointments to prevent infection, will heal with time

**Dry eyes:** a result of decreased tear production or abnormal tear content concentrations, or blepharitis

**Corneal ulcers:**

**Corneal edema:**
- could be from bad cataract surgery or high blood pressure
- presentation: dull light reflex

**HSV keratitis:**
- investigation: stain eyes and shine blue light to look for branching pattern
- treatment: topical or oral antivirals

☐ **Nerve Palsy**
- A semi-emergency
- Presentation:
  - deviation will be greater in direction of action of the weak muscle
    - horizontal diplopia → weak lateral or medial rectus
    - vertical diplopia → weak superior rectus, inferior rectus, superior or inferior oblique
  - oculomotor affected → ptosis and a dilated, unreactive pupil, eyes positioned down and out
    - could be a posterior communicating artery aneurysm
    - or if pupil is normal it could be microvascular palsy of CN III (whatever that means)
    - common with HTN and diabetes
  - abducens affected → lateral rectus weakness with esotropia
    - caused by increased ICP, tumor, trauma, stroke, microvascular
  - trochlear affected → vertical or oblique diplopia
    - hypertropic eye on affected side
    - diplopia and deviation increase on gaze to opposite side
    - head tilt to side to compensate
    - caused by congenital defect or trauma

☐ **When to Get an Ophthalmology Consult**
- When funduscopic exam shows blood, inflammatory cells, tumor cells, or foreign body

☐ **Causes of Blindness in the US**
- Cataracts, age-related macular degeneration, glaucoma, diabetic retinopathy, retinal detachment, CRA/VO

☐ **Red Eyes**

**Common Dental and Oral Mucosal Disorders**

☐ **Tooth Anatomy**
- Crown is what you see, and is covered in enamel
- Root is covered in cementum, which fuses it to the periodontal ligament
  - PDL attaches tooth to alveolar bone and limits extent of biting by sensing pressure
- Each tooth contains a neurovascular bundle
  - pulp only senses pain = any sensation that stimulates it will be felt as pain

☐ **Caries**
- More common in children
- Late manifestation of a bacterial infection
  - bacterial biofilm produces acid that demineralize and dissolve the enamel
  - infection progresses through dentin, cementum, and pulp
  - may create fistulas into the gums for drainage
  - can reach periodontal region and beyond to the bone and soft tissue
- Prevention: brushing with fluoridated toothpaste, flossing, drinking water fluoridation, sealants, fluoride treatments
  - systemic fluoride supplements only if needed
-Treatment
   -remineralization if early
   -later, drill and fill with synthetic material

☐ Periodontal Disease
- More common in adults
- Also caused by biofilm infections
- Gingivitis occurs in the gums = soft tissue only
   - biofilm is mostly anaerobes
   - appears as red gingiva near tooth
   - after brushing, spit out blood-tinged toothpaste
   - may or may not have pain
   - leads to destruction of the attachment of gums to teeth
   - can cause gum overgrowth
   - commonly caused by medications (phenytoin, cyclosporin, Ca channel blockers) or hormonal changes
   - can be aggravated by immunosuppression
   - reverse with brushing and flossing to prevent progression to periodontitis
- Periodontitis occurs in the soft tissue or bone supporting the teeth
  - causes loss of periodontal attachment and bone → gingival pockets that harbor infection
    - depth correlated to severity
  - more common in adult males
  - not always preceded by gingivitis
  - aggravated by smoking, diabetes, osteoporosis, AIDS

☐ Oral Mucosal Infections and Conditions
A.) Oral candidiasis (thrush): yeast infection that occurs when host flora is altered or with immunocompromised host
   - forms:
     • pseudomembranous candidiasis: most common, white plaques that you can scrape off with underlying red mucosa
     • erythematous candidiasis: no white component
       - red path under the tongue = median rhomboid glossitis
       - from continual denture wear = denture stomatitis
     • hyperplastic candidiasis: thick white patches that can’t be scraped off
     • angular cheilitis: forms externally, on corners of mouth
       - infection can be mixed, so need to cover both bacteria and fungi
   - caused by altered normal flora
   - associated with xerostomia, endocrine dysfunction, immunosuppression, medications, trauma, blood diseases, and tobacco
   - presentation: mouth burning or soreness, sensitivity to acidic and spicy foods, foul taste, or asymptomatic
   - treat with oral antifungals, wash appliances in nystatin
B.) HSV infection
   - presentation: may have lymphadenopathy, fever, chills, oral lesions, nausea, irritability
     - kids: gingivostomatitis
     - adults: pharyngotonsillitis
   - can get secondary infections
   - treatment: best time to give antivirals is during prodrome
     - late treatment → magic mouthwash (Maalox, Kapectate, Benadryl, viscous lidocaine), fluids
C.) Recurrent aphthous ulcers
   - ulcers of unattached mucosa = not herpes! = not contagious
   - caused by immune dysfunction creating breaks in mucosa + varying individual causes (stress, allergies, etc)
   - different severities:
     - minor = most cases, 1-2 ulcers that are small
     - major (Sutton’s disease) = 6-7 ulcers that are larger
     - clusterform = 10-40 large ulcers
-investigation: must rule out Celiac, cyclic neutropenia, malnutrition, immunosuppression, IBD
-treatment: topical steroids + antibiotic ointment to ease pain until it heals

D.) Bisphosphonate-related osteonecrosis of the jaw
-occurs with patients taking IV bisphosphonates (chemo, etc)
-leads to development of bone that can’t repair itself = limited ability to respond to injury → necrosis
-mucosa on top of bone then dies
-prevention: any patient on bisphosphonates or antiresorptives needs to have a thorough dental exam and cleaning before starting treatment
-treatment: debridement, pain management, antibiotics, may need to stop chemo

**Oral Cancer and Precancerous Lesions**

-If you are uncertain about an ulcer/bump, watch it for 3 weeks max, then cut it out!

-**Oral Lesions**
  - **Torus**: benign osteoma of the hard palate or mandible
    - no surgery unless they interfere with dentures or eating
  - **Leukoplakia**: any mucosal condition that produces whiter than normal coloration (a catch-all term!)
    - includes hyperkeratosis, actinic cheilitis, heat lesions, dysplasias, carcinomas, candidiasis, hairy leukoplakia, lichen planus, lupus, white sponge nevus, hairy tongue, geographic tongue
    - can’t be scraped off
    - considered to be precancerous mucosa = always biopsy
  - **smokers and chewers**
  - **have different stages of dysplasia**
  - **Oral hairy leukoplakia**: caused by a virus, just watch
  - **Erythroplakia**: red, irregular mass
    - frequently dysplastic or carcinomatous = always biopsy
  - **Lichen planus**: autoimmune disorder that appears similar to leukoplakia but has a lacy pattern
    - no treatment
    - can biopsy to rule out leukoplakia

-**Squamous Cell Carcinoma**
  - Risks: tobacco, alcohol, viruses, genetic, immune dysfunction
  - if a smoker develops cancer and has it removed but continues smoking, the chances of developing a 2nd lesion skyrocket
  - Prevention: limiting alcohol and marijuana use, quitting tobacco use, use of sunscreen on lips, HPV vaccine
  - Presentation: may have many different appearances, including leukoplakia, erythroplakia, ulceration, mass, papillary growth, induration, loose teeth, paresthesias
    - could be on lower lip, lateral or ventral tongue, floor of mouth, soft palate, gingival ridge, buccal mucosa
    - with metastasis, swollen submandibular or superficial/deep cervical chains
  - Investigation
    - differentiation
      - called carcinoma in situ if entire thickness of epithelium is involved with intact basement membrane
      - problem with oral cancers is that dysplasia is not as organized and invasion can occur without going through all the steps of dysplasia
  - Prognosis: disparity in survival rates between whites and blacks