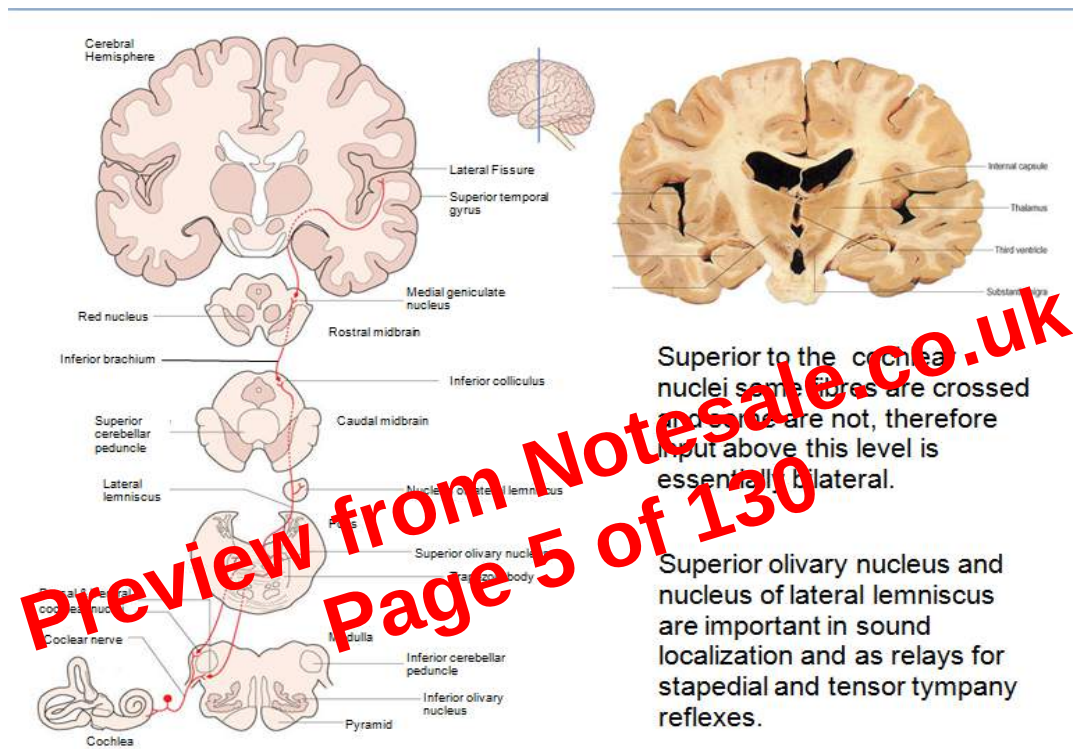


Auditory pathway

- Superior to the cochlear nuclei (in the medulla) some fibres are crossed and some are not, therefore input above this level is essentially bilateral.
- The transverse **temporal gyri** (also called **Heschl's gyri – he shall hear!**) are found in the area of primary auditory cortex. It is the first cortical structure to process incoming auditory information.
- Auditory information received by Heschl's gyrus (auditory cortex) is bilateral receiving input from both ears.
- Therefore unilateral hearing loss is very unlikely to be due to a cerebral lesion. **Unilateral hearing loss occurs due to a problem within the ear or CN VIII.**



Summary

- **External ear:** The auricle and external acoustic meatus (external auditory canal) compose the external ear. The external ear functions to **collect and amplify sound**, which then gets transmitted to the TM of the middle ear. Blockage of external auditory meatus can cause conductive HL.
- **Middle ear:** The tympanic membrane (TM), malleus, incus, and stapes compose the middle ear. The tympanic cavity (middle ear) extends from the tympanic membrane to the oval window and contains the bony conduction elements of the malleus, incus, and stapes. The primary functionality of the middle ear is that of **conduction and amplification** of sound via transference of sound waves in the air collected by the auricle to the fluid of the cochlea in



Aetiology:

- **Swimming in water** is a common way to contract external otitis but it is also possible to contract it from water trapped in the ear canal after a shower, especially in a humid climate.
- Even without exposure to water, the use of objects such as cotton swabs or other small objects to clear the ear canal is enough to cause breaks in the skin, and allow the condition to develop.
- The majority of cases are due to **Pseudomonas aeruginosa** (commonly found in water), followed by a great number of other gram-positive (e.g. **Staph aureus**) and gram-negative species.
- **Candida albicans** and **Aspergillus** species are the most common fungal pathogens responsible for the condition. Fungal ear canal infections, also known as **otomycosis**, range from inconsequential to extremely severe.
- Tx for bacterial if persistent/severe: Amoxicillin

Acute otitis media (AOM)

- Common in children, not adults
- Otitis media is the medical term for **middle ear infection**
- The common cause of all forms of otitis media is **blockage of the Eustachian (auditory) tube**. This is usually due to swelling of the mucous membranes in the nasopharynx, which in turn can be caused by a **viral upper respiratory infection (URTI)** or by **allergies** e.g. hayfever.
- Because of the blockage of the Eustachian tube, the air volume in the middle ear is trapped and parts of it are slowly absorbed by the surrounding tissues, leading to a mild vacuum in the middle ear (**-ve pressure occurs if persists to OME**). Eventually the vacuum can reach a point where **fluid from the surrounding tissues is sucked in to the middle ear's cavity** (also



Normal Eardrum



Acute Otitis Media (ear infection)

Otitis media with effusion (OME) – GLUE EAR

- Otitis media with effusion (OME) (also called **glue ear**) is simply a collection of fluid that occurs within the middle ear space due to the **negative pressure** produced by altered Eustachian tube function.
- **No active infection**
- This can occur purely from a viral URTI, with no pain or bacterial infection, or it can precede and/or follow acute bacterial otitis media.
- Otitis media with effusion (OME) does NOT usually present with otorrhoea as the TM prevents any discharge from passing. **However the TM can rupture leading to discharge as is seen in chronic suppurative otitis media (CSOM).**
- Fluid in the middle ear **can cause conductive hearing impairment**, but only when it interferes with the normal vibration of the TM by sound waves.
- Over weeks and months, middle ear fluid can become very **thick and glue-like** (thus the name glue ear), which increases the likelihood of its causing **conductive hearing impairment**.
- Early-onset OME is associated with feeding while lying down, parental smoking, too short a period of breastfeeding and greater amounts of time spent in group child care
- **The TM is retracted due to the negative pressure**
- **OME = effusion of the middle ear that may be either mucoid or serous (serous otitis media)**
- **Key features:**
 - DULL TM
 - Fluid levels
 - Retracted
 - Conducting HL

Benign positional paroxysmal vertigo (BPPV)

- **Very common**
- It is the commonest cause of vertigo on looking up
- BPPV is characterised by a **positional trigger of the vertigo** on:
 - Looking up
 - Turning in bed - often worse to one side
 - First lying down in bed at night
 - On first getting out of bed in the morning
 - Bending forward
 - Rising from bending
 - Moving head quickly – often only in one direction
- **Brief episodes of vertigo (few seconds to several minutes).** This is in contrast to Menieres disease which is characterized by longer periods of vertigo (>20 mins to hours)
- **No associated tinnitus, sensorineural hearing loss or aural fullness** (these symptoms are associated with the excess fluid in the inner ear in Ménière's Disease)
- **Patients do not experience other neurological deficits such as numbness or weakness,** and if these symptoms are present, a more serious aetiology such as posterior circulation stroke, vertebrobasilar insufficiency, or ischemia, must be considered.
- **The spinning sensation (vertigo) experienced from BPPV is usually triggered by movement of the head, will have a sudden onset, and can last anywhere from a few seconds to several minutes.**
- The most common movements patients report triggering a spinning sensation are tilting their head upwards in order to look at something, and rolling over in bed
- Patients with BPPV will report a history of vertigo as a result of fast head movements. Many patients are also capable of describing the exact head movements that provokes their vertigo
- **Nystagmus may also be present during vertigo spells**
- **It is important to remember that the DD includes vertebro-basilar insufficiency (VBI) and Carotid Sinus Hypersensitivity.** Vertigo, the sensation of spinning even whilst a person is still, is the most recognizable symptom of decreased blood flow in the vertebra-basilar distribution. **However, for a diagnosis of VBI we need other posterior circulation features with the vertigo e.g. visual disturbance, diplopia, dysarthria, dystaxia, etc.**
- Carotid sinus hypersensitivity (CSH) is an exaggerated response to carotid sinus baroreceptor stimulation. Causes pre-syncope/syncope NOT vertigo. **The assessment must be part of a thorough assessment of recurrent dizziness, syncope and falls.** Typical trigger factors are shaving, head turning, neck extension or tight collars.

Aetiology:

- **Otolithiasis:** Within the labyrinth of the inner ear lie collections of calcium crystals known as otoliths. In patients with BPPV, the otoliths are dislodged from their usual position within the utricle (vestibule) and migrate over time into one of the semicircular canals. When the head is reoriented relative to gravity, the gravity-dependent movement of the heavier

- Brow lift
- Botulinum toxin
- Blepharoplasty
- Skin rejuvenation
- Skin peel and laser resurfacing

Reconstruction

- Primary intention (closure): When wound edges are brought together so that they are adjacent to each other (re-approximated). Minimizes scarring. **Most surgical wounds heal by primary intention healing.** Wound closure is performed with **sutures (stitches), staples, or adhesive tape.** Examples include well-repaired lacerations, well reduced bone fractures, and healing after flap surgery
- Healing by secondary intention: Secondary intention wounds usually involves some loss of tissue. They are left open to allow the free drainage of exudate and the formation of **granulation tissue** to fill the cavity left by dead or excised tissue. The wound is allowed to **granulate.** Surgeon may pack the wound with a gauze or use a drainage system. Granulation results in a **broader scar.** Healing process can be slow due to presence of drainage from infection. Wound care must be performed daily to encourage wound debris removal to allow for granulation tissue formation. Examples: gingivectomy, gingioplasty, tooth extraction sockets, poorly reduced fractures, burns, severe lacerations, pressure ulcers.
- Tertiary intention (delayed primary closure or secondary suture): Where there is great loss of tissue, the wound must heal by contraction of the wound edges, and the formation of granulation tissue. In some cases, the presence of a foreign body or infection may be suspected, and these wounds are left open deliberately for several days until the potential complication has resolved. When resolution has occurred, the wound edges can be brought together (approximated) and the wound proceeds to heal. Wound healing by this approach is termed "healing by tertiary intention" or "delayed primary closure", and is most commonly used in surgical wounds complicated by infection. The wound is initially cleaned, debrided and observed, typically 4 or 5 days before closure. The wound is purposely left open. **Examples = healing of wounds by use of tissue grafts.**
- Skin grafts: blood supply from donor area is cut off
- Skin flaps: intact blood supply from donor area

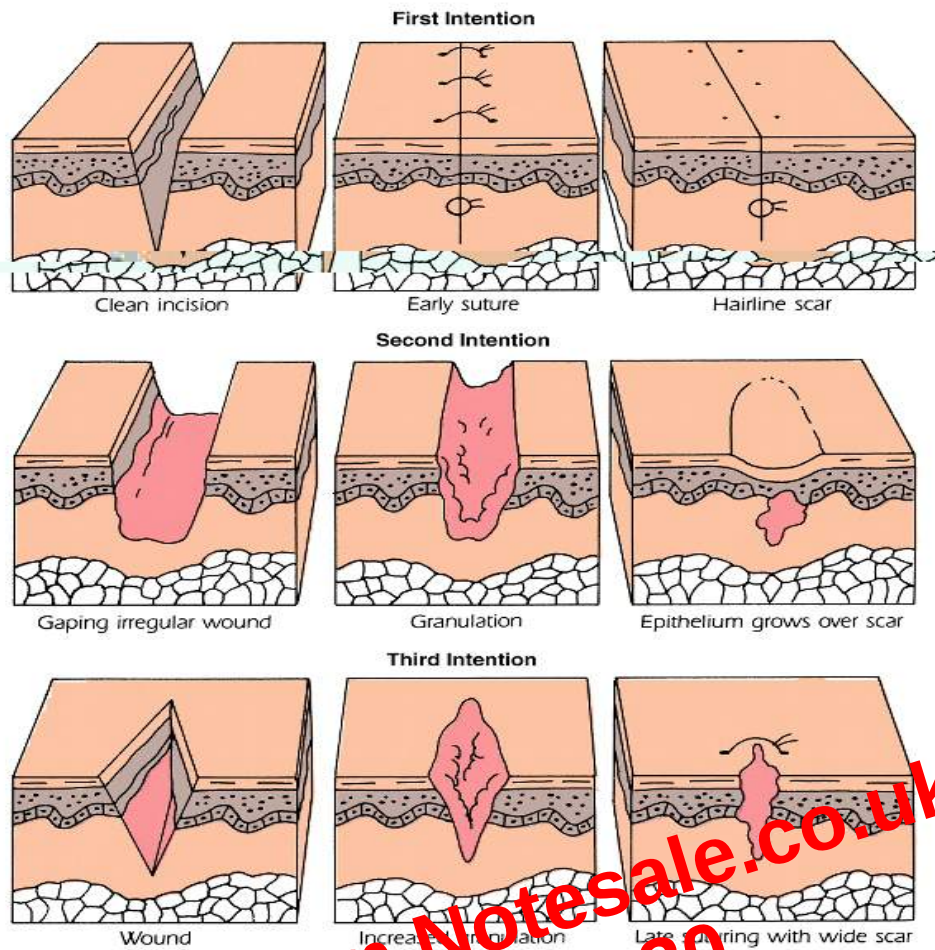
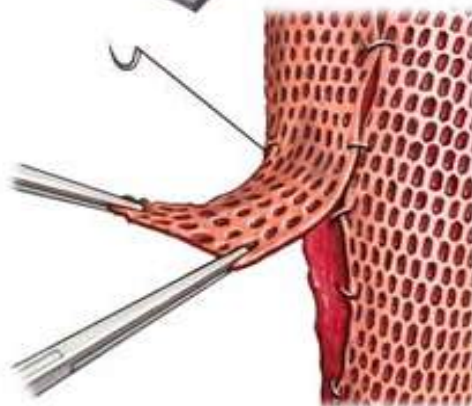


Figure 20-7 Types of wound healing: first intention healing, second intention healing, and third intention healing

Graft taken from patient's healthy skin



Skin is meshed to cover a large wound



Ear Lacerations

- Blunt trauma
- Avulsion
- Dog bites => co-amoxiclav, tetanus
- Tissue loss

Management:

- **Debridement:** medical removal of dead, damaged, or infected tissue to **improve the healing potential of the remaining healthy tissue and prevent infections**
- Closure of wound
 - Primary e.g. suturing
 - Reconstruction
- Usually LA (local anaesthetic) is administered
- Prophylactic antibiotics (if cartilage exposure)

Temporal bone fractures

- The temporal bone is a very complex bone in the human body
- It houses many vital structures, including the cochlear and vestibular end organs, CN VII (the facial nerve), CN VIII (the vestibulocochlear nerve), the carotid artery, and the jugular vein.
- A temporal bone fracture can involve none or all of these structures.

History:

- Injury mechanism
- Hearing loss
- Vertigo
- Facial palsy (due to damage to CN VII)
- Fluid leak including blood (bloody otorrhoea) and CSF (CSF otorrhoea) into the external auditory meatus
- Associated injuries

Examination:

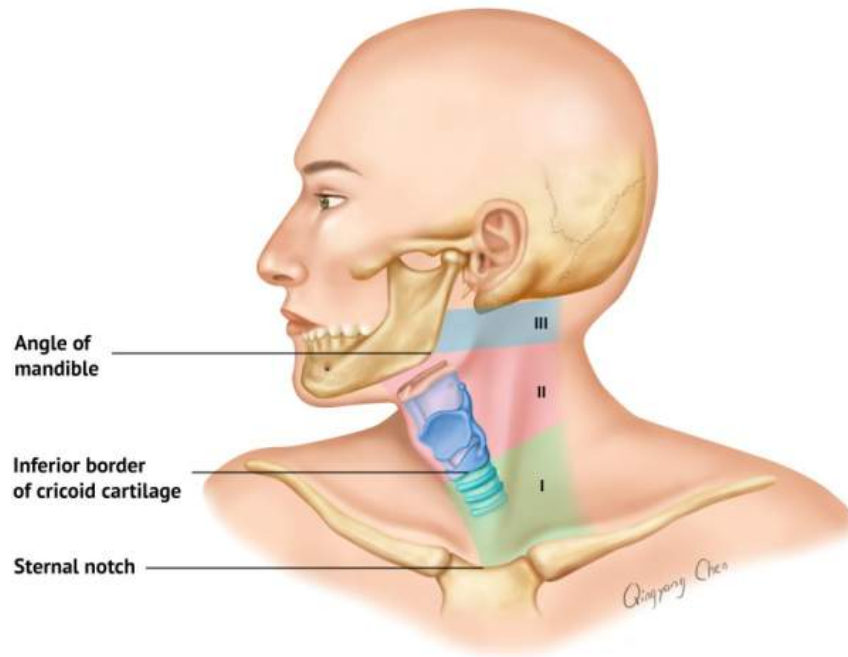
- **Bruising: Battle sign (bruising over the mastoid process, a sign of middle cranial fossa fracture)**
- TM (tympanic membrane) and ear canal
- CN examination (in particular CN VII and CN VIII)
- Hearing tests (Weber and Rinne tests)
- Fluid leak including blood and CSF in the external auditory meatus

Webber and Rinne test

- **Normal Hearing: air conduction (AC) should be greater than bone conduction (BC)**
- This is because the ears have evolved to hear noises which travel as sound waves **through the air** into the ear.
- Air conduction uses the apparatus of the ear (pinna, eardrum and ossicles) to **amplify** and direct the sound to the inner ear (cochlea)
- **In contrast bone conduction bypasses some or all of these and allows the sound to be transmitted directly to the inner ears albeit at a reduced amplitude**
- Webber test: in patients with no hearing defects the sound should NOT be localized to either ear (e.g. should be heard equally in both ears, or not heard at all)
- Rinne test: in patients with no hearing defects $AC > BC$ in both ears i.e. +ve Rinnes (however note that sensorineural loss can also result in $AC > BC$ – as both AC and BC are equally decreased maintaining their relative difference). Rinne's main use is for identifying conducting HL ($BC > AC$ Rinne –ve) or ruling out conducting HL (Rinne +ve $AC > BC$)

Webber test

- The Weber test is a quick screening test for hearing. It can detect unilateral (one-sided) conductive hearing loss (external/middle ear air hearing loss) and unilateral sensorineural hearing loss (inner ear or neural hearing loss).
- **A 512 Hz** tuning fork is placed on top of the head equidistant from the patient's ears on top of thin skin in contact with the bone. The patient is asked to report in which ear the sound is heard louder.
- A normal Weber test has a patient reporting the sound heard equally in both sides (or not hearing it at all)
- **A patient with a unilateral conductive hearing loss would hear the tuning fork loudest in the affected ear.** This is because the conduction problem of the external or middle ear diminishes the conduction of sound through the external and middle ear (**so that ambient noise received by the affected ear is decreased**), while the well-functioning inner ear (cochlea) picks the sound up via the bones of the skull (bone conduction) causing it to be perceived as a louder sound than in the normal ear (as sensed ambient noise is reduced in defected ear).
- **In contrast, a patient with a unilateral sensorineural hearing loss in an affected ear would hear the sound louder in the unaffected ear.** This situation is because the affected ear is less effective at picking up sound even if it is transmitted directly by bone conduction into the inner ear.
- An abnormal Weber test is only able to tell the clinician that there is either a conductive loss in the ear which hears the sound louder or that there is a sensorineural loss in the ear which does not hear as well.
- **If Weber lateralises (sound heard loudest) to the left => either conductive loss in left or sensorineural loss in right (or combined losses)**
- **If Weber lateralises (sound heard loudest) to the right => either conductive loss in right or sensorineural loss in left (or combined losses)**



History

- Site: including zone
- Onset, evolution – when did it happen? How has the injury symptoms progressed with time?
- Character - mechanism of injury
- Associated symptoms:
 - Pain: site, onset and timing, character, radiation, associated symptoms, exacerbating and alleviating factors, severity
 - Aerodigestive tract: dyspnoea, hoarseness, dysphonia, dysphagia
 - CNS problems: paraesthesias, weakness
 - Bleeding

SAMPLE history in acute situations

- S : Symptoms
- A : Allergies
- M : Medications (Anticoagulants, insulin and cardiovascular medications especially) – **always ask about before administering medication**
- P : Previous medical/surgical history
- L : Last meal (time) – **important for surgery**
- E : Events /Environment surrounding the injury e.g. exactly what happened

Examination

- A (airway): check for patent airway, stridor, hoarseness

- B (breathing): Assess RR, SpO₂, ABGs, use of accessory muscles, sternal/intercostals recession, palpate trachea, respiratory exam, administer O₂
- C (circulation): Assess BP, HR, skin temperature, core temperature, capillary refill time, palpable pulses, precordial exam, secure IV access, administer fluids if necessary, take bloods (U&Es, LFTs, G&S, X match, etc)
- D (disability): Check BM (blood glucose), neurological exam including pupil assessment, GCS
- E (full exposure): Check for any lacerations, haemorrhages, cuts etc
- Secondary survey: This assessment is a complete examination of the patient from top to toe, both front and back.
- **Continually reassess (ABCDE) – particularly after every intervention**
- **CALL FOR HELP if compromised SEWS score**

Secondary survey

- Top to toe examination
- Further Ix
- Zones of neck
- Bleeding/haematoma
- Aerodigestive injuries
- Neurological: power, tone, reflexes, sensation (upper and lower limbs)

Investigations

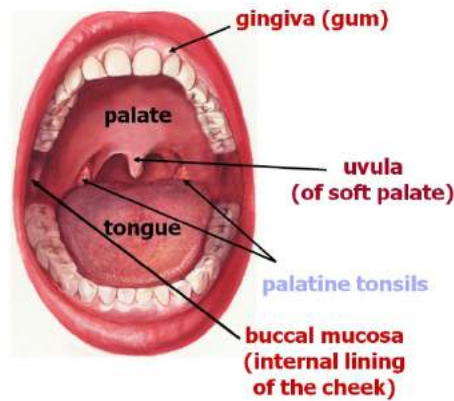
- Bloods: FBC, group and save (G&S) (<20% chance need of blood transfusion, or cross match (XM) if >20% requirement)
- Imaging: A full lateral of neck (full body e.g. CT head, body, abdomen and pelvis may be indicated depending on mechanism of injury)
- CXR: to assess for haemo-pneumothorax, surgical emphysema
- CT Angiogram: vascular, pseudoaneurysm, laryngeal, aerodigestive tract
- MRA (magnetic resonance angiography) if neurological symptoms
- NB: The aerodigestive tract= the combined organs and tissues of the respiratory tract and the upper part of the digestive tract (including the lips, mouth, tongue, nose, throat, vocal cords, and part of the esophagus and windpipe).

Management

- Laryngoscopy, bronchoscopy, pharyngoscopy, and oesophagoscopy
- Angiography: embolize, occlude
- Urgent assessment for airway obstruction, expanding haematoma, hypovolaemic shock, blood in aerodigestive tract

Facial trauma

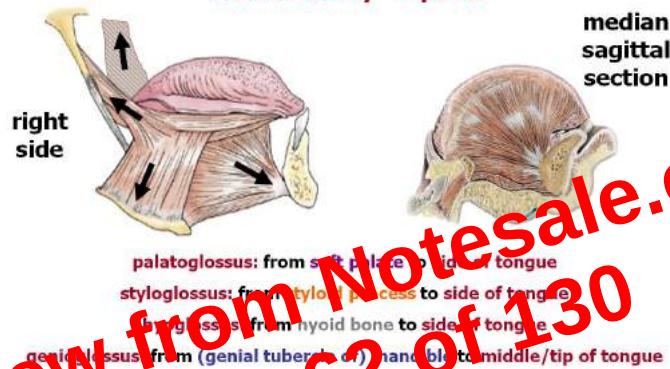
Normal Surface Anatomy



THE SKELETAL (VOLUNTARY) MUSCLES of the TONGUE

ALL supplied by the hypoglossal nerve CN XII
(except palatoglossus: muscle of the soft palate)

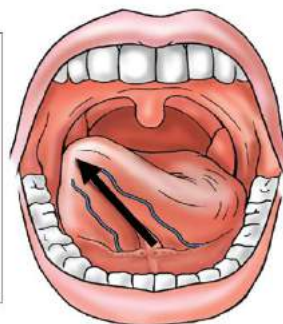
muscles alter position of tongue within the oral cavity - 4 pairs:



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CLINICAL TESTING OF CN XII, HYPOGLOSSAL NERVE Ask your patient to 'stick their tongue straight out'

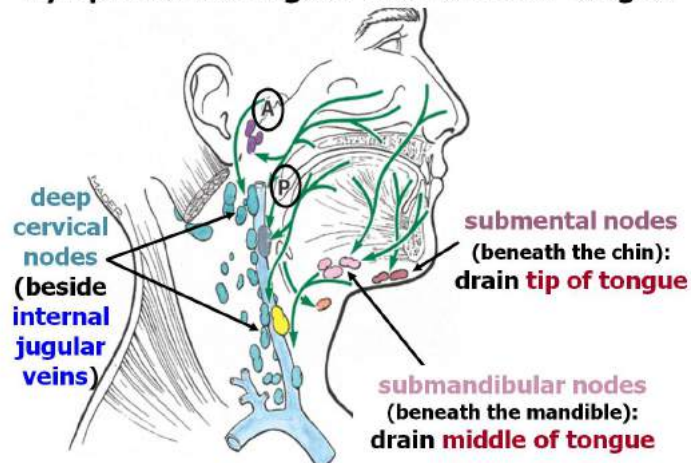
If both CN XII's are normal, the tongue tip should stick out and remain in the MIDLINE



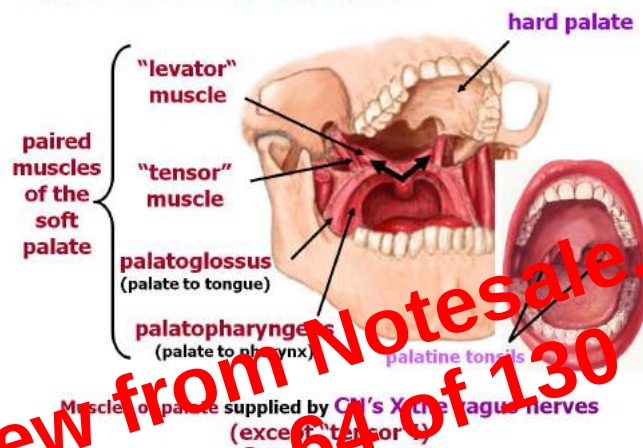
If one CN XII is damaged, the tongue tip will POINT TOWARDS the side of the injured nerve

this image shows RIGHT hypoglossal nerve damage

Lymphatic Drainage of the Tonsils & Tongue



Skeletal Muscles of the Soft Palate

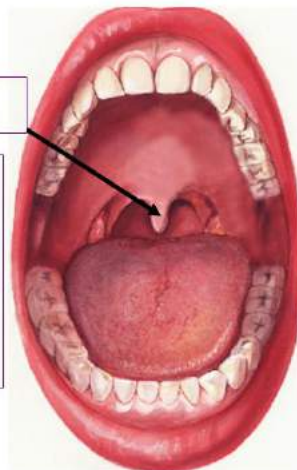


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Clinical Testing of CN's X the vagus nerves
Ask the patient to say "ah"

watch the uvula

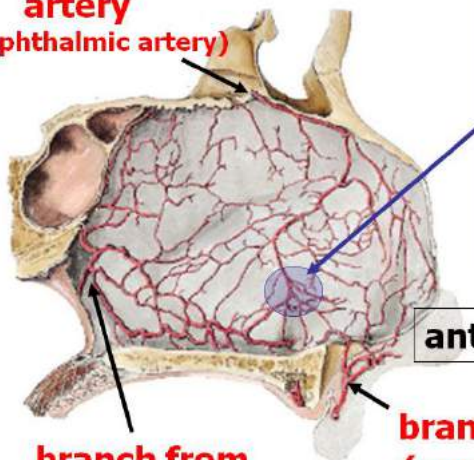
If both CN X's are normal the uvula should lift straight up in the MIDLINE



If CN X is damaged on one side the uvula will be pulled AWAY FROM the injured side by the good side

Medial Wall of the Right Nasal Cavity (Nasal Septum)

branch from
internal carotid
artery
(via ophthalmic artery)



Little's or
Keisselbach's area:

site of arterial
anastomoses and
common bleeding
point in epistaxis
(nosebleed)

branch from
maxillary artery
(deep artery of
the face)

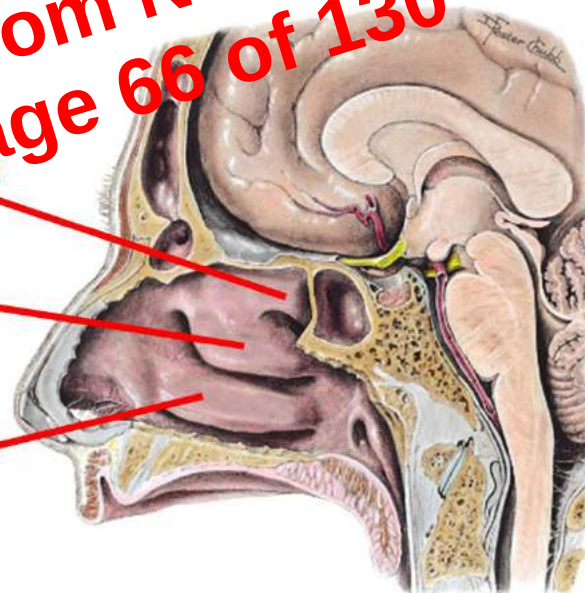
branch from facial artery
(superficial artery of the
face)

The Three Nasal Conchae of the Lateral Wall of The Right Nasal Cavity

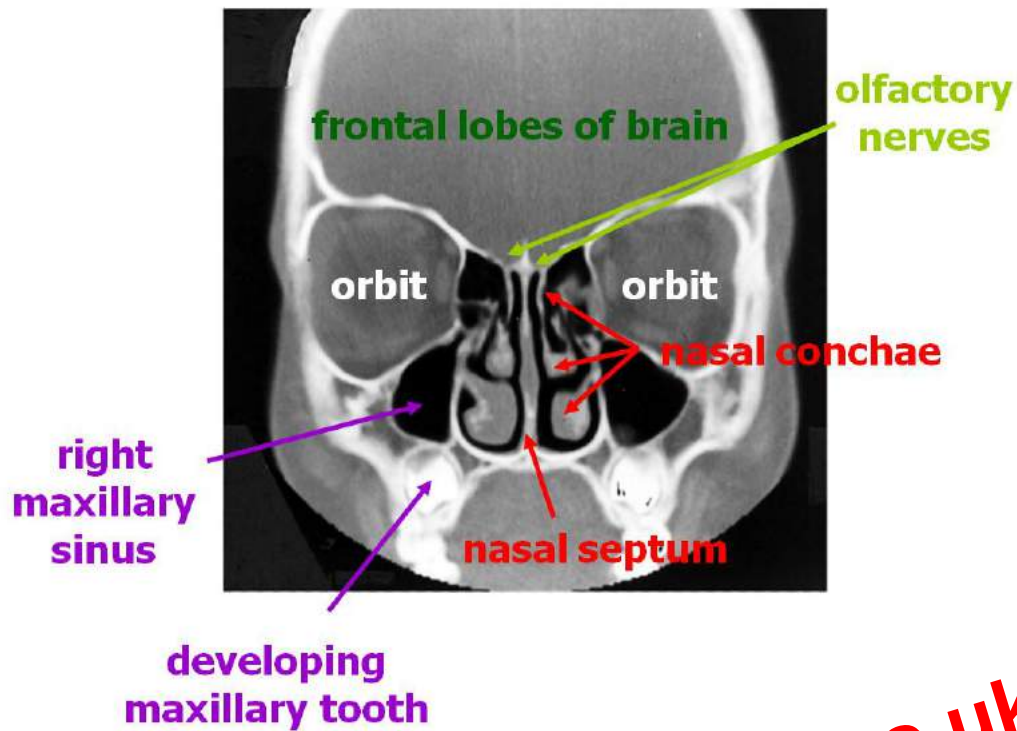
superior concha

middle concha

inferior concha

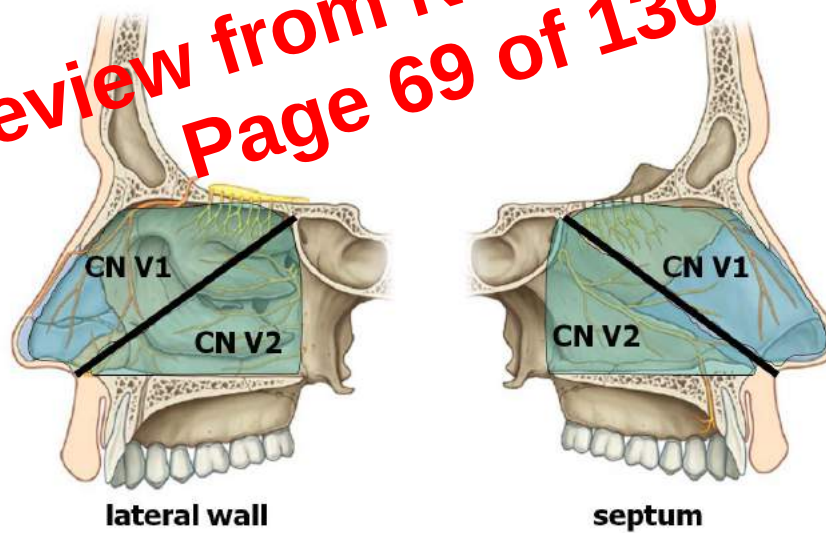


coronal CT scan



(Somatic) Sensory Nerve Supply to the Nasal Cavities

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THE ANATOMY OF COUGHING

- breathe in using the **diaphragm** (**phrenic nerves**)
- close the vocal ligaments (**vagus nerves**)
- contract the **antero-lateral abdominal wall muscles** (**intercostal/subcostal nerves**) to build up pressure beneath the closed vocal ligaments
- suddenly open the vocal ligaments (**vagus nerves**)
- tense and raise the **soft palate** to direct the stream of air through the mouth (**vagus nerves**)
- arguably **THE** most important protective reflex in humans
- the inside of the **larynx** is ACUTELY **sensitive to touch** (**vagus nerves**)

The **sensory and motor supply to the pharynx and larynx:**
CN's X: the vagus nerves

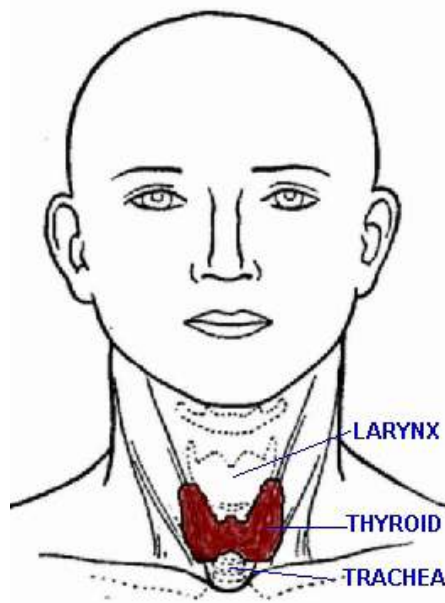


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CLINICAL TESTING OF CN's X

1. Ask the patient to swallow (a sip of water). Observe the larynx moving up and down normally. Wait. Do they splutter?
2. Listen to the patient speaking. Is the voice hoarse?
3. Ask the patient to cough. Is the cough powerful?
(NB also requires functioning **diaphragm** and **phrenic nerves**)

- The gag reflex tests the afferent pathway (glossopharyngeal CN IX) and the efferent pathway (vagus CN X)
- To test vagus also ask patient to say "ahhhh" and watch the soft palate and uvula

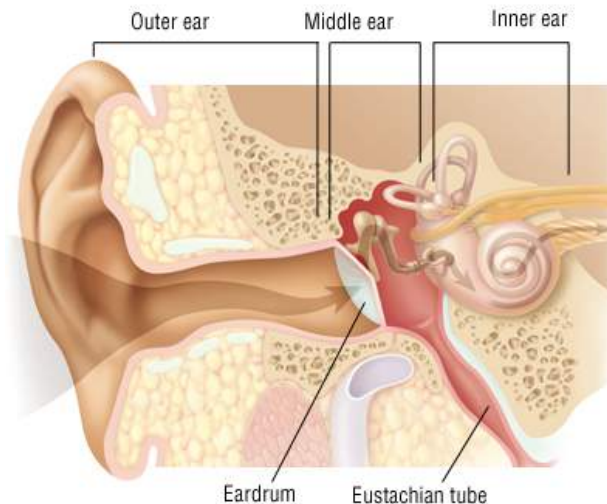


- Appreciate how far down the thyroid gland is – it is at the bottom the neck!

ADENOTONSILLAR DISEASE AND OTTIS MEDIA WITH EFFUSION (OME)

Tonsils

- Three sets of tonsils:
 - “Tonsils” (palatine tonsils)
 - Adenoids (pharyngeal tonsils): close proximity to opening of Eustachian tube
 - Lingual tonsils
- Together they form **Waldeyer's Ring**: a ring of lymphoid aggregation in the subepithelial layer of oropharynx (palatine tonsils and lingual tonsil) and nasopharynx (adenoids)
- Primarily consist of **lymphocytes** (B cells and T cells)
- Main functions
 - Trap bacteria and viruses on inhalation
 - Expose to **immune system**
 - Produce ABs
 - Help to prime immune system and help to prevent subsequent infections
- Significant adenotonsillar enlargement is unusual under 2
- **After early teenage years, the tonsils & adenoids decrease in size**



Symptoms of OME

- **Conductive hearing loss and deafness:** over weeks and months, middle ear fluid can become very thick and glue-like (hence the name glue ear), which increases the likelihood of it causing conductive hearing impairment
- Hearing problems can result in: **poor school performance, behavioural problems, and speech delay** (if chronic => chronic hearing loss)
- Balance problems
- **NO OTALGIA, FEVER OR IRRITABILITY (this triad of signs is characteristic of AOM)**

Signs of OME

- **TM retraction:** retracted eardrum occurs when a person's eardrum, or tympanic membrane, gets sucked or pulled into the space behind it. This happens when the pressure in this space, known as the middle ear, is too low (**-ve pressure**) **which occurs due to Eustachian tube blockage. TM retraction is opposite to TM bulging (which is often seen in AOM)**
- **Reduced TM mobility** (due to build up of fluid in middle ear)
- **Altered TM colour** (but no erythema which is a sign of acute inflammation e.g. AOM) – **grey and dull**
- **Visible middle ear fluid/bubbles**
- Conductive hearing loss (in affected ear) demonstrated by tuning fork tests: Webber will lateralise to affected ear and Rinnes will show BC>AC (-ve result) in affected ear

Symptoms and signs useful for distinguishing between different types of otitis media

- Acute otitis media (AOM): pain (otalgia) and/or fever and feeling unwell
- AOM with perforation: Ear discharge (otorrhoea)
- Otitis media with effusion (OME): Conductive hearing loss with no signs of acute infection (e.g. no fever or pain)
- Chronic suppurative otitis media: Same as AOM but with ear discharge (otorrhoea)

Otoscopy

- OME:
 - **TM retraction:** retracted eardrum occurs when a person's eardrum, or tympanic membrane, gets sucked or pulled into the space behind it. This happens when the pressure in the middle ear is too low (-ve pressure) which often occurs in OME due to Eustachian tube blockage.
 - **Reduced TM mobility** (due to build up of fluid in middle ear and -ve pressure)
 - Altered TM colour (but no erythema indicative of AOM) – **grey and dull TM**
 - Visible middle ear **fluid/bubbles** – if completely filled the TM will appear opaque (loses its translucency)
 - **Conductive hearing loss** (in affected ear) demonstrated by tuning fork tests (Webber will lateralise to affected ear; Rinnes will show BC > AC (-ve result) in affected ear)
- AOM:
 - In contrast, the appearance of the eardrum in acute otitis media (AOM) is red (**erythema**) or yellow (**due to acute inflammation**) in appearance – **RED ANGRY LOOKING TM**
 - TM is also **opaque** (opposite to transparent) with indistinct landmarks (due to acute inflammation)
 - It can appear to be **bulging** towards the viewer (in contrast to the -ve pressure induced retraction in OME)
 - Insufflation (inhaling a substance) may show **decreased mobility of TM**
 - **Perforation may be present:** Sometimes in AOM the eardrum will burst, allowing the pressure (and the pain) to reduce. Then one can often see the tear in the drum, as well as the discharge in the outer ear. In the majority of people, such a tear will heal completely.

OME versus AOM summary

- Allergic rhinitis may cause additional symptoms, such as sneezing and nasal itching, coughing, allergic conjunctivitis, headache, fatigue, malaise, and cognitive impairment.
- The allergens may also affect the eyes, causing watery (epiphora), reddened (forniceal injection) or itchy eyes and puffiness around the eyes (**allergic conjunctivitis**)
- Allergic rhinitis occurs when an allergen such as pollen or dust is inhaled by an individual with a sensitized immune system, triggering antibody (IgE) production. These antibodies mostly bind to **mast cells**, which contain **histamine**. When the mast cells are stimulated by allergens => histamine (and other chemicals such as leukotrienes) are released. This causes itching, swelling, and mucus production. Eosinophils also play a role in the inflammation process.
- This inflammatory process that can cause symptoms outside the nose, such as fatigue and malaise
- Symptoms vary in severity between individuals. Very sensitive individuals can experience hives or other rashes.
- **In summary: allergic rhinitis is a type 1 HS (IgE mediated) resulting in activation of mast cells (and release of histamine and leukotrienes), eosinophils, and other inflammatory cells in the nose +/- eyes (conjunctivitis)**

Intermittent triggers

- Grass pollen
- Tree pollen
- Fungal spores

Persistent trigger

- House dust mite
- Cat
- Dog

O/E:

- Lateral crease on the nose
- **Swollen nasal turbinates (nasal conchae)**
- **Nasal congestion or rhinorrhea (runny nose)**
- **Middle ear effusion (OME) – due to blockage of Eustachian tube**
- **Conjunctivitis** (if eyes affected): conjunctival swelling, erythema (forniceal injection) and watering (epiphora)
- Eyelid swelling

ALWAYS EXAMINE THE WHOLE ENT SYSTEM – not just one part in isolation

ARIA (allergic rhinitis and its impact on asthma) classification

Intermittent

- Symptoms <4 days per week or symptoms for <4 weeks

Persistent

- Tongues relative large and larynx relatively high up => just behind tongue is the larynx => simian position => babies can breath and suckle at same time
- Neck muscles are weak in young children
- Subglottis (cricoid area of larynx, just below vocal cords which are contained in thyroid cartilage) has a very small diameter
- **The cricoid areas is the narrowest part of airway:** very narrow => croup (laryngotracheobronchitis) is very concerning in young children as just a tiny bit of mucus swelling can cause blockage
- In Down's syndrome children, the cricoid area is even narrower

Fluid dynamics of the airways

- Airflow is co-axial flow (smooth). If you narrow any part of airway => flow will break => increased resistance => decreased airflow
- Poiseuille's Law states that the resistance exerted by a tube is proportional to $1/r^4$
- **Therefore if we ½ the diameter of the airway => we get a 16 times increase in resistance => 16 times decrease in airflow (but an increase in velocity, remember)**
- Bernoulli principle: when air flows through a tube, the pressure on the lateral wall drops. If you have an airway which is not strong (e.g. laryngomalacia- floppy larynx, or decreased tone during sleep) => flow of air through it => decrease in pressure on lateral wall => airway collapses. **Therefore in laryngomalacia, inspiration can result in collapse of airways.**
- **Laryngomalacia** (literally, "soft larynx") is the most common cause of stridor in infancy, in which the soft, immature cartilage of the upper larynx collapses inward **during inhalation**, causing airway obstruction and **STRIDOR**

Laryngomalacia

- **Laryngomalacia** (literally, "soft larynx") is the most common cause of stridor in infancy, in which the soft, immature cartilage of the upper larynx collapses inward during inhalation, causing airway obstruction
- Laryngomalacia results in **partial airway obstruction**, most commonly causing a characteristic high-pitched squeaking noise on inhalation (inspiratory stridor). Some infants have **feeding difficulties** related to this problem. Rarely, children will have significant life threatening airway obstruction. The vast majority, however, will only have stridor without other more serious symptoms such as dyspnea (difficulty breathing).
- Infants present within the **first few weeks of life** (not necessarily at birth, typically at 4-6 weeks) with **noisy respiration and inspiratory stridor which are worse in the supine position, when feeding or when agitated**. Infants may also have gastro-oesophageal reflux but they are otherwise generally well, happy babies. Importantly, the cry is normal (if not, there may be an abnormality at or near the vocal cords). The abnormal sounds may best be heard just above the sternal notch.

Upper airway during sleep

If this infection had presented in the first few days of life, what other possible causes of infection should you consider?

See above – Chlamydia and N gonorrhoeae can present in the neonate as transferred from the mother during a vaginal deliver

Case 6

This young boy presents to A&E with a swollen eye and double vision. There is no history of any injury but he is pyrexial at 38°C. On examination the eyelid is very swollen and the eye movements are restricted.

What is your working diagnosis?

Orbital cellulitis – serious infection that is a MEDICAL EMERGENCY
Presents with fever, swollen eye and restricted movement
Proptosis (exophthalmos) – forward displacement of eye can occur
SIGHT THREATENING infection

What microbiological and non-microbiological investigations would you order and why?

CT scan to identify orbital abscesses
Try to get a swab of pus to culture

Which specialists would you ask for help?

Ophthalmology and microbiology

What treatment would you start?

Requires emergency drainage
Organisms: HIB, Staph, Strep, anaerobes
Antibiotics: Cefuroxime/ceftriaxone IV + high dose Flucloxacillin + metronidazole

What complications may occur?

Loss of sight, abscess
Cavernous sinus thrombosis

Must know ENT conditions

- Rhinitis (both allergic and non-allergic types)
- Nasal trauma – exclude septal haematoma, DON'T image for fractures
- Acute and Chronic Sinusitis
- Presbycusis (age related SN hearing loss): cumulative effect of aging on hearing. It is a progressive bilateral symmetrical age-related sensorineural hearing loss caused by multiple factors e.g. deterioration of cochlear hair cells. **HIGH FREQUENCY LOSS.**
- Noise-induced hearing loss
- Tinnitus
- Otitis Externa
- Acute Otitis Media
- Otitis Media with Effusion
- Tympanic Membrane Perforation
- Cholesteatoma: destructive and expanding growth (cyst like growth) consisting of keratinizing squamous epithelium in the middle ear and/or mastoid process.