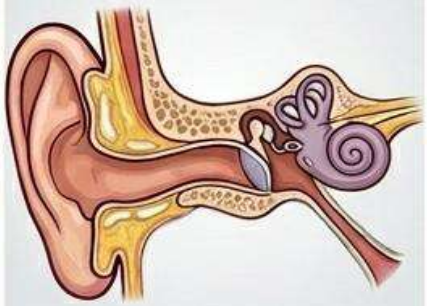
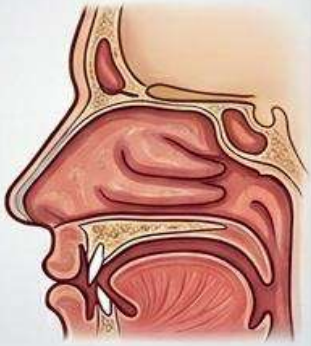
Stellenbosch University

ENT DISEASE

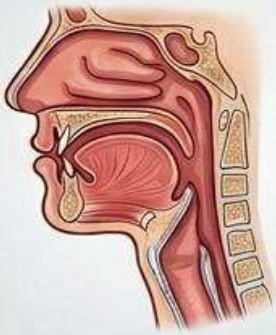
THE EAR & HEARING



THE NOSE & SINUSES



THE THROAT & VOICE



Faculty of Medicine and Health Sciences,
Department of Surgical Sciences, Division of ENT

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Terms of use

1. Nature of Content

The information contained in these notes (the "Content") is provided for **educational and informational purposes only**. It is intended to support medical students in their studies and General Practitioners in their continuing professional development. For Stellenbosch University medical students, the text in light grey font is “nice to know” information and is not considered as core knowledge. However, some questions might be included from the text in examinations.

2. No Physician-Patient Relationship

The Content does not constitute professional medical advice, diagnosis, or treatment. Accessing or reading these notes does not establish a physician-patient relationship. Always seek the advice of a qualified healthcare provider regarding a medical condition.

3. Clinical Accuracy and "Living" Knowledge

Medicine is a rapidly evolving field. While every effort has been made to ensure the accuracy of the information at the time of publication:

- **Guideline Variations:** Clinical protocols vary by region, institution, and governing body (e.g., NICE, WHO, or local Health Departments).
- **Updates:** Drug dosages, contraindications, and "gold standard" treatments may change.
- **Errors:** Users are encouraged to cross-reference all clinical data, especially **medication dosages and surgical anatomy**, with official pharmacopeia and peer-reviewed literature.

4. Limitation of Liability

The authors and contributors shall not be held responsible for any clinical errors, omissions, or adverse outcomes resulting from the use of this information. The final responsibility for patient care lies solely with the **treating clinician**, who must integrate this knowledge with the patient’s specific clinical presentation, history, and local resource availability.

5. Professional Responsibility (For Practitioners)

General Practitioners are reminded that these notes are a supplemental resource. They do not supersede the clinician’s duty to exercise independent professional judgment or to adhere to the standard of care required in their specific jurisdiction.

1) Ear examination

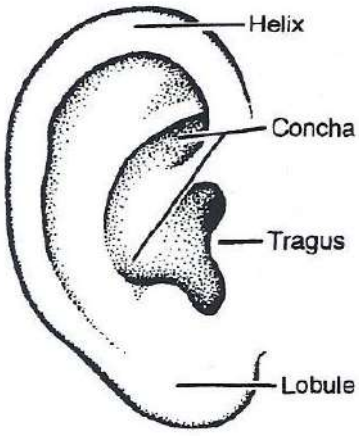
Overview

- Review of the basic anatomy
- Ear examination
- Videos on
    - Normal hearing and the three types of hearing loss
    - How to examine the external ear, ear canal, and tympanic membrane
    - How to do clinical voice tests and tuning fork tests (bedside evaluation of hearing)
- Limitations – what can your ENT do?

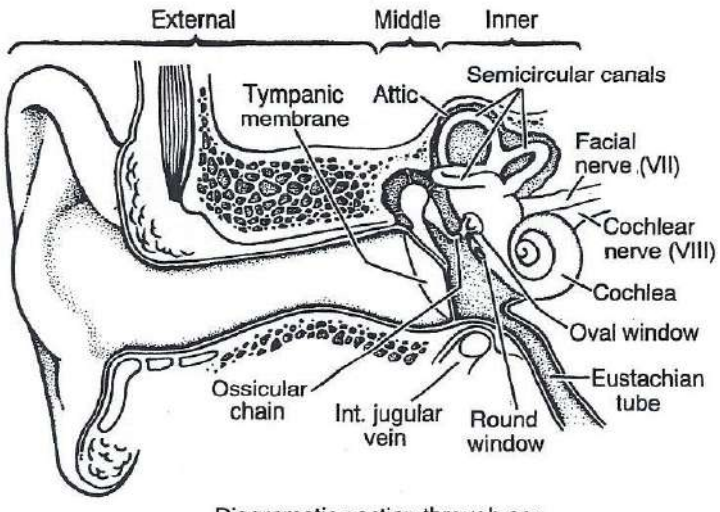
General points

- Personal protective equipment
- Proper light
    - Whether using a head light or otoscope, it should be bright
    - Preferable the ambient light should not be overly bright
- Use two hands
- Be sure to examine all the nooks and crannies
- Be sensitive regarding the position in which you examine a patient
    - Sitting in front or to the side of a patient
- Be sensitive regarding the examination of a child
    - First try to make a bond

Normal anatomy

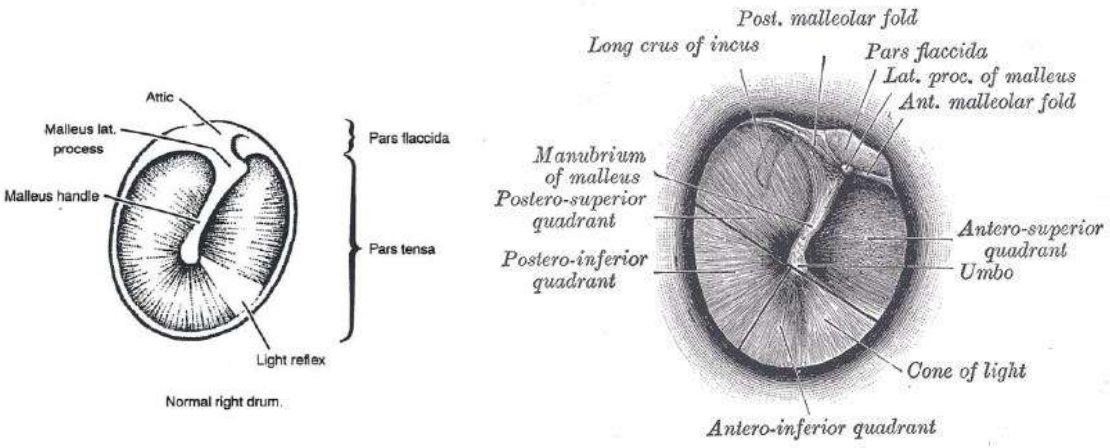


Parts of the external ear

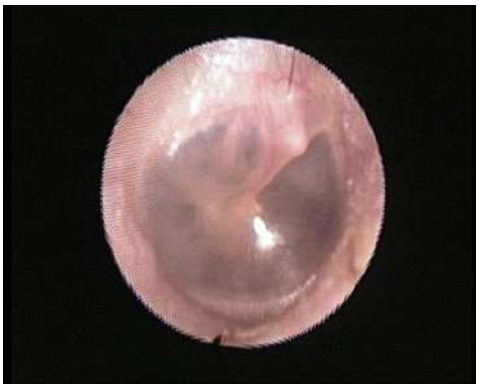


Diagramatic section through ear

Normal pinna (left picture) and external, middle and inner ear (right picture)



Normal right tympanic membrane



Right ear

Left ear

Videos (Medel®)

- Hearing - https://youtu.be/_h9FjGTFMFY
- Conductive hearing loss - https://youtu.be/Kn2gX78M_aQ
- Sensori-neural hearing loss - https://youtu.be/O0CmzXZ1sEo
- Mixed hearing loss - https://youtu.be/8SrusMbjEC4

History

- The initial history is usually kept simple and to the point
- The history is geared to the presenting compliant
- The traditional rule of first history; then physical examination; then special investigation is sometimes bent slightly in the interest of time-efficient examination - e.g.: the identification of wax in the external canal in a patient complaining of hearing impairment may obviate a detailed history
- Certain problems (e.g. vertigo, tinnitus) require extensive history taking
- The important points in history are dealt with under each problem

Examination

You will need to examine the:

- Structure
    - Pinna
        - Inspect and palpate

                - Two mastoids
                    - Antrum / Tip
                - Skin incisions
                - Pinna
                - Concha
                - Meatal skin
                - Tragus
            - (Percussion)
            - Auscultation
        - External ear canal
            - Curve and diameter
        - Tympanic membrane and Middle ear
            - Malleus
            - Look at full diameter
            - Remember pars flaccida
            - Assess if it is
                - Normal / Inflamed / Infected
                - Normal position / Retracted / Bulging
                - Normal mobility / Impaired / Hyper
        - Inner ear
- Function
    - Hearing
    - Balance
- Special investigations
    - Audiology
        - Tympanometry
        - Pure tone audiometry
        - Speech reception thresholds
        - Speech detection thresholds
        - Speech discrimination
        - Stapes reflex
    - Balance
        - Nystagmography
            - Video / Infra-red / Frenzel glasses
        - Caloric testing
        - MRI / CT
    - Radiology
        - CT
        - MRI
    - Bloods

Structure

Outer Ear:

- Scalp
    - Skin conditions causing periauricular lymphadenopathy, especially skin squamous cell carcinomas
- Pinna
- Mastoid antrum (posterior-superior to pinna helix) and process (inferior-posterior to lobule)

    - Signs of inflammation
    - Abscess presents with:
        - Fluctuant swelling over the mastoid process
        - Loss of auricular crease
        - Pinna displaced anterior-inferiorly
        - Rarely the abscess can spread into the zygoma, posterior belly of digastric, or sternocleidomastoid muscle
- Periauricular
    - Pre-auricular pits / sinuses / cysts
    - Lymphadenopathy / abscess formation
- External meatus:
    - Wax
    - Foreign body
    - Narrowing of lumen
    - Otitis externa: Diffuse, Localized e.g. Furunculosis (refer later)
    - Tenderness on manipulation of ear (otitis externa)

Practice removing any wax / pus / foreign body (FB) under supervision in the clinic (also see section "practical tips in otology").
Remember:

- Use a headlight to be able to work with two hands.
- You will rarely have access to a microscope.
- Make sure you have proper equipment such as a Jobson-Horne, Ear Syringe, Small Tiley's, and / or Crocodile forceps.
- The only REAL emergency is a corrosive FB, such as a battery, in the external ear canal. This should be referred as an emergency if you are unable to remove it.
- Otherwise, do not risk injuring the ear canal and middle ear structures in an attempt to remove a FB.

Doctors practicing in the public sector will see a high number of children with chronic suppurative otitis media. You will need to be able to do a proper ear mop (toilet) before instilling any drops or powders into the ear. The videos below are links to an ear toilet and boric powder installation:
https://youtube.com/shorts/SNXWIkNz0jE
https://youtube.com/shorts/fGeZzGU8oQA

Middle ear:

- Eardrum (window to middle ear)
    - Pars tensa
        - Light reflex
        - Colour
        - Malleus
    - Pars flaccida (over "attic")
    - Mobility: pneumatization or Valsalva

Make specific notes regarding:

- Suppurative process
- Cholesteatoma
- Perforation / retraction pocket
- Tuning fork tests (Rinne and Weber)
- Hearing tests

Inner ear:

Can't be visualised.
See functional: hearing / balance assessment.

Function

Hearing:

- During the examination (room testing is limited, but useful).
    - Assess response to your speech
    - Assess patient's speech
    - Assess school progress in relation to child's age
- Adults / Older children can recognize hearing at:
    - Whisper 30dB
    - Speak 60dB
    - Shout 90dB
- Tuning fork tests
    - Rinne
    - Weber
- Young children: Rattle / cup and spoon (distraction) tests

Tuning fork tests:

Differential diagnosis of tuning fork tests

		Weber central	**Weber to the left**	**Weber to the right**
Rinne Positive	**Right ear**	Normal ear	Non-diagnostic Redo	Conductive hearing loss > 5 dB and < 25 dB (mild)
	Left ear	Normal ear	Conductive hearing loss > 5 dB and < 25 dB (mild)	Non-diagnostic Redo
Rinne Negative	**Right ear**	Non-diagnostic Redo	False negative Rinne – severe / dead ear on the right	Conductive hearing loss >25 dB (moderate – severe)
	Left ear	Non-diagnostic Redo	Conductive hearing loss >25 dB (moderate – severe)	False negative Rinne – severe / dead ear on the left

Balance:

Balance is a complex interplay of inputs from the:

- visual system
- vestibular system
- neck
- proprioceptive system

This information is integrated in the brainstem and analysed by the cerebellum by comparing it to previous inputs. At random interval the cerebrum "checks in" on these two. Balance is therefore mainly an automatic

process. Think about running, you can't "compute" the process, it just happens. Ironically some balance disorders, such as PPPD, are based on over-computing normal bodily movements and wants to willingly correct this.

The ears are responsible for producing quick changes in the position of the eye in relation to space to keep an object focused on the fovea. This process is far quicker than eye movements produced by the cerebrum, also known as neurological eye / oculo-motor movements. These quick movements allow for clear vision. If the system fails, vision becomes blurry, and a patient complains about an imbalance / dizziness. Vestibular disease can be either uni- or bi-lateral and either a hypo- or hyper-function. More details with regards to a differential diagnosis will be discussed later.

Balance examination (please refer to the chapter about vertigo):

- History
    - Classical approach
    - Alternative approach
    - Million-dollar questions
- Examination
    - General
        - One can form an immediate clinico-pathological picture by assessing the patient's posture and walking when coming into the consulting room. The physician should also look for signs of anaemia, lymphadenopathy, weight loss and any other obvious signs.
    - Head and neck
    - Ear, nose and throat (ENT)
    - Neuro-otological
        - Nystagmus
            - Spontaneous / Induced
            - Fixation – Gaze straight
            - Fixation – gaze in different positions
            - Without fixation (ideal in dim room with Frenzel lenses)
        - Central oculo-motor signs
            - Smooth pursuit
            - Saccades
            - Vergence
            - Visual fixation / gaze holding
            - Optokinetic nystagmus
        - VOR battery
            - Dix Hallpike and Lateral semi-circular canal testing
            - Dynamic visual acuity test (DVAT)
            - Head thrust / Head impulse test
            - Head shake
            - Caloric test
            - Rotation testing
            - Fistula test
            - Fixation suppression test
            - Subjective visual vertical
    - CNS
        - Higher functions
            - GCS
            - Orientation
            - Intellect

        - Communication
        - Emotional status
    - Cranial nerves
    - Cerebellum
        - Fast tongue movements
        - Ataxia
        - Dysmetria
        - Finger-nose test
        - Dysdiadochokinesia
        - Romberg
        - Heel-shin test
        - Pendular reflexes
        - Nystagmus
    - Motor
        - Inspection
        - Palpation
        - Strength
    - Sensory
        - Pain, temperature and general sensation
        - Light touch, position and vibration
        - Stereognosis
    - Reflexes
    - Gait
    - Coordination
    - Posture
- It is important to also examine the following systems
    - CVS
        - Arrhythmias
        - Reduced cardiac output
        - Hypovolemia
        - Pericarditis
        - Orthostatic hypotension
        - Autonomic dysfunction
        - Vasovagal syncope
    - Hyperventilation
    - Hypoglycaemia
- Special examinations
    - VNG
    - VEMP's
    - Posturography
    - Scans
    - Neurologist / Cardiologist / Physician / GP
    - Audiology / audiologist
    - Physiotherapist
    - Bloods

Special investigations

Ordering these tests is usually (not always) in the realms of an ENT specialist, but often GPs will receive the results of the tests described below.

Audiology:

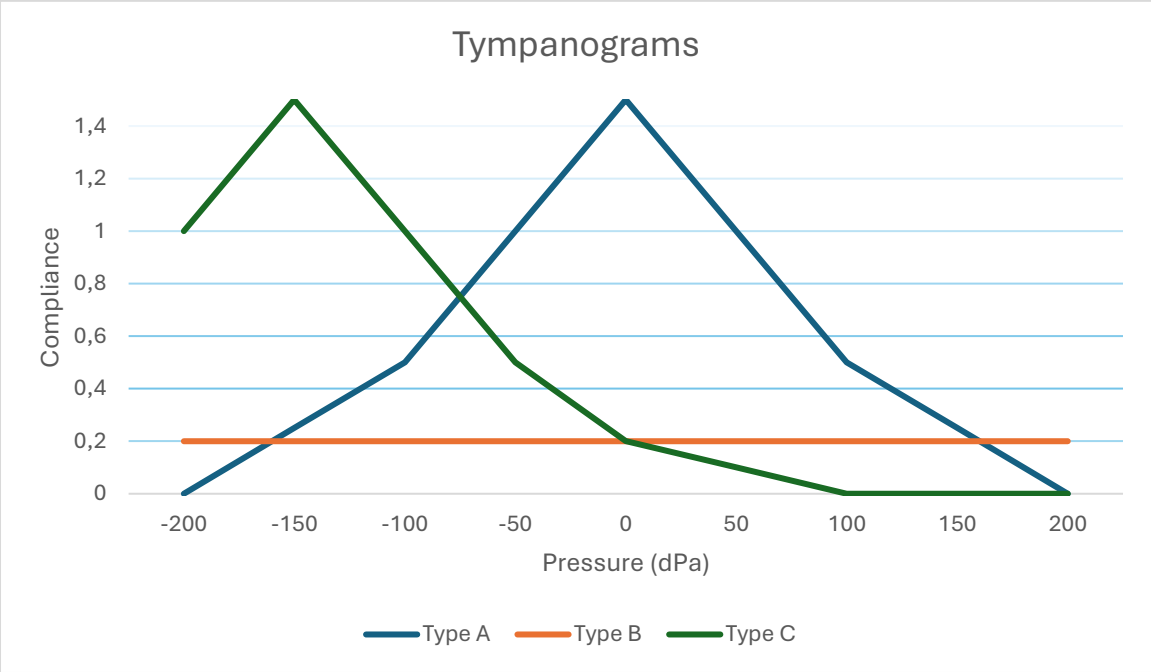
- Tympanometry
- Pure tone audiometry
- Speech reception thresholds
- Speech detection thresholds
- Speech discrimination
- Stapes reflex

Tympanometry

- A probe is placed into the external ear canal and seals it off. The probe sends a sound wave into the external ear canal and measures the amount of sound energy that is reflected back. This all happens while manipulating the pressure in the external ear canal between positive and negative. In a normal ear, the maximal sound should be admitted through to the middle ear when ear canal and middle ear pressures are equal and at atmospheric pressure. Therefore, implying a pressure whereby the least amount of sound energy is reflected back to the probe. By definition this is at zero (atmospheric pressure) and is called a type A tympanogram. A graph is printed and shows a peak at zero, and frequently this is explained as the "movement" of the tympanic membrane. In reality, it is also an indication of the ossicular chain, middle ear pressure, and function of the Eustachian tube
- Before interpreting a tympanogram, one should first look at the external ear canal volume. The normal ranges are 0.5 – 2.0 ml
- A tympanogram with a low volume and type B curve (explained below) points to the probe pressing against the external ear canal skin and is NOT a reflection of the middle ear system
- Otherwise, a type B tympanogram with a volume larger than normal points to a tympanic membrane perforation. In fact, ENT surgeons can use the volume to indirectly predict the successful outcomes of tympanic membrane reconstructions
- There are three types of tympanograms
    - Type A
        - Peaks at between -100 and +100 daPa
        - Implies middle ear pressures reflect atmospheric pressures
        - Two variations are
            - As ("stiff")
                - Has a low peak (compliance)
                - Seen in
                    - Otosclerosis
                    - Malleus fixation
                    - Tympanosclerosis
            - Ad (hypermobile)
                - High compliance
                - Seen with
                    - Ossicular chain dislocations
    - Type B
        - The graph shows that no peak is produce. Therefore, despite varying the pressure of the external ear canal, at no point is there an absolute "sweet spot" where sound energy is admitted through to the middle ear system. This is almost exclusively seen when the middle ear cavity is filled with fluid or a mass. Depending on the type of fluid / mass one might see a dull / yellow / red appearance behind the tympanic membrane
    - Type C
        - The graph shows a peak in the negative pressure range, usually at -150 daPa and more. It implies that the middle ear is at a negative pressure and points to a Eustachian tube that cannot compensate for the loss of middle ear gases (remember there is a constant loss

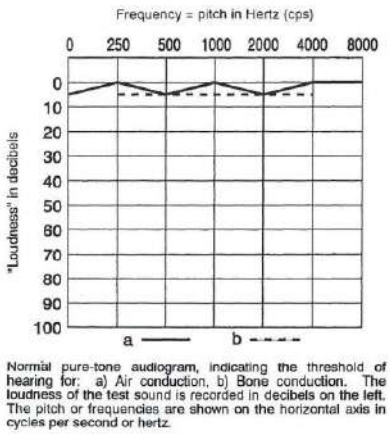
of middle ear gases absorbed by the middle ear mucosa). Clinically one will see retractions for the pars flaccida and / or pars tensa and in severe cases this may lead to adhesive middle ear diseases. Type C tympanograms point to Eustachian tube dysfunction

- Lastly, compliance of TM is 0.39 – 1.3 ml. As explained above this inherently implies the stiffness / laxity of the TM

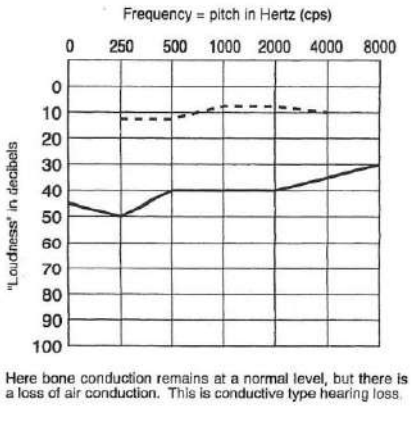


Pure tone audiometry

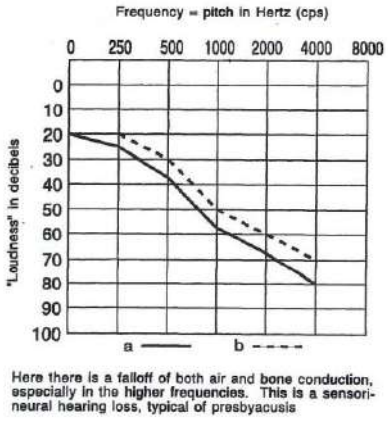
- Pure Tone Audiometry (PTA) is a common hearing test used to assess a person's hearing sensitivity. It's one of the most basic and essential tests in audiology and measures the softest sounds (dB) a person can hear at various frequencies (pitches)
- The test is usually done in a soundproof booth and test both the air and bone conduction
- The patient wear headphones or ear inserts, and a machine called an audiometer plays pure tones (beeps) at different pitches (frequency) and volumes (dB)
- The patient indicates (usually by pressing a button or raising your hand) whenever they hear a sound, even if it's very faint. The audiologists usually start loud and then decrease the loudness by 5 dB increments until the tester responds correctly 50% or more. Patients that consistently respond incorrectly may be malingering
- Frequencies tested are typically between 250 Hz and 8000 Hz
- The results are plotted on a graph called an audiogram, showing the quietest level of sound you can hear at each frequency
- It is used to determine the type (conductive, sensorineural, or mixed) and severity (normal, mild, moderate, severe) hearing loss



Normal pure-tone audiogram, indicating the threshold of hearing for: a) Air conduction, b) Bone conduction. The loudness of the test sound is recorded in decibels on the left. The pitch or frequencies are shown on the horizontal axis in cycles per second or hertz.



Here bone conduction remains at a normal level, but there is a loss of air conduction. This is conductive type hearing loss.



Here there is a falloff of both air and bone conduction, especially in the higher frequencies. This is a sensorineural hearing loss, typical of presbyacusis

Speech reception threshold (SRT)

- Lowest dB where pt can repeat 50% of spondee words – Uses voice but is also measured in SPL dB
- Spondee is bi-syllable words, with equal weight like "railroad"
- Can be used to confirm PTA
- Should be within 10 dB of average of PTA at 0.5, 1, 2 kHz (REMEMBER an audiometer uses Hearing level dB)
- Tested in groups of 6 words
- If the subject has more than 3 incorrect, then increase dB by 10

Speech detection threshold

- Also uses sound pressure level as reference
- Lowest dB where a subject responded to sound
- Commonly used in children when unable to perform SRT

Speech discrimination (SD)

- Percentage "phonetically balanced" words that a subject can repeat
- Single syllable words
- Output is SPL dB, but remember it is measured for the subject's ear, therefore SENSATION LEVEL for him
- Start at 30-40 dB SL (which with normal hearing = SPL dB)
    - Groups of 25 words. If you get 3 wrong, then increase the dB with 5
    - Test at least 3 frequencies
    - Normal is 100% SD 40 dB above the average of the PTA thresholds
    - 50% at 25 dB above average of PTA thresholds
- Interpretation
    - 90-100% - normal
    - 76-89% - Slightly abnormal
    - 60-74% - Moderate
    - 40-59% - Severe
    - < 40% - Very severe
- Remember it tests for sensation level, so if you already have a 25 dB hearing loss, and your 100% SD is at 40dB, the PTA value would be at 65 dB

Balance:

- Nystagmography
    - Video / Infra-red / Frenzel glasses
- Caloric testing

- MRI / CT
- Bloods

Radiology:

- CT (high resolution)
- MRI

Bloods:

Many auto-immune conditions can affect the ears. Of the well-known ones include Granulomatosis with Polyangiitis (GPA) (previously known as Wegener's), Cogan syndrome, Rheumatoid arthritis, SLE etc.

Syphilis remains something we see in the public sector in the Western Cape, but it will be exceedingly rare to see in most settings.

HIV is another infective pathogen known to cause hearing loss and for which patients should be tested.

2) Examination of the Nose and Sinus cavities

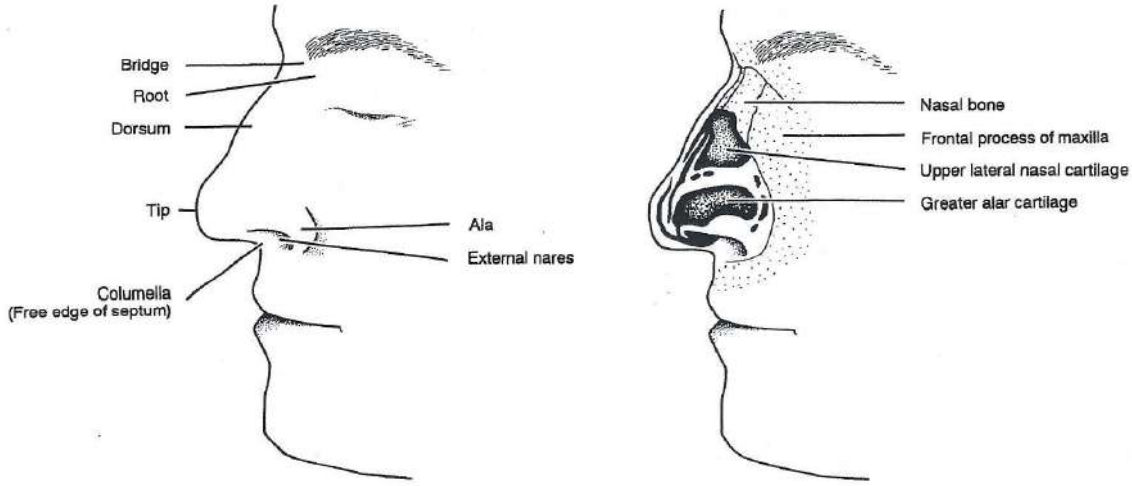
- Review of the basic anatomy
- How to examine the external and internal nose
- Limitations – what can your ENT do?

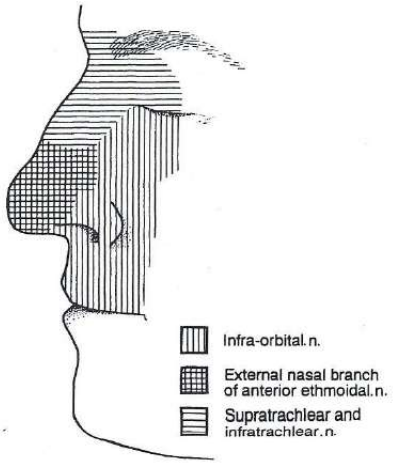
General points

- Personal protective equipment
- Proper light
    - Whether using a head light or otoscope, it should be bright
    - Preferable the ambient light should not be overly bright
- Use two hands
- Be sure to examine all the nooks and crannies
- Be sensitive regarding the position in which you examine a patient
    - Sitting in front or to the side of a patient
- Be sensitive regarding the examination of a child
    - First try to make a bond

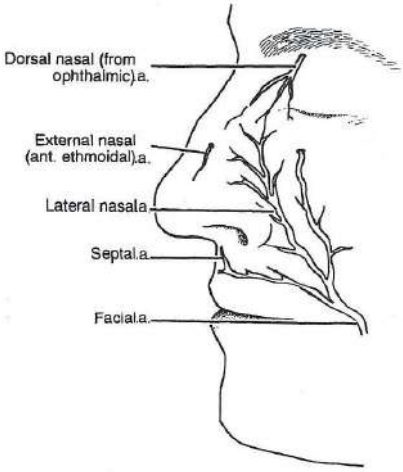
Normal anatomy (also under anatomy and physiology of the nose and PNS)

Nose – External



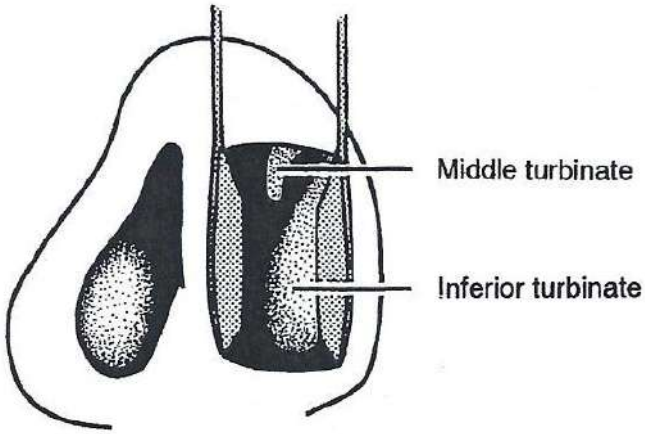


Innervation of the skin of the nose



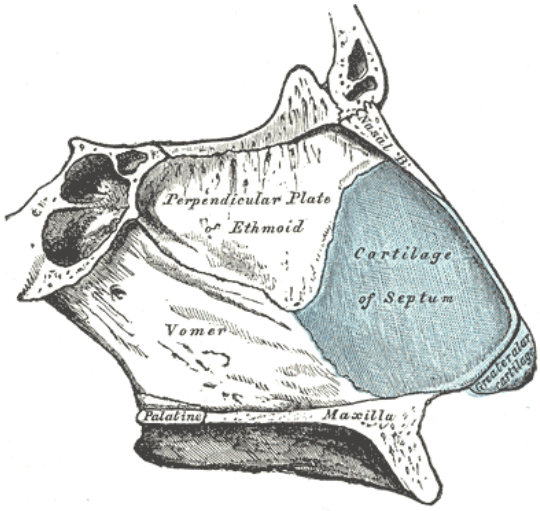
Chief arteries about the external nose

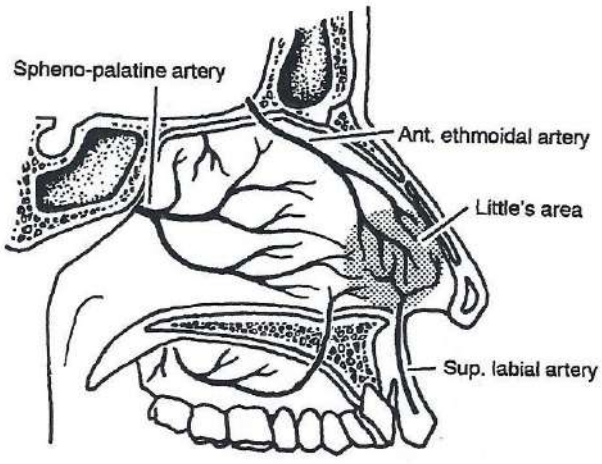
Nose – Bottom view (dog's view)



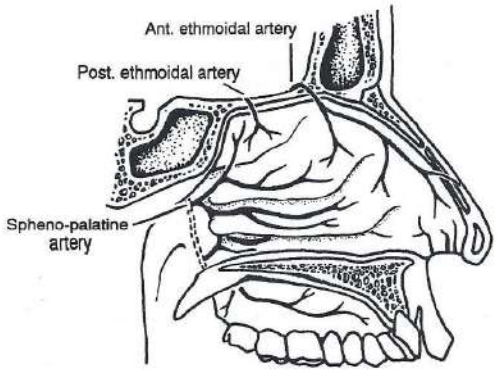
The turbinates are frequently misdiagnosed as polyps (see normal videos below).

Nose – Internal midline

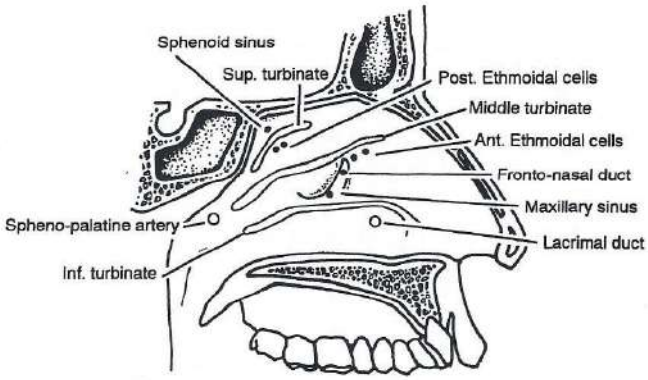




Nose – Internal lateral wall

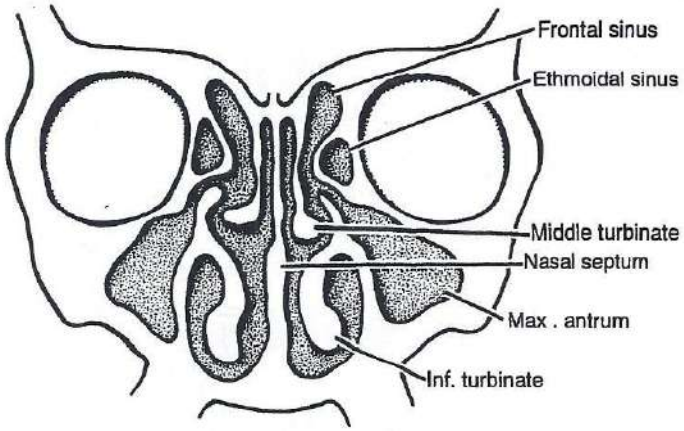


Blood supply to the lateral nasal wall



Lateral wall of nose illustrating important apertures and the attachment of the turbinates

Nose – sinus cavities and adjacent structures



Examination

- Inspection
  - Front
  - Side
  - Bottom
- Palpate
  - Skin
    - Loose over bony part
    - Fixed over cartilage part
- Structure
  - Symmetry / asymmetry
- Internal (Use a speculum or otoscope. If using an otoscope close to ventilation port on the side)
  - Vestibule
  - Septum
  - Floor of nose
  - Inferior turbinate anterior end
  - Middle turbinate anterior end – not always possible
  - Airway – space between septum and turbinates
- Function
  - Misting test
    - Use any metallic object, like a spatula, and hold it below the nostrils and check for misting
    - Bilateral misting implies that both nostrils are open

    - Maximum inspiration through nose with mouth closed
        - Look for collapse of ala and external valve
- Adjacent structures
    - Eyes
    - Sinuses
        - Percuss
    - Teeth
    - (Brain)

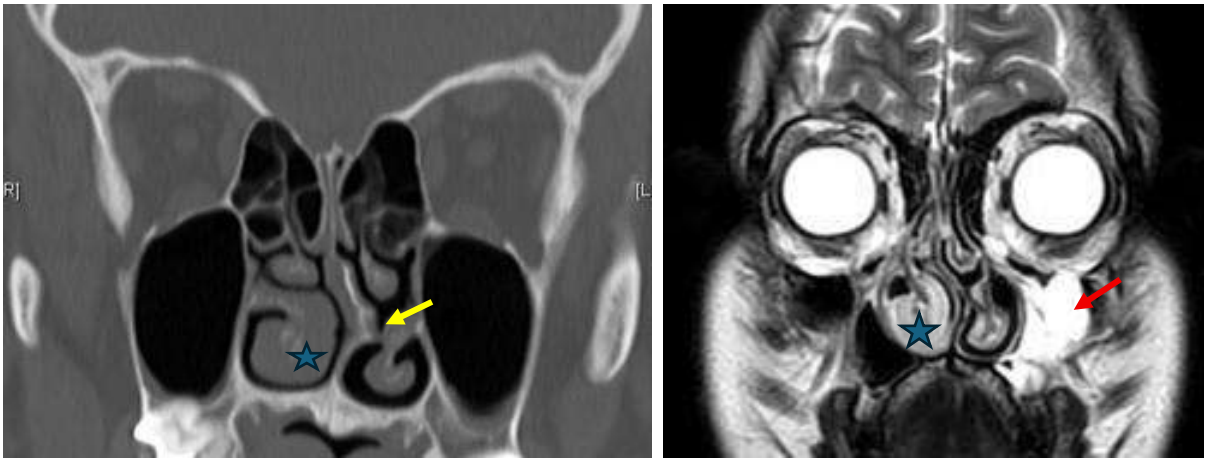
Limitation

- The nose is like an iceberg – you can only examine the external nose and a small portion of the anterior internal part
- With endoscopy an ENT can examine the nose and nasopharynx
    - The video below shows a rigid endoscopy:
        - https://youtu.be/6ttFlcb4Ybo
    - The video below shows a flexible scope:
        - https://youtu.be/tSJFTdh_hLk

Special investigations

Special investigations in patients with rhinological symptoms are frequently requested. Below follows a brief description of some of the tests:

- Allergy testing
    - Type
        - Inhalant
        - Foods
        - Preservatives
        - Colourants
    - Methods
        - Skin prick testing
        - Blood
        - In vivo (nasal stimulation)
- Radiological
    - XR
        - Mostly of no value but unfortunately frequently requested
    - Ultrasound
        - Especially for sinusitis in European countries
    - CT / Cone beam CT
        - Best for bony delineations
        - CTs are generally indicated if
            - An acute RS does not respond to medications in 48-72 hours
            - Any sign(s) of extra-sinus complications
            - Suspected tumours
            - Pre-operative
    - MRI
        - Better than CT for possible dural / brain involvement as well as nerve involvement



The image on the left shows a CT demonstrating a septal spur (yellow arrow) to the left and an enlarge inferior turbinate on the right (star). On the right is a T2 weighted MRI demonstrating a fluid filled left maxillary sinus on the left (red arrow) and also an enlarge inferior turbinate on the right (star).

3) Examination of the Head and Neck

Topics discussed:

- Review of the basic anatomy
- History
- Throat / Neck examination
    - Videos on
        - How to examine the oral cavity, oropharynx, and neck (throat and neck) (https://youtu.be/_dp8iCTsDUc)
    - Limitations – what can't you see and what can your ENT do?
        - https://youtu.be/tSJFTdh_hLk
- Special investigations

General points (applicable to any ENT examination):

- Use personal protective equipment as required
- Remember that a proper light is indispensable
    - Whether using a head light or otoscope, it should be bright
    - Preferable the ambient light should not be overly bright
- Use two hands (Bimanual)
- Be sure to examine all the nooks and crannies
- Be sensitive regarding the position in which you examine a patient
    - Sitting in front or to the side of a patient
- Be sensitive regarding the examination of a child
    - First try to make a bond

Examination follows the classically taught dictum:

- Inspection
- Palpation
- (Percussion)
- Auscultation

Remember to report using standard medical terminology such as:

- Size, Shape, Surface, Symmetry, Skin
- Consistency
- Cystic, Soft, Firm (Hard / Rubbery)
- Fixed

Basic anatomy

Oral cavity and oropharynx

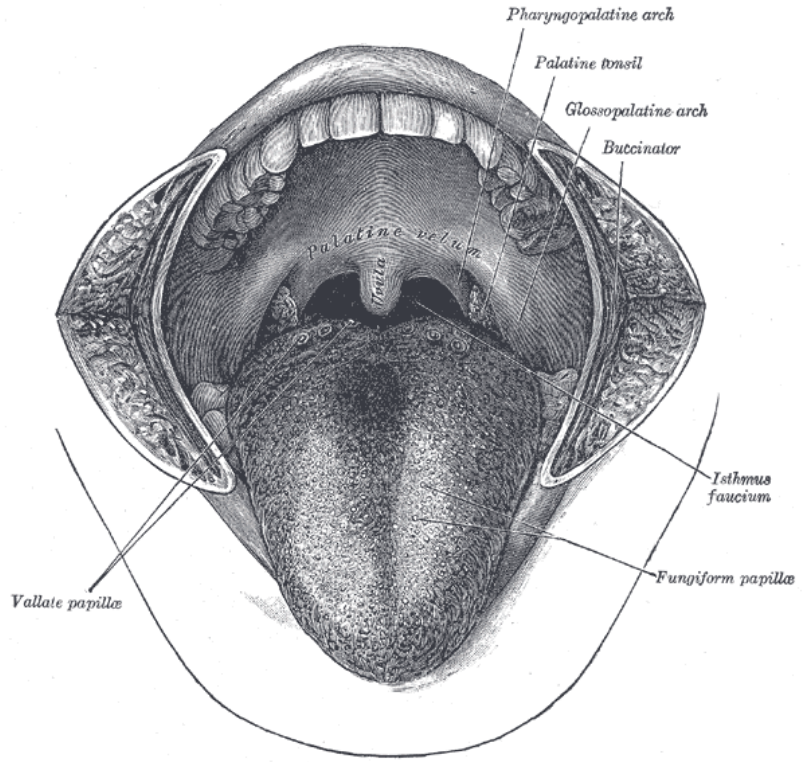
During your career, you will examine thousands of mouths / throats. A thorough history and a meticulous examination of the mouth pharynx, larynx and neck will greatly assist the doctor in making the correct diagnosis. Knowledge of a strategy for the investigation of patient complaints and a functional knowledge of the underlying anatomy is of fundamental importance. It allows the investigator to ask meaningful questions, obtain maximum benefit from clinical examinations and limits the number of invasive and expensive special investigations required. For us it is important that you establish a firm understanding of the different areas. The two areas that

you can see is the oral cavity and oropharynx. The oral cavity is divided into seven subsites and the oropharynx into four. It is not that important that you can recite this off by hand, but it will teach you to systematically "look and feel" in all the areas. The oral cavity and oropharynx are lined by stratified epithelial mucosa.

- Oral cavity – seven areas
    - Lips
    - Buccal mucosa
    - Dento-alveolar ridges (gums)
    - Floor of mouth (FOM)
    - Anterior 2/3 of the tongue
    - Hard palate
    - Retromolar trigone (coffin's corner)
- Oropharynx – four areas
    - Base of tongue – largely blind to your view
    - Palatine tonsils and anterior and posterior tonsillar pillars
    - Soft palate
    - Posterior pharyngeal wall (second and third vertebra lies behind the mucosa)

The mouth is also sometimes described as:

- The vestibule
    - Lies between the lips and cheeks, and the teeth and alveoli.
- The mouth proper
    - The space enclosed by the teeth and alveolus.



Also make a note to identify the parotid and submandibular salivary gland openings. The parotid glands open opposite the upper second molar on the buccal mucosa and the submandibular gland in the floor of the mouth. Make notes on the dental status, excessive dryness, and trismus. Areas that you can't routinely examine includes the nasopharynx, hypopharynx, and larynx. Your ENT specialist can examine these areas in a consulting

room using rigid and flexible scopes. Therefore, if a patient has new onset symptoms and signs for more than **3 weeks** in these areas that you can't examine, you need to refer them to an ENT specialist.

Remember, the oropharynx forms part of the pharynx, which is the upper part of the respiratory and digestive passages. It is about 10cm in length in the adult and extends from the base of the skull to the level of the sixth cervical vertebra, at the lower border of the cricoid cartilage. The pharynx is roughly funnel-shaped and divided, superiorly to inferior, into the nasopharynx, oropharynx, and hypopharynx (also referred to as the laryngopharynx). The nasopharynx opens anteriorly to the nose, the oropharynx to the oral cavity, and the hypopharynx to the larynx.

Subepithelial lymphoid tissue deposits are scattered widely beneath the pharyngeal mucosa. Collectively they form Waldeyer's ring. They have efferent vessels but no afferent vessels and they consist of the palatine tonsils, adenoids, lingual tonsils, tubal tonsils and multiple small discrete lymphoid nodules.

The palatine tonsils lie between the anterior and posterior pillars (palatoglossal and palatopharyngeal arches), on each side of the oropharynx. The free surface is covered by stratified, squamous epithelium. 12-15 crypts open on this surface, and each is lined with squamous epithelium. The intra-tonsillar cleft or crypta magna is the largest. The lymphoid tissue is arranged in follicles. The deep surface is separated from the constrictor muscles of the pharynx by a connective-tissue capsule. This makes complete removal by dissection possible. This is referred to as an extra-capsular tonsillectomy. Other techniques involve intra-capsular tonsillotomy options. A peri-tonsillar (Quincy) abscess also forms in this space.

The adenoid lies between the roof and upper part of the posterior wall of the nasopharynx. It is a single midline structure. The free surface exhibits about five vertical fissures. The deep surface has no capsule, therefore complete enucleation by dissection is therefore not possible. The lingual tonsils are on the base to the tongue, and often continuous with the palatine tonsils.

Lymphatic drainage is primarily to the deep cervical nodes either directly or indirectly. The retropharyngeal nodes situated between the buccopharyngeal and prevertebral fasciae are said to atrophy in childhood. Efferent vessels pass to the upper deep cervical nodes. The tonsil node also known as the jugulodigastric node is part of the upper deep cervical group and is situated around the internal jugular vein, where it is crossed by the posterior belly of the digastric muscle. The adenoids drain into the upper deep cervical nodes, either directly or indirectly through the retropharyngeal nodes. It is common for a malignancy in the post-nasal space to drain to the upper deep cervical nodes, specifically at the apex of the posterior triangle where it may be the presenting feature. The tonsil also sends efferent vessels to the upper deep cervical group. Most of them end in the jugulodigastric node. The epiglottis drains to the infrahyoid lymph nodes, and the remainder of the pharynx drains to the deep cervical nodes, either directly or indirectly through the retropharyngeal and paratracheal nodes.

Nasopharynx

The nasopharynx opens anteriorly into the nasal fossae. It is bound above by the base of the skull, below by the soft palate. The first cervical vertebra is separated from its posterior wall by the prevertebral fascia and the underlying longus capitis and cervicis muscles. The lower opening of the Eustachian Tube is situated in the lateral wall of the nasopharynx, about 1-1/2 cm behind the posterior end of the inferior turbinate. The pharyngeal recess (fossa of Rosenmuller) lies behind and above the tubal elevation. The adenoid is situated submucosally at the junction of the roof and posterior wall of the nasopharynx. The nasopharyngeal isthmus leads from the nasopharynx into the oropharynx. It is closed during swallowing by raising of the soft palate and contraction of the palatopharyngeal sphincter.

Hypopharynx

The hypopharynx opens anteriorly into the larynx through the sloping laryngeal inlet. It is bounded above by the upper border of the epiglottis, below by the lower border of the cricoid cartilage. The third, fourth, fifth and sixth cervical vertebrae lie behind it. The pyriform fossae are small recesses lying on each side of the laryngeal inlet. Each is bound by the aryepiglottic fold medially, and the thyroid cartilage and thyrohyoid membrane laterally. The internal division of the superior laryngeal nerve runs beneath the mucous membrane of its floor. The valleculae are paired shallow recesses lying between the base of the tongue anteriorly and the anterior surface of the epiglottis posteriorly.

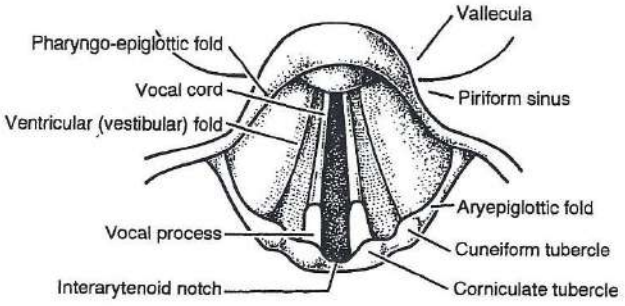
Larynx

The larynx is situated in the midline of the neck and located at the junction of the digestive and respiratory passages. It lies in front of the hypopharynx from the level of the third to the sixth cervical vertebrae. The laryngeal cartilages form the main framework of the larynx (see below). The larynx's cartilaginous framework is completed by muscles, fibrous membranes and ligaments. The boundaries are:

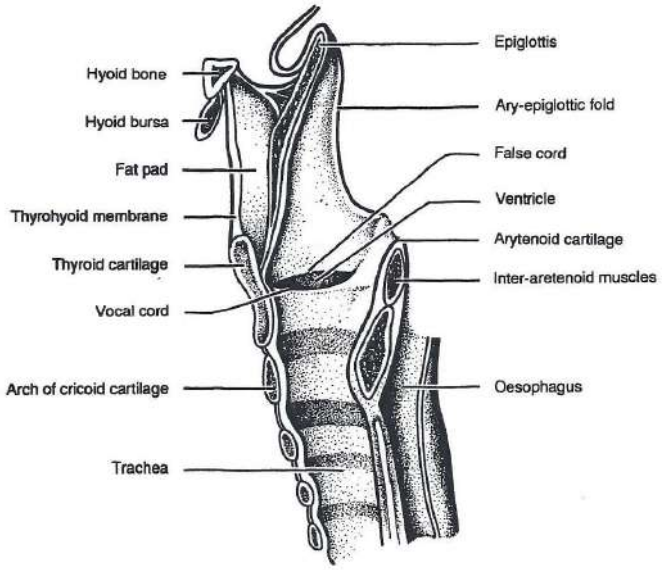
- Above - the laryngeal inlet made up of the free edge of the epiglottis, aryepiglottic folds, arytenoids and inter-arytenoid band.
- Below - the inferior border of the cricoid cartilage. The free edge of the true vocal cord on each side encloses an area called the rima glottidis, with the supraglottis above and the subglottis below.

The larynx has two important joints:

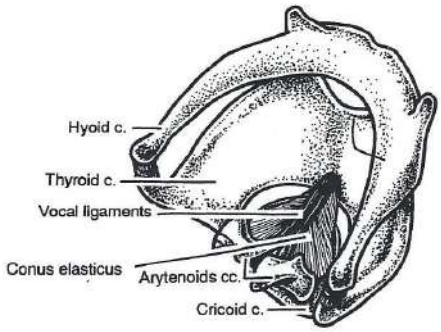
- The cricothyroid joint
  - Lies between the inferior cornu of the thyroid cartilage and the facet on the cricoid cartilage at the junction of arch with lamina.
  - It is a synovial joint with a capsular ligament.
  - Two movements occur
    - Rotation through the transverse axis.
    - Gliding movement.
- Crico-arytenoid joint
  - Lies between the base of the arytenoid cartilage and facet of the upper border of the lamina of the cricoid cartilage.
  - It is also a synovial joint with a capsular ligament.
  - Two movements occur
    - Rotation of arytenoid, on a vertical axis. The vocal process moves medially or laterally.
    - Gliding the arytenoid move forward or away from each other.



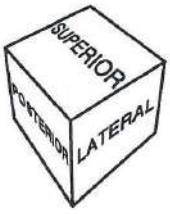
Schema of the laryngeal aditus, from above.



Section of larynx



Larynx : Perspective view

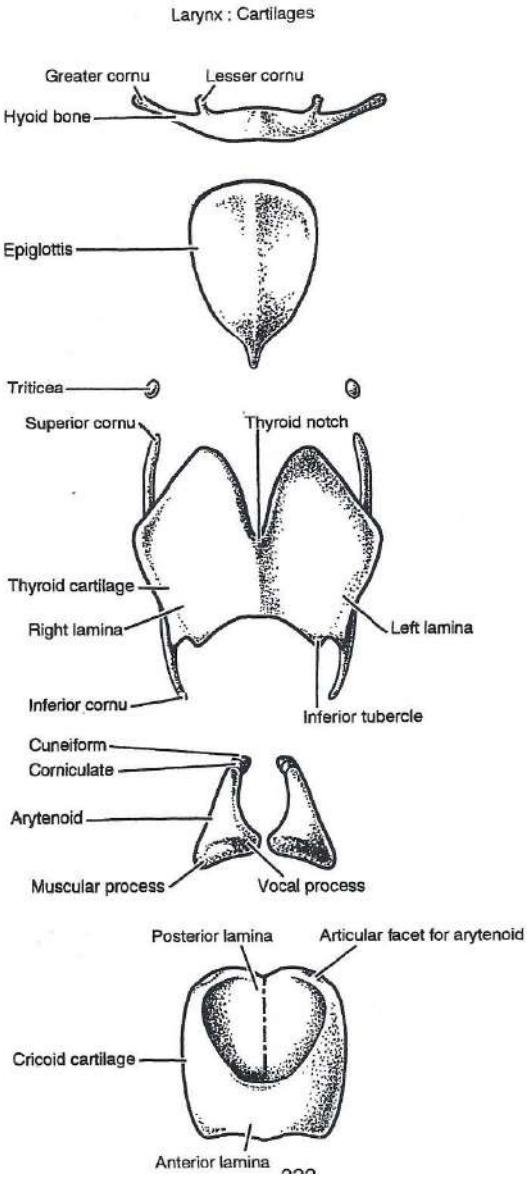


Cartilages

- Unpaired
  - Thyroid
    - Each half consist of the Ala. Medially related to the glottis & pyriform fossa. The point of junction of the upper border of the alae, is indented by the V-shaped thyroid notch. An oblique line, the site of muscular attachments, runs on the side of each lamina. There is a superior and inferior cornu. There is a facet on the inner surface of the inferior cornu for articulation with the cricoid cartilage.
  - Cricoid
    - Thicker and stronger than the thyroid cartilage. Resembles a signet ring, narrow in front, broad behind. It provides attachment for the upper fibres of the oesophagus on the posterior surface. There is a facet for articulation with the arytenoid cartilage.

- Epiglottis
  - Rises up behind the tongue. It is a thin leaf-like sheet of elastic fibrocartilage. The stem is directed downwards. It is attached to the posterior surface of the thyroid alae at their junction. The free border, directed upwards, is broad and rounded from side to side. The anterior surface is free in its upper part but is separated from the hyoid bone and thyrohyoid membrane some fatty tissue in its lower part. This is called the pre-epiglottic space. The tubercle of the epiglottis projects backwards in its lower part.

- Paired
  - Arytenoids
    - They are pyramidal in shape and have four surfaces namely posterior, anterolateral, medial and inferior. The posterior surface is triangular and concave. It extends laterally into a muscular process. The anterolateral surface is convex. It extends forwards into a vocal process. The medial surface is narrow, smooth and flat. The inferior surface or base is concave. It articulates with the cricoid cartilage. The apex curves backwards to articulate with the corniculate cartilage.
  - Corniculates
    - They articulate with the apices of the arytenoid cartilages and prolong them backwards and medially
  - Cuneiforms
    - They are small bars of yellow elastic cartilage. There is one in each aryepiglottic fold, which acts as a passive prop. They do not articulate with any other cartilage.



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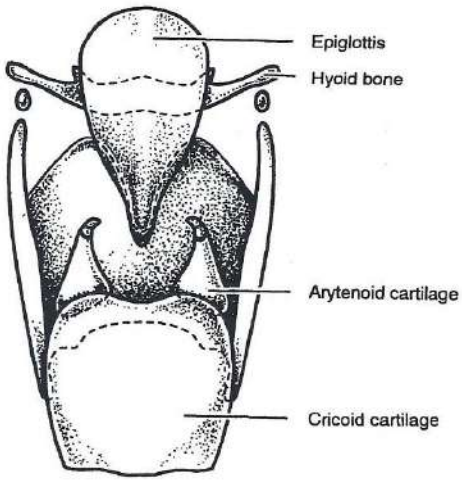
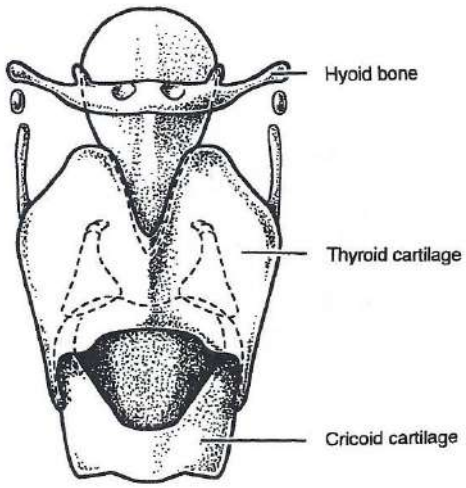


Larynx : Posterior view



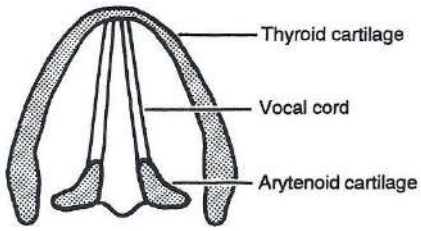
Larynx : Anterior view

Muscles

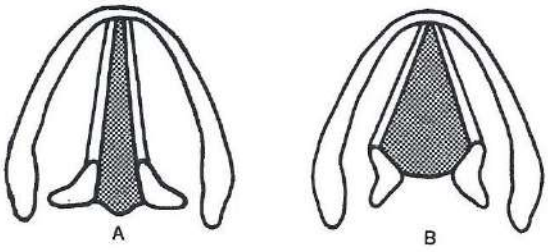
- Intrinsic
    - Abductors
        - Posterior cricoarytenoids
    - Adductors
        - Lateral cricoarytenoids
        - Interarytenoids
        - Thyroarytenoids
    - Tensors
        - Cricothyroid
        - Vocalis
- Extrinsic
    - Stylopharyngeus
    - Palatopharyngeus
    - Sternothyroid
    - Thyrohyoid

Movement of the vocal cords results from rotation or sliding of the arytenoids on the cricoid cartilages brought about by the intrinsic muscles of the larynx.

Vocal cord positions



Attachments of the vocal cords

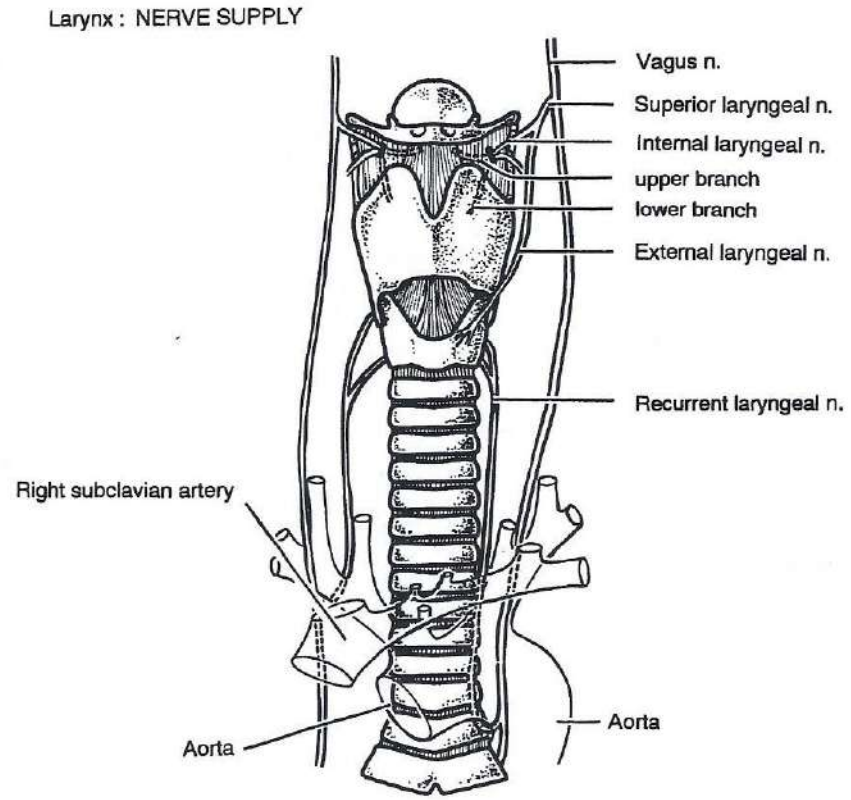


Glottis in (A) quiet respiration
(B) forced inspiration
Note : Abduction and lateral rotation of arytenoid cartilages

Nerve Supply

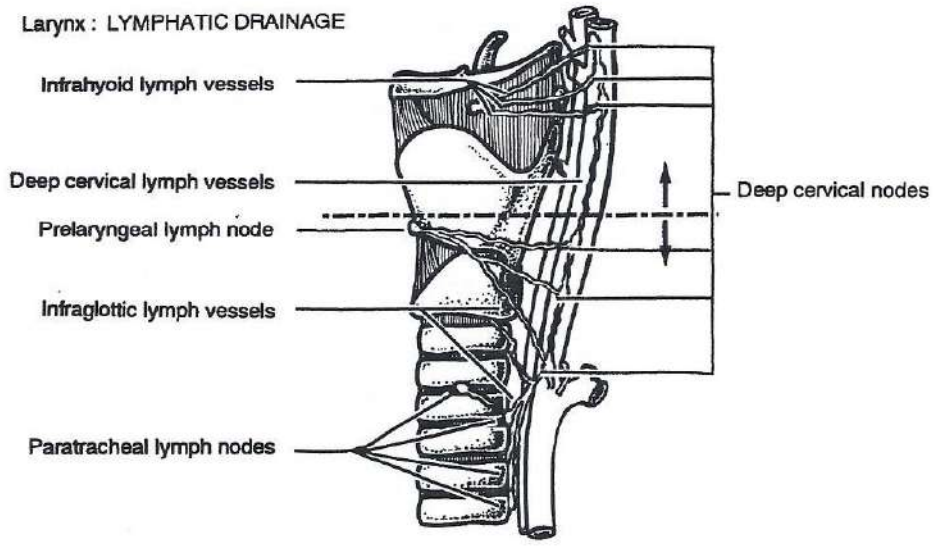
- Motor
  - All the intrinsic muscles of the larynx are supplied by the recurrent laryngeal nerves
  - EXCEPT the cricothyroids which are innervated by the external branches of the superior laryngeal nerves
- Sensory
  - Supraglottis
    - Internal branches of superior laryngeal nerve
  - Glottis and subglottis
    - Recurrent laryngeal nerves

The superior laryngeal nerve has two laryngeal branches. The internal branch which is entirely sensory and the external branch. The internal branch pierces the thyrohyoid membrane with the superior laryngeal artery and vein. It supplies the cavity of the larynx as far down as the level of the vocal cords. The external branch travels down on the inferior constrictor muscle and supplies the cricothyroid muscle.
Recurrent laryngeal nerve has a much longer course on the left side than on the right. On the left side it turns round the arch of the aorta. On the right side it turns round the subclavian artery. In the neck it lies between the trachea and oesophagus as it approaches the larynx. Its terminal part passes upwards, under cover of the ala of the thyroid cartilage, immediately behind the inferior cricothyroid joint. It then divides into an anterolateral (motor) branch which supplies all the intrinsic muscles of the larynx except the cricothyroid muscle and the posteromedial (sensory) branch supplies the cavity of the larynx below the level of the vocal cords.



Lymphatic drainage

The edges of the vocal cords divide the lymphatic system of the larynx into two parts namely the supraglottic and subglottic. The supraglottis, above the vocal cords, drain into the pre-epiglottic and upper deep cervical nodes. After piercing the thyrohyoid membrane, the vessels pass to these nodes accompany the superior thyroid artery. The subglottis, that is below the vocal cords, drain to the pre-laryngeal and pre-tracheal nodes after piercing the cricothyroid ligament. The vessels also drain to the lower deep cervical nodes after emerging from below the cricoid cartilage. The glottic region where the vocal cords lie there are practically no lymphatic vessels, this is why malignant tumours limited them do not spread readily.



Laryngeal ligaments and membranes

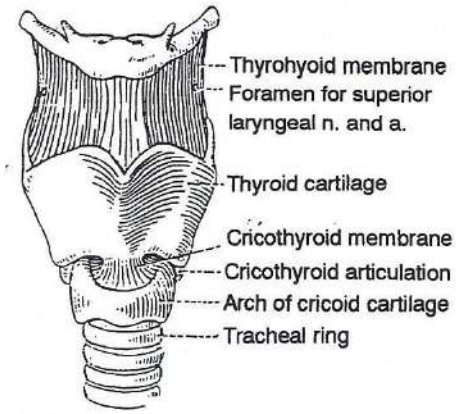
The intrinsic ligaments include the conus elasticus or cricovocal membrane, as well as the quadrangular membrane. The conus elasticus is attached in front to the deep surface of the angle of the thyroid cartilage. The second attachment is behind to the vocal process of the arytenoid cartilage. The vocal ligament is the free upper edge of the conus between these points of attachment. The quadrangular membrane runs from the lateral edges of the epiglottis to the arytenoid cartilages. Its free inferior edge forms the false cords.

The extrinsic ligaments or membranes of the larynx include the thyrohyoid membrane and cricothyroid membrane. The thyrohyoid membrane is attached to the thyroid cartilage below and the hyoid bone above. The membrane is pierced on each side by a superior laryngeal vessel and the internal branch of the superior laryngeal nerve. The triticeal cartilage is a small cartilage often found in each ligament. These are sometimes visible on x-ray and may be confused with a foreign body.

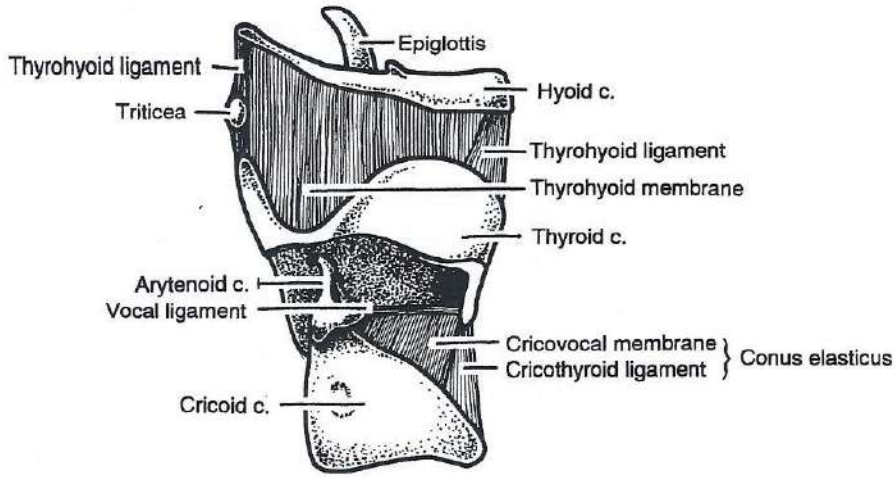
The cavity of the larynx extends from the inlet of the larynx, where it opens into the hypopharynx to the lower border of the cricoid cartilage, where it is continuous with the trachea. It is divided into three parts by two folds of mucous membrane. The false vocal cords, which are formed by the mucous membrane covering the ventricular ligament and the upper part of the external portion of the thyro-arytenoid muscle. The true vocal cords project further into the cavity than into the false cords and lie at a lower level. Parts of them can therefore be seen by inspection from above. The covering epithelium is closely bound down to the underlying vocal ligament, and the blood supply is poor, hence the pearly white appearance of the vocal cords.

The mucosal folds divide the cavity into the following parts. The vestibule, ventricle of larynx, and subglottic space. The vestibule lies between the inlet and the edges of the false cords. It is deeper in front than behind. It is bounded by the posterior surface of the epiglottis in front, the interval between the arytenoid cartilage behind, and the inner surface of the aryepiglottic folds and upper surfaces of the false cord on each side. The pre-epiglottic space is a wedge-shaped space lying in front of the epiglottis and bounded anteriorly by the thyrohyoid membrane and hyoid bone. It is bounded above by a deep layer of fascia connecting epiglottis to the hyoid bone. This is called the hyo-epiglottic ligament. The ventricle of the larynx is a recess between the false and true vocal cords. Numerous mucous glands open on to the surface of its lining mucosa. The glottis (rima glottidis) is the interval between the true vocal cords in its anterior three-fifths and the vocal processes of the arytenoid cartilages in its posterior two-fifths (and 1 cm inferiorly). Its average length in the adult male is about 2.5 cm, and in the adult female about 1.6 cm. The subglottic space lies between the true vocal cords (and 1 cm inferiorly) and the lower border of the cricoid cartilage.

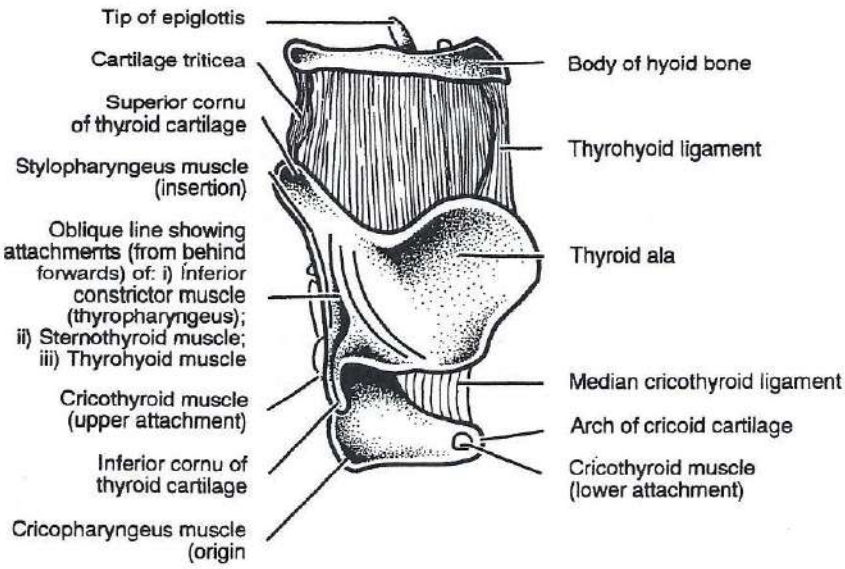
A mucous membrane lines the whole cavity. It is tightly attached to the walls over the true vocal cords, the epiglottis, and the cartilages of Santorini (corniculate cartilages) and Wrisberg (cuneiform cartilages). Elsewhere is it loosely attached and therefore liable to become swollen from effusion. Reinke's layer of connective tissue lies immediately under the epithelium of the glottis and superficial to the elastic layer. There are no glands beneath it and no lymph vessels in it. Stratified squamous epithelium is found over the vocal cords and other parts of the vestibule of the larynx. Ciliated columnar epithelium lines the remainder of the cavity. This transition zone between stratified squamous epithelium and ciliated columnar epithelium is often when laryngeal papillomatosis occurs.



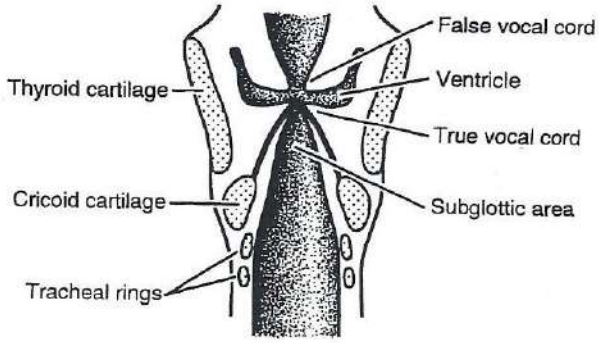
Cartilages of the larynx from the front



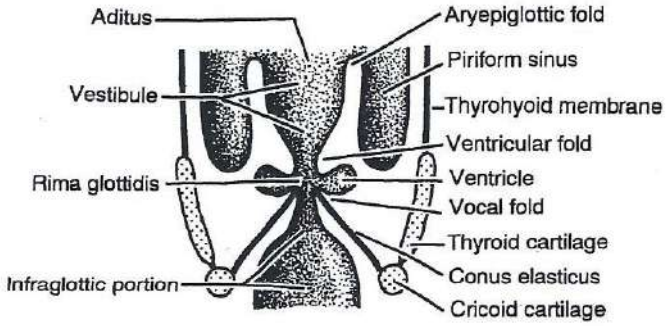
Larynx : Lateral view
Right lower lamina of thyroid removed



Cartilages of larynx and hyoid bone - lateral view



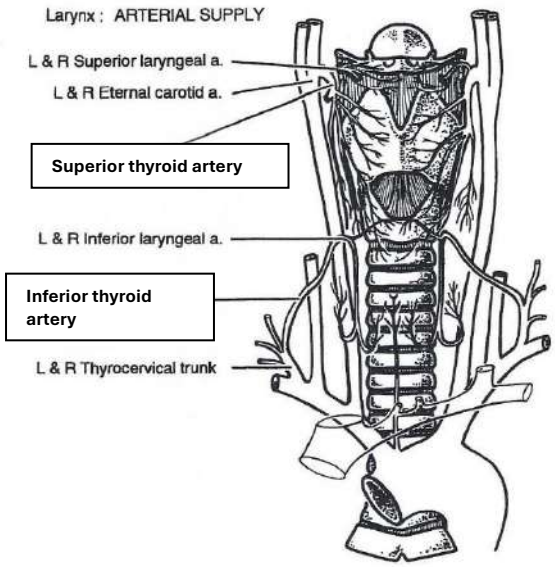
Longitudinal section through larynx.
Oedema of the subglottic area follows laryngeal infection thereby compromising the airway.



Cavity of the larynx and its subdivisions in a frontal section.

Blood supply

Blood supply is via laryngeal branches of superior thyroid artery, laryngeal branches of inferior thyroid artery, and cricothyroid branches of superior thyroid artery. Veins accompany the arteries.

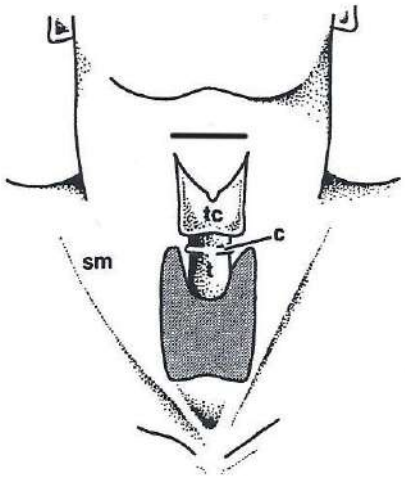


The infantile larynx.

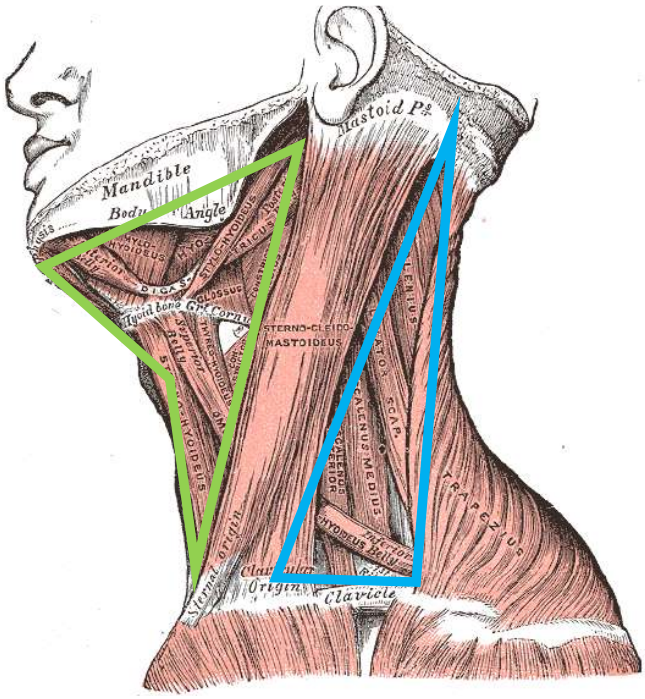
The larynx is both absolutely and relatively smaller. The lumen is therefore disproportionately narrower. The shape of the infant larynx is more funnel shaped. Its narrowest part is at the junction of the subglottic larynx with the trachea. A very slight swelling of the lax mucosa at this point may produce serious obstruction to breathing. The consistency of the laryngeal cartilages is much softer in the infant. They therefore collapse more easily in forced inspiratory efforts or oedematous conditions. The position of the infantile larynx lies high up under the tongue, but with development assumes an increasingly lower position. The plane of its inlet is less oblique, and the axis of air entry is straighter than in the adult.

Neck

As with the oral cavity and oropharynx, basic anatomy is very important in the neck. Firstly, think about the constant landmarks palpable in the anterior midline of the neck. If we move down from the mandible, you encounter the hyoid bone, thyroid cartilage, cricoid cartilage, thyroid gland, and the supra-sternal notch. To the sides are the two prominent sternocleidomastoid (SCM) muscles. The area below the mandible superiorly, SCM muscle laterally, and the midline is known as the anterior triangle on each side. The area between the SCM anteriorly, trapezius (TPZ) posteriorly, and clavicle inferiorly is known as the posterior triangle on both sides.



Position of the thyroid gland c. cricoid: t. trachea: tc. thyroid cartilage: sm sternomastoid muscle

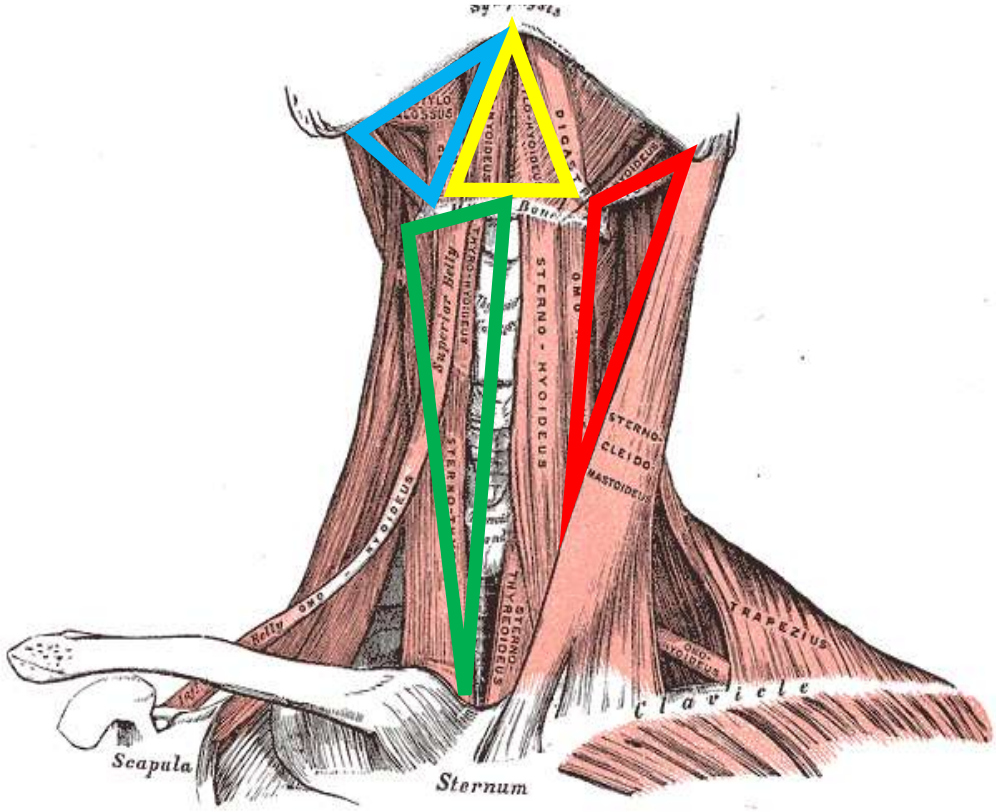


(Green – anterior triangle; Blue – posterior triangle)

The anterior triangle can be divided into:

- Submental triangle

- Submandibular triangle
- Muscular triangle
- Carotid triangle



(Yellow – submental triangle; Blue – submandibular triangle; Red – carotid triangle; Green – muscular triangle)

In head and neck cancer surgery, we use levels which will briefly be discussed here. Also remember that in trauma surgery, they divide the neck into three zones. Neck levels refer to specific lymph nodes in a defined anatomical boundary. Remember, the lymph nodes are in the fat and neck dissections removes the fat with the lymph nodes of that specific level. (See more of this under head and neck cancers)

Neck levels:

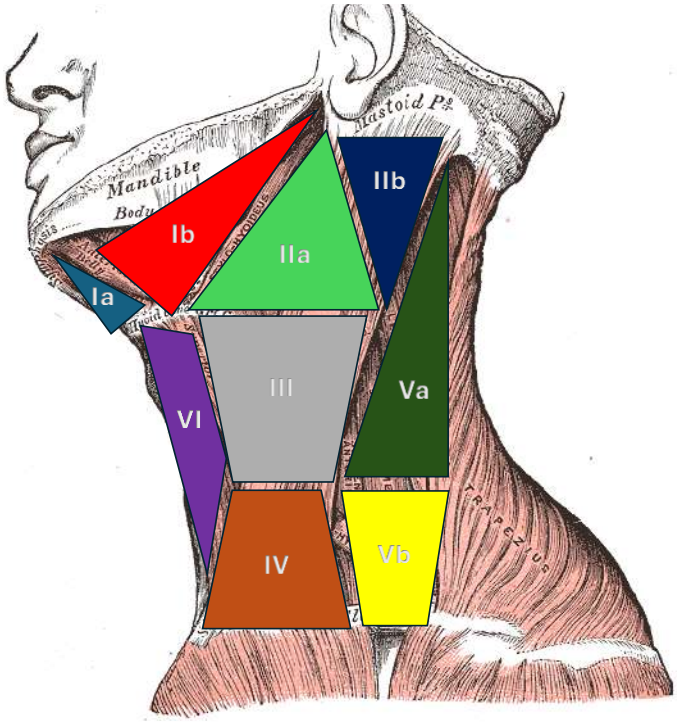
Level	Clinical name	Boundaries	
Ia	Submental	S	Mandible
		I	Hyoid bone
		L	Anterior belly digastric muscle
		L	Contralateral anterior belly of digastric muscle
Ib	Submandibular	S	Mandible
		I	Posterior belly digastric muscle
		A	Anterior belly digastric muscle
		P	Stylohyoid muscle
IIa	Upper jugular	S	Skull base
		I	Horizontal line at level of hyoid bone
		A	Stylohyoid muscle
		P	NXI nerve
IIb	Upper jugular	S	Skull base
		I	Horizontal line at level of hyoid bone

		A	NXI nerve
		P	Posterior border of SCM
III	Middle jugular	S	Horizontal line at level of hyoid bone
		I	Horizontal line at level of cricoid bone
		A	Sternohyoid muscle
		P	Posterior border of SCM
IV	Lower jugular	S	Horizontal line at level of cricoid bone
		I	Clavicle
		A	Sternothyroid
		P	Posterior border of SCM
Va	Posterior triangle	S	Skull base
		I	Horizontal line at level of cricoid bone
		A	Posterior border of SCM
		P	Anterior border of TPZ
Vb	Posterior triangle	S	Horizontal line at level of cricoid bone
		I	Clavicle
		A	Posterior border of SCM
		P	Anterior border of TPZ
VI	Anterior compartment	S	Hyoid bone
		I	Sternal notch
		L	Common carotid artery
		L	Common carotid artery
VII	Superior mediastinum	S	Sternal notch
		I	Innominate artery
		L	Common carotid artery
		L	Common carotid artery

S – superior; I – inferior; A – anterior; P – posterior; L – lateral
(*There is some more detail than what is given above, but at GP level this is more than adequate)

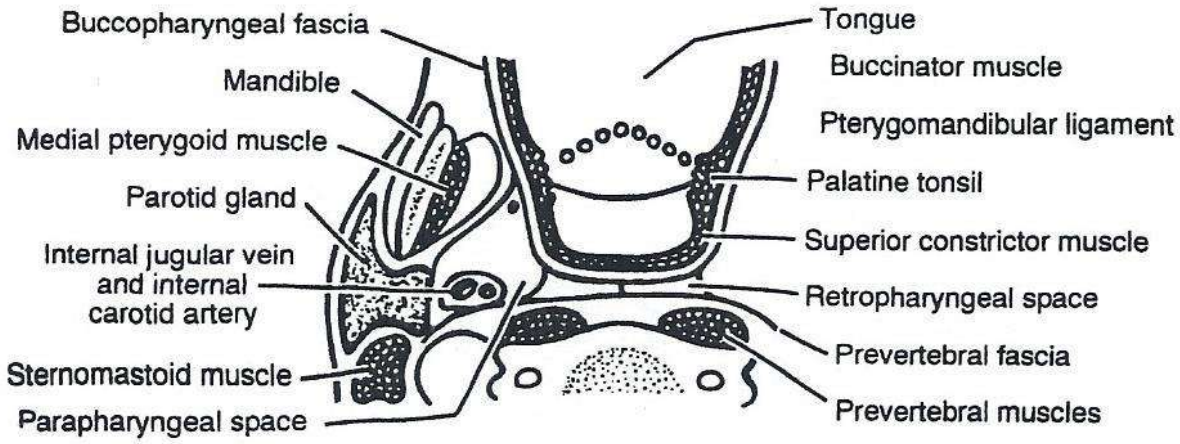
The posterior part of the neck is referred to as the suboccipital area.

The picture below shows the level in relation to the neck.



The neck contents can be studied from an anatomical textbook, but important concepts are:

- The investing fascial layers
- Parapharyngeal space
    - Pre- and post-styloid
    - Carotid compartment
        - Carotid artery
        - Jugular vein
        - Vagal nerve
- Lower cranial nerves
- Cervical plexus
- Brachial plexus



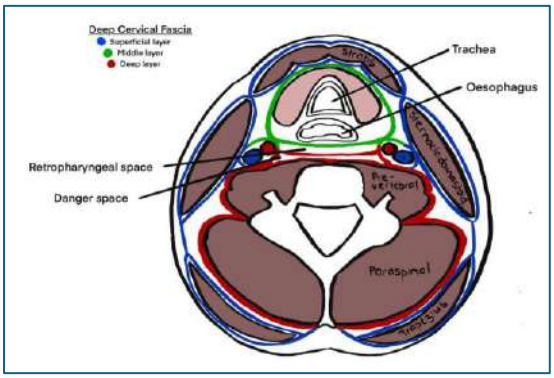
Fascial compartments of neck at level of C.2

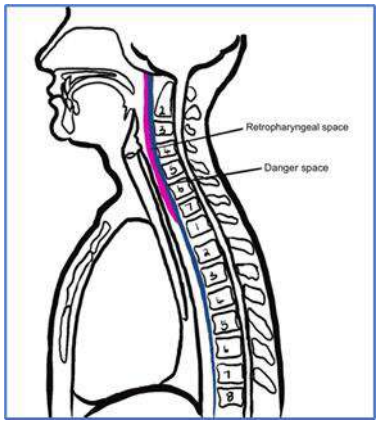
Investing layers and potential spaces

Deep cervical fascia
This completely encircles the neck. It lies deep to the platysma muscle. It splits to enclose the trapezius and sternomastoid muscles and the parotid and submandibular glands.
Prevertebral fascia
This lies on the prevertebral musculature. The brachial plexus and subclavian artery lie deep to it and acquire a sheath from it which becomes the axillary sheath surrounding the branchial plexus, axillary artery and vein.
Pretracheal fascia
This extends from the hyoid to the aortic arch and envelopes the thyroid gland. It fuses loosely laterally with the carotid sheath.

The retropharyngeal space (RPS) is a potential space that lies posterior to the pharynx and oesophagus and anterior to the pre-vertebral muscles. Posterior to this is another potential space called the "danger space". The two spaces are differentiated by their lower limit. The RPS extends from the skull base to the level of C7-T1, whereas the "danger space" extends from the skull base to the diaphragm. The anterior and posterior boundaries of the RPS are the middle and alar layers of the deep cervical fascia respectively, and the lateral limit is the carotid sheath.

Upper respiratory tract infections may spread to the retropharyngeal lymph nodes via capillary lymphatic drainage pathways. Infection begins with the pre-suppurative phase with enlarged reactive lymph nodes. Progression leads to the suppurative phase with liquefactive necrosis and surrounding oedema. Finally, nodal breakdown may result in spread of infection into the surrounding space with retropharyngeal abscess formation.





Lymph nodes

Lymph nodes are discussed in the head and neck section. Here we will be discussing the important concept with regards to the drainage pattern of lymph nodes. Whether the aetiology is infective of neoplastic, by establishing the position of the affected lymph node(s), one is prompted to examine the primary area that drains to those lymph nodes. The site and clinical names have been discussed above, so the drainage patterns are shown in the pictures below.

History

Symptoms relate to the site / organ affected. In general, common symptoms of pathologies in the head and neck area include:

- Asymptomatic lump / mass
- Voice changes (larynx)
- Aspiration (larynx)

- Dysphagia / Odynophagia (hypopharynx or oesophagus)
- Nasal obstructions (nasopharynx)
- Epistaxis (nasopharynx)
- Bleeding (rare)
- Pain
- Referred pain
    - Especially otalgia in adults with hypopharyngeal / laryngeal cancers
- Ulcers in mouth (common)
- Loss of weight

Always think in term of chronology, aetiology (infective, neoplastic, congenital etc.), constant versus episodic. Always enquire about risk factors in head and neck cancers. Smoking and alcohol use is the most important, but please see the head and neck cancer section.

Examination

Perform a thorough examination of the oral cavity and oropharynx (remember the subsites listed under basic anatomy). Use two spatulas and a head light. Be careful to not to elicit a gag reflex when putting the spatula too deep. A useful trick is to first place it gently onto the oral tongue and have the patient breath through their mouth. If this does not relax the base of tongue (BOT), gently pinch the nostrils close. This will force the patient to breathe through their mouth and will facilitate the inspection of the oropharynx (use this even in children when examining the tonsils). Look into both floor of mouth ("gutters") and anterior floor of mouth with the openings of the submandibular salivary glands (Wharton's ducts). The sublingual glands also open into the floor of the mouth with multiple small openings. The "gutter" is the space between the under surface of the oral tongue and the dento-alveolar ridges (all the way posteriorly) and a common place for cancers to develop. Lift the cheek laterally and inspect the opening of the parotid ducts (Stenson's duct), buccal mucosa, and the retromolar trigone area. The retromolar trigone is known as coffin's corner because of the late presentation and poor outcomes for patients with cancer there. The easiest way to think of the retromolar trigone is the mucosa posterior to the last maxillary and mandibular molars, over the mandibular ramus (medial and lateral). Remember that advanced cancers here typically present with trismus.

Next, perform a bimanual examination of all the areas, especially the base of tongue area. Also palpate both submandibular areas (with submandibular salivary gland) with one finger inside the floor of mouth and the other on the outside. As a general rule, with regards to head and neck cancers, any lump you can feel between your fingers is already bigger than 1 cm. Remember that some cancers are mainly submucosally, and if you don't feel with your fingers you are going to miss it.

The neck is inspected from the front, and depending on pathology, a patient is asked to swallow and / or protrude their tongues to elicit signs (see thyroid / thyroglossal duct cyst). A brief palpation can be performed from the front, but classically the neck is examined by standing behind the patient. Examine the neck systematically, ask the patient to gently tilt their neck forward and sometimes it helps to turn the neck ever so slightly to the side being examined. Establish your own preference, either from superior to inferior, midline to lateral, or variations. Also auscultate the neck for possible bruits.

See the video below on how to examine the head and neck area.
https://youtu.be/_dp8iCTsDUc

The limitations of what you can't see include the:

- Nasopharynx

- Larynx
- Hypopharynx and oesophagus

The videos below show how easy it is for ENTs to examine these patients in the rooms.
Flexible endoscopy ending with a view of the larynx– https://youtu.be/tSJFTdh_hLk
Rigid endoscopy showing inferior and middle turbinate and ending with the nasopharynx - https://youtu.be/6ttFlcb4Ybo

The last video shows a patient with cancer in the BOT / gutter of the FOM. It is easy to miss if you fail to examine the "gutter" all the way posteriorly. REMEMBER that you need to specifically examine all the nooks and crannies in the oral cavity and oropharynx!

Retromolar trigone cancer - https://youtube.com/shorts/uK81776G0cw

Special investigations

Various special investigations can be ordered in head and neck pathologies. The more common ones include:

- Radiology
  - CXR
  - CT
  - MRI
  - PET-CT
  - Ultrasound
  - Barium swallow
  - Angiography
- Histology / Cytology
  - Biopsies
  - FNAC and cell blocks
  - Flow cytometry (lymphomas)
- Blood investigations
- Laryngeal investigations
  - Stroboscope
  - High speed video photography
  - Ultrasound

Although not truly a special investigation, in head and neck cancer we need to establish a performance status of the patient. Patients on the lower end of the performance scales are usually not offered curative options for a variety of reasons. Any curative treatment, whether surgery or chemo/radiotherapy, is intensive with substantial physiological demands, carrying substantial side effects and imposing considerable strain on an already compromised system. Some of the more common scales are shown below.

ECOG		**WHO**		**Karnofsky**	
0	Fully active, able to carry on all pre-disease performance without restriction	0	Normal	100	Normal, no complaints, no signs of disease
1	Restricted in physically strenuous activity	1		90	Capable of normal activity, few symptoms or signs of disease

	ambulatory and able to carry out work of a light or sedentary nature, e.g., light housework, office work		Symptoms but nearly fully ambulatory	80	Normal activity with some difficulty, some symptoms or signs
2	Ambulatory and capable of all selfcare but unable to carry out any work activities. Up and about more than 50% of waking hours	2	Some bedtime but needs to be in bed< 50% of normal daytime	70	Caring for self, not capable of normal activity or work
				60	Requiring some help, can take care of most personal requirements
3	Capable of only limited selfcare, confined to bed or chair more than 50% of waking hours	3	Needs to be in bed > 50% of normal daytime	50	Requires help often, requires frequent medical care
				40	Disabled, requires special care and help
4	Completely disabled. Cannot carry on any selfcare. Totally confined to bed or chair	4	Unable to get out of bed	30	Severely disabled, hospital admission indicated but no risk of death
				20	Very ill, urgently requiring admission, requires supportive measures or treatment
				10	Moribund, rapidly progressive fatal disease processes
5	Death	(5)	Death	0	Death

Charlson comorbidity index	**+1**	**+1**	**+2**	**+3**	**+4**	**+6**
	50-59	MI	60-69	70-79	>80	AIDS
	CHF	COPD	Localized solid tumour	Moderate - severe liver disease		Metastatic solid tumour
	Peripheral vascular disease	Connective tissue disorder	DM with end organ damage			
	CVA / TIA	Peptic ulcer	Hemiplegia			
	Mild liver disease	Uncomplicated DM	Chronic kidney disease			
	Dementia		Lymphoma			
			Leukaemia		**TOTAL:**	

4) The Past, Present, and Future of ENT

Otorhinolaryngology is a product of the early 20th century, when the disciplines of otology and laryngology were joined together. Otologists were surgeons and laryngologists were physicians dealing with throat and chest problems. The first clinic was established in 1870 in Vienna. Rhinology became part of the discipline during the early 1900's

Worldwide milestones in ENT

Important dates – Otology

- 500 BC – Hippocrates refers to the tympanic membrane as a "thin web"
- 1649 – Mastoidectomy
- 1724 – Eustachian tube dilatation
- 1860 – Mumps association with deafness
- 1861 – Meniere's syndrome
- 1867 – Myringotomy – Politzer
- 1878 – Stapes mobilization – Kessel
- 1900 – First hearing aid
- 1901 – Vestibular nerve sectioning – Perry
- 1912 – Tympanoplasty
- 1920 – Otoplasty
- 1921 – Monocular microscope
- 1932 – Facial nerve decompression
- 1940 – Electrical burr
- 1953 – Microscope – Zeiss
- 1954 – Grommet
- 1960's – Stapedectomy
- 1967 – ABR
- 1977 – First cochlear implant - Brazil
- 1978 – OAE's

Important dates – Laryngology

- 3600 BC – Tracheostomy
- 350 BC – Aristotle – Larynx and the windmill
- 350 BC – Alexander the great – tracheotomy
- 240 – Tonsillectomy / Quincy – Cornelius Celsus
- 1854 – Mirror laryngoscopy – Manuel Garcia
- 1860 – Laryngeal fissure
- 1873 – Laryngectomy – Billroth
- 1906 – Thompson – association between laryngeal cancer and smoking
- 1900's – Guillotine tonsillectomy
- 1954 – Fibre optic scope – Hopkins
- 1998 – Laryngeal transplant

Important dates – Rhinology

- 3500 BC – Sekheténach – King Sahura – Made his nostrils well
- 3000 BC – Egyptians – removing the brain through the nose as part of mummification
- 600 BC – Hindus – nasal reconstruction

- 500 BC – Hippocrates – detailed description of nasal trauma and remedies. Also the first to remove polyps transorally
- 1489 – Da Vinci – detailed drawings
- 1597 – Treaty of Rhinoplasty – Tagliacozzi
- 1765 – attempt at a maxillary antrostomy
- 1768 – opening of the maxillary sinus through the oral cavity or tooth socket
- 1886 – opening of the maxillary sinus through the inferior meatus
- 1893 – through the canine fossa
- 1900's – decline in rhinology surgery due to antibiotics
- 1912 – trans sphenoid approach – Harvey Cushing
- 1950's – Messerklinger / Stammberger – endoscopic nasal surgery
- 1969 – CT scan

Important dates – Other

- 1846 – Anaesthesia
- 1867 – Antisepsis
- 1879 – Thomas Edison – light
- 1903 – Radiation
- 1950's – Chemotherapy

In general, ENT was

- First to use local anaesthesia
- First to use microscopes and endoscopes
- Pioneer in using prosthesis

ENT mentioned in

- Conception of Virgin Mary arose from the breath of the Holy Ghost in her ear
- 3 Feb – Benediction of the throat in memory of St. Blaise
    - "Piece of bone or thorn, whatever thou are, just as Jesus Christ caused Lazarus to come forth from the tomb and Jonah from the belly of the whale (patient should be seized by the throat) in the name of St. Blaise, martyr and servant of Christ, I order thee to come up or go down."
- Folklore
    - Size of the ears / nose
    - Tingling of the ears
    - Shakespeare believed that one can be poisoned through the ear
- Fabricius - 1560
    - "Of all the surgical operations which are performed on man for the preservation of his life by the physician, I have always judged to be the foremost that by which man is recalled from a quick death to a sudden repossession of life, a feat which raises the surgeon nearest to the level of Æscalapius; the operation is the opening of the aspera arteria, by which patients, from a condition of almost suffocating obstruction to respiration, suddenly regain consciousness, and draw again into their heart and lungs that vital ether, the air, so necessary to life, and again resume an existence which had been all but annihilated."

Presently, otorhinolaryngology has met other disciplines as it expands into "other territories", leading to subspecialties such as:

- Neuro-otology

- Skull base surgery
    - Lateral
    - Anterior
- Facial plastics
- Head and neck surgery – cancer (robotics included)
- Paediatric ENT
- Aesthetic surgery
- Immunology
- Reconstructive surgery (flaps)

Therefore, ENT as a speciality is certainly expanding its boundaries. Studies have also proven that 30-40% of GP consultations are ENT related. ENT knowledge and training is of vital importance. Despite this, ENT remains one of the smaller blocks taught at undergraduate level. Furthermore, opportunities to upskill in ENT are limited with most referral / draining hospital not having dedicated ENT divisions.

The future of ENT will see the incorporation of AI models to more accurately predict nodal metastases, hearing restoration using DNA / RNA vector viruses, and more use of robotics such as in cochlear implants.

ENT at Stellenbosch University / Tygerberg Hospital

The Division of Otorhinolaryngology was established in 1956 in the hospital named after Dr Karl Bremer, who was himself an ear, nose and throat specialist who had formerly practiced in Cape Town.

As was the case with all the other divisions, the ENT division had quite humble beginnings with part-time staff as its only resource. The first head, Dr Harry Wykerd, was a part-timer who practiced in Cape Town, and he was assisted in the beginning by Dr Jack de Villiers, another private specialist from Cape Town.

Professor Carel du Toit was the first full-time head of the division. He was appointed in 1973 and retired from the post in 1981. The first Registrar in the Division, Dr P. Olivier, later settled in Port Elizabeth and he was followed in 1961 by Dr PK de Villiers. Dr de Villiers later became the head of ENT at the State Hospital in Windhoek, Namibia. The third Registrar was Dr Johnny Nell who completed his training under Prof Carel du Toit and later joined the practice of Prof Du Toit which, interestingly, was also the former practice of Dr Karl Bremer. Dr Nell was the first ENT specialist in the Northern suburbs of Cape Town.

Prof Carl du Toit, first Head, 1973 – 1981
Ahead of his time, Professor Carel du Toit had the vision to start a programme for hearing impaired children to develop their residual hearing to enable them to develop spoken language and enter mainstream education. He undertook several study trips overseas to acquire information to develop a model for such a programme. He was influenced by the work of Edith Whetnall in the UK, Dr Roskjawer in Denmark and especially by the Central Institute for the Deaf in Missouri, USA. In order to achieve this, he had special training organized overseas for three individuals, viz. Lida Müller, Frikkie van Zyl and Izelle Heunis. Together they started the Hearing and Speech Clinic at Tygerberg Hospital where hearing impaired children and adults were seen. The Paedo-Audiology Unit was developed under Mr Frikkie Van Zyl as part of this Clinic, and it established an early screening programme in the Cape Province that was conducted by public health nurses. Most significantly, Professor Du Toit started the pre-school for hearing impaired children at Tygerberg Hospital, which was later, in 1986, was named after him. The children at the school were trained in the auditory-oral method to facilitate spoken language development. Izelle Heunis was the first teacher, and she set up a parent guidance programme. The posts for the teachers at this school were jointly funded by the Health and Education Departments. Prof Du Toit was also instrumental in setting up the Acoustic Laboratory, under Prof Guelke, and this laboratory focused on research into the physiology of hearing and in fitting and maintaining hearing aids.

Prof Derrick Wagenfeld (1982 - 1987) was the next head and he had the foresight to establish, in association with Mrs Lida Müller, the First Cochlear Implant Programme in South Africa in the Dept of Otorhinolaryngology at Tygerberg Hospital. This programme has gained international recognition. Its first multi-channel cochlear implant was done in 1986 and since then 264 adults and children have received a cochlear implant. Prof Wagenfeld continues to be involved in this Programme. Prof Wagenfeld was a charismatic and astute Head of department with a sharp intellect and skills in all three ENT disciplines, including Head and Neck.

Prof Johan (Zan) Reyneke, Head of the ENT division from 1988 to 1994, continued the interest in Otology, and he focused particularly on the teaching of both undergraduate and postgraduate students. He proved to be a fine educator and led many a young colleague to discover his/her full potential. His efforts ensured that the teaching of undergraduate ENT in the final clinical year, came to full fruition. Prof Reyneke was beloved by all his students and by his registrars. His dedication to education is appropriately symbolized in the special seminar room he established for academic meetings in the Department. He successfully negotiated for the establishment of a single G5 ward in place of the separate West (“White”) and East (“Non- White”) wards in 1990.

Prof R T (Theo) Gregor, the fourth Head, was appointed in 1995 and retired in 2000. He had a special interest special in Head and Neck Surgery. With his excellent surgical skills and insight into cancer of the head and neck, as well as an outgoing personality and international connections, Prof Gregor significantly advanced the reputation of the Division. Under his leadership, the scientific publications of the Division flourished.

Prof James Loock was the Head from 2001 to 2022. He first joined the Division under Professor Reyneke and has interests in Otology, hearing and paediatric airway surgery. He runs an annual Cape Temporal Bone Course in association with Professor George Browning of Scotland, who has become a good friend to the Department.

The current Head is Dr J Grobbelaar.

5) AB stewardship in ENT

Antimicrobial Resistance: A crisis

Antibiotic resistance is a global health threat. Antimicrobial-resistant infections currently claim 700,000 lives a year globally. By 2050, antimicrobial resistance could result in a loss of 10 million lives a year. A greater mortality than that of cancer (Reference: The Review on Antimicrobial Resistance: Tracking Drug-resistant infections globally: Final report and recommendations O'Neill J. May 2016. Antimicrobial stewardship across 47 South African hospitals: an implementation study. Brink AJ. et al. *Lancet Infect Dis* 2016; 16: 1017–25)

Below shows a trend of AB use in South Africa

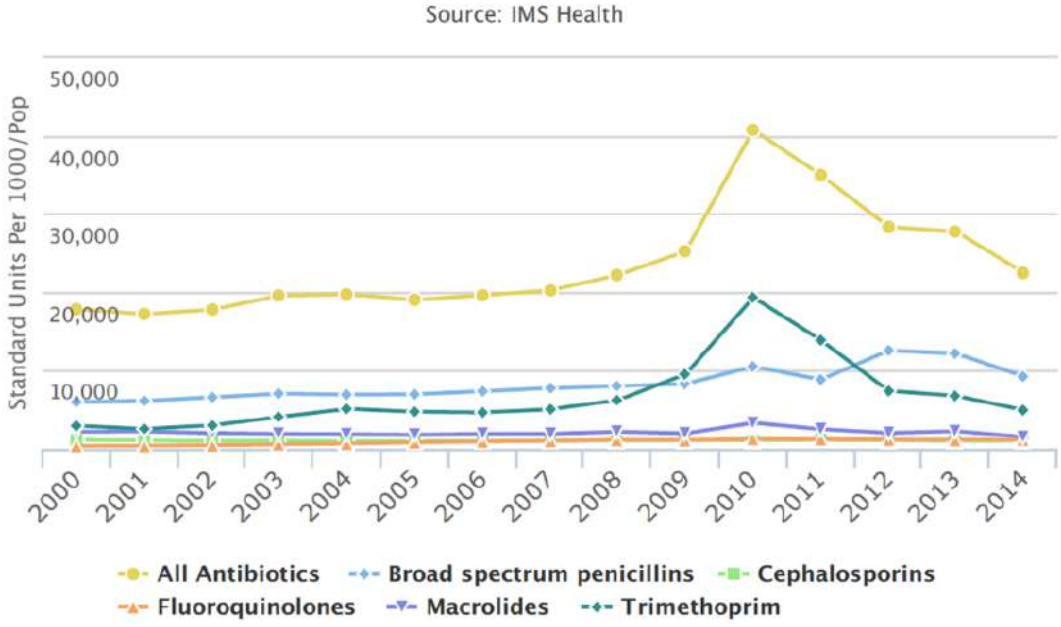
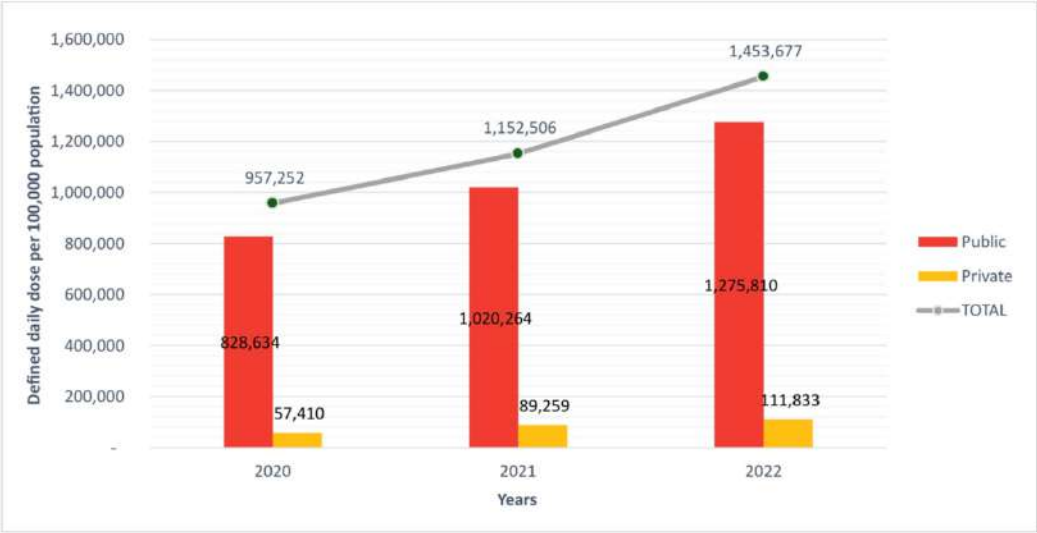


Figure 19: *Total antibiotic consumption and procurement results for South Africa by sector in DDD/100,000 population.*



(Reference: 1. Antibiotic Use in South Africa, Center for Disease Dynamics, Economics & Policy. https://resistancemap.cddep.org/AntibioticUse.php
2. Antimicrobial Stewardship: The South African Perspective. Precious Matsoso Director General; National Department of Health; South Africa 13th November 2015. http://www.who.int/phi/implementation/Precious_Matsoso_MoH_South_Africa.pdf?ua=1)

SURVEILLANCE FOR ANTIMICROBIAL RESISTANCE and CONSUMPTION - SA, 2021
SOUTH AFRICN ANTIMICROBIAL RESISTANCE NATIONAL STRATEGY FRAMEWORK; A ONE HEALTH APPROACH 2017-2024 (https://knowledgehub.health.gov.za/)

Data below comes from the government website.

- Klebsiella pneumoniae
    - 70% Blood specimen isolates (BSIs) are non-susceptible to 3rd generation cephalosporins
    - 40% BSIs are non-susceptible to 1st generation carbapenems
- Pseudomonas aeruginosa
    - 33% BSIs are non-susceptible to carbapenems
    - 17% BSIs is non-susceptible to 3rd and 4th generation cephalosporins and to piperacillin-tazobactam
- Staphylococcus aureus
    - 17% BSIs are non-susceptible to cloxacillin (MRSA)
- Acinetobacter baumannii
    - 80% BSI are resistant to carbapenems
- Enterococcus faecalis / faecium
    - 1.3% BSIs are resistant to vancomycin
- Escherichia coli
    - 25% BSIs non-susceptible to 3rd generation cephalosporins
    - 33% BSIs are non-susceptible to ciprofloxacin

Klebsiella pneumoniae is the commonest organism isolated from blood in both the public and private sectors followed by Staphylococcus aureus, Escherichia coli and then Pseudomonas aeruginosa and Acinetobacter baumannii. The prevalence of extended spectrum beta-lactamase (ESBL)-producing Klebsiella has increased from 65 to 70% over the past five years, which limits the use of cephalosporins as treatment options.

E. coli had showed increasing resistance to quinolones with 33% of isolates resistant to ciprofloxacin, a common empiric treatment for urinary tract infections (UTIs). 25% of E. coli is an ESBL-producer, resistant to 3rd generation cephalosporins.

P. aeruginosa isolates regarded as hospital acquired infections (HAIs) are showing resistance of 20% to piperacillin-tazobactam and 33% to carbapenems, which are commonly used as first and second line treatments, respectively.

Carbapenem resistance in A. baumannii is 80%, with consistent findings across the country, as well as increasing levels of resistance over time. 20% of isolates show non-susceptibility to tigecycline. This limits treatment options to last resort antimicrobials such as colistin.

Methicillin resistance in Staphylococcus aureus (MRSA) is the only major bacterial resistance mechanism to show a decline over the past five years from 23% to 18%.

Enterococcus faecalis susceptibility to ampicillin is greater than 95% but this is not unusual, however the majority of E faecium have always been resistant to ampicillin. The growing concern is of vancomycin resistance (one of the last resort antimicrobials).

The GERMS-SA2 surveillance programme showed that resistance to penicillin of 4% for Streptococcus pneumoniae occurs mainly in children under five years of age and young adolescents and originates from community settings.

Antimicrobial Use:

The most used antibiotics in the public sector in 2020 were extended-spectrum penicillin, accounting for 28% of total antibiotics used. This was followed by oral trimethoprim-sulfamethoxazole and metronidazole at 13% and 12% respectively. By contrast, in the private sector extended spectrum penicillin, carbapenems and 3rd generation cephalosporins accounted for 41%, 20%, 13% respectively. Macrolides have more than doubled in proportionate use from 5% to 11% between 2018 and 2020, which may reflect possible increases in use during the COVID-19 pandemic in South Africa from March 2020 and ongoing at the time of writing this report. The private sector used more carbapenems as an overall percentage compared to the public sector; but both have high broad-spectrum penicillin usage.
Use of antibiotics has the possibility of driving resistance amongst ESKAPE pathogens and should be a target for future monitoring. (ESKAPE = Enterococcus faecalis and Enterococcus faecium, Staphylococcus aureus, Klebsiella pneumoniae, Acinetobacter baumannii, Pseudomonas aeruginosa and Escherichia coli)

The drivers of antibiotic resistance include:

- Antibiotics play a vital role in the management of bacterial infections, reducing morbidity and preventing mortality. They are estimated to increase life expectancy by 20 years. However, the extensive use of antibiotics in animal and human health, agricultural, and environmental sectors has resulted in drug resistance that threatens to reverse the life-saving power of these medicines. A tipping point has been reached for the international community, where we find ourselves entering a “post-antibiotic era”.
- In South Africa, the identification and publication of the first untreatable, pan-resistant Klebsiella pneumoniae from the urine of a patient admitted for cardiac surgery, and the emergence of colistin resistant genes in poultry and humans represents the extreme end of the spectrum of increasingly common multi-drug resistant (MDR) bacterial infections in this country.
- AMR or the ability of a microorganism to withstand treatment with an antimicrobial medicine, is a significant and multifaceted public health problem and a direct threat to human and animal health, food security and the continued use of available antimicrobials. The societal and financial costs of treating antimicrobial resistant infections in humans and animals will place a significant human and economic burden on society and compromise food security.
- Sixty percent of the human pathogens come originally from animals and therefore it is clear that AMR poses a serious global threat to both animal and human disease treatment. From an animal health perspective antimicrobial agents are essential tools for protecting animal health and welfare, and also contribute to satisfying the increasing world demand for safe food of animal origin,

Amount of antibiotics used:

- The total volume of antibiotics used by humans and animals according to international estimates suggest that half of all antibiotics prescribed in humans are unnecessary, either as no infection exists, the infection is not caused by a bacterium, or antibiotics are prescribed for too long a duration. Approximately, 80% of all antibiotics used globally are for animal health, agriculture and aquaculture to prevent or treat infection, or for growth promotion in the feed of animals.
- Reliance on broad-spectrum antibiotics, which have activity against a wide range of different bacteria will select out a greater range of resistant bacterial populations as opposed to narrow-spectrum antibiotics, which target the specific bacteria causing infection.
- Poor infection control practices leading to the acquisition and spread of hospital acquired infections.
- Hospitalized patients are at high risk of developing a MDR bacterial infection, as they are often immune-compromised, may have MDR bacteria transferred to them as a result of poor hand hygiene practice by

health care professionals, and may have MDR bacteria introduced into the body as a result of invasive procedures and devices.
- Lack of veterinary health professionals, weak regulations and enforcement mechanisms to oversee antimicrobial use and control of its appropriate application in animals.

The strategic framework consists of five strategic objectives:

- Strategic objective 1: Strengthen, coordinate and institutionalize interdisciplinary and intersectoral efforts through national and provincial One Health governance structures which encompasses human, animal, and environmental health experts.
- Strategic objective 2: Diagnostic Stewardship to improve the appropriate use of diagnostic investigations to identify pathogens and guide patient and animal treatment and antimicrobial management whilst strengthening quality laboratory systems for the detection of disease.
- Strategic objective 3: Optimize surveillance and early detection of AMR and antimicrobial use to enable reporting of local, regional, and national resistance patterns to optimize empiric and targeted antibiotic choice.
- Strategic objective 4: Enhance infection prevention and control and biosecurity to prevent the spread of resistant microbes to patients in healthcare settings and between animals, farms and countries. Reduced use of antimicrobials by disease prevention and community measures includes wide-reaching vaccination programs, improvements in water and sanitation, and improved biosafety.
- Strategic objective 5: Promote appropriate use of antimicrobials in human and animal health through AMS practices and controlled access to antimicrobials to ensure availability.

South African Antibiotic Stewardship Programme (SAASP):
https://cct.mycpd.co.za/fidssa/SAASP_Antibiotic_Guidelines_2015.pdf

Antibiotics in ENT

ENT related infections:
- It is common – **30-40%** of all primary care visits are **ENT related**
- Bacteria
    - Most common bacteria, for the majority of ENT conditions, are
        - ***Streptococcus pneumonia, Haemophilus influenza, and Moraxella catarrhalis***
    - *Haemophilus influenza* has replaced *Streptococcus pneumonia* as the most frequently isolated pathogen following routine vaccination of children with PCV-7 (2009) and PCV-11 (2011)
- Systemic reviews suggest that in high-income countries the benefit of antibiotic use for acute pharyngotonsillitis, acute otitis media (AOM), and acute bacterial rhinosinusitis (ABRS) is extremely limited (NNT 1/13)
- However, there is **limited data** from low- and middle-income countries, where rheumatic fever and complicated mastoiditis are common

When to prescribe antibiotics?
- When a diagnosis of a bacterial infection is made
    - Four D's
        - Correct Diagnosis
        - Correct Drug
        - Correct Dose
        - Correct Duration
- When to use a higher dose as first line treatment?

- When there are risk factors for *Streptococcus pneumonia* such as
    - Recent anti-biotic use
    - <3 and >65 years
    - Immunocompromised
    - Day-care attendees or siblings of children attending day-care centres
    - Health care workers / environment
- Severely ill, toxic patient
- Complicated disease

<u>B-lactam allergy</u>

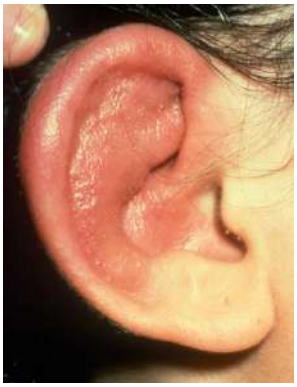
- Differentiate between the immediate type I IgE hypersensitivity and the rest
    - Type I IgE hypersensitivity symptoms and signs includes
        - **Anaphylaxis, Angio-oedema, Urticarial rash, Bronchospasm**
    - These patients should avoid penicillin / amoxicillin / ampicillin
- Patients with lesser reactions may tolerate second and third generation cephalosporins
- To exclude or confirm a B-lactam allergy (penicillin) order a Skin prick test or CAST test

Individual sites
Below follows a very short description of ENT conditions with the appropriate management. More details with regard to the pathologies are discussed under their separate headings.

Ears

Pinna

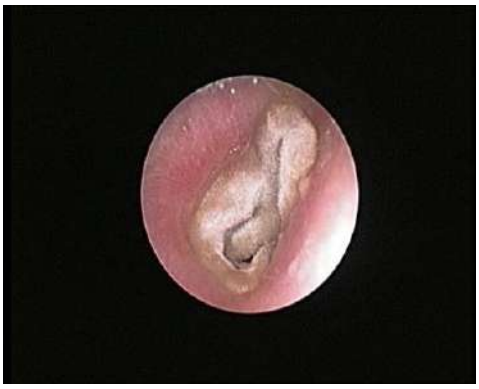
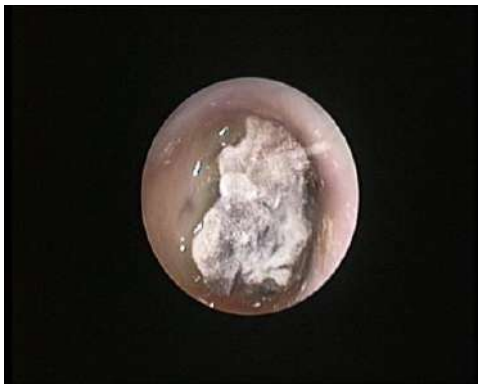
- Cellulitis / chondritis
- Amoxycillin / clavulanic acid
- Cephalosporin
    - 2nd / 3rd



Right ear with perichondritis involving most of the pinna except the lobule.

Fungal otitis externa

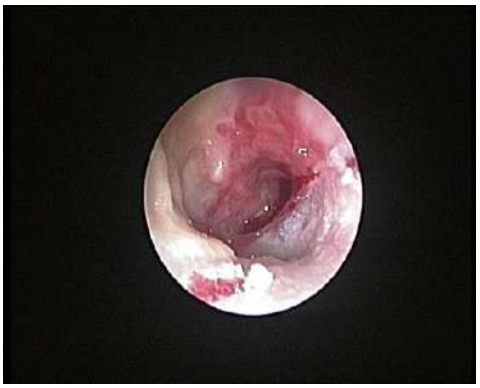
- Difficult
- Needs cleaning of external ear canal
- Boric acid
- Alcohol / Acetic acid
    - Swimmers drops
- Anti-fungal cream
- **Never anti-bacterial topical drops**

Two different ears (difficult to say which side). Hyphae can be seen and the one on the right has the typical "wet newspaper" appearance.

Bacterial otitis externa

- Keep the ear dry
- Ciprofloxacin topical drops
    - In combination with steroid drops
- Anti-bacterial creams
- Boric acid
- Alcohol / Acetic drops



Left ear with puss, erythema and some keratinised skin between 3 and 6 o'clock.

Treatment of diffuse Otitis Externa

- Clean ear canal
    - Ear bud – mopping = Ear toilet
    - If it's swollen shut, you need to insert some type of plug – see below
- Bacterial
    - Ofloxacin and steroid ear drops
        - Cilodex®
        - Safe in external and middle ear cavity
    - Boric acid powder
    - Gentamycin and others
        - Sofradex®, Covomycin D® etc
        - Toxic to the inner ear. Therefore, **unsafe if there is a tympanic membrane perforation.**
- Fungal
    - More difficult to treat

        - Canestan cream®
        - Swimmers drops, Acetic acid, Gentian violet, Methiolate, Boric acid powder
    - Combination
        - Quadriderm cream®
        - Mupirocin / Clotrimazole / steroid
    - Ear canal swollen shut
        - Creams as above on a ribbon gauze, and then inserted into the external ear canal

SEE VIDEOS OF EAR CANAL CLEANING AND BORIC POWDER!
https://youtube.com/shorts/SNXWIkNz0jE
https://youtube.com/shorts/fGeZzGU8oQA

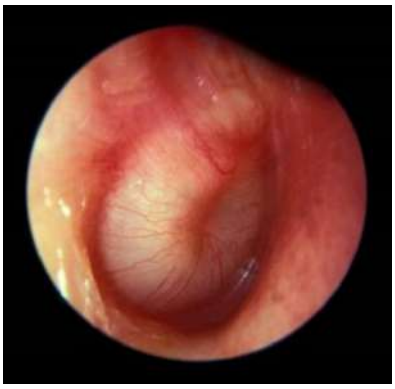
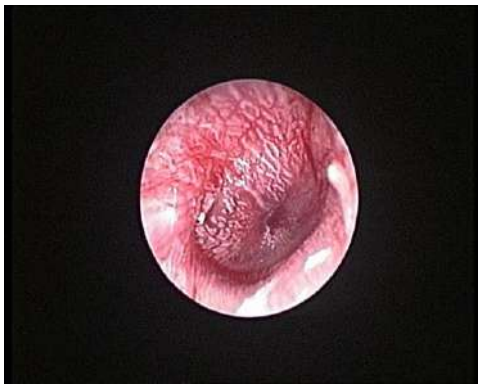
Acute otitis media – without perforation

Background:

- It is estimated that 75% of children will have at least one episode of AOM by the age of three
- Mostly due to viruses, and bacterial AOM has a high spontaneous resolution rate
- Therefore, anti-biotics are deferred for 48 hours **except** for
    - Bulging tympanic membrane and fever > 38^0C (controversial)
    - The very young (< 1-2 years)
    - Immunocompromised
    - Recurrent AOM
    - Symptoms and signs of complicated AOM
    - Pain > 48 hours
    - Limited access / follow up capability (controversial)
- It has been proven in large meta-analysis that pain relief is the most important aspect in the treatment of AOM

Antibiotics:

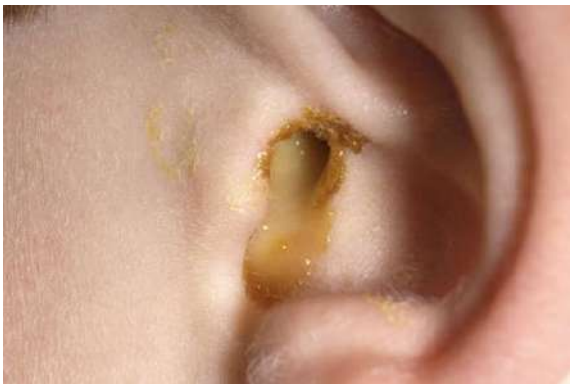
- High incidence of β-lactamase producing *Haemophilus influenza*
- Therefore, consider **Amoxicillin-clavulanate as first choice** instead of amoxicillin
- No β-lactam allergy
    - Children
        - (Amoxicillin 80-90 mg/kg/d in two divided dosages x 5-7/7)
        - Amoxicillin-clavulanate 90 mg/kg/d in two divided dosages x 5-7/7
        - Cefuroxime 30 mg/kg/d in two divided dosages x 5-7/7
        - Cefpodoxime 16 mg/kg/d in two divided dosages x 5-7/7
    - Adults
        - (Amoxicillin 1 gr q8h po x 5/7)
        - Amoxicillin-clavulanate 1-2gr bd po x 5/7
        - Cefuroxime 1000 mg q12h po x 5/7
        - Cefpodoxime 400 mg q12h po x 5/7
        - Rarely Ceftriaxone 50 mg/kg OD IM/IV x 3/7
- B-lactam allergy
    - Children
        - Azithromycin 10 mg/kg OD po x 3/7
        - Clarithromycin 15-30 mg/kg/d in two divided dosages x 5/7
        - Erythromycin 40 mg/kg/d in four divided dosages x 5/7
        - Rarely Levofloxacin 20 mg/kg/d in two divided dosages x 5/7
    - Adults
        - Levofloxacin 500 mg q12h po x 5/7
        - Moxifloxacin 400mg OD po x 5/7

Two pictures, both of right ears. The one on the left shows a building TM with obvious puss behind it (yellow). The righthand picture shows the haemorrhagic phase of AOM with also a building TM.

Acute otitis media with a perforation or post grommet otorrhea

- Clean ear – dry mopping
- Keep ear dry
- Ciprofloxacin topical drops
    - Cilodex® / Exocin® / Ciloxan®
- NEVER any other drops
- If persistent – oral antibiotics



Left ear with puss draining into the concha from the external ear canal.

Nose

The diseases below will be discussed in more detail under rhinology, but in the "acute rhinosinusitis" patient one needs to differentiate the following conditions.

	Acute viral rhinosinusitis (RS) (AVRS)	**Acute post viral RS (APVRS)**	**Acute bacterial RS (ABRS)**	**Recurrent ARS**
Symptoms	Two or more symptoms, of which one should be either **Blockage / obstruction / congestions** OR **nasal discharge** (anterior / posterior), and +/- facial pain pressure +/- reduction / loss of smell (cough in children)			

Additional symptoms	Fever, Cough, Toothache, Halitosis, Otalgia, Tiredness, Sore throat, Dysphonia, Pain on bending forward			
Time frame	< 10 days No severe fever No lasting purulence No worsening	Increase in symptoms after 5 days Persistent symptoms > 10 days Less than 12-week duration	Can occur early after an acute viral RS but is rare Chances of it being secondary bacterial infection correlates with days after onset and severity of symptoms	≥ 4 / year

When is it bacterial AND antibiotics will make a difference in acute bacterial rhinosinusitis (ABRS)?

Two different guidelines are available. The European based EPOS and American guidelines are given below. There is definite overlap between them, and we think that both can be used.

- European / EPOS
    - At least 3 of the following 5
        - Discoloured discharge
        - Severe local pain (unilateral)
        - High fever (380C)
        - Double sickening
        - Elevated CRP / ESR
- American guidelines:
    - Any / all the following
        - Worsening of symptoms at day 5-7 after initial improvement
        - Symptoms persist for more than 7 days
        - Purulence and fever present for 3-4 days

What else points more to a bacterium?

- Acute onset
- Temperature above 38^0C
    - Strongly associated with *Streptococcus pneumonia and Hemophilus influenzae*
    - Particularly in conjunction with more severe symptoms (VAS)
- CRP and ESR
    - Low – Avoid AB
    - High
        - Correlates with bacterial disease on MCS / sinus puncture
        - Correlates CT changes
- Procalcitonin
    - Showed a reduction in AB prescriptions, without detrimental outcomes, if it was normal

Treatment:

AVRS

- Education
- Decongestants < 10 days
- NSAIDS / Paracetamol
- Zinc
- Vitamin C
- Nasal rinses

- Herbal medicine
    - BNO1016 (e.g. “Sinupret®”)
    - Cineole
    - Andrographis paniculata

APVRS
- Intra-nasal cortisone spray (INCS)
- Decongestants
- Herbal medicine
- Nasal rinses
- Follow up plan
- Special investigations

ABRS
- As the other
- Antibiotics after 7-10 days

Antibiotics and dosages:

First line dosages in children
- Amoxycillin / clavulanic acid 45 mg/kg/d
- Cefpodoxime 4-8 mg/kg/d
- Cefuroxime 15 mg/kg/d

Failure / Step up dosages in children
- Amoxycillin / clavulanic acid 90 mg/kg/d
- Cefpodoxime 16 mg/kg/d
- Cefuroxime 30 mg/kg/d

First line dosages in adults
- (Amoxycillin 1 - 2 gr bd)
- Amoxycillin / Clavulanic acid 1 gr bd
- Cefuroxime 500 mg bd
- Cefpodoxime 200 mg bd
- (Cefprozil 500 mg bd)

Failure / Step up dosages in adults
- Amoxycillin / Clavulanic acid 2 gr bd
    - 1 gr bd with added amoxicillin
- Cefuroxime 500 – 750 mg tds
- Cefpodoxime 200 mg tds
- (Cefprozil 500 mg tds)

Throat

Acute pharyngotonsillitis (APT)

Causes include:
- Infective
    - Viral most common
        - Adenovirus, Coxsackie A virus, Influenza virus, Parainfluenza virus, Epstein Barr virus
    - Bacteria (5-30%)

        - Group A β-haemolytic *Streptococcus pyogenes* (GABHS)
        - Other
    - Fungi
        - Candida
    - Granulomatous
- Other
    - Irritant
    - Reflux
    - Tumours
    - Auto-immune
    - Trauma
    - Neuralgias

Most of the time, acute pharyngotonsillitis is of viral origin. However, Group A β-haemolytic *Streptococcus pyogenes* (GABHS) remains the most common bacterial cause. Because of its ability to cause rheumatic fever, glomerulonephritis, and other septic complications it needs to be diagnosed from viral and other aetiologies. Treatment with antibiotics have been shown to prevent the development of rheumatic fever and septic complication, but not the development of glomerulonephritis. Fortunately, there has never been a GABHS isolate that showed resistance to penicillin.

Symptoms of infective pharyngotonsillitis are not specific enough to differentiate between the different infective causes. Symptoms in general include sore throat, sever, dysphagia, and halitosis. Symptoms in favour of acute viral pharyngitis include rhinorrhoea, cough, conjunctivitis, hoarseness, stomatitis, ulcer(s), diarrhoea. Therefore, the question remains how can we diagnose a bacterial pharyngitis and GABHS in particular? We advise using the modified Centor criteria. It can only be applied in patients with recent onset (≤3 days) acute pharyngitis. A score of more than 3-4 points correlates with a bacterial infection, and thus antibiotic use.

Modified Centor criteria

Criteria		**Points**
Age	< 3	0
	3-14	+1
	15-44	0
	> 44	-1
Cough	Present	0
	Absent	+1
Tonsillar exudates / swollen		+1
Temp > 38°C		+1
Anterior cervical lymphadenopathy		+1

Other options to diagnose a possible GABHS include:

- Rapid antigen testing
- Throat swab (PCR as well)
- Nucleic Acid Amplification Techniques (NAATs)

The following link gives a nice overview of subject (https://www.frontiersin.org/journals/cellular-and-infection-microbiology/articles/10.3389/fcimb.2020.563627/full).

Treatment of acute bacterial tonsillitis:

- Penicillin / Amoxicillin
    - Pen VK (30 minutes before meals)
        - 250 mg BD po x 10/7 (<27 kg)
        - 500 mg BD po x 10/7 (>27kg)

        - Amoxicillin
            - Children
                - 50 mg/kg/d in two divided dosages x 10/7 (max 1000 mg)
            - Adults
                - 500-1000 mg BD po x 10/7
- B-lactam allergy
    - Children
        - Azitromycin 10-20 mg/kg/d OD x 5/7
        - Claritrhomycin 15 mg/kg/d in two dosages x 10/7
    - Adolescents and adults
        - Azitromycin 500 mg OD po x 3/7
        - Clarithromycin 500 mg BD x 10/7

Acute Laryngitis

- Can either be infective or non-infective
- Infective laryngitis
    - Associated with an upper respiratory tract infection
    - Voice changes, throat pain
    - Spontaneous resolution
    - Voice rest, Steam inhalation, Steroids
    - NO ANTIBIOTICS!

Chronic laryngitis

- Dysphonia for weeks
- Smoking, gastro-oesophageal reflux (GERD), laryngopharyngeal reflux (LPR), and alcohol use are risk factors
- May induce secondary epithelium changes that can be pre-malignant
- Vocal hygiene principles are important
    - Lots of water
    - Avoid caffeine
    - Voice rest
    - Do not whisper or force your voice
    - Do not raise your voice over the background noise
    - Avoid smoking and alcohol
    - Empirical treatment for GERD / LPR
- Needs endoscopy by an ENT

Laryngotracheobronchitis / Croup

- Typically presents between ages 6 months – 6 years and is preceded by a viral URTI
- Keeping the child and the parent calm
- Steroids
    - Oral / IV / nebs
    - Oral and IV equally effective
        - Oral prednisolone 1mg/kg
        - Oral dexamethasone 0.15mg/kg
        - IV / IM dexamethasone 0.6mg/kg
    - Low vs high dose studies have been equivocal
- Adrenaline
    - Nebs

    - Remember potential rebound swelling, therefore children should be observed for at least 3-4 hours
- Humidified air
    - Heliox
- Securing the airway
    - Intubation vs tracheostomy (only in cases of Grade 3-4 stridor where there is concern of acute airway compromise)

Supraglottitis / Epiglottitis

- Cephalosporin, steroids, HIB vaccine

Anaphylaxis guideline of South Africa:

EMERGENCY MANAGEMENT OF ADULT & CHILD ANAPHYLAXIS

1 RECOGNIZE THE SUDDEN ONSET OF EITHER:

AFSA ALLSA

EXPOSURE TO KNOWN OR UNKNOWN ALLERGEN

- **SKIN/MUCOSAL INVOLVEMENT** (rash, swelling) **AND ANY OF:**
- **RESPIRATORY COMPROMISE** (dyspnoea, wheeze), **OR**
- **CARDIOVASCULAR DYSFUNCTION, OR**
- **SEVERE GASTROINTESTINAL SYMPTOMS** (abdominal pain, repetitive vomiting)

AFTER EXPOSURE TO KNOWN ALLERGEN

- **RESPIRATORY DIFFICULTY** (stridor, voice change, wheeze, hypoxaemia, distress)

AND/OR:

- **CARDIOVASCULAR DYSFUNCTION** (shock, hypotension, syncope, collapse)

(No need for skin or mucous membrane involvement)

2 IMMEDIATE TREATMENT:
- ⊘ REMOVE EXPOSURE
- ⓘ CALL FOR HELP

ADRENALINE

1mg/ml (1:1000) - 0.01mg/kg IM (Max 0,5ml IM) anterolat aspect of thigh
Repeat every 5-15 minutes if no improvement or use an **auto-injector**
<6yrs - 0,15ml IM; 6-12 yrs - 0,3ml IM; >12 yrs - 0,5ml IM

3 ASSESS VITAL SIGNS: OXYGEN - MONITORS - IV ACCESS

High flow oxygen, maintain patent airway (Intubate/Cricothyrotomy if necessary)
High flow IV line, BP, Sats, ECG monitoring
Lie patient supine with legs elevated if hypotensive

4 ADJUNCTIVE TREATMENT IF NECESSARY

H1 ANTIHISTAMINE
Promethazine
2-6 yrs - 6,25mg IM or slow IV
6-12 yrs - 12,5mg IM or slow IV
>12 yrs - 25mg IM or slow IV
(Avoid if <2yrs old and low BP)

CRYSTALLOID
(e.g. Ringers/Balsol)
Rapid infusion of 20ml/kg (max 1-2 litres)
Repeat IV infusion as necessary
Adrenaline infusion (0,1 - 1 ug/kg/min)
ONLY if unresponsive to IM adrenaline & fluids

NEBULISED BRONCHODILATORS
Every 15-20 mins if severe bronchospasm
Salbutamol 5mg
WITH
Ipratropium 0,5mg

H2 RECEPTOR ANTAGONIST
Cimetidine
IM or Slow IV
5mg/kg (Max - 300mg)
Diluted in 20ml over 2 min

CORTICOSTEROIDS
Hydrocortisone
IM or Slow IV
<1 yr - 25mg; 1-6 yrs - 50mg;
6-12 yrs - 100mg; >12 yrs - 200mg

GLUCAGON
20ug/kg (Max 1-2mg)
IM or slow IV every 5 mins if unresponsive to adrenaline (Look out for vomiting and hyperglycaemia)

RISK REDUCTION STRATEGIES

- Only discharge patient if clinically stable 4-6 hours after resuscitation (may need longer if at risk of biphasic reaction)
- Provide a written anaphylaxis emergency action plan, including how to administer IM adrenaline
- Refer to specialist for investigation and management
- Provide patient eduction (www.allergyfoundation.co.za) and medic-alert bracelet

FAQ's:

When is it appropriate to initiate treatment for Anaphylaxis?
Treat anaphylaxis at diagnosis with IMI adrenaline even if severe respiratory or cardiovascular symptoms are not (yet) present.
Why are Antihistamines considered adjunctive treatment?
H1-antihistamines may relieve itching and urticaria but do not prevent or relieve life-threatening symptoms of anaphylaxis. Antihistamines should not be used alone, or instead of adrenaline, for anaphylaxis.

Version May 2024

Ref: Cardona et al. World allergy organization anaphylaxis guidance. WAOJ;13(10):2020.

6) Practical tips in Otology

Wax

Background
The commonly taught philosophy that, if there is wax obstructing the external auditory meatus, the ear must be normal behind it is false. Examination of the ear is inadequate unless the eardrum has been seen. The presence of even a little wax in the ears tends to distract one's eye and make it difficult to focus on the tympanic membrane.

Methods of removing wax

Headlamp and Jobson-Horne or wax hook:
Allows quick and easy removal of superficial wax. Be careful not to hurt the patient or go too deep (damage the ear). This is mostly only possible in adults and older children that cooperate. Start by pulling the ear posterior-superiorly (to straighten the external ear canal). Under direct vision gently pass the hook past the wax (usually superior to it) and rake the wax outwards by applying gentle downward pressure. MAKE SURE NOT TO INJURE THE EXTERNAL EAR CANAL! The skin is tightly adherent to the underlying periosteum leading to severe pain when touched and is easily lacerated with subsequent bleeding. We would strongly suggest that you buy yourself any decent outdoor camp headlight. At the end there are also examples of Jobson-Hornes and various wax hooks. You can also use a hair pin or paper clip as a hook.

Syringing:
You would need a headlamp, bowl to collect water, and a gentle stream of body temperature water (big syringe and a blunt/soft nozzle e.g. sheath removed from a drip needle). The idea is to try and aim for a gap in the wax if possible and get the returning stream of water to push the wax out. Otherwise, one should aim at the posterior-superior quadrant and let the stream come back at you from antero-inferiorly. REMEMBER, if you use water that is either cooler or warmer that body temperature, you will induce a caloric reflex. This will cause nystagmus and frequently violent nausea and vomiting.

(Caloric reflexes are frequently used to assess if a patient is “brain dead”. In that setting, you will on purpose use water that is either below or above body temperature. Classical teaching stipulates 7°C above or below, but a mini-caloric test involves rinsing the ear with tap water for 10 seconds in the summer and 5 seconds in the winter. Furthermore, caloric testing is one of the few vestibular tests that stimulates only one vestibulum at a given time.)

Dissolving / softening wax:
By far the best wax solvent is simple water with or without Savlon®. It works very quickly and by syringing the ear at +/-15-minute intervals or giving your patient a dropper to pump into his ear each few minutes. You will be able to clear the ear of wax within 30 minutes by syringing it after the water has softened it.

Cotton wool ear buds:
Generally, it causes the wax to impact more by pushing it deeper in with its blunt end. It may be useful on occasion for wax lying on the external auditory meatus wall, if one can pass the "bud" beyond it and then rake it out. REMEMBER, don’t confuse this with ear mopping in the case of a discharging ear.

Foreign bodies

Background
Usually occur in little children who are generally uncooperative in the removal of the FB. Try not to rush in on the FB, first establish a “bond” with the child. This is especially important if previous attempts have failed (or impossible). If cooperation is obtained from the child, the first attempt at removal affords the best (often the

only) opportunity. Get help in securing the child's arms and head by usually wrapping the child in a blanket. In general, try to do this with the child on the caretaker's lap.

Techniques
Most FB can be removed with a small crocodile of Tilley's forceps. Most casualties will have these instruments and if in private practice you will find joy in investing in these instruments and helping many patients.

If the foreign body is hard or sharp, it may cause damage if pushed into the ear. Rather refer to your local ENT specialist.

If the object is round and has no means of getting purchase, the use of a crocodile or other forceps will only push it deeper. If the child cooperates and lies still, and there is a gap through which a Jobson-Horne or right-angled wax hook may be passed, the FB may be raked out. Preferably rake out down and forwards: the antero-inferior external auditory meatus is less sensitive to pressure and touch.

If there is a little gap and one can selectively syringe water through this with a 20-cc syringe with a "plastic" cannula on its end (see wax technique), this may force the FB out.

There have been reports of the successful use of cyanoacrylate glue ("superglue") applied to a probe and touched to the FB and then allowed to set (quickly, as it does). Make sure the child can't move!

Suction tips applied on a FB can also successfully suck out a FB.

Often one is confronted with an insect in the ear. These patients experience huge discomfort as the insects' usually ends up close to the tympanic membrane. The reason being that the natural instinct is to try and get rid of the insect by turning the ear downwards. In reality, the insects crawl upwards and therefore deeper into the ear canal. In the acute situation one only needs to kill the insect by putting IV lignocaine or an oily solution into the ear.

REMEMBER, the only true emergency in the ear canal is a battery! These should be referred immediately if you can't remove it. In general, if the situation looks difficult, rather refer to your local ENT specialist.

Ear mopping and installing drops or boric powder

Technique of dry mopping
Ear mopping, also known as ear toilet, refers to the process of manually clearing the discharge from the external ear canal before applying topical agents. This is usually done in the acute process with acute post-grommet otorrhoea or in chronic suppurative otitis media. By using the back end of a Jobson-Horne current (or a blunt ended orange wooden stick), cotton buds, or simply rolling cotton wool around any applicator the ear is cleaned. This step should be repeated until the ear is dry. It is also imperative to dry mop the ear before installing any drops or powders into it. The video link demonstrates how to dry mop the ear:
https://youtube.com/shorts/SNXWIkNz0jE

Technique
Topical agents include antibiotic drops, acetic acid, and / or boric powder. Historically gentamycin-based drops were used but has now been replaced by ciprofloxacin-based drops which is not ototoxic. Frequently the ciprofloxacin drops are combined with a topical steroid, and combination products are available. Before installing drops, shake the bottle well and warm it to body temperature (otherwise you will elicit a caloric response). The easiest is to carry it against your own body for a couple of minutes before installing it into the ear. With the affected ear facing up, install the number of drops prescribe and lightly apply a pumping action on the tragus (see video). Sometime patients report "tasting" the drops which is due to some reaching the pharynx via the Eustachian tube.

The Division of Otorhinolaryngology prefers the application of boric powder in cases with chronic discharge. Please click in the link to see the video on installing boric powder in the ear:
https://youtube.com/shorts/fGeZzGU8oQA

Instruments frequently used: A Tilley forceps on the left, Jobson-Horne in the middle, and a crocodile forceps on the right.

7) Ear Conditions

Must know

- Appearance of normal tympanic membrane
- Wax removal
- Foreign body removal
- Ear syringing / mopping
- Infections
    - OE / AOM / COM
- Complications
    - Mastoiditis
    - Cholesteatoma
- Bell's palsy
- Acute idiopathic nerve deafness
- Approach to acute dizziness

Nice to know

- Inner ear problems
    - Tinnitus
    - Complicated vertigo
- Causes of hearing loss
- Skull base trauma

External Ear

External ear canal infections

Risk factors

- Trauma
    - Ear buds
    - Chemical
- Wet ear canal
    - Swimmers
    - Hearing aids
    - Climate
- Skin conditions
- Anatomical obstructions
- Immune suppression

Types

- Diffuse
    - Otitis externa – Swimmer's ear
    - Cellulitis
- Localized
    - Furuncle
- Cartilage
    - Chondritis

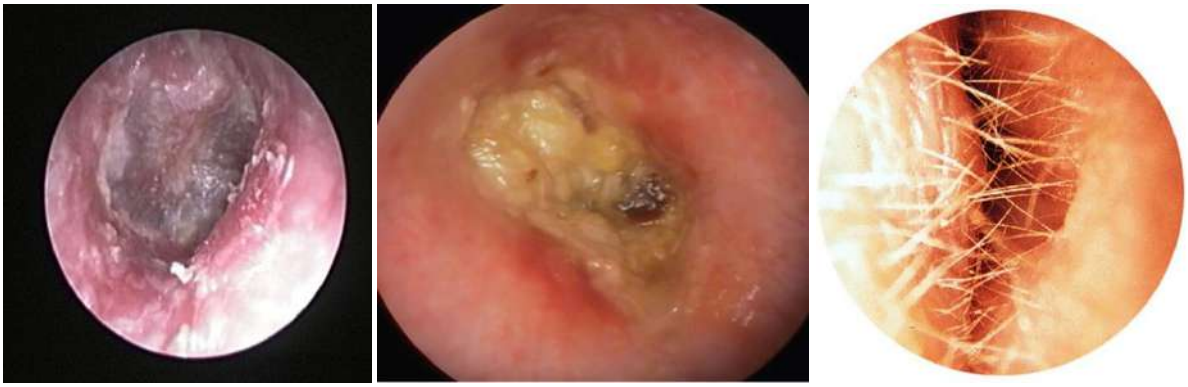
- Skull base
    - Malignant OE / Necrotizing OE
    - Skull base osteomyelitis

Aetiology

- Bacterial
    - Staph / Pseudomonas / E. Coli
- Fungal
    - Candida / Aspergillum
- Viral
    - Herpes zoster
    - Very rare
- Combination

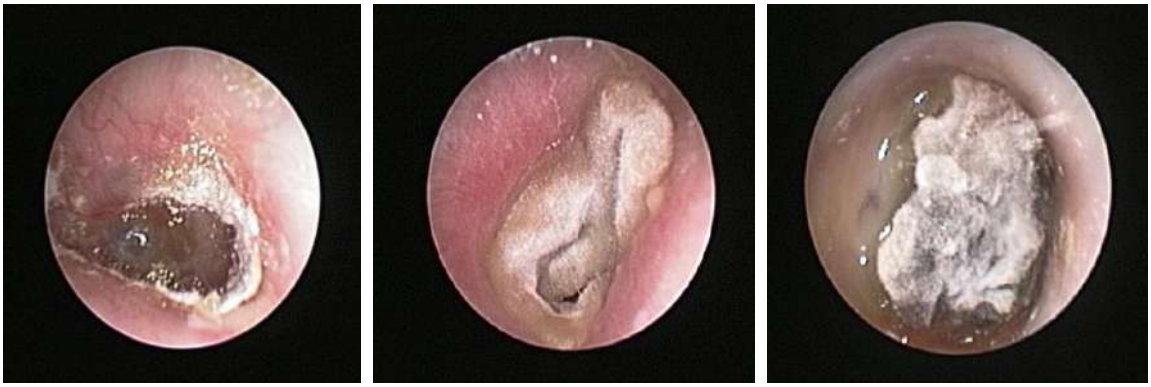
Treatment

- Clean ear canal
    - Ear bud – mopping = Ear toilet
    - If it's swollen shut, you need to insert some type of plug – see below
- Bacterial
    - Ofloxacin and steroid ear drops
        - Cilodex®
        - Safe in external and middle ear
    - Boric acid
    - Gentamycin and others
        - Sofradex®, Covomycin D® etc.
        - Toxic to the inner ear. Therefore, unsafe if there is a tympanic membrane perforation!
- Fungal
    - More difficult to treat
    - Canestan cream®
    - Swimmers drops, Acetic acid, Gentian violet, Methiolate, Boric acid
- Combination
    - Quadriderm cream®
    - Mupirocin / Clotrimazole / steroid
- Ear canal swollen shut
    - Creams as above on a ribbon gauze, and then inserted into the external ear canal



Three examples of bacterial OE. The picture on the left might fool you in not thinking the OE is severe. Sometimes there is minimal erythema of the ear canal. The picture in the middle shows think pussy secretion medial in the

ear canal. The picture on the right shows an ear canal completely shut. It is best to insert an antibiotic cream on the ribbon gauze in ears like this.



Three examples of fungal OE. Typiccally, one can see hyphae or the “wet newspaper” debris.

Videos:
Boric acid - https://youtube.com/shorts/fGeZzGU8oQA
Ear mopping - https://youtube.com/shorts/SNXWIkNz0jE

How to use ear drops?

- Shake the bottle
- Warm it in your hands
- Clean the ear of secretions (ear toilet / mopping)
- Lie on your side, and insert the required number of drops, in the uppermost ear
    - Pull the ear up and out in an adult
    - Pull the ear down and out in a child
- Apply a pumping action on the tragus
- Lie for 2-3 minutes with the ear facing upwards
- If you want to, put a cotton wool in the ear, but only temporarily

Prevention of OE

- Keep ear dry
    - Avoid water
    - Plugs
        - Various
        - Can sometimes cause OE
    - Hair dryer
- Avoid ear buds – trauma
- Drops
    - After exposure
        - Acetic acid, Swimmers drops
    - Before exposure
        - Swim seal, Oily drops

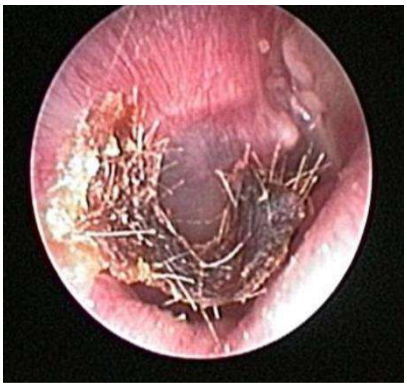
Treatment of other EAC infections / conditions

- Furuncle
    - Local or oral antibiotics

    - Open if abscess
- Viral
    - Supportive – PAIN relief
    - Prevent secondary infection
- Cellulitis
    - Oral anti-biotics
- Chondritis
    - Oral anti-biotics
- Malignant OE
    - Extreme pain
    - Purulent otorrhoea
    - Granulation tissue and / or exposed bone
    - NVII palsy
    - Other lower cranial nerve palsies
    - **Always in the background of**
        - **immune suppression**
    - **REFER ASAP**

Wax

- Varies from easy to impossible
- Different types
    - Soft / Flaky / Sticky / Hard
- Options
    - Syringing – blind
    - Drops
        - Cerumol / Waxsol / Oily drops
    - Under vision – two hands
        - Suction
        - Curette
- Instrumentation
- Refer
- See syringing an ear

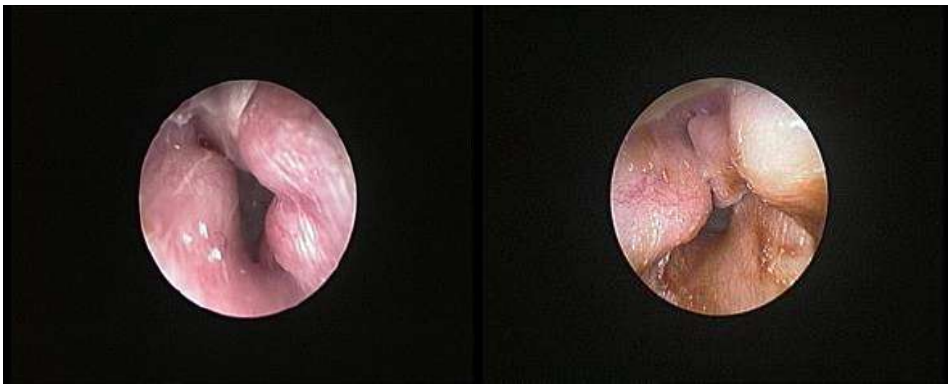


Wax in the external ear canal. Note that was can completely obstruct the ear canal.

Exostosis

- Bony overgrowths
- When to refer
    - Repeated infections
    - Hearing loss

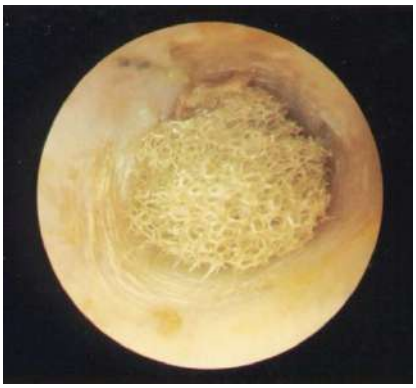
    - Wax
    - Progressing to complete closure
- Extremely common in areas with cold water exposure



Two pictures both demonstrating severe exostosis.

FB

- You only have one go!!
- There is no need to risk injuring the ear to remove the FB
- Oily solution / Lignocaine 1% for insects
- Instrumentation and Headlight
- Decide beforehand
    - **To grab FB**
    - **Pass an instrument behind it**
    - **Syringing**
    - **Suction**
- Restrain?
- Refer



Sponge in the ear canal.

Pinna

- Infections
    - Mostly streptococcus
    - Anti-biotics
        - Penicillin / Quinolones
- Haematoma
    - Small
        - Aspiration with pressure bandage

    - Other
        - Refer
- Treatment
    - Aspirate x2
    - N.B. Sterile conditions & use a large bore needle
    - Compression bandage
    - Review in 24 hours
    - Consider re-aspiration
- If re-accumulates again:
    - Proceed to formal drainage
    - Quilting stitch
- Cover with antibiotic conditions to prevent infection

Tumours of the EAC

- Rare
- Squamous cell carcinomas, Basal cell carcinomas, Melanomas
- Presents with
    - Skin changes
    - Bleeding
    - Ulceration

Middle Ear

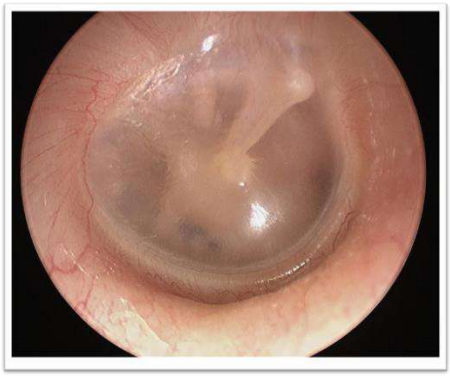
Conditions

- Tympanic membrane
    - Bullous myringitis
    - Granular myringitis
- Middle ear
    - Otitis media with effusion (OME)
    - Acute otitis media (AOM)
    - Chronic otitis media (COM)
- Various
    - Trauma
    - HIV / TB
    - Other

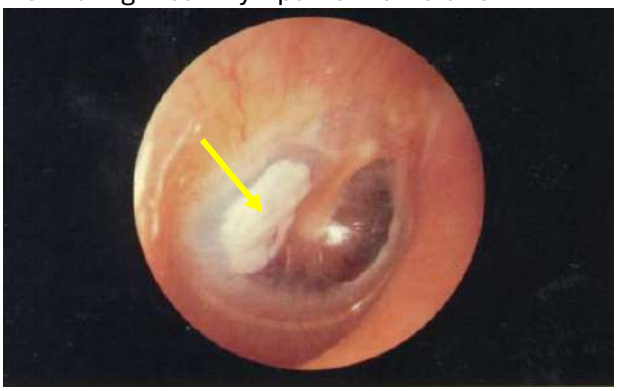
Normal TM

- Variation
    - Child
    - Adult
- Factors influencing the appearance of a TM
    - Previous infections
    - Previous grommets
    - Previous operation
    - Screaming child
    - External ear canal
        - Wax

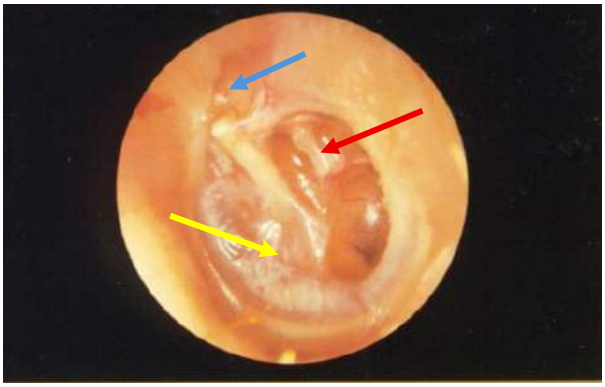
- Exostosis
- Foreign bodies



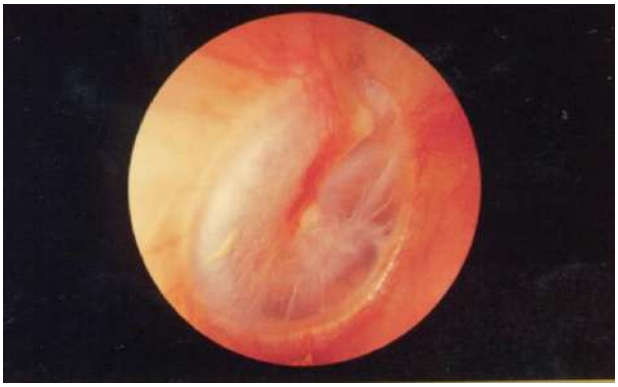
Normal right ear / tympanic membrane.



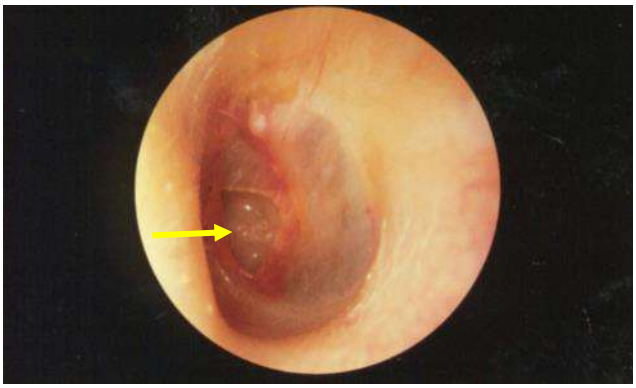
Right ear with myringosclerosis in posterior part (yellow arrow).



Left ear with retraction of pars flaccida (blue arrow) and pars tensa (yellow arrow). Also, incudo-stapedeopexy (red arrow) with retraction of TM onto incus / stapes.



Dull right TM. May be due to previous ear disease.

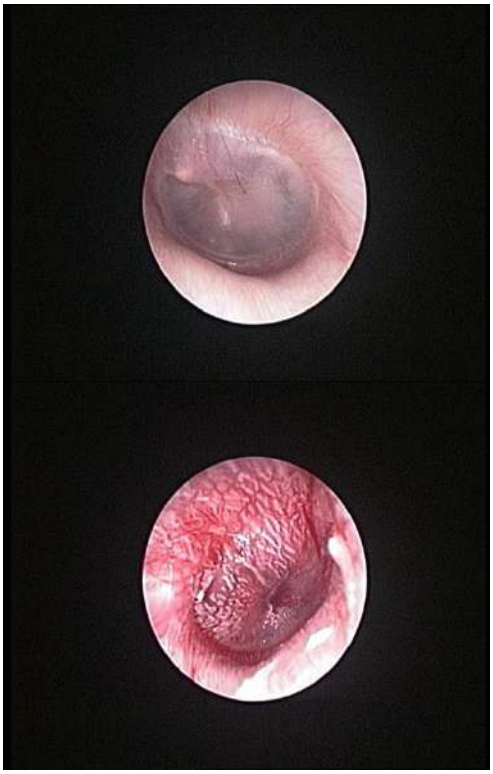


Left ear with pars tensa central perforation (yellow arrow).

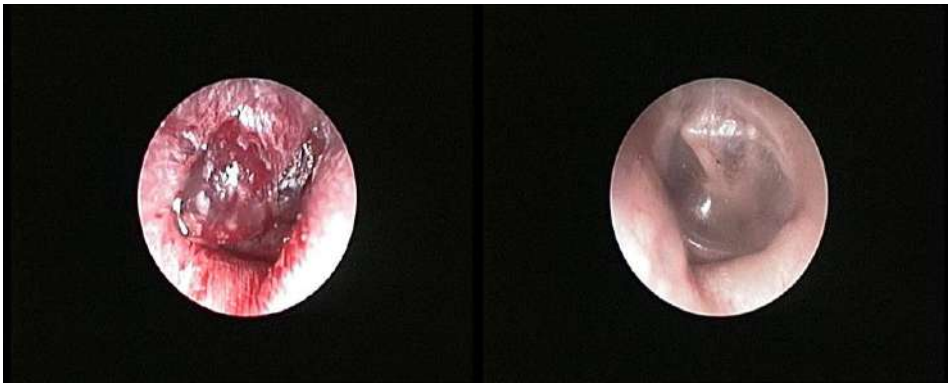
Tympanic membrane conditions

Bullous myringitis

- Acute pain
  - 11/10
- Mostly unilateral
- Bloody discharge
  - Pain improves
- Aetiology
  - Previously thought to be Mycoplasma / Viral
  - Now bacterial
- Treatment
  - PAIN RELIEF
  - Oral quinolone
  - Topical ofloxacin / steroids
- Can have a middle ear effusion afterwards
  - Clears up over weeks



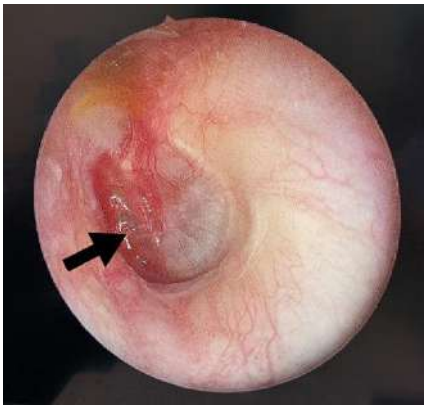
Same patient with a normal left ear (top picture) and a haemorrhagic bulging right TM.



Pictures demonstrating a bigger haemorrhagic bulla on right TM and a normal left TM.

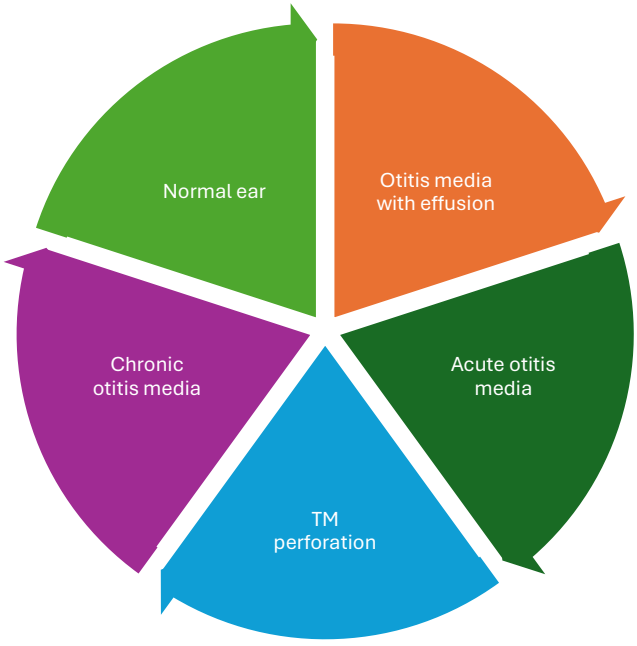
Granular myringitis

- Aetiology poorly understood
- Presents with
    - Itching
    - Otalgia
    - Aural fullness
    - Otorrhoea
- Treatment
    - Acetic drops / Boric acid
    - Cauterise with silver nitrate
    - Resection of affected TM and repair with graft



Picture demonstrating a left TM with the black arrow showing the myringitis.

Middle ear disease



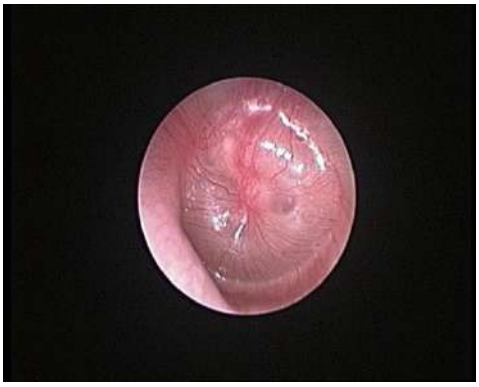
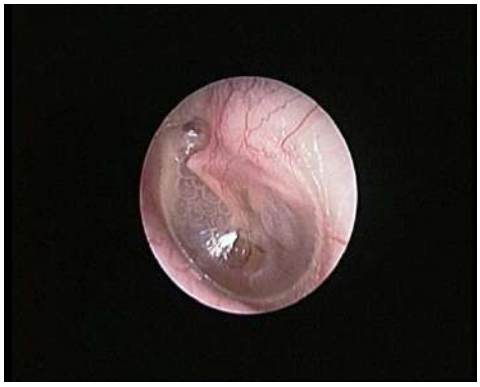
Incidence

- Extremely common – bimodal peak
  - **6/12 to 2 years**
  - **4 to 7 years**
- Risk factors
  - Passive smoking
  - Repeated AB use
  - Day care
  - Adenoid hypertrophy
  - Eustachian tube dysfunction
  - Ciliary diseases
  - Family history
  - Winter months
  - Boys
  - Skull base abnormalities – includes conditions such as Down syndrome
- Protective factors
  - Strep pneumonia vaccine

    - ?? Probiotics / Prebiotics
- EXTREMELY UNCOMMON IN ADULTS!!
    - If an adult presents with middle ear disease for the first time, nasopharyngeal pathology should be excluded

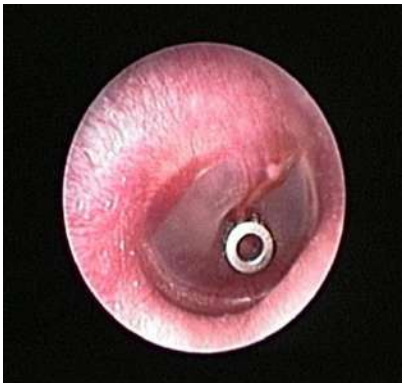
Otitis media with effusion (OME)

- Can arise de novo or follow after acute otitis media (AOM)
- Can cause AOM
- Difficult to diagnose
    - **Under diagnosed**
    - Tympanometry
- Can present with
    - Irritable child
    - Disturbed sleeping pattern
    - Pulling on ear / chewing finger
    - Hearing loss / Poor speech development
    - Minimal symptoms

Both pictures are of the left ear. The picture on the left demonstrates a bulging TM with thick (glue) OME behind (medial) to the TM. The picture on the right show bubbles and fluid behind the TM.

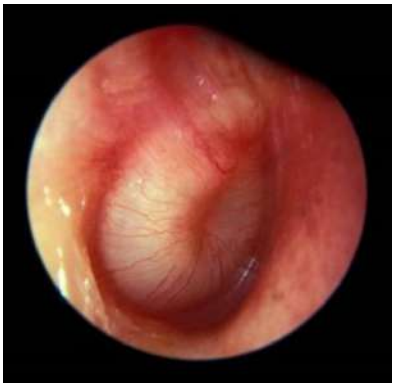
- Treatment
    - Medical
        - Steroids
            - Intra-nasal
        - Nasal sprays
            - Saline / Sea water
        - Antibiotics – not advised!
        - Wait and see
    - *Grommets / Ventilation tube*
        - Effusion that's not clearing up
            - 8-12 weeks
        - Hearing loss / Poor speech development
        - Associated with repeated AOM episodes
            - ≥3 in 6 months
        - Handicapped child
            - Learning disabilities
            - Already a hearing loss



Right ear with a grommet in place.

Acute otitis media (AOM)

- Easier to diagnose
    - **Unfortunately, commonly over / missed diagnosed**
- Usually, a normal child which develop severe pain and high fever in a short period
- Common symptoms include
    - Otalgia (holding, tugging, rubbing of ear)
        - The very young can't localize the pain
    - Mostly unilateral
    - Hearing loss
    - Fever, nausea, and dizziness
    - Rarely children present with otorrhea without any other symptoms
- You need to visualize the tympanic membrane
    - Red / Puss
    - Oedematous
    - Immobile
    - Bulging



Right ear with a bulging TM and obvious pus (yellow) behind the TM.

- It is estimated that 75% of children will have at least one episode of AOM by the age of three
- Mostly due to viruses, and bacterial AOM has a high spontaneous resolution rate
- Therefore, anti-biotics are deferred for 48 hours except for
    - Bulging tympanic membrane and fever > 38^0C (controversial)
    - The very young (< 2 years)
    - Immunocompromised
    - Recurrent AOM
    - Symptoms and signs of complicated AOM
    - Pain > 48 hours
    - Limited access / follow up capability (controversial)

- It has been proven in large meta-analysis that pain relief is the most important aspect in the treatment of AOM

AB guidelines for AOM

- High incidence of β-lactamase producing *Haemophilus influenza*
- Therefore, consider Amoxicillin-clavulanate as first choice instead of amoxicillin
- No β-lactam allergy
    - Children
        - (Amoxicillin 80-90 mg/kg/d in two divided dosages x 5-7/7)
        - Amoxicillin-clavulanate 90 mg/kg/d in two divided dosages x 5-7/7
        - Cefuroxime 30 mg/kg/d in two divided dosages x 5-7/7
        - Cefpodoxime 16 mg/kg/d in two divided dosages x 5-7/7
    - Adults
        - (Amoxicillin 1 gr q8h po x 5/7)
        - Amoxicillin-clavulanate 1-2gr bd po x 5/7
        - Cefuroxime 1000 mg q12h po x 5/7
        - Cefpodoxime 400 mg q12h po x 5/7
        - Rarely Ceftriaxone 50 mg/kg OD IM/IV x 3/7
- B-lactam allergy
    - Children
        - Azithromycin 10 mg/kg OD po x 3/7
        - Clarithromycin 15-30 mg/kg/d in two divided dosages x 5/7
        - Erythromycin 40 mg/kg/d in four divided dosages x 5/7
        - Rarely Levofloxacin 20 mg/kg/d in two divided dosages x 5/7
    - Adults
        - Levofloxacin 500 mg q12h po x 5/7
        - Moxifloxacin 400mg OD po x 5/7

Complications

- Infective
    - Treatment failure
        - 48 hours
        - Change antibiotic
        - Grommets – drainage
    - Mastoiditis ± abscess formation
    - Other Abscesses – soft tissue / brain
    - Brain – meningitis / sigmoid sinus thrombosis
    - Skull base – petrositis
    - CHRONIC OTITIS MEDIA
- Non-infective
    - 1/3 can develop an ear drum perforation despite antibiotics
        - Puss draining with immediate relief of pain
        - Clean ear canal and use topical drops
    - Damage in middle ear cavity
        - Ossicles, NVII, Labyrinth, Sclerosis, Adhesions, Adhesive ME
    - Sensori-neural hearing loss and conductive hearing loss

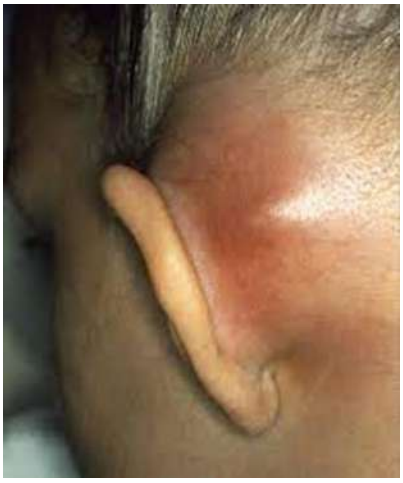
Differential diagnosis between diffuse OE and AOM

	Otitis externa	**Acute otitis media**
Prodrome	Local risk factor	Upper airway infections

Pain	Extremely painful	Moderate to severe pain
Localization	Painful when pressing on tragus	Can be painful over mastoid process with skin changes
Otorrhoea	Rarely otorrhoea Minimal if present	Can have otorrhoea only if tympanic membrane ruptures Puss / mucoid
Tympanic membrane	External ear canal swollen - can't visualize TM	Can see tympanic membrane but it's abnormal

Mastoiditis

- Complication of AOM
- Can develop a subperiosteal abscess the presents with
    - Skin changes
    - Tenderness over mastoid
    - Loss of skin crease
    - Pinna displaced down and out
    - Fluctuation
- Start with antibiotics and refer to your ENT colleague



Picture of a left ear with signs of a subperiosteal abscess.

Chronic otitis media (COM)

Classification (simplified)

- Chronic suppurative otitis media (CSOM)
- CSOM with a cholesteatoma
- CSOM due to tuberculosis
- Healed COM
    - Tympanosclerosis
    - Healed perforation
        - Thinning and/or local or generalized opacification of the pars tensa without perforation or retraction

Classification (more complex)

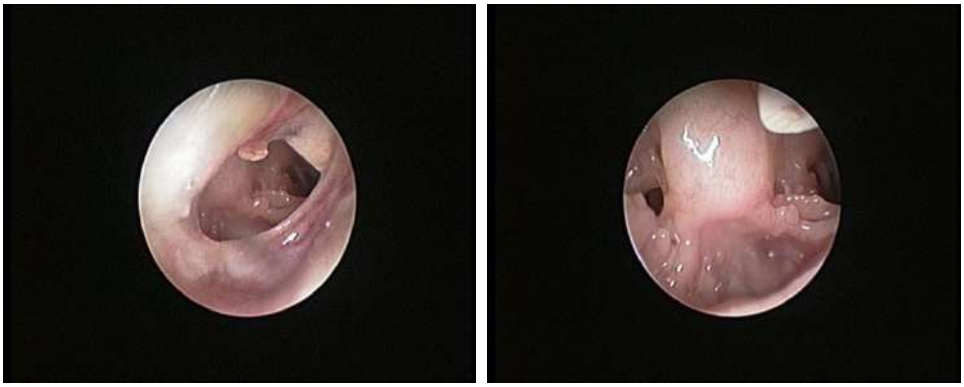
- Inactive mucosal COM = **Dry perforation**

    - Permanent perforation of the pars tensa but the middle ear mucosa is not inflamed
- Active mucosal COM = **Chronic suppurative OM**
    - Permanent defect of the pars tensa with an inflamed middle ear mucosa which produces mucopus that may discharge
- Inactive squamous COM = **Retraction pockets**
    - Retraction of the pars flaccida or pars tensa (usually posterosuperior) which has the potential to become active with retained debris
- Active squamous COM = **Cholesteatoma**
    - Tympanic membrane perforation with skin that has grown into the middle ear cavity (cholesteatoma)
        - This is mostly due to retraction pockets, but can be due to perforations, operations, metaplasia, or congenital
    - There is retained squamous epithelial debris (keratin) and is associated with inflammation and the production of foul smelling mucopus, and often a granuloma / granulation tissue

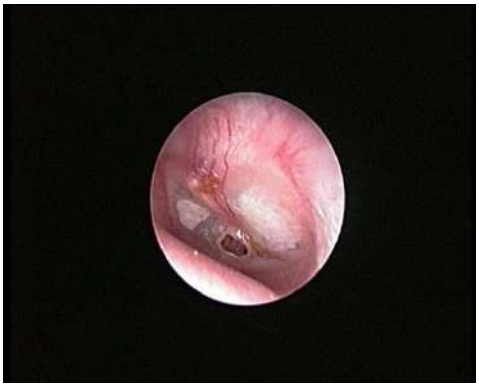
	Inactive	**Active**
Mucosal	Perforation	Chronic suppurative otitis media
Squamous	Retraction pockets	Cholesteatoma

Inactive mucosal COM = Dry perforation

- Presents with
    - Conductive hearing loss
    - Mostly dry
        - Can have episodic discharge
    - Asymptomatic
- Refer
- Surgical repair preferred in most cases
    - Tympanic membrane reconstruction
    - Depends on
        - Age
        - Hearing loss
        - Status of opposite ear

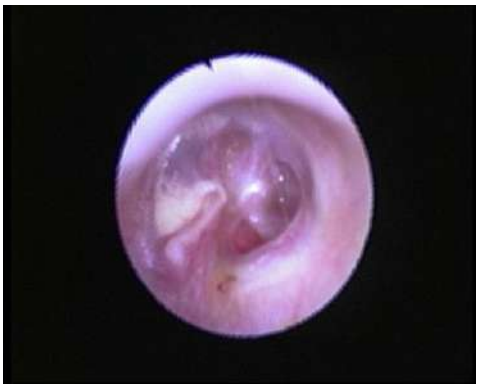


Same ear demonstrating a perforation in the left TM and a close up through the perforation with the promontory and floor of the middle ear.



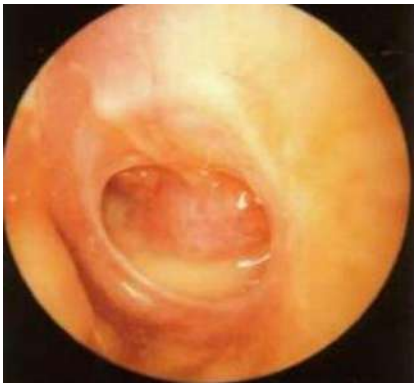
Small (pinhole) perforation anterior to the malleus in the left ear.

Inactive squamous COM = Retraction pockets



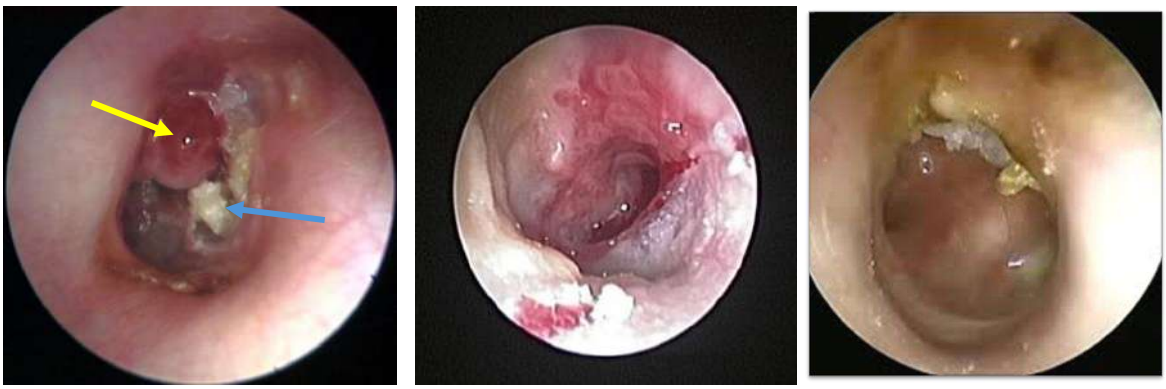
Retraction of most of the pars tensa.

Active mucosal COM / CSOM



Left ear with a central perforation and pus draining.

Active squamous COM / CSOM with Cholesteatoma



Three pictures of CSOM with cholesteatoma. The left picture shows the classical sentinel polyp (yellow arrow) and keratin (blue arrow).

Symptoms and signs of CSOM / Cholesteatoma

- Chronic otorrhoea
- Hearing loss
- Vertigo
- NVII paralysis
- Granulation in EAC
- Foul smelling otorrhoea
- Keratin visible (looks like dandruff)
- Not responding to treatment

Treatment of COM

- **CSOM**
  - Ear toilet
  - Topical ofloxacin / steroids or boric acid
  - Targeted anti-biotics according to MCS
  - Surgery
  - Optional for CSOM
- **Cholesteatoma**
  - As for CSOM, except always for surgery

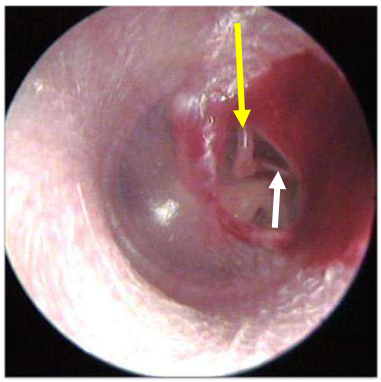
Reasons being that the cholesteatoma sac keeps on expanding and will erode through adjacent structures like

- Ossicles with CHL
- Tegmen with brain problems
- NVII
- Otic capsule with vertigo

Traumatic perforation

- History
  - Trauma history
  - Bloody discharge
  - Hearing loss
  - Minimal balance problems
- Treatment
  - Conservative
  - Majority will close within 4 weeks
  - Assault case

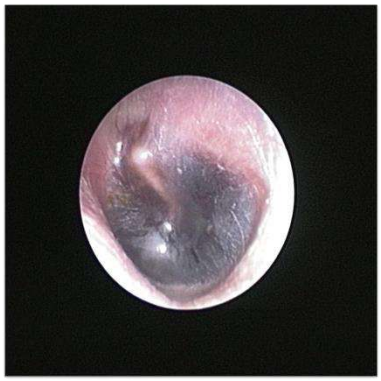
- Photo documentation
- Hearing test



Traumatic perforation of the right TM and blood in the external ear canal. Incus (yellow arrow) and stapes tendon (white arrow) are visible through the perforation.

Hemotympanum

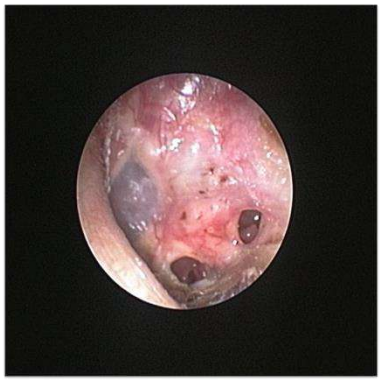
- Follow trauma
- Treatment
    - Wait
    - Grommet



Left ear with blood (old) behind TM.

TB

- Multiple perforations
- Exposed bone
- NVII palsy / paralysis
- Not responding to treatment
- Gene Xpert on discharge



Left ear with two perforations in the tympanic membrane.

HIV and the ear

- External ear canal
  - Otitis externa
  - Seborrhoea dermatitis
- Middle ear
  - Otitis media with effusion
  - Acute otitis media
  - Chronic otitis media
- Inner ear
  - Sensorineural hearing loss
  - NVII

Inner ear

Pathologies include

- Cochlea
  - Nerve deafness
  - Acute idiopathic nerve deafness (AIND)
  - Tinnitus
- Vestibulum
  - Vertigo
  - BPPV
  - Meniere's disease
  - Vestibular neuritis
- Other
  - NVII palsies / Paralysis
  - Trauma

Deafness

- Conductive
  - External ear canal
  - Tympanic membrane
  - Ossicles
  - Middle ear
- Nerve / Sensori-neural
  - Cochlea
  - Nerve
  - Brain
- Mixed
  - Tuning fork
- Rattle test
- Hearing test

Deafness in children (see hearing loss chapter)

- Can be classified as congenital or acquired
- Incidence of congenital hearing loss

    - 3/1000
- Screening – Otoacoustic emissions **(OAE)**
- Neonatal hearing screening may not identify children with progressive hearing loss, which accounts for 15–20% of preschool children with sensorineural hearing loss
- When these children and those with late onset hearing loss are taken into account, the overall incidence of paediatric SNHL is probably 50% higher than the figures quoted above

Hearing loss in adults

- Extremely common
- As a general rule one will lose 1 dB per year after the age of 35
- Causes
    - Age related
    - Noise exposure
    - Medications
- Refer semi-urgently if
    - **Unilateral with no other reasons to explain it**
    - **Associated with balance problems**
    - **Fluctuating**
    - **Associate with pulsatile tinnitus**
- Audiologist or ENT
    - Both

Acute idiopathic nerve deafness (AIND)

- **Definition**
    - **30 dB loss in three consecutive frequencies in less than 3 days**
- How does it present?
    - Sudden “blocked” feeling in one ear
    - THE PATIENT DOES NOT TELL YOU THAT HE / SHE CAN’T HEAR
    - Patient tries to open ear by
        - Fiddling with finger in the ear canal
        - Valsalva / popping the ear
- Commonly missed diagnosed with “middle ear effusion” or “acute otitis media”
    - Exclude middle ear effusions in your practice by asking the patient to Valsalva and seeing the movement of the tympanic membrane (or pneumo-otoscopy)
        - Examination of the ear will be normal
- Caused by a decrease in the blood supply to the inner ear or viral attack
- Needs urgent treatment
    - Oral steroids – 1 mg/kg for 7-10 days and then taper it over next 7-14 days
    - Anti-viral only if early in the disease (48 hours)
    - Refer to ENT. They might consider
        - Intra-tympanic steroids
        - MRI scan

Tinnitus

- Extremely common
- Can be divided into
    - Subjective / Objective
    - Constant / Episodic

        - If episodic
            - Pulsatile or non-pulsatile
    - Pitch
        - High / Middle / Low
    - Side
        - Unilateral / Bilateral / Whole head
    - Aetiology
        - Idiopathic versus definite cause
- In general
    - Mostly subjective
    - Mostly idiopathic
        - Implies cochlea / nerve / brain
- Can arise from external or middle ear pathologies like wax or middle ear effusions
    - ENT examination can exclude external or middle ear as a cause in the majority of patients
- Refer if
    - **Unilateral**
    - **Pulsating**
    - **Associate with other inner ear symptoms such as hearing loss and / or balance problems**
    - **Other central nervous symptoms**

Vertigo

See separate chapter

NVII palsies / paralysis (see NVII chapter)

- Most common pathologies are
    - Viral
    - Traumatic
- Always try to differentiate between
    - UMN vs LMN
    - Degree of involvement
    - Complete vs Incomplete vs Single affected branch
    - Other CN involvement or neurological fallout
- Always also examine
    - Middle ear status
    - Parotid status
- Always provide eye protection
- Document progression or resolution

Bell's palsy

- Aetiology = Idiopathic
    - Theories
        - Viral (Herpes simplex)
        - Ischemia
        - Auto-immune
- Pathophysiology = Entrapment Neuropathy
- Prognosis = very good
    - Complete paralysis= 75% will fully recover

    - Incomplete paralysis = 99% will fully recover
- Symptoms
    - LMN Affecting all branches
    - No other CNS diagnosis
    - No other cerebro-pontine angle lesions
    - No other ear or parotid lesions
    - Sudden onset (within 3/7)
    - Maximal weakness by 3/52
    - Improvement by 3/12
- Variable
    - Viral prodrome
    - Facial / Retro-ocular pain
    - Hyperacusis
    - Facial numbness
    - Dysgeusia
- Management
    - Eye care
    - Steroids
        - 1mg/kg for 10 days and taper up to 3 weeks
    - Addition of an antiviral
        - No evidence to support this
    - Surgery
        - In general, no role
- Follow up
    - Document degree (House Brackmann scale)
- Referral
    - If not improving at 4 weeks despite steroids
    - If fluctuating

Ramsey Hunt Syndrome

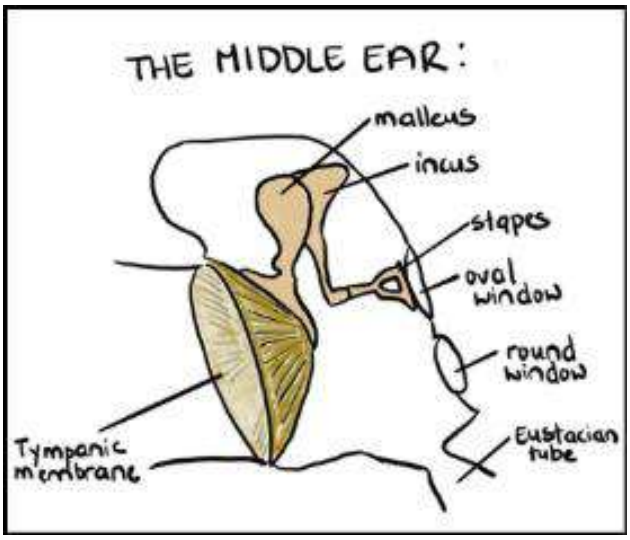
- A.K.A Herpes zoster oticus
- Definition
    - Herpetic vesicular rash of concha, EAC, Pinna
    - Ipsi-lateral CN7 LMN paralysis
- Pathology
    - Reactivated VZV ganglionitis
        - Geniculate ganglion
- Prognosis much poorer than Bell's palsy
    - Complete Palsies = 10% will fully recover
    - Incomplete Palsies = 66% will fully recover
- Clinical picture
    - Deep seated otalgia
    - Vesicular rash
    - Poly cranial nerve neuropathy
        - CN7, CN8, CN5
- Treatment
    - Acyclovir
    - Prednisone
    - Analgesia
    - Hearing and Vertigo rehabilitation

Traumatic CN7 injury

- Blunt vs Penetrating vs Iatrogenic
- Rarely isolated due to force needed to injure
- Big Question = Immediate vs Delayed onset
- Treatment
    - Life threatening injuries get preference
    - Immediate onset
        - Warrant early ENT referral for assessment
            - We will consider HRCT of temporal bone
            - Nerve decompression
    - Delayed onset
        - Steroids

8) Common middle ear conditions

Acute Otitis Media



Definition

Acute otitis media (AOM) is an inflammatory condition affecting the middle ear (ME). The incidence is high among children, especially between the ages of 6 months and 2 years. It is usually preceded by an upper respiratory tract infection (URTI). In adults, AOM is less common.

Subgroups:

- **SPORADIC episodes:** Associated with URTIs
- **RESISTANT AOM:** Persistence of signs & symptoms of ME infection beyond 3-5 days of antibiotic treatment
- **PERSISTENT AOM:** Recurrence of AOM within 6 days of finishing treatment
- **RECURRENT AOM:**

≥

3 AOM episodes over 6 months (OR) 4-6 episodes in 1 year

Aetiology

Between sixty to ninety percent of cases are associated with viral infections. The most implicated viruses include RSV, Influenza A, Parainfluenza, Rhinovirus or Adenovirus. Bacterial causes, ranked from highest to lowest incidence, include Streptococcus pneumoniae, Haemophilus influenzae, Moraxella catarrhalis, Streptococcus pyogenes, and Staphylococcus aureus.

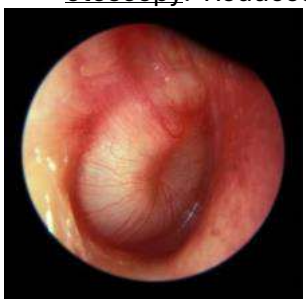
Infections can spread via the Eustachian tube due to negative middle ear pressure, which facilitates the movement of bacteria up the Eustachian tube; it can also occur via a perforation in the tympanic membrane or a grommet which is commonly seen after exposure to water; and lastly, spread can also be via haematogenous spread.

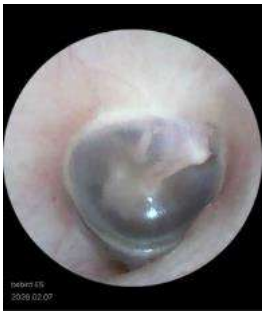
Risk Factors can broadly be classified in 4 main groups, this includes:

- **Genetic:** A family history of AOM (e.g. siblings or parents with a history of ear infections) increases the likelihood of developing this condition.

- **Immunological:** Immunological factors play a significant role in the pathogenesis and susceptibility to AOM. Studies indicate that children prone to AOM often exhibit specific humoral deficits, such as low levels of the IgG2 subclass or primary immunodeficiencies characterized by defective complement-dependent opsonization, which impairs bacterial clearance. At the molecular level, the aberrant expression of critical cytokines, specifically Tumour Necrosis Factor (TNF) and Interleukins, contributes to the inflammatory cascade, while the over-expression of mucin genes may alter mucociliary transport, leading to stasis and infection. Allergic predispositions also contribute to risk; specific dietary sensitivities like cow's milk allergy have been identified, and there appears to be a synergistic link between viral respiratory tract infections and allergies that predisposes the middle ear to disease. Furthermore, systemic immunocompromise alters the pathogen profile significantly; for example, children with advanced HIV and low CD4 counts show a notably higher incidence of *Staphylococcus aureus* infections compared to immunocompetent children.
- **Environmental risk factors:** Children attending daycare (crèche) are exposed to more infections due to close contact with others. The risk of AOM is higher in the winter months, which coincide with an increase in URTIs. The use of pacifiers, exposure to second-hand smoke, and certain dietary factors, such as a cow's milk allergy, can also predispose children to ear infections. Prematurity, not being breastfed, and living in low socioeconomic conditions are additional risk factors linked to a greater susceptibility to AOM.
- **Syndrome associated:** Syndromic children often experience Eustachian tube dysfunction, which can lead to otitis media with effusion (OME) and potentially AOM. However, it is debated whether AOM is due to the OME or underlying immunological deficiencies.

Signs & Symptoms

Symptoms	**Signs**
• Rapid onset otalgia • Headache • Hearing Loss • Otorrhea – mucopurulent (OR) blood-stained • Fever • Irritability/Excessive crying (OR) General malaise • Coryzal Symptoms • Vomiting • Poor feeding • Ear pulling • Clumsiness	• Appears unwell • Rubs ears • Otoscopy: ▪ Colour: opaque/yellow (OR) pink/red ▪ Tympanic membrane: Bulging (OR) Perforation with mucopururlent discharge ▪ Pneumatic otoscopy: Reduced mobility 



Normal right tympanic membrane.

Management

Management can broadly be divided into conservative and medical approaches:

Conservative management:

Primarily involves the use of analgesics and antipyretics to relieve pain and fever, which is recommended in uncomplicated cases. Without antibiotic treatment, the natural history of AOM shows that approximately 60% of children experience symptom relief within 24 hours, 80% within 2-3 days, and 88% within 4-7 days.

Medical Management:

Includes the use of antibiotics under specific situations; these specific situations are:

- Children under the age of 6 months.
- Children under the age of 2 years with recurrent AOM.
- Cases where there is no improvement after 2 days of watchful waiting.
    - Consider writing a script for antibiotics to be used if symptoms persist.
    - When symptoms are severe, such as high fever, vomiting, an atypical course, or complications.
    - Lastly, antibiotics are also advised for high-risk children, including those with syndromic conditions, craniofacial anomalies, congenital inner ear defects, or immunodeficiencies.
    - In a context where pt return to the medical services is not feasible, err on the side of prescribing antibiotics rather than not.

Appropriate antibiotic choice:
First line: Amoxicillin (80-90mg/kg/day) in 2 divided doses or Amoxicillin-Clavulanate (90mg/kg/day) in 2 divided doses – A 5-to-7-day course is typically sufficient.
B-lactam allergy: Cefuroxime (30mg/kg/day) in 2 divided doses or alternatively the macrolides – Azithromycin (10mg/kg/d) daily dose for 3 days or Clarithromycin (15-30mg/kg/day) in 2 divided doses for 5 days.

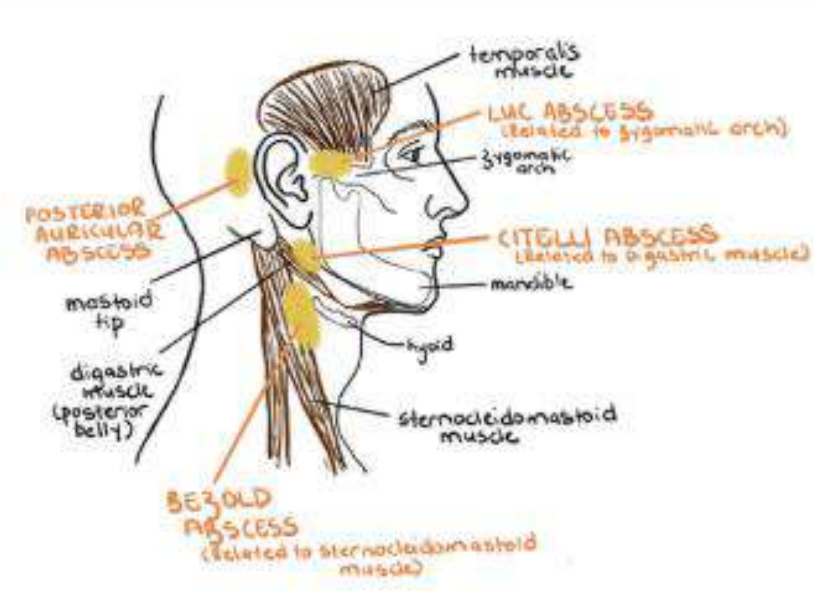
A **myringotomy and grommet** may be considered in cases involving complications, severe pain, or when a microbiological diagnosis is needed.

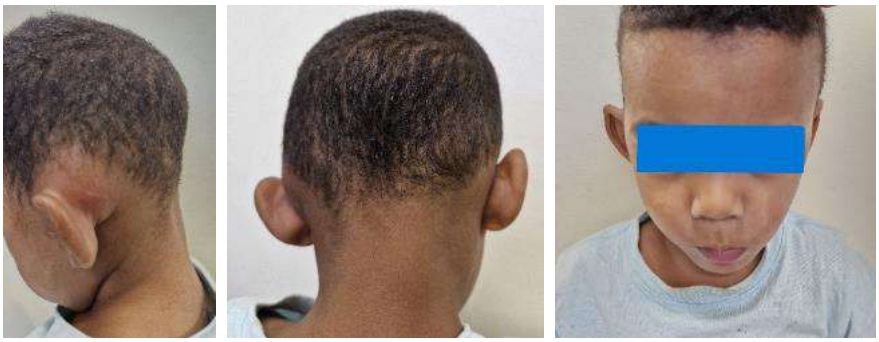
If symptoms do not improve after 2-3 days of treatment, consider changing the antibiotic therapy.

Complications

- Tympanic membrane perforation: Development of a purulent, bloody ear discharge and is often accompanied by immediate pain relief. Often these perforations heal spontaneously, however there is a risk that this may lead to chronic otitis media.

- Acute mastoiditis: Characterized by infection spreading from the middle ear to the mastoid air cells. The complications can be divided into temporal and extra-temporal complications
    - Temporal Complications:
        - **Labyrinthitis** – Infection spreads to the inner ear and can lead to hearing loss and balance disorders.
        - **Facial nerve paralysis** – This is a rare complication and is usually a neuropraxia caused by oedema leading to nerve compression or due the effects of bacterial toxic metabolites. These cases tend to resolve spontaneously.
        - **Pertrous apicitis** – Infection that spreads to the petrous portion of the temporal bone, presenting with otorrhea, retro-orbital pain and a sixth cranial nerve palsy (Gradenigo's syndrome).
    - Extra-temporal Complications:
        - **Posterior auricular subperiosteal abscess** – The most common type of abscess associated with acute mastoiditis. It typically presents with swelling and erythema located posterosuperior to the pinna (above the level of the tragus), accompanied by loss of the posterior auricular crease and displacement of the pinna forward and downwards. Other less common abscesses: **Citelli, Luc and Bezold**
        - **Intracranial complications**:
            - Meningitis
            - Abscesses – Extradural, Subdural and Intracranial





Note the left subperiosteal abscess with the forward and downward displacement of the pinna.

References

1. Peter A. Rea & Natalie Ronan. *Acute Otitis Media*. In Scott-Brown's Otorhinolaryngology and Head and Neck Surgery, Eight Edition, edited by John C Watkinson and Raymond W Clarke, pg. 137 – 153, CRC Press, 2018
2. Anil Banerjee. *Acute Otitis Media and Otitis Media with Effusion in Adults*. Otorhinolaryngology and Head and Neck Surgery, Eight Edition, edited by John C Watkinson and Raymond W Clarke, pg. 971 – 976, CRC Press, 2018

Chronic Otitis Media

Introduction and Definition

Chronic: > 12 weeks duration
Otitis: inflammation (with or without infection) of the ear
Media: in the middle ear

Chronic otitis media (COM) implies a **permanent abnormality of the tympanic membrane (pars tensa or flaccida**) most commonly resulting from earlier acute otitis media, negative middle ear pressure, or otitis media with effusion.

The disease is a major global health issue, with the World Health Organization estimating that it affects between **35 million to 45 million people in Sub-Saharan Africa alone.** A disproportionate amount of this burden falls on **children**.

While COM is prevalent globally, there are distinct racial variations; for instance, Inuit Eskimos have a significantly lower annual incidence of cholesteatoma—approximately 5 per 100,000—compared to Northern European Caucasian populations

Classification of COM

Classification (simplified)

- Chronic suppurative otitis media (CSOM)
- CSOM with a cholesteatoma
- CSOM due to tuberculosis
- Healed COM
  - Tympanosclerosis
  - Healed perforation
    - Thinning and/or local or generalized opacification of the pars tensa without perforation or retraction

Clinical assessment aims to categorise the ear into one of five diagnostic categories based on otoscopic findings (Table 1).

Table 1: Clinical Classification of COM

Category		Synonyms	Otoscopic Findings
Healed COM		Tympanosclerosis; healed perforation	Thinning and/or opacification of the pars tensa; no perforation or retraction.

Mucosal COM	Inactive	Perforation	Permanent perforation of the pars tensa; middle ear mucosa is not inflamed.
	Active	'CSOM'	Permanent defect of the pars tensa with inflamed, discharging mucosa.
Squamous COM	Inactive	Retraction	Retraction of the pars flaccida or tensa with potential to become active.
	Active	Cholesteatoma	Retraction that retains squamous debris (keratin), often with inflammation and pus.
Tuberculous COM			Double perforations (rarely seen), pale granulation tissue, (facial nerve palsy)

Aetiology and Pathogenesis

The development of COM is multifactorial, influenced by:

- **Eustachian Tube Dysfunction:** Poor aeration of the middle ear leads to negative pressure and retraction of the tympanic membrane.
- **Infection:** Common pathogens in active COM include ***Pseudomonas aeruginosa***, ***Proteus*** spp., and ***Staphylococcus aureus***.
- **Socioeconomic Factors:** Prevalence is higher in lower socioeconomic groups and manual workers.
- **Environmental Factors:**
    - lack of breastfeeding
    - passive smoking
    - daycare attendance

Nice to know: Molecular studies suggest that innate immunity (toll-like receptors), cytokines (TNF-α, IL-1β), and even gastro-oesophageal reflux (GORD) may play roles in the persistence of middle ear inflammation.

Subtypes of Chronic Otitis Media

Mucosal COM (Inactive and Active) - (CSOM without cholesteatoma)

- **Inactive mucosal COM**
    - Permanent **dry perforation**.
    - Main symptom: hearing loss (conductive).
- **Active mucosal COM**
    - Chronic inflammation of the mucosa
    - Main symptoms:
        - **Recurrent otorrhoea** (discharge)
        - Hearing loss (conductive).
        - **Aural polyps** (granulation tissue)
            - May protrude through the perforation and are indicative of active disease.
            - Discharge may be blood stained

Squamous COM (Cholesteatoma) - (CSOM with cholesteatoma)

Cholesteatoma is a **benign keratinizing epithelial-lined cystic structure** found in the middle ear and mastoid. It is "active" when it retains keratin debris and becomes infected.

- **Pathogenesis:** Most cholesteatomas arise from **retraction pockets** in the pars flaccida or the posterosuperior quadrant of the pars tensa.
- **Bone Destruction:** Cholesteatomas can destroy ossicles, otic capsule, facial nerve canal, and skull base - leading to serious complications.

A rare but distinct subgroup is eosinophilic otitis media, which occurs primarily in patients with bronchial asthma and is characterised by a thick, yellow, viscous effusion and a T-helper type 2 dominant inflammatory predisposition

Tuberculous COM – (CSOM with TB)

See more below

Clinical Assessment

History

The primary symptoms are **hearing loss** (~80%) and **ear discharge** (~70%).

- Pts with COM should not have otalgia – If present - think of complications or referred otalgia

Also ask about:

- The nature of the discharge (foul-smelling discharge is common in cholesteatoma).
- Symptoms of complications, such as **otalgia, vertigo**, **facial weakness** or **severe headache**.

Examination (Otoscopy)
(see also chapter on ear examination)

NB: wax or discharge must be cleared to visualise the TM. Aural toilet ('syringing') is the safest method to this and **may be done in actively discharging ear.** Do not do aural toilet for a dry perforation or ear trauma (with a dry or blood stained perforation).

- **Perforations:** Note the size and position (site of pathology).
- **Retractions:** if they are adherent to middle ear structures or eroding the bone. Ask patient to perform Valsalva while doing otoscopy to see if retraction is mobile or not.

Investigations

- **Tuning fork tests** (Weber, Rinne)
- **Audiology:**
  - Preferable to have a dry ear prior to hearing test
  - Pure-tone audiometry is essential to assess the degree of conductive and/or sensorineural hearing loss.
- **Imaging:**
  - Not routine
  - May be requested by ENT in specific situations. Patients with suspected complications (e.g. mastoiditis) will require contrasted CT scans.
- **Ear swabs**
  - Not routine
  - May be useful if not responding to topical treatment or in the presence of complications
  - Washing for GXP (see note on TB COM later)

Management

The principle of management for the GP is to get the ear dry/inactive and refer to ENT for surgical management. An ear with cholesteatoma may never become dry with medical management alone

Medical Management (Primarily for Active Mucosal COM)

- **Aural Toilet:** Regular cleaning of the ear is the first step.
  - Ear syringing
  - Dry mopping – can include video here
- **Topical Medication:**
  - Acetic acid (ineffective)
  - Boric acid powder – Video link https://youtube.com/shorts/fGeZzGU8oQA
  - Topical antibiotic-steroid drops (gold standard)
- **Quinolones** (e.g. ciprofloxacin) are highly effective and superior to systemic antibiotics.
  - May be combined with steroid (dexamethasone) – either as a combination drop (not available in state) or combination drops.
  - Instil 4–6 drops into the affected ear twice daily. The patient should lie on the unaffected side for 5–10 minutes after instillation. Gentle tragal pressure (tragal pumping) should be applied to facilitate penetration into the middle ear (ME).

NB: systemic antibiotics are not indicated for COM, unless there is a complication or otorrhoea which does not resolve on the correct treatment described above.

Surgical Management

- **Myringoplasty:** Reconstruction of the tympanic membrane perforation to prevent further infection and potentially improve hearing.
- **Ossiculoplasty:** Reconstruction of the ossicular chain using prostheses or the patient's own remodelled ossicles.
- **Mastoidectomy:** Mandatory for **cholesteatoma** to eradicate disease and create a safe, dry ear.
  - *Canal Wall-Down (CWD):* Removes the posterior canal wall, resulting in a mastoid cavity.
  - *Canal Wall-Up (CWU):* Preserves the canal wall but has a higher risk of recurrent cholesteatoma.

Complications

Complications are more likely in patients with active squamous disease (cholesteatoma).

Extracranial Complications

- **Mastoid Abscess:** The most common extracranial complication.
- **Facial Nerve Paralysis:** Caused by erosion of the bony facial canal and infection of the nerve.
- **Labyrinthitis & Labyrinthine Fistula:** Infection spreading to the inner ear, causing sensorineural hearing loss and vertigo.

Intracranial Complications

- **Meningitis:** Presents with fever, headache, and neck stiffness.
- **Brain Abscess:** Most commonly occurring in the temporal lobe or cerebellum.
- **Lateral Sinus Thrombosis:** Infection spreading to the intracranial venous sinuses.

The annual risk of a 30-year-old patient with COM developing an otogenic brain abscess is estimated at 1 in 10,000.

Notes on Tuberculous COM

While TB COM is a rare form of COM, in the South African setting where TB is endemic, the astute doctor must always consider it, especially in COM that does not respond to conventional treatment.

'In South Africa, any patient with a runny ear and a facial nerve palsy has TB until proven otherwise'

- **Classical triad:**
- **Multiple TM perforations**
- **Painless otorrhoea**
- **Facial nerve palsy**

In reality, the findings are often more subtle.

Things to look out for:
Otoscopic Signs
- Abundant "Pale" Granulations:
- Eroded Ossicles: happens much faster in TB than in mucosal COM.

Disproportionate Finding
- Hearing Loss Disproportionate to Exam: hearing loss that seems far worse than the physical damage seen on otoscopy. *This is due to the toxins or the infection directly affecting the inner ear (labyrinthitis) early in the disease course.*
- Resistance to Treatment: to respond to standard antibiotic ear drops or systemic antibiotics.

Investigations:
- Washings of ear discharge – send for GeneXpert
    - 1ml sterile saline
    - Aural toilet
    - Collect the turbid fluid
    - Send in sterile specimen container for GXP and TB MC&S.
- CXR and sputum GXP – concurrent PTB is estimated to affect 50% of the patients with TB ear

Management:
- Systemic TB treatment as per national guidelines for extrapulmonary TB.
- Perforation often heals on treatment.

Key Points

- COM is a **permanent abnormality** of the tympanic membrane.
- **Cholesteatoma** requires surgical intervention because of its bone-eroding nature.
- **TB** COM is a rare but important type of COM in South Africa
- **Topical quinolones** are the treatment of choice for active mucosal discharge.
- Any patient with COM and **vertigo, facial palsy, or severe headache** must be treated as a medical emergency.

References

Browning, G.G., Weir, J., Kelly, G. and Swan, I.R.C. (2018) 'Chronic Otitis Media', in Watkinson, J.C. and Clarke, R.W. (eds) *Scott-Brown's Otorhinolaryngology and Head and Neck Surgery*. 8th edn. Boca Raton: Taylor & Francis Group, pp. 977–1014.

World Health Organization (2021). *World report on hearing*. Geneva: World Health Organization. Available at: https://iris.who.int/handle/10665/339913 (Accessed: 15 January 2026).

Otitis media with effusion (OME)

Introduction

The terms "Glue ear", "Middle ear effusion (MEE) and Otitis media with effusion (OME) are all used interchangeably to describe the presence of non-purulent fluid in the in middle ear and mastoid air cell system WITHOUT signs of acute infection. It is very common in children, and it often occurs after an episode of acute otitis media (AOM). It may persist for a few weeks, but generally it resolves spontaneously within 3 months. Unilateral OMEs are more common, while bilateral are less common and tend to persist.

Epidemiology

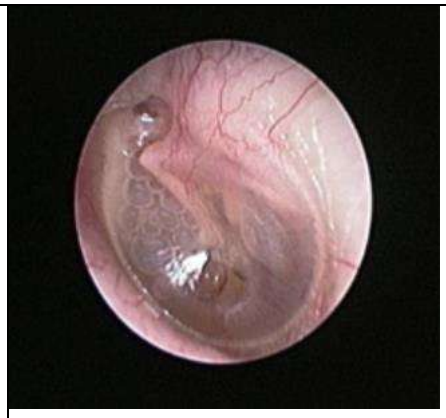
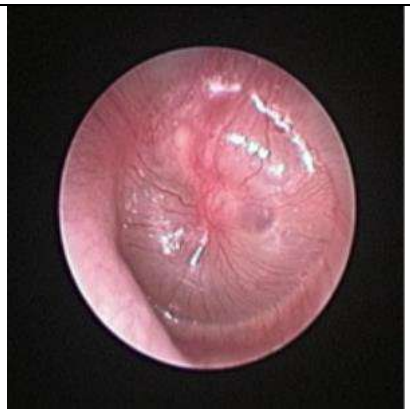
OME shows a bimodal age distribution, with the first and largest peak at 1-2 years of age. This is typically when children start attending creche or expand their social contact at playgrounds. The second peak is at 3-7 years when they start attending primary school. Episodes are more common in winter months, likely due to an increased incidence of upper respiratory tract infections (URTI) and AOM, along with closer household contact. Thus, the main determinants of OME are age and the season of the year.

Aetiology

Proposed contributors include post-viral URTI-related Eustachian tube dysfunction, allergy, craniofacial abnormalities (notably cleft palate and Down syndrome), and possible contributions from adenoids (due to mechanical obstruction and biofilms). Gastro-oesophageal reflux disease (GORD) has been proposed as a contributor (via refluxate reaching the nasopharynx and travelling up the Eustachian tube to the middle ear), but the evidence is still limited, thus anti-reflux treatment is not recommended purely for OME. Other risk factors overlap with those for acute otitis media (e.g., age/season/URTI exposure).

Signs & Symptoms

Symptoms	**Signs**
Hearing loss	**Otoscopy:** Often difficult to diagnose Dull (OR) Retracted tympanic membrane Fluid level in middle ear Reduced movement with pneumatic otoscopy or Valsalva

	  **Tuning fork tests:** Could be suggestive of conductive hearing loss (CHL) – Rinne negative & Weber localizing to affected ear. Although, this may be difficult to elicit in a child.

Investigations

Tympanometry typically shows a type B (flat) or type C tympanogram. Audiometry often shows a conductive hearing loss.

Management

Counselling and Communication Strategies:

Counselling should primarily offer reassurance that OME is a benign condition with a high rate of spontaneous recovery and no serious long-term sequelae. Most cases resolve within 3 months, but recurrence is common.

It is vital to extend this guidance beyond the parents to include teachers and child minders. Practical communication tactics should be adopted to assist the child, such as gaining their attention before speaking, minimizing background noise, and facing the child directly to facilitate lip-reading. Speakers should maintain a normal volume, speed, and emphasis, but position themselves as close to the child as possible.

Medical Management:

Current medical evidence suggests that pharmaceutical interventions are generally ineffective for OME. Intranasal corticosteroids, systemic steroids, nasal decongestants, and mucolytics have shown no benefit and are not recommended. Furthermore, antibiotics are unlikely to be beneficial and are discouraged due to the risk of increasing antibiotic resistance.

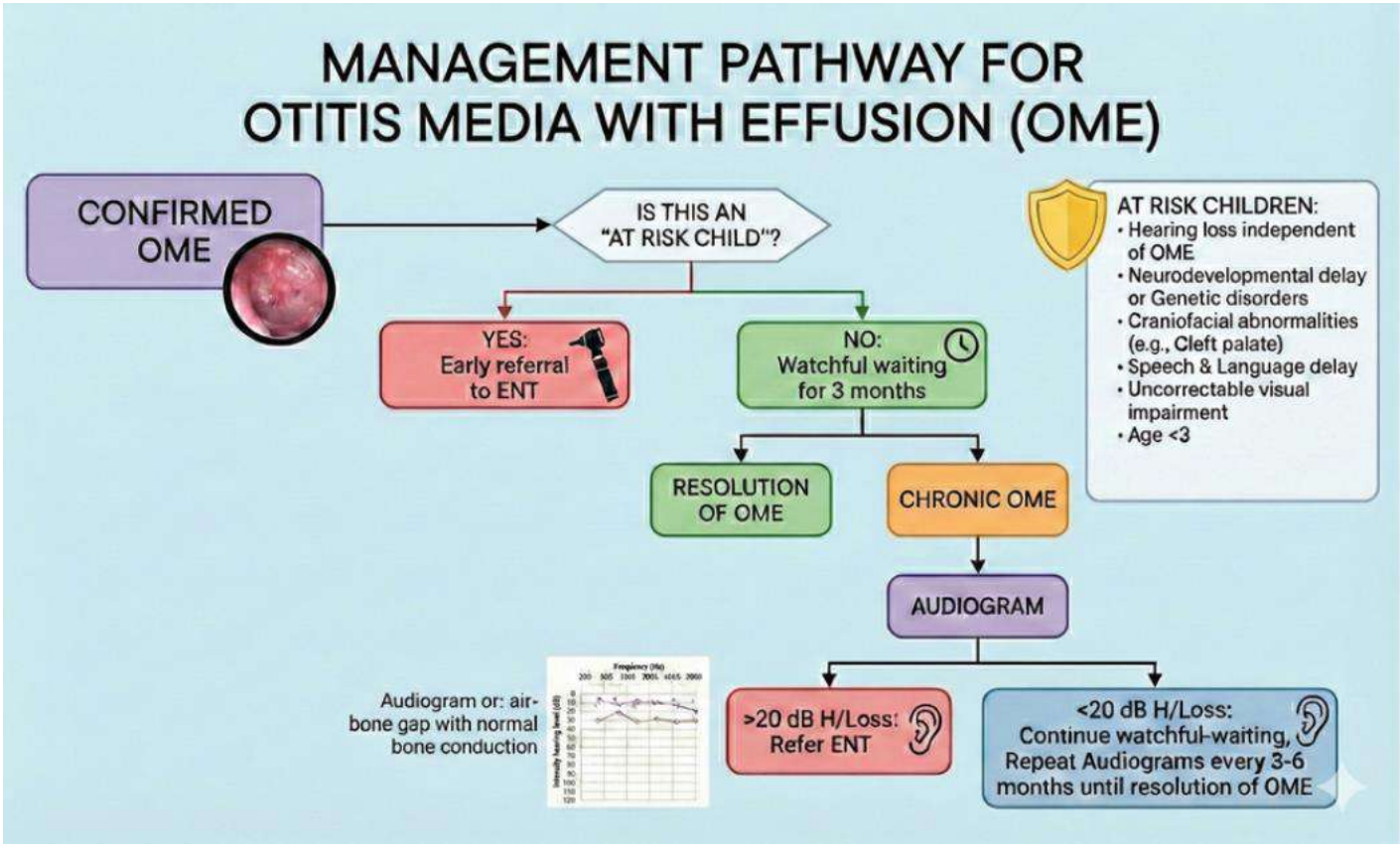
Non-Surgical Approaches:

Auto-inflation (Valsalva / Otovent®) is considered a great tool to augment "watchful waiting". If the child is too young to perform a Valsalva, the Otovent® is a useful alternative. Instruct the subject to put the balloon nozzle snugly into one nostril, take a deep breath through the mouth, then close mouth and other nostril before blowing through the nose to blow up the balloon. The ears should make a soft ‘pop’.

Hearing aids, specifically softbands, can be used as an alternative to grommets if parents prefer, or for syndromic children (such as those with Down's syndrome or Cleft palate). However, this approach is more time and cost-intensive than surgery.

Surgical Approaches:

If surgery is indicated, myringotomy alone is ineffective as incisions close rapidly, therefore ventilation tubes (grommets) are required. Grommet extrusion usually occurs within 6 to 12 months. Some children may need repeat procedures. Complications can include displacement, infection (best managed with topical quinolones), myringosclerosis and tympanic membrane perforation. An adenoidectomy is often considered at the time of grommet insertion because adenoids contribute to OME through biofilms and physical obstruction of the eustachian tube.



References

1. Peter J. Robb & Ian Williamson. *Otitis Media with Effusion*. In Scott-Brown's Otorhinolaryngology and Head and Neck Surgery, Eight Edition, edited by John C Watkinson and Raymond W Clarke, pg. 115 – 135, CRC Press, 2018

9) Hearing Impairment

Introduction

Hearing impairment is a relative term, ranging from a mild loss of hearing to complete deafness. Hearing loss has a profound effect on the human being. Total deafness isolates the individual from this environment. Even mild hearing loss may severely impact a child's development. Hearing impairment may occur at any age, from the neonate to the geriatric patient.

Hearing loss in the elderly

In our setting, the commonest cause is presbycusis which translates to hearing loss due to age. All people will have worsening of hearing thresholds as they get older. In general, most people will lose on average 1 dB / year after the age of 35 years. The approach is therefore to do an ENT examination, tuning fork tests, and to exclude other simple, reversible causes.

If your patient complains of progressive bilateral hearing loss usually with constant tinnitus, your ENT examination is normal and Rinne tests are positive, you have confirmed the diagnosis of presbycusis. Typically, one can differentiate the patient who continues to struggle despite an increase in the loudness versus the patient who struggles to hear and then says don't speak so load. The former group is due to retro-cochlear loss, and they would typically have poor speech discrimination. The latter group is due to cochlear loss, and the complaint is known as recruitment.

Remember that the three inner ear symptoms are hearing loss, tinnitus, and vertigo. The presence of all three should raise the suspicion of a more sinister underlying problem, especially when the symptoms are unilateral, the tinnitus pulsatile in nature, and there is presence of vertigo. These patients should be referred to an ENT specialist.

Once the diagnosis of presbycusis is established, provide the patient counselling and refer for a hearing aid trial. It is important to establish a working relationship with your audiologist. Frequently, older patients are fitted with hearing aids, but they struggle to find benefit in real world situations. Rather have them pay for the ear mould and use a demo hearing aid for 2-4 weeks before committing to pay for them. Recently, older patients are also considered for cochlear implants, especially if their thresholds at 1 kHz exceed 70dB.

Other common causes are wax impaction and middle ear effusions. REMEMBER that in adults who present with a new onset middle ear disease / effusion, nasopharyngeal pathology should be excluded. This is usually done by an ENT specialist by performing a nasal endoscopy. Nasopharyngeal carcinoma typically presents with middle ear disease in an adult, as well as cervical lymphadenopathy.

Hearing loss in the child

The early diagnosis of hearing impairments in children is vital so that the resultant speech, communication, educational and psychological handicap can be minimised through amplification and intensive education. It is better to over-suspect hearing loss in a child rather than to miss the diagnosis. The testing of hearing is very difficult in young children and increasing reliance is being placed on Otoacoustic Emissions (OAEs) and Auditory Brainstem Response Audiometry (ABR), which are objective tests.

The capacity of the human brain to learn to interpret sound and speech (plasticity) is unique to the child below 6 years of age. If they are not exposed to sound before the age of 18-24 months, they can never learn to understand speech. If hearing loss is corrected before the age of 18 months, educational outcomes are the same as normal hearing children. However, if hearing remains reduced or not corrected at all, they will never catch up with normal

hearing children. Cochlear implant outcomes correlate directly with the age of hearing correction, so much so that implanting a completely deaf child after the age of 2-3 years will not lead to speech development. Therefore, hearing loss in a child, especially < 18 months, is an EMERGENCY and should be referred immediately! Ideally every child should undergo newborn hearing screening.

Approach to hearing loss – all ages

Milestones and history
Normal speech milestones are given in the table one below.

Table 1. Speech milestones.

Age	Task
0 – 4 months	Startled by sounds (even Moro reflex) Quietens when hearing sounds ("listen")
4 – 6 months	Turns to localise sounds once neck control is achieved
9 months	Listens Respond to name Mimics environmental sounds
12 months	Uses one word – girls a bit earlier, boys a bit latter
18 months	Babble rhythmically Understands instructions
24 months	Uses 20 words

When taking the history, always believe the mother (caretaker)! An easy acronym to assess risk factors for hearing loss in a young child is HEARING. It stands for;
H – Heredity
E – Ear abnormalities / syndromes
A – Asphyxia
R – Intra-venous or other ototoxic medications
I – Infections such as TORCH / Meningitis
N – Neonatal ICU (also ask about oxygen requirement and jaundice)
G – Growth. Babies below 1500 grams.

Older children might have other risk factors, and ones that influence the Eustachian tube are the most important. These include the adenoids, skull base deformities (Down syndrome), passive smoking, and structural problems with the muscles of the soft palate and Eustachian tube (cleft palates). Otherwise, ear problems are more common in boys, winter months, family history of ear disease, and primary and secondary immune diseases.

The doctor would need to think if it is congenital or acquired and whether it is sensori-neural, conductive, or mixed hearing loss. Congenital and acquired is discussed under aetiology below in more detail. When referring to a deafness as sensori-neural, conductive or mixed one can use the following approach:

- Sensori-neural
    - Cochlea
        - Outer hair cells / Inner hair cells / Synapse
        - Cochlear / Retro-cochlear
    - Nerve
        - Cochlear nerve
    - Brain
        - Cochlear nuclei / Olivary complex / Lateral lemniscus / Inferior colliculus / Medial geniculate body / Cortex (ECOLI mnemonic)
    - Auditory nerve spectrum disorder

        - Inner hair cells / Synapse / NVIII
    - Mechanical inner ear deafness
        - Acoustic neuroma
- Conductive
    - EAC
        - Obstructions / Wax / Exostosis / Etc
    - TM
        - Perforation / Stiffness / Anterior blunting / Myringosclerosis
    - Middle ear
        - Ossicles
            - Tympanosclerosis / Dislocation / Subluxation / Erosion / Fixation / Fracture
            - Congenital (Dysplasia, Hypoplasia, Aplasia)
            - Increased mass – Paget's disease / Osteopetrosis
        - ME cavity
            - Pressure / Fluid / Mass / Fibrosis / Atelectasis
        - Oval / Round window obliteration
            - Congenital / Otosclerosis / Tympanosclerosis
        - Third window
            - Superior SSC / Lateral SSC dehiscence / Disease associated (cholesteatoma / TB / Syphilis)
- Mixed

While taking a history, it is important to note factors mentioned in table two.

Table 2. Characteristics of hearing loss.

Characteristics		
Onset	Pre-lingual	
	Post-lingual	
Severity	Mild	21-40 dB
	Moderate	41-60 dB
	Moderately severe	61-80 dB
	Severe	81-100 dB
	Profound	>100 dB
Frequency	Low	<500 Hz
	Middle	501-2000 Hz
	High	>2000 Hz
Pattern	Sloping	Typical pattern in presbycusis
	Rising	Meniere's
	Cookie-bite	Think congenital
	Notched	Noise induced hearing loss if at 4 kHz
	Saucer	
	Falling	Acute idiopathic nerve deafness
	Flat	Metabolic / Metabolic presbycusis
Clinical	Progressive / Acute idiopathic nerve deafness / Fluctuating / Stable	

Aetiology

A differential diagnosis for congenital hearing loss is given in table three. It will never be expected of you at GP level to known this and it only serves for reference value.

Table 3. Congenital hearing loss

Congenital hearing loss				
Genetic (50%)	Syndromic (30%)	Mono-gene	Autosomal dominant (AD)	Waardenburg Stickler NFII Treacher Collins
			Autosomal recessive (AR)	Pendred Usher Jervell
			X-linked	Alport Norries
			Other	Osteogenesis imperfecta Crouzon Apert
		Chromosomal		Down syndrome Turner syndrome
		Mitochondrial		
		Heterogeneous		Goldenhaar Klippel-Feil
	Non-syndromic (70%)	Mono-gene	AD (18%)	
			AR (80%)	Connexin 26 (50%) Connexin 30,31,43 Other
			X-linked (<2%)	Usually with Stapes fixation
			Other	
		Chromosomal		
		Mitochondrial		Aminoglycosides DM
		Heterogeneous		
Acquired (25%)	Pre-natal	TORCH – Toxoplasmosis, Rubella, Cytomegalovirus, Herpes		
	Natal	Asphyxia / Medication / Infections (meningitis) / NICU / Low birth weight / Jaundice		
	(Post-natal)	See later for full differential diagnosis		
Idiopathic (25%)				

A differential diagnosis for acquired hearing loss is given in table four. This can also be applied to adults.

Table 4. Acquired hearing loss.

Acquired hearing loss				
Infections	Viral – HZ / VZ / Measles / Mumps / CMV / EBV / Influenza / Rubella / Hep / HIV	Bacterial – Meningitis / AOM Strep / Staph / H Infl	Labyrinthitis – Viral / Via AOM	Other – Syphilis / Lyme / Rocky Mountain spotted fever / Rickettsia / Malaria / Parasites / Toxoplasmosis
Granulomatous	TB / Histiocytosis / Fungal / Sarcoidosis / Syphilis / Wegener's (GPA)			
Auto immune	Cogan / PAN / Relapsing polychondritis / GPA / Primary inner ear AI disease / Para-neoplastic / Temporal arthritis / SLE / RA / Sarcoidosis / Scleroderma / HIV			
Vascular / Haematological	Emboli / Anaemia / Coagulation problems / Migraine / Vertebrobasilar occlusions / Waldenstrom / Cryoglobulinemia / Sickle cell / Leukaemia / Lymphoma / Loops			

Noise	Constant / NIHL / Blast trauma				
Neurological	MS / Friedreich ataxia / ALS /				
Medication	Aminoglycoside / Loop diuretics / Quinine / Salicylate / NSAIDS / Vanco / Erythromycin / Cisplatin / Vincristine / Vinblastine / Eflornithine / Deferoxamine				
Metabolic	DM / Hypothyroidism / Mucopolysaccharidosis				
Tumours	Acoustic neuroma / Meningioma				
Trauma	Head (fractures, concussion, penetrating) / NIHL / Barotrauma / Perilymph fistulas / Radiation				
Toxins	Mustard gas / Heavy metals				
Temporal bone diseases	Bone diseases / Metabolic / Granulomatous / Auto immune / Neoplastic				
Presbycusis	Neural – Normal STO and reduced SD	Sensory – Classical high tone loss, and later progress to other frequencies	Metabolic – Flat HL in young	Mechanical – Inner ear conductive component	
Inner ear diseases	Meniere's				
Oxygen	Hypoxia				

Congenital ear abnormalities

The inner ear embryologically develops separate from the middle ear and external ear canal. In roughly 25% of cases inner ear congenital abnormalities will overlap with middle ear and ear canal abnormalities. Isolated middle ear abnormalities are extremely rare and occur in less than 1% of cases. Any congenital abnormality can be divided into:

- Isolated (75%)
    - Teratogens
- Genetic
    - Syndromic
    - Non-syndromic

Table five below gives a more detailed description of congenital abnormalities, but this is just for reference and too detailed for a general practitioner.

Table 5. Congenital ear abnormalities

Congenital ear abnormalities				
Inner ear	Membranous	Complete	(Siebenmann-Bing)	Jahrsdorfer 10 points: • Stapes – 2 • Oval window 2 mm – 1 • Round window 1 mm – 1 • ME space > 3mm from prom to atretic plate – 1 • NVII – 1 • Malleus-incus – 1 • Incus-stapes – 1 • Mastoid pneumatisation – 1 • EAC – 1
		Limited	Cochleo-saccular (Scheibe) / Cochlear basal turn	
		Complete	(Michel)	

<table>
<tr><td rowspan="7"></td><td rowspan="7">Membranous and osseus</td><td>Cochlear</td><td colspan="3">Aplasia / Hypoplasia / Incomplete partition (Mondini) / Common cavity</td></tr>
<tr><td>Labyrinthine</td><td colspan="3">Aplasia / Hypoplasia / Dysplasia</td></tr>
<tr><td rowspan="2">Aqueduct</td><td>Vest aqueduct (VA)</td><td>Measure against post SSC >1.5-2mm</td><td>Associated with:
Isolated – Large VA syndrome
With cochlear abnormalities (Mondini)
With syndromes – Pendreds / BOR</td></tr>
<tr><td colspan="3">Cochlear aqueduct</td></tr>
<tr><td>IAM</td><td colspan="3">Narrow / Wide</td></tr>
<tr><td>NVII</td><td colspan="3">Aplasia / Hypoplasia</td></tr>
<tr><td>ME</td><td>25% also inner ear abnormalities 25% due to syndrome <1% in isolation</td><td>Stapes, Incus, Malleus, oval or round window – hypo, dys-, aplasia</td><td>Abnormalities of stapedial artery, carotid, jugular, NVII</td><td colspan="2">Types of stapes fixation:
Genetic
Syndromic – Treacher Collins / Branchio-Oto-Renal / Klippel-Feil / Crouzon
X-linked
Isolated
Trauma / Post surgery / Tympanosclerosis / Infections</td></tr>
<tr><td>EAC</td><td colspan="5">Schuknechts classification A – Fibro-cartilage narrow, B – A + TM / Malleus abnormalities, C – Total atresia, no TM, Malleus-Incus fixed, Stapes mobile, D – A + B+ C + extreme ossicle abnormalities, NVII abnormalities, Poor pneumatisation</td></tr>
<tr><td>Pinna</td><td colspan="5">Minor (Bat, Cupped, Lop, Constricted) / Microtia / Anotia</td></tr>
<tr><td>Hillocks</td><td colspan="5">Pre-auricular sinus / fistulas / pits</td></tr>
<tr><td>Branchial cleft</td><td colspan="5">Works I – Anterior to lobule, parallel to EAC, blind ending, only skin. Works II – Mandible angle, curves superior, true duplication because contain skin and cartilage</td></tr>
</table>

In practice

Take a thorough history, do a proper ENT examination which includes the tuning fork tests. In children always think about other neuro-developmental milestones and record that in your notes. Be careful of the very young child with pre-lingual hearing loss. Arrange for urgent otoacoustic emission (OAEs) to confirm or exclude congenital sensori-neural hearing loss and when in doubt rather refer to an ENT specialist / Audiologist. Children between the ages of 2 – 9 years of age, with post-lingual onset deafness is usually due to middle ear conditions (OME / AOM / Perforation). Remember that AOM has two peak incidences namely between 6 – 24 months and between 4 – 7 years of age. The first peak coincides with the immaturity of the ET due to its fairly horizontal orientation in the young skull. The latter peak is due to the adenoid and / or tonsil lymphoid tissue being at its most active. This then leads to middle ear conditions and mostly conductive hearing losses. Lastly, you will frequently see older people, and this has been discussed above.

Special investigations

Hearing tests

The following can be used:

- PTA
- Tympanometry
- Acoustic reflexes
- OAEs
- ABR
- In children also
    - Rattle test
    - Behavioural observational audiometry (0-6 months of age)
    - Visual reinforcement audiometry (6 months – 2 years)
    - Play – conditioning audiometry (2 years – 5 years)

Bloods

Genetic testing is becoming more available and also as panel tests. However, the clinical implications are still limited. Recently the first congenital genetically deaf children were given a reverse transcriptase viral vector to restore hair cells in the cochlea with positive results.

Radiology

The following can by useful

- MRI for pulsatile tinnitus, AIND, cerebro-pontine angle pathology, pre-cochlear implantation, congenital inner ear abnormalities
- CT for tinnitus, congenital inner ear abnormalities

10) The Facial Nerve: Anatomy, Approach to Palsies & Management

The facial nerve, also known as the **seventh cranial nerve (CN VII)**, plays a critical role in both motor and sensory functions of the face. It is responsible for the movement of facial muscles, enabling expressions such as smiling, frowning, and blinking. Beyond its motor functions, the nerve also provides sensory input for taste from the anterior two-thirds of the tongue and contributes to the secretion of tears and saliva through its parasympathetic fibres.

Facial nerve palsy refers to a condition in which there is weakness or paralysis of the facial muscles due to damage or dysfunction of the nerve. This condition can arise from a variety of causes, ranging from viral infections (e.g. Bell's palsy) to trauma, tumours or neurological diseases. It can have significant physical and emotional impacts on patients, affecting both facial function and appearance.

This chapter aims to provide an in-depth review of:

1. The anatomy and functions of the facial nerve.
2. The clinical presentation and classification of facial nerve palsies.
3. Management strategies

By the end of this chapter, readers will gain an understanding of the facial nerve's importance, the implications of its dysfunction, and the principles of managing facial nerve palsies.

Anatomy:

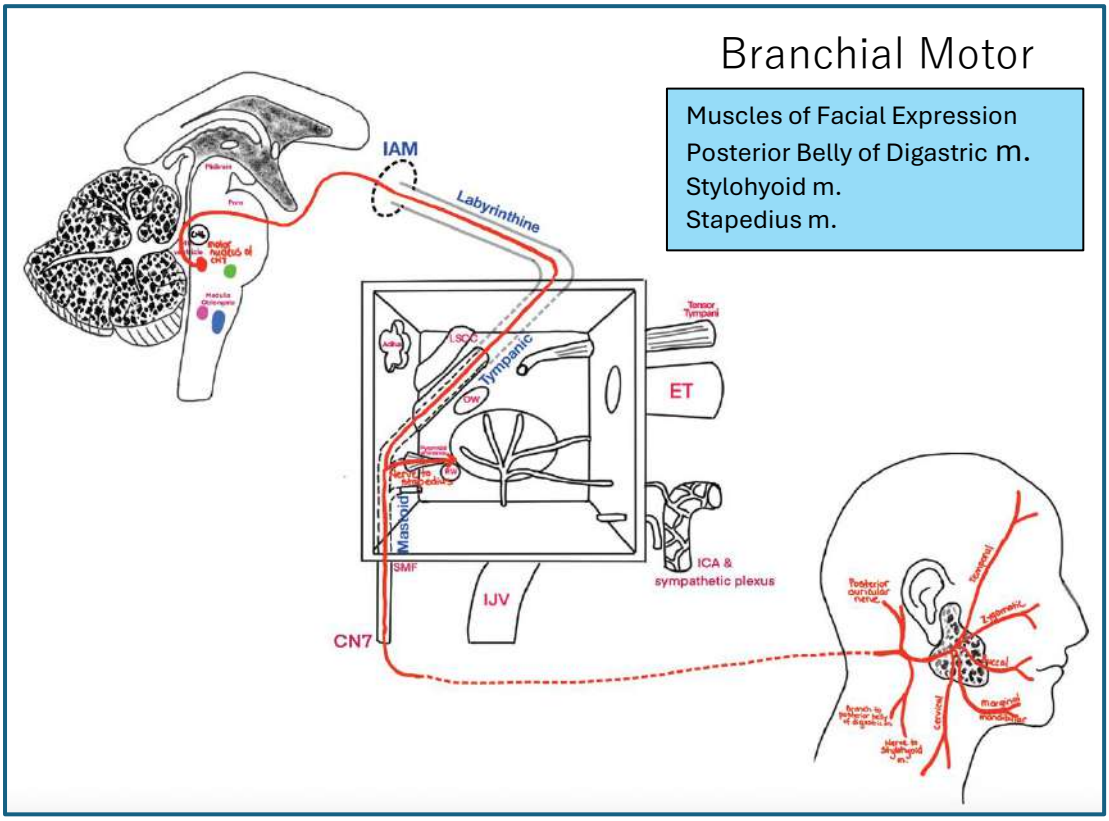
To understand facial nerve palsies, it is important to first understand the anatomy of the nerve in order to localize the site of injury. The facial nerve has a complex course – it begins at the brainstem and travels through the temporal bone via the internal acoustic meatus (IAM) to reach the facial canal. The facial canal has 3 parts, this includes the labyrinthine, tympanic and mastoid segments. Once the facial nerve exits the temporal bone via the stylomastoid foramen, it gives rise to its external branches.

Brainstem – 4 Components:

Efferents (Located in pons):	Afferents (Located in medulla)
Branchial Motor (Motor nucleus of CN 7)	General Sensory (Spinal nucleus of trigeminal)
Visceral Motor (Superior salivatory nucleus)	Special Sensory (Gustatory nucleus)

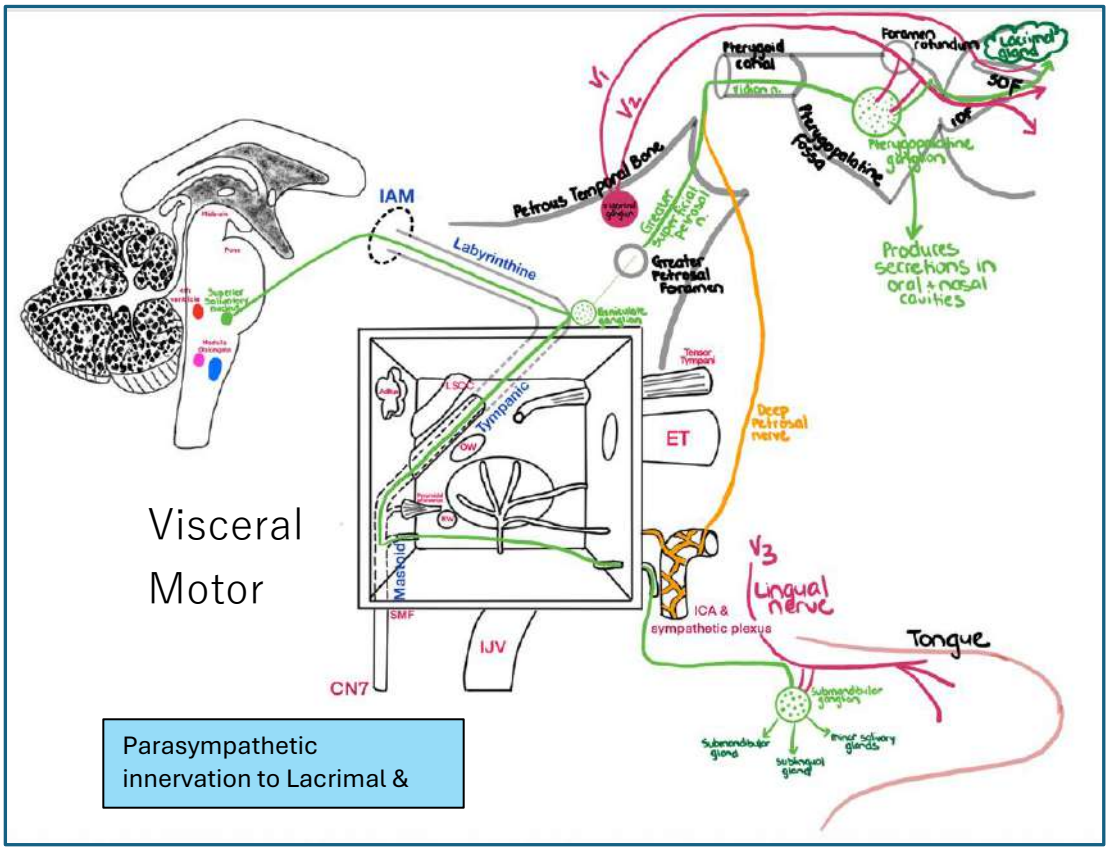
Branchial motor:

Begins at the Motor nucleus of CN 7 where it courses backwards towards the floor of the 4th ventricle and loops around the CN 6 nucleus. The significant branches that this component gives off include:
Within the temporal bone: Nerve to the stapedius muscle
External branches:
Immediately after exiting the stylomastoid foramen: Posterior auricular nerve, Branch to posterior belly of digastric muscle & Branch to stylohyoid muscle
Branches given off within the parotid gland: Temporal, Zygomatic, Buccal, Marginal mandibular and Cervical nerve.



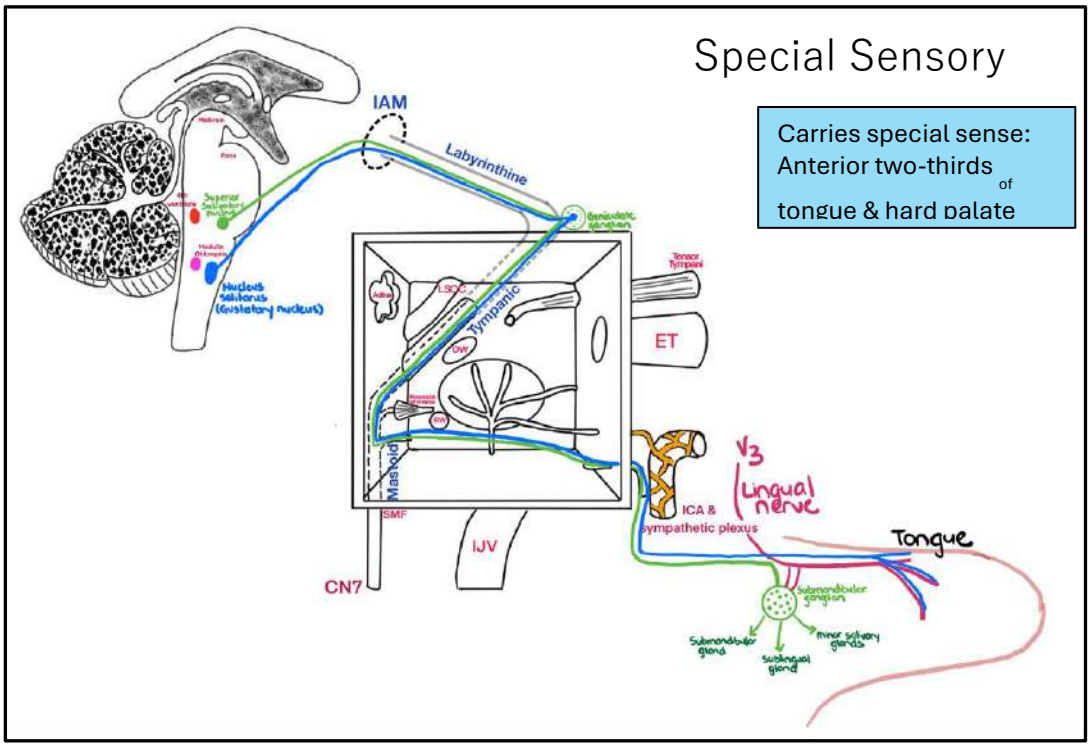
Visceral motor:

Pre-ganglionic fibres arise from the superior salivatory nucleus and travels via the IAM to reach:
Geniculate ganglion
Submandibular ganglion (via the chordae tympani nerve)
They provide parasympathetic innervation to:
Lacrimal gland
Salivary glands – this includes, the minor salivary glands of nasal and oral cavity and the submandibular and sublingual glands



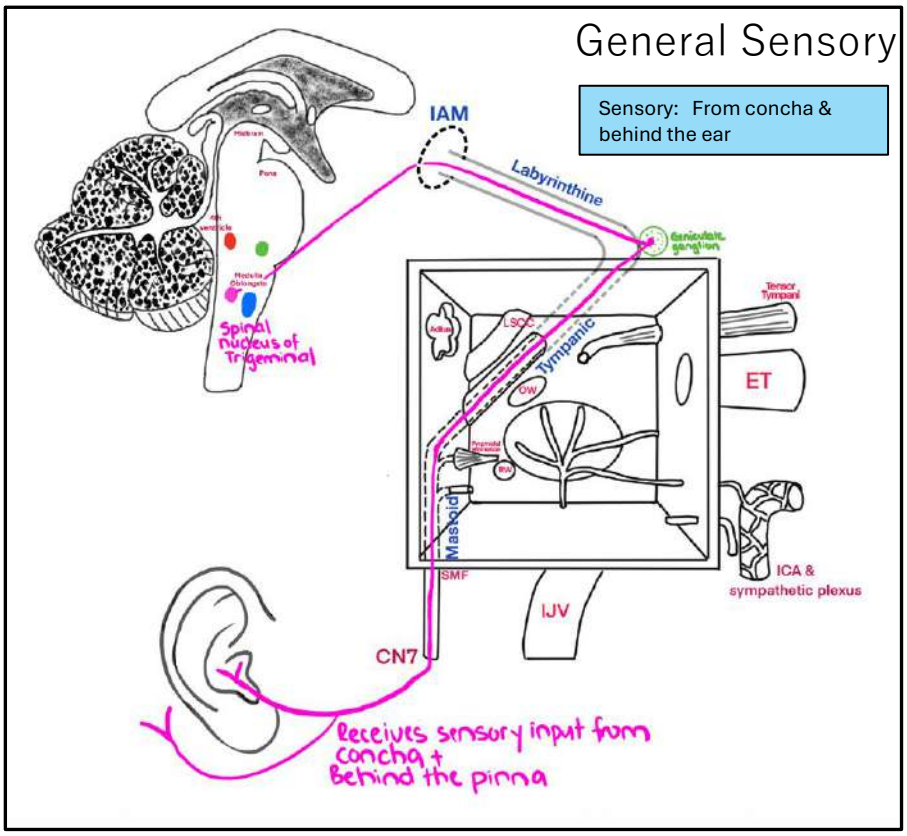
Special sensory:

Information from the taste buds in the anterior two-thirds of tongue is carried via lingual nerve, then chordae tympani towards its cell body in the geniculate ganglion. This information then travels via the IAM towards the gustatory nucleus in the medulla oblongata.



General sensory:

Sensory information from the skin of concha and behind the ear travels through the stylomastoid foramen and the facial canal to reach the geniculate ganglion, where the nerve cell body is located. From there, the information continues through the internal acoustic meatus (IAM) to the spinal nucleus of the trigeminal nerve.



Facial nerve palsies

Facial nerve palsies can be classified as:
Peripheral (lower motor neuron): Affects the entire side of the face, including the forehead.
Central (upper motor neuron): Spares the forehead due to bilateral cortical innervation.

Nerve injury classification

Nerve injuries can be classified according to Seddon or Sunderland Classification

Seddon	Sunderland	Pathology	Prognosis
Neuropraxia	Grade 1	Area of blockage of nerve conduction. May have segmental demyelination, but no axonal damage.	Complete recovery
Axonotmesis	Grade 2	There is loss of axons, however, endoneurium, perineurium and epineurium remain intact.	
	Grade 3	As above but endoneurium is also disrupted	Partial recovery
	Grade 4	As above but perineurium is also disrupted	Spontaneous nerve recovery is unlikely and will likely require surgery.
Neurotmesis	Grade 5	As above but epineurium is also disrupted – i.e. Nerve transection.	

Common causes of facial nerve palsies

Birth	Infective	Traumatic	Neoplastic
Forceps delivery Dystrophia myotonica Moebius syndrome Facial nerve agenesis	Otitis media – Acute or Chronic Necrotizing otitis externa Encephalitis Herpes zoster oticus (Ramsay hunt syndrome) Chicken pox Mumps Infectious mononucleosis/ Epstein-Barr virus HIV Tuberculosis Lyme's disease	Base of skull fractures Facial nerve trauma – Penetrating injury to face or middle ear	Parotid tumours Facial nerve tumour Glomus jugulare Glomus tympanicum Meningioma Carcinoma Haemangioblastoma Leukaemia Cerebral Lymphoma
Metabolic	**Idiopathic**	**Iatrogenic**	**Toxins**
Diabetes Mellitus Hyperthyroidism Pregnancy Hypertension Acute porphyria Vitamin A deficiency	Bell's Palsy Guillain-Barré syndrome Melkerson-Rosenthal syndrome Sarcoidosis Granulomatosis with polyangiitis	Middle ear & Mastoid surgery Parotid surgery CPA angle tumour surgery Use of local anaesthesia for blocks Embolization	Tetanus Diphtheria Carbon monoxide Thalidomide

History

1. Onset & Duration
When did the weakness start?
Was it sudden or gradual and is it progressing?
Have you had similar episodes before? If yes, was it the same side that was affected last time?

2. Distribution & Symptoms
Is the weakness affecting the entire side of the face or just part of the face?
Have you noticed changes in taste, hearing or tearing?
Any drooling or difficulty eating?

3. Associated Symptoms
Any pain around the ear or face?
Any rash or vesicles? (Suggestive of Ramsay Hunt Syndrome)
Any associated dizziness, hearing loss or tinnitus?
Any recent infections, fevers, or upper respiratory tract infections?
Any trauma or recent surgery – especially around the ear or parotid gland?
Any recent travel or tick bites? (Concern for Lyme disease in endemic areas)
Any history of headaches or visual disturbances? (Possible neurological cause)

4. Medical History
History of Diabetes mellitus or Hypertension? (Risk factors for ischemic cranial nerve palsies)
Any history of strokes?
Any autoimmune conditions? (e.g. **sarcoidosis, Guillain-Barré syndrome, myasthenia gravis**)
History of cancer? (concern for metastatic or parotid malignancies)

Focused Examination of a patient with Facial nerve palsy

Differentiate between upper motor neuron (UMN) and lower motor neuron (LMN) facial nerve palsy. Forehead sparing is generally related to an UMN facial nerve palsy.
Record the degree of facial weakness – Most commonly used grading system is the House-Brackmann Staging System.

House-Brackmann Staging System						
Grade	**Degree of injury**	**Function**	**Eyes**	**Mouth**	**Forehead**	**Re-innervation**
I	Normal	Normal	Normal	Normal	Normal	Normal
II	Mild	Mild weakness	Complete closure with minimal effort	Slight asymmetry with maximal effort	Reasonable function	Synkinesis barely noticeable Contracture/Spasm absent
III	Moderate	Obvious weakness, not disfiguring	Complete closure with maximal effort	Asymmetric movement with maximal effort	Slight to moderate movement	Non-disfiguring synkinesis, mass movement or spasm
IV	Moderately severe	Obvious disfiguring weakness	Incomplete closure	Asymmetric movement with maximal effort	None	Severe synkinesis, mass movement & spasm
V	Severe	Motion barely perceptible	Incomplete closure	Slight movement of corner of mouth	None	Absent
VI	Total paralysis	No movement	Total paralysis			Absent

Examine the head & neck and the ears
If patient has a partial facial nerve palsy – check for a parotid gland lesion
Examine for any other cranial nerve fallout
Check for any associated symptoms:
Impaired vision or iritis -? Sarcoidosis
Look for secondary effects complicating facial nerve injuries: Synkinesis, Contractures, Crocodile tears, Epiphora, Dysgeusia, Pain and Hyperacusis

Investigations

Electrophysiological tests

These tests are not done in cases of incomplete paralysis. It is done to assist with making decisions regarding facial reanimation surgery.

Options available include:

Electroneurography (ENoG)	Electromyography (EMG)
Measured between >3 days to 3 weeks	**Complementary to ENoG after 2 weeks**
Wallerian Degeneration is the process of denervation of the neural fibres; this takes about 72 hours to be completed. This is why we wait 3 days before the study is done. Two electrodes are used for this test: The **stimulation electrode** is placed near the stylomastoid foramen where the facial nerve exits the skull, and the **recording electrode** is placed near the nasolabial fold to record the electrical responses of the facial nerve muscles. The response is recorded as a compound muscle action potential (CMAP) at the nasolabial fold. The test compares the amplitude of the CMAP from the affected side to the healthy side. The percentage response is calculated by dividing the amplitude of the paralysed side by the amplitude of the normal side. A low percentage degeneration (<25%) suggests a good prognosis with a high likelihood of recovery and a high percentage degeneration (>90%) suggests a poor prognosis with a reduced chance of recovery. The ENoG may be repeated at intervals of 3 to 5 days to monitor nerve recovery or deterioration. After 21 days, the predictive value of an ENoG is inaccurate as nerve regeneration and collateral nerve development will begin.	It is often used after several months of paralysis to assess muscle re-innervation. Small needles are inserted into the facial muscles – usually on the orbicularis oculi and orbicularis oris. Electric activity is measured during rest and voluntary contraction. Interpretation of results: **Reinnervation:** Muscles show polyphasic innervation potentials. **No Reinnervation:** Fibrillation potentials are seen which suggest muscles are alive but not receiving nerve signals. Can consider surgical exploration in such cases. **Chronic denervation:** Silence on EMG, thus suggesting irreversible muscle denervation

Bloods

Are not usually necessary in truly idiopathic cases. However, if one wanted to exclude less common causes can consider: ACE/ANCA/HIV/Lyme disease serology/Syphilis serology

Imaging

CT Scan:
HRCT Temporal bone can follow the facial nerve from the internal auditory meatus (IAM) to the stylomastoid foramen (SMF).
Contrasted CT Neck is useful if the pathology is intra-parotid

MRI:
An MRI can provide better soft tissue detail than a contrasted CT scan and can show enhancement of the facial nerve to suggest underlying pathology

Management

Proper eye care is essential:
Lubrication: Use artificial tears during the day and ocular lubricants and eye tape at night
Eye patch

Other option: Upper eyelid weighting
Physiotherapy: Facial exercises help to relax the facial muscles and help to improve the asymmetry
Botox: Helps to release over-tightened facial muscle and can be used to weaken more active "normal" facial muscles in order to achieve a more balanced facial appearance
Facial reanimation surgery is only considered if there is a lack of recovery after 1 year. This procedure is done by Plastic surgery.
Psychological counselling and support groups may be useful

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11) Tinnitus

Definitions

The sensation of hearing a sound that originates involuntarily in the head of its owner, in the absence of an external stimulus

- May be **pulsatile** or **non-pulsatile**
- Pulsatile tinnitus may have an intracorporeal sound source
- **Subjective** or **objective**
- There is some ambiguity as the definition technically includes auditory hallucinations of mental illness, which is **not** considered to be a type of tinnitus
- Sound can take any form: usually simple (e.g. humming, whistling, ringing, notes), but may be complex (e.g. indistinct music)

Non-Pulsatile Tinnitus

- Idiopathic Tinnitus = commonest form of non-pulsatile tinnitus
- *[Prevalence: 10.1% UK adults (spontaneous persistent tinnitus lasting ≥5min)*
    - *Prevalence increases with age, but decreases in severity*
    - *Male: Female = 1:1]*
- Predictors of Tinnitus:
    - Hearing loss (especially high frequency Hearing Loss)
    - Noise exposure (up to 3x increase in tinnitus compared to no noise exposure)
    - Advancing age (although tinnitus seems to plateau at 70 years of age)
- [Risk Factors:
    - Smoking and ETOH use
    - Previous Head injuries
    - Cardiovascular disease / Hypertension (HPT)
    - Otologic conditions: Meniere's disease, otosclerosis, vestibular schwannoma
    - Drugs: salicylates, quinine, aminoglycosides, platinum based anti-neoplastic drugs
    - Diet: higher caffeine consumption may be **protective against tinnitus**
- Associated Comorbidities: Depression and Anxiety; Disorders of sound tolerance (e.g. hyperacusis: 40% tinnitus sufferers report hyperacusis; 86% hyperacusis sufferers report tinnitus)

Pathophysiology of Tinnitus

1. **Ignition site** = peripheral or central location in the auditory system where the initial tinnitus signal is generated
2. **Central promotion** = central auditory mechanisms which must be present for the generated signal to be misconstrued as a sound

[Numerous theories exist outlining mechanisms which lead to tinnitus:

- ***Peripheral mechanisms:***
    - *Discordant damage of cochlear hair cells: outer hair cells damage while inner hair cells remain intact*
    - *Calcium channel dysfunction: drugs (salicylates and quinine) and noise exposure affect intracellular calcium levels, which may cause depolarization*
- ***Central mechanisms:***
    - *Increased spontaneous neural activity in the auditory cortex*
    - *Central neural synchrony and reorganization of the cortical auditory map: Peripheral auditory damage results in increased synchrony of spontaneous cortical activity → may result in tinnitus*

- *Habituation: the central auditory system should habituate tinnitus over time, although this may not happen when there is high autonomic arousal (basis of tinnitus retraining therapy → working with autonomic nervous system, limbic system, reticular system)]*

Investigations

1. Audiometry with pure tone audiogram and tympanometry
2. Imaging: **Not for everyone.** Consider an **MRI** when concern of retro-cochlear pathology
    a. Unilateral tinnitus
    b. Asymmetrical sensorineural hearing loss (with no clear cause)
    c. Associated neurological symptoms (vertigo, cranial nerve fallout)
3. *[Tinnitus Questionnaires: useful in research and assessing response to management*
    *a. Tinnitus Handicap Questionnaire; Tinnitus Handicap Inventory (THI); **Tinnitus Functional Index***
    b. Visual analogue scales
    c. Hospital Anxiety Depression Scale (HADS)]

Treatment

- Multiple options available, but poor evidence base supporting these due to a lack of RCTs (randomized control trials)
- Probably placebo effect involved
- There tends to be natural improvement in tinnitus over time due to habituation

1. Explanation and Reassurance
    - Avoid negative counselling i.e. "there is nothing we can do"
    - Education and information about the condition, natural history etc. supported by RCTs
2. Hearing Aids
    - Useful in cases where there is associated hearing loss (intervention of choice)
    - Amplification of external sounds may mask tinnitus
    - Indirect benefits: enhance communication, reduce associated anxiety and stress
3. Sound Therapies (part of TRT)
    - Sound can mask tinnitus in 95% patients in a clinic setting
    - Complete masking of tinnitus is counterproductive as it limits habituation
    - Low level sound (just below tinnitus threshold) may aid in habituation to tinnitus signal
4. *Ultrasound*
    - *High-frequency sound applied by a bone conduction transducer → meant to stimulate cochlea without interfering with hearing sounds within the normal auditory spectrum*
    - *Promising initial study results*
5. Combination Therapies: Tinnitus Retraining Therapy (TRT)
    - Combining sound therapies with counselling, cognitive behavioural therapy (CBT) promote habituation and seem to show good results
6. Alternative Medicine
    - Relaxation techniques have proven benefit: Acupuncture, Aromatherapy, Herbal medicine, Massage, Hypnotherapy, Meditation, Yoga, Thai Chi etc. (seem to promote relaxation)
    - Ginkgo biloba 480mg daily dose shows some promising results
    - Vitamin supplementation: Vitamin Bs (theoretically protect cochlea against noise trauma); zinc (highest concentration of zinc in the human body is in the cochlea → but limited evidence of response to supplementation)
7. Systemic Medication
    - Tricyclic antidepressant; Serotonin reuptake inhibitors: useful in cases with coexisting depression / anxiety
    - Benzodiazepines: limited use due to dependence. May have some benefit in treating tinnitus
    - No strong evidence: anticonvulsants, antispasmodics, betahistine, hyperbaric oxygen therapy

- Intravenous Local Anaesthetic: invasive but provides temporary central suppression of tinnitus (not practical long-term solution)

8. ***Regional Drugs***
    - *Botulinum toxin: has shown non-paralytic benefits in treating migraines and neuropathic pain (blocks acetylcholine release as well as other neurotransmitters in the autonomic pathway). One small study showed benefit to Botulinum toxin injection around the ear (not statistically significant as only 26 participants)*
9. ***Intratympanic Drugs***
    - *Direct labyrinthine drug absorption with theoretically improved labyrinthine metabolism - but will only target the peripheral pathways (i.e. cochlear pathology)*
    - *Potential use in cases of **sudden onset sensorineural hearing loss, acute noise trauma, acute otitis media***
    - *Drug options: Steroids, local anaesthetic, non-ototoxic NMDA antagonists]*
10. Surgery
    - For specific conditions associated with tinnitus:
        - otosclerosis (stapedectomy eradicates associated tinnitus in 80-88% patients)
        - Cochlear implantation for profound hearing loss (86% tinnitus improvement on implanted side; 67% on contralateral ear)

Pulsatile Tinnitus

- Refers to sound that is not continuous (clicking, pulsation, fluttering)
- Classified as: **Synchronous** or **Non-synchronous** (based on timing with patient's pulse)

Synchronous Pulsatile Tinnitus

Causes of Synchronous Pulsatile Tinnitus		
PATHOLOGY TYPE		EXAMPLES
Vascular	**Arterial**	Carotid artery atherosclerosis / dissection / stenosis Arteriovenous malformation / fistula Intracranial aneurysm Vascular anomalies of the ear Vascular compression of CNVIII (lateral)
	Venous	Jugular bulb abnormalities Dural venous sinus stenosis / diverticulum Abnormal emissary veins of mastoid Idiopathic tinnitus
Microvascular		Glomus tumour Paget's disease Cholesterol granuloma of middle ear Meningioma of middle ear Cavernous haemangioma
Circulatory		Increased Cardiac Output: anaemia, pregnancy, thyrotoxicosis
Perceptual		Conductive hearing loss Cochlear trauma

Other	Benign Intracranial Hypertension Superior Semicircular Canal Dehiscence

INVESTIGATION:

- Bloods: FBC; Thyroid function; bHCG (especially when bilateral)
- Imaging: depends on underlying aetiology
    - Contrasted CT temporal bone, brain: if retrotympanic mass seen
    - Duplex Carotid Ultrasound: Atherosclerosis
    - MRA/MRV "Time of Flight": Intracranial venous and arterial anomalies
    - Gold standard = formal angiography but this is invasive, therefore decision of imaging depends on clinical picture

TREATMENT

- Supportive
    - Counselling and Reassurance
    - Sound therapy
- Surgery
    - Targeted at identified cause: Microvascular decompression of vascular loops; decompression of sigmoid sinus dehiscence; coiling / tying off vascular anomalies
    - Variable results: 40-77% improvement of tinnitus

Non- Synchronous Pulsatile Tinnitus

= buzzing / fluttering sounds that are not related to the patient's pulse

PATHOLOGY	FEATURES
Middle Ear Muscle Myoclonus	- Always subjective tinnitus - May have impedance changes on audiogram over time - Tensor tympani / Stapedius muscle

Palatal Muscle Myoclonus	- Can be objectively audible to others
	- Irregular clicking
	- Associated with involuntary palatal muscle movements which can be seen (transorally or transnasally)
	- 2 forms:
	  - Symptomatic palatal myoclonus = associated with brainstem lesions
	  - Essential palatal myoclonus = idiopathic, isolated condition

Otologic: Middle ear	- Patulous eustachian tube - Ossicular chain pathology; Middle ear effusion - Otosclerosis - Semicircular canal dehiscence
Joint Disorders	- Temporomandibular joint disorder

- ***INVESTIGATION:***
    - *Audiogram to assess impedance changes over time (middle ear myoclonus)*
    - *MRI if palatal myoclonus: to exclude brainstem lesions*
- ***TREATMENT:***
    - *Conservative therapies as for non-pulsatile tinnitus*
    - *Pharmacotherapy: Benzodiazepine, Orphenadrine; Carbamazepine; Botulinum toxin*

- *Failed above: can consider surgical division of stapes and tensor tendons]*

SUMMARY:

12) Vertigo

In general, we will not expect of you to have a thorough knowledge of Vertigo. This fairly comprehensive review aims to serve as a reference source for you.

Part I – History and examination

Introduction

Vertigo and dizziness are complex problems, which is poorly understood by most practitioners. In a busy practice or casualty setting, attending to a dizzy patient can be difficult to say the least. Traditional teaching places an extreme emphasis on distinguishing vertigo from dizziness, unsteadiness, oscillopsia, and presyncope. The problem however is that patients often have no clue as to what this means and would use these terminologies completely incorrectly and interchangeably.

It is estimated that approximately 90% of individuals over 65 years of age have visited their physician at least once for vertigo as their primary complaint, and the lifetime incidence is 30%. A diagnosis can be made in 70% of patients on history alone. Physical examination and special tests will only contribute 10-20% respectively. Vertigo can be successfully treated in 90% of patients. Unfortunately, patients are often over investigated, misdiagnosed and given a cocktail of medications.

My aim is to provide you with some basic anatomy and physiology knowledge, but more importantly a stepwise approach to a dizzy patient. Of course, it is very easy to over-complicate or simplify this topic which is of no value to you. At the end you will be able to distinguish vertigo from other forms of dizziness. You will also be able to differentiate vertigo further into peripheral and central pathologies and advise the correct treatment options. Complex cases should however be identified and referred to a specialized unit.

Anatomy and Physiology

Each labyrinth consists of the vestibular apparatus and the cochlea. The vestibular apparatus can be divided into three semi-circular canals, and the otolith organ which is composed of the utriculus and sacculus (figure 1).

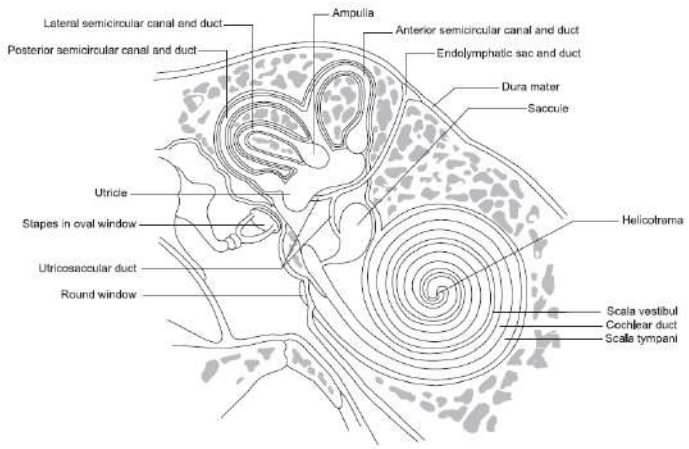

Figure 1. Inner ear anatomy

The semi-circular canals register angular rotations, and the otolith organs linear changes and gravitational direction. The information from the ears, along with visual and proprioceptive information forms the major input to the balance system as depicted in figure 2.

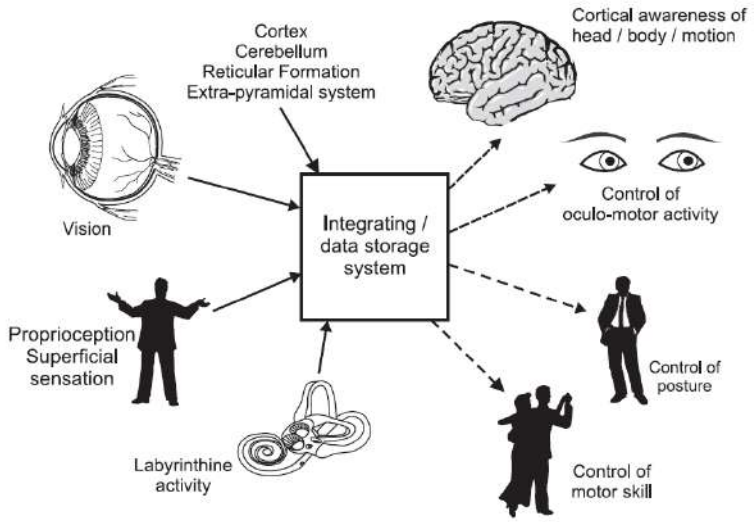

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Figure 2. Input and output of vestibular system

The vestibular end organs are dynamic structures. They are not silent until stimulated but constantly discharge at a resting pattern of signals to the brain. The two sets (right and left) of vestibular apparatus are mirror images of one another. With any acceleration / deceleration movement the opposite but equal change in firing rate takes place on the contra-lateral side. The cerebral cortex interprets the change in firing rate as movement of specific direction and speed. The vestibular nuclei have important connections to various regions in the brain, but three of the most important are to the ocular nuclei, cerebellum and spinal tracts. In response to vestibular stimulation, the eyes will move in the opposite direction to retain the field of last gaze or otherwise known to reduce retinal slip as not to cause blurred vision (see figure 3). This allows for movement without becoming dizzy or feeling off-balance while still maintaining clear vision. This is the basis of what is referred to as the vestibular-ocular reflex (VOR). Examination of the VOR is of vital importance in the dizzy patient as will be later shown. So, at its core, the vestibular organs change mechanical energy to electrical energy and activate the VOR. This produces extremely fast (< 80 milliseconds) compensatory eye movements to keep focus on a target. It is far quicker than conscious eye movements, known as neurological eye movements or ocular-motor signs.

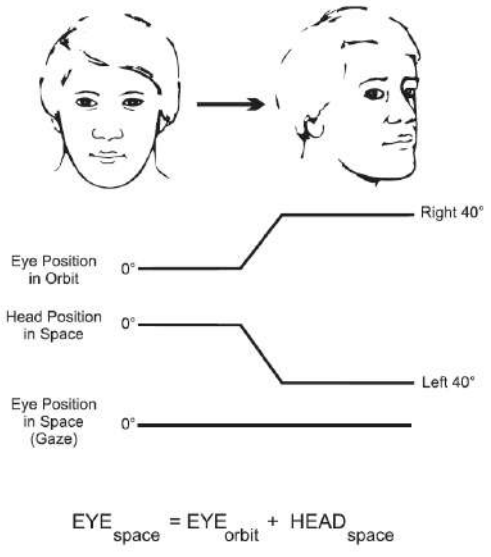


Figure 3. Vestibular-ocular reflex

Vestibular-spinal tracts will adjust trunk and limb muscles, and the cerebellum adjust muscle tone to meet the new situation. Over time the brain has learned exactly what to expect from the vestibular organs, partly instinctual but mostly learned as a baby learns to walk etc. The vestibular organs are therefore in constant dynamic balance, one checking the other and working as a team.

Disease strikes

With a sudden pathological dysfunction of one vestibular organ (mostly hypofunction), the two sides discharge at unequal rates. This unbalanced information is interpreted by the brain as a condition of constant motion. This is the basis of our definition of vertigo, namely a hallucination of movement which will be rotatory in most cases, but can be describe as pitching, yawing or rolling in character. The same unbalanced information arrives at the ocular nuclei and spinal tract. The eyes move in response to the stimulation to the last field of gaze and the slow phase of nystagmus is born. Because the eyeball can't turn 360°, it reaches a point of maximal deviation and a signal from the reticular formation return the eyes to their starting position, which forms the fast phase of nystagmus. By international convention, nystagmus is reported in the direction of the fast phase. The same unbalanced information in the spinal tract causes staggering and ataxia.

In a matter of minutes, the cerebellum imposes a shutdown of the unbalanced information from the vestibular nuclei. This alleviates but certainly does not eliminate the immediate problem. Fortunately, over a few days the spontaneous nystagmus and vertigo abate because of plasticity and adaptation within the central nervous system, but the asymmetric VOR persist as long as the impaired labyrinth remains depressed (important to note in diseases like vestibular neuritis). Taking this explanation into consideration there are two important rules; (1) a vestibular crisis of any severity will cause vestibular nystagmus of which the characteristics will be explained later on; (2) if the symptoms last continuously for more than 2 to 3 weeks, the cause is not vestibular.

Bilateral vestibular loss will cause severe oscillopsia with minimal vertigo. Oscillopsia refers to a sensation of bobbing up and down and blurred vision when moving. Patients struggle to read signs while moving, and once they stand still, they are able to do so. These patients tend to make slower head movements by turning the head and body together to avoid this sensation.

In contrast, unilateral loss of otolith input causes postural instability and deviations in upright stance. During acute otolith loss, the head tilts and the body leans towards the impaired side because of loss of extensor tone on the involved side. Usually, the ipsilateral eye counter roll inferiorly and the contralateral eye superiorly. Because proprioceptive input is intact, the patient can still stand. Over time the deviation lessens, and the patient shows little sign of injury. With bilateral loss of otolith input, the patient is deprived of his or her internal sense of gravity. He or she becomes more dependent on proprioceptive and visual cues and has trouble walking in the dark or on unpredictable surfaces (sand, grass, inclines). This disability persists although most patients learn to ambulate under most conditions quite well.

The ability to maintain quiet stance is driven mainly by proprioceptive input. Pressure receptors in the soles of the feet and joint stretch receptor information in the feet, legs, trunk, and neck, all combine to create a rich network of multilevel subcortical and cortical reflex pathways designed to maintain the body's centre of gravity over its base of support at the feet. During vestibular dysfunction, this system becomes more important to make up for loss of labyrinthine information regarding gravity. Diseases such as peripheral neuropathies and corticospinal degeneration interrupt these pathways and create difficulties with posture control and ambulation.

Definitions

Table 1. Symptoms and Definitions.

Symptom	Definition
Vertigo	Vertigo implies a hallucination of movement. That means a sensation of movement when no movement is occurring. Usually, it is a rotatory sensation with the room spinning around the patient, or rarely the patient spinning around the room. Rarely the sensation of movement can be an up/down or side-to-side.
Dizziness	The sensation of disturbed or impaired spatial orientation without a hallucination of movement
Unsteadiness / Disequilibrium	The feeling of being unstable while seated, standing, or walking without a particular directional preference
Oscillopsia	A bobbing sensation (close your one eye, and press up and down on the lower eyelid of the open eye)
Presyncope	The sensation of impending loss of consciousness
Syncope	Transient loss of consciousness due to transient global hypoperfusion characterized by rapid onset, short duration, and spontaneous complete recovery
Non-specific dizziness	Any other balance related sensation not fitting the prior categories

Approach to a patient

There are some excellent articles on the examination of dizzy patients [2,3,4]. I would urge all general practitioners to read through these, as they explain specific tests in more detail which I did not include here due to space limitation. We know that a diagnosis can be made in 70% of patients on history alone and physical examination and special tests will contribute 10-20% respectively. History therefore is of vital importance, and there are three approaches to this. The **classical approach** is to differentiate between "vertigo" and "the rest". It assumes that patients are able to describe their symptoms to a high degree. In reality, quite frequently, patients find it very difficult to describe their symptoms. Thus, the **alternative approach** can be used, especially in a casualty setting. Lastly, one can focus on only the **million-dollar questions** to establish a diagnosis.

Below is an outline of the history and examination. It is neither fully comprehensive nor will everything be discussed. However, as you will read along, it will build on the outline given below.

- History
    - Classical approach
    - Alternative approach
    - Million-dollar questions
- Examination
    - General
        - One can form an immediate clinico-pathological picture by assessing the patient's posture and walking when coming into the consulting room. The physician should also look for signs of anaemia, lymphadenopathy, weight loss and any other obvious signs.
    - Head and neck
    - Ear, nose and throat (ENT)
    - Neuro-otological
        - Nystagmus
            - Spontaneous / Induced
            - Fixation – Gaze straight
            - Fixation – gaze in different positions
            - Without fixation
        - Central oculo-motor signs
            - Smooth pursuit

- Saccades
- Vergence
- Visual fixation / gaze holding
- Optokinetic nystagmus

▪ VOR battery

- Dix Hallpike and Lateral semi-circular canal testing
- Dynamic visual acuity test (DVAT)
- Head thrust / Head impulse test
- Head shake
- Caloric test
- Rotation testing
- Fistula test
- Fixation suppression test
- Subjective visual vertical

○ CNS

▪ Higher functions

- GCS
- Orientation
- Intellect
- Communication
- Emotional status

▪ Cranial nerves

- I
  - Smell
- II
  - Vision (Snellen chart)
  - Vision (counting fingers at 1 meter)
  - Eye fields
  - Colour vision
  - Ophthalmoscopy
  - Pupil reflex
    - Direct
    - Indirect
- III, IV, VI
  - Ptosis
  - Movement
  - Cover up
  - Diplopia
- V
  - Sensory
  - Motor
  - Corneal reflex
  - Jaw reflex
- VII
  - Sensory
  - Motor
  - Taste
  - Lacrimation
  - Hyperacusis
- VIII
  - Cochlear

        - Vestibular
    - IX, X
        - Soft palate sensation
        - Gag reflex
        - Larynx
    - XI
        - Inspection
        - Palpation
        - Motor
    - XII
        - Inspection
        - Palpation
        - Motor
- Cerebellum
    - Fast tongue movements
    - Ataxia
    - Dysmetria
    - Finger-nose test
    - Dysdiadochokinesia
    - Romberg
    - Heel-shin test
    - Pendular reflexes
    - Nystagmus
- Motor
    - Inspection
    - Palpation
    - Strength
- Sensory
    - Pain, temperature and general sensation
    - Light touch, position and vibration
    - Stereognosis
- Reflexes
- Gait
- Coordination
- Posture

- It is important to also examine the following systems
    - CVS
        - Arrhythmias
        - Reduced cardiac output
        - Hypovolemia
        - Pericarditis
        - Orthostatic hypotension
        - Autonomic dysfunction
        - Vasovagal syncope
    - Hyperventilation
    - Hypoglycaemia
- Special examinations
    - VNG
    - VEMP's
    - Posturography
    - Scans

- Neurologist / Cardiologist / Physician / GP
- Audiology / audiologist
- Physiotherapist
- Bloods

History:

Classical approach

The key history points are:

- Is it vertigo?
- What is the time course?
- Precipitating / Exacerbating factors
- Accompanying symptoms

Vertigo can be an hallucination that the external world is moving relative to an individual or the individual relative to space. Rotational vertigo or other hallucinatory sensations of motion indicate vertigo (vestibular symptoms), whereas a sensation of light headedness, giddiness, drowsiness, or impending fainting implies dizziness of non-vestibular origin. Non-spinning dizziness only when standing or walking usually indicates a neurological gait problem rather than vestibular vertigo.

Vertigo onset is usually sudden and comes in spells varying from seconds or minutes to hours. The offset is less clear with patients feeling unwell for a variable time. Some types of peripheral vertigo are brought on by a change in position, and most will improve by lying still. Tinnitus, hearing loss, and aural fullness frequently accompany peripheral disease. Central disease can rarely also have hearing loss and tinnitus if the anterior inferior cerebellar artery is involved.

Unlike peripheral vertigo, central causes of dizziness produce a more variable picture. Patients may describe it as spinning, tilting, forced to one side, light headedness, clumsiness or blacking out. If loss of consciousness is documented, a peripheral aetiology for dizziness is rarely – if ever – at fault. The following symptoms also points to a central cause namely dysarthria, dysphagia, diplopia, hemiparesis, severe localized cephalgia, seizures or memory loss.

Alternative approach

The new approach places an emphasis on categorizing timing and triggers, and four key patterns are recognizable:

- Acute, spontaneous, prolonged symptoms (acute vestibular syndrome)
- Episodic, positional symptoms
- Episodic, spontaneous symptoms
- Chronic unsteadiness
    - With oscillopsia
    - Without oscillopsia

Acute, spontaneous, prolonged symptoms:
This is seen in patients with vestibular neuritis, labyrinthitis (which is extremely rare and will present with hearing loss), or posterior fossa stroke. Differentiating between vestibular neuritis and posterior fossa stroke can be difficult. Only 50% of patients with a posterior fossa stroke will have symptoms such as diplopia, dysarthria, dysphagia, and dysmetria. On the other hand, audiological symptoms such as tinnitus and hearing loss usually point to an ear problem but can also occur in anterior inferior cerebellar artery stroke. The most sensitive way to discriminate between the two conditions on clinical grounds is explained by the acronym HINTS. This stands for head impulse test (HI), nystagmus pattern (NT), and alternate cover test for skew deviation (S). An abnormal

head impulse test and unidirectional direction fixed nystagmus will point to vestibular neuritis. Direction changing or weird patterns of nystagmus, and skew deviation will point to a central problem. (HINTS describe below again).

Episodic, positional symptoms:
This is seen in patients with BPPV and central mimics such as central positioning nystagmus. BPPV can be diagnosed almost instantaneously on history taking. Patients will complain of a severe spinning sensation for seconds, when lying down, turning over in bed, looking up, or bending over. As doctor you need to be specific about questioning these patients, as they would often misinterpret the spinning sensation with the unwell, nauseated feeling that follows. BPPV can be confirmed with positional testing and will cause a unidirectional specific type of nystagmus. Central positioning nystagmus will cause a direction changing nystagmus depending on the position.

Episodic, spontaneous symptoms:
Ninety percent can be explained by six disorders (discussed in more detail under diseases):

1. Meniere's disease is characterized by vertigo attacks lasting 20 minutes to hours. The patients would typically complain about tinnitus, aural fullness and hearing loss with an attack. With time the hearing loss becomes permanent (especially the lower tones).
2. Vestibular migraine can cause vertigo lasting minutes to days. There is a female predominance, and most patients will have a history of previous migraine. Most patients will develop migraine symptoms during a vertigo attack as time goes on.
3. Vertebrobasilar TIAs affect older patients with vascular risk factors. Most attacks will last less than 1 hour and can be accompanied by other symptoms of the posterior fossa circulation.
4. Vestibular paroxysmia is caused by vascular compression of the eight' cranial nerve. It is characterized by numerous brief attacks of vertigo lasting seconds, especially when turning the head left or right.
5. Orthostatic hypotension causes brief attacks of dizziness lasting seconds to minutes after standing up. In older patients it may be accompanied by supine hypertension.
6. Panic attacks usually last minutes, occur in specific situations, and are accompanied by choking, palpitations, tremor, heat, and anxiety symptoms.

Less common causes are labyrinth fistulas such as superior semi-circular canal dehiscence, cardiac arrhythmia, otosclerosis, autoimmune inner ear disease, and medication side effects.

Chronic unsteadiness (with or without oscillopsia):
This is seen in patients with persistent postural perceptual dizziness (3PD) (previously chronic subjective dizziness (CSD) and phobic postural vertigo), cerebellar degeneration, bilateral vestibular failure (will produce oscillopsia), spinal cord compression, metabolic disease, or psychiatric problems.

3PD is commonly reported as the second most common diagnosis, after BPPV, among patients with vestibular symptoms. It is characterized by persistent unsteadiness and non-vertiginous dizziness, usually worse when walking, less severe when standing, and absent or minimally present when they are lying down. Other traits have been associated with the disease over the years such as being more common in patients with obsessive compulsive behaviour, it improves with mild intake of alcohol, and the patients can typically still produce complex balance tasks (which is usually automatic) such as running or riding a bicycle.

Million-dollar questions (short cut)

As mentioned, some questions in the history can point to as diagnosis, so to speak "Million-dollar" questions.

- Do you get dizzy just rolling over in bed?
    - Benign paroxysmal positional vertigo (BPPV)
- Are you light sensitive during your dizzy spell or / and had a previous diagnosis of migraine?

    - Vestibular migraine
- Does one ear feel full before or during an attack?
    - Meniere's disease
- Does a loud sound make you dizzy or make your world jiggle?
    - Superior semi-circular canal dehiscence
- Was your first attack severe vertigo lasting hours with nausea and vomiting?
    - Vestibular neuritis
- Are you lightheaded when you get up from a bed or chair for a few seconds?
    - Blood pressure / Cardiovascular disease (CVS)
- Do you pass out completely with your dizziness?
    - CVS

Other symptoms

Presyncope and syncope would prompt a search for cardiovascular, metabolic or central causes. ***Unsteadiness*** without dizziness or vertigo is most commonly seen in patients with sensory loss (e.g., peripheral neuropathy), spinal cord diseases (e.g., transvers myelitis, cord compression), and slowly progressive, bilateral cerebellar or vestibular failure. ***Gait unsteadiness*** only with eyes closed, or when walking on an uneven surface or in the dark, is usually bilateral vestibular failure. ***Oscillopsia*** present at rest usually indicates the presence of spontaneous nystagmus (brainstem lesions, drug overdose, or alcohol intoxication). Oscillopsia that occurs only when walking (head motion) usually indicates bilateral vestibular failure. Nonspecific dizziness would prompt a search for psychiatric or metabolic causes.

Examination:

As mentioned earlier a general, ENT and neuro-otological examinations are done. I will mainly focus on the neuro-otological part. The section below does not follow the natural sequence of events in a consulting room. That is discussed under the heading **"how do I do it"** that follows this section. I will also discuss **"Some HINTS on differentiating between vestibular neuritis and posterior fossa stroke"**. This is certainly repetitive but can be consulted later on as single sections.

Firstly, look for nystagmus with fixation with the gaze straight and then in different positions. Repeat the same without fixation usually using Frenzel glasses (20+ dioptre), although if you do not have access to these, a dimly lit room will aid in reducing fixation. Lastly, look for nystagmus in positional testing (discussed later). Peripheral nystagmus (vestibular nystagmus) will cause a unidirectional, direction fixed nystagmus which will increase when looking in the direction of the fast phase and with loss of fixation. In almost all cases it will be horizonto-rotatory with the eye in the neutral position (important, because patients often would look in the direction you are turning them). All other forms points to a central problem such as direction changing, disconjugate, seesaw, pendular, congenital and gaze nystagmus. Purely vertical and / or tortional nystagmus always points to a central problem. A differential diagnosis for these types of nystagmus is given below.

Next examine specific oculo-motor eye movements. For **smooth pursuit** testing, ask the patient to track your finger / a light in the horizontal and vertical planes. Make sure as not to exceed 40^0/s and more than a 60^0 arc. As a general rule horizontal smooth pursuit is better than vertical, and both will diminish with age. Next ask the patient to look back and forth between two fingers about 20 cm apart without moving their head. This is known as **saccade** testing. Observe the eyes for either under or over correction and also conjugate movement. Abnormalities in smooth pursuit or saccade testing point to a central problem. The other oculo-motor tests won't be discussed but two of them, namely **fixation** and **vergence** are covered when testing the cranial nerves.

The VOR battery of testing follows: The **Dix Hallpike** and **lateral semi-circular canal** testing are specifically aimed at diagnosing the different forms of BPPV. Because posterior canal BPPV causes more than 90% of problems the Dix Hallpike test is discussed in more detail (see figure 4). With the patient sitting up in bed the

head is turned 45^0 to the side and the patient is brought into a supine position with the head barely hanging over the edge of the bed. This is not a simultaneous movement and there is also no need to do this briskly. The patient is instructed to keep their eyes in the neutral position and on instruction by the physician to the left and right. Observe the eyes using Frenzel glasses and note any type of nystagmus. In posterior canal BPPV there is usually a brief latency, followed by a geotropic (beating towards the ground) horizonto-rotatory nystagmus lasting less than 60 seconds. Sometimes there is a reversal of the nystagmus pattern when coming up again. Lateral canal testing is done with the patient lying prone with the head flexed 30^0 and then turning their head to the left and right. Again, observe for any nystagmus using Frenzel glasses. Because of the different variations associated with lateral canal BPPV, it is best to refer these patients.

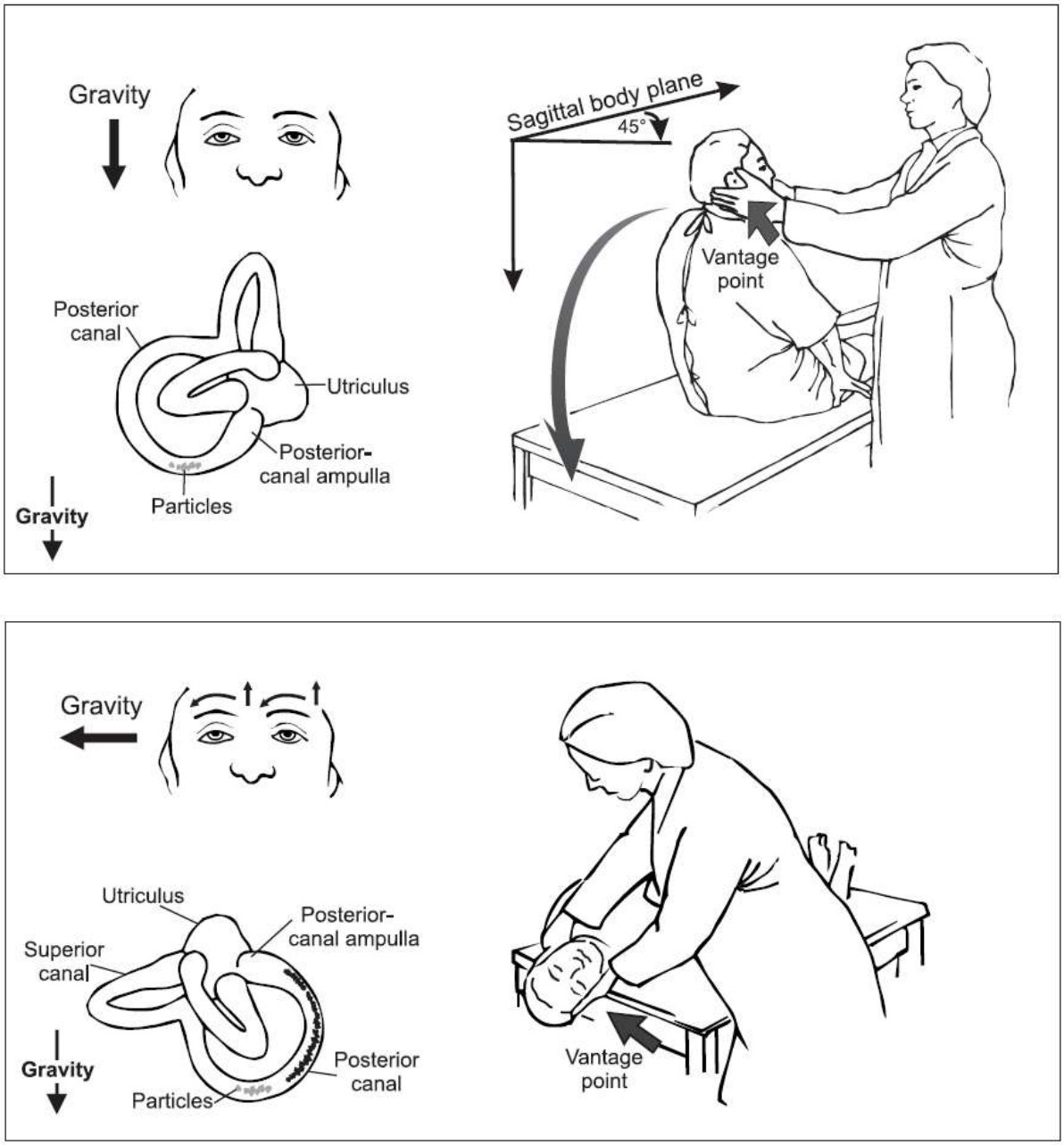


Figure 4. Dix Hallpike test for BPPV showing "crystals" in the posterior SCC.

The most common modification of this is to have the patient sitting up in bed with the feet over the edge. The head is turned 45^0 to a side and the patient is brought into a side lying supine position opposite to the side in

which the head was turned. This is commonly used in elderly patients or morbid obese patients. The same process follows to note any nystagmus.

DVAT is an extremely useful test to confirm a peripheral lesion. With the best corrected vision, the patient reads the smallest line possible on a Snellen eye chart (handheld is acceptable). The procedure is repeated shaking the patient's head at 2 Hz by the examiner and record the number of lines lost during headshake. A more than 2-line drop indicates a bilateral vestibular loss or a poorly compensated unilateral loss.

The **head thrust** test is also known as **head impulse test** (HIT). This test has become one of the most important bedside tests for the evaluation of the vestibular ocular reflex (VOR). The patient is instructed to look at the examiner's nose while he quickly turns the head randomly around the horizontal axis between 20^0 - 30^0. Normal patients will have no problem keeping his gaze on the nose. With unilateral vestibular loss the VOR will fail to keep the gaze on the nose and there will be a catch-up saccade. A patient who presents with acute vertigo and has a normal HIT **strongly** points to a central problem. The HIT is illustrated in figures 5-6.

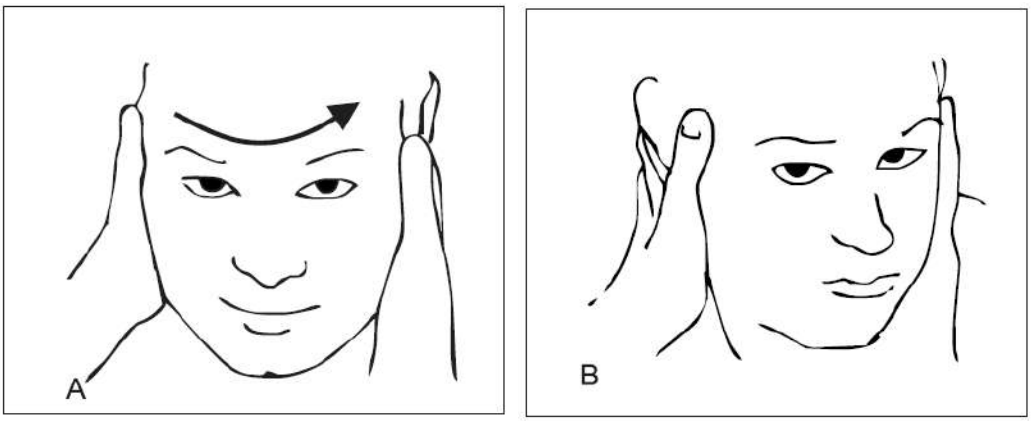
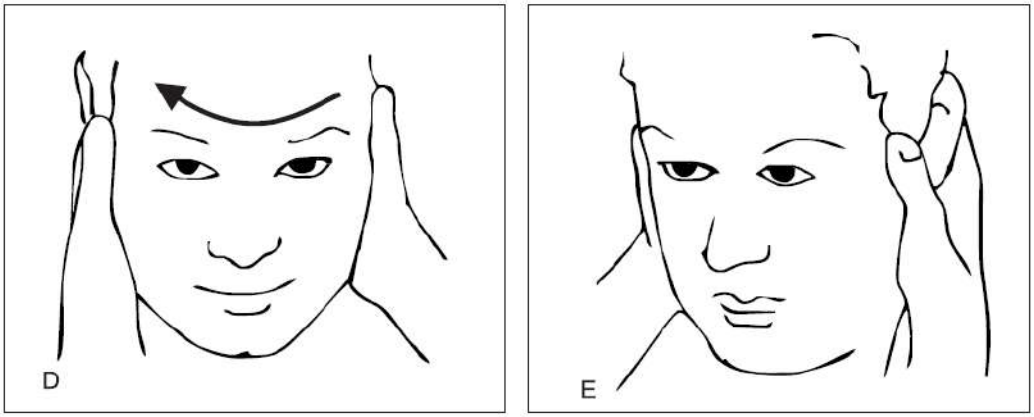


Figure 5. Normal HIT. When turning the head to the left (A), the eyes stay focused on the examiners nose (B).



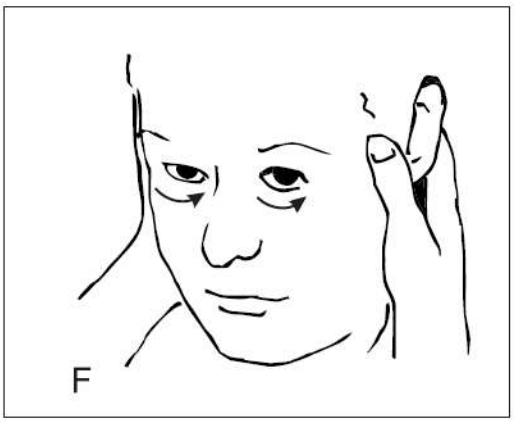


Figure 6. Abnormal HIT. When turning the head to the right (D), the eyes follow the movement of the head (E), and a corrective saccade is necessary to fixate on the examiners nose (F).

In the **head shake test**, tilt the patients head 30^0 forward and shake at 2 Hz for 20 seconds. Observe the eyes with Frenzel glasses for any abnormal eye movements. A peripheral problem will cause post head shake nystagmus with a small reversal component. Central problems may cause prolonged, disconjugate or cross coupling nystagmus (vertical nystagmus following horizontal shaking).

Caloric testing stimulates mainly the lateral semi-circular canal. Various irrigating regimes are described, but I suggest using the mini-caloric test. In this test tap water is used to irrigate the ear for 10 seconds in summer and 8 seconds in winter. The most important aspect of caloric tests is to visualize the tympanic membrane when irrigating. Frenzel glasses are used, and the durations and amplitude of nystagmus are recorded. The caloric test is very important seeing that it is the only vestibular test that can test one side at a time. The acronym COWS stands for cold water opposite, warm water same side, indicating the direction of nystagmus produced with water. Sometimes, ice water is used in a suspected brain-dead patient to elicit an extreme caloric response. It is important to note that nystagmus occurring in the opposite direction of the cold-water injection, is a **normal** response and indicated a normal functioning vestibular system.

The Romberg test, tests primarily for somatosensory and proprioception and not vestibular input. There are however two variations to make it more sensitive for vestibular input. Firstly, instructing the patient to do a tandem stance Romberg (standing with one foot in front of the other) and secondly to do a Romberg test while standing on 10 cm foam. Observe for any sway with the eyes open and then closed.

The subjective visual vertical test will be abnormal 95% of patients with peripheral or central problems. In an abnormal test patients are unable to vertically align an object like a ruler. It is an extremely easy test to perform in a casualty setting. Close the curtains and dim the lights and ask the patient to align a ruler vertically. If they are out by more than 15 degrees, it confirms a problem. Variations of this test is known as the “bucket test”.

The other neurological tests such as cranial nerves examination, cerebellar function, posture and gait will not be discussed but are of extreme importance.

Summary of results:
There are **three main** groups after the examination. Firstly, the group with definite **peripheral vertigo** will have positive signs with some of the following tests: Dix Hallpike test, lateral SCC test, caloric test (absent or abnormal response on affected side), HIT, and DVAT. Patients with **central vertigo** will have positive signs with some of the following: smooth pursuit, saccades, and cerebellar tests. Lastly **patients with dizziness** will have none of the above or sometime bizarre combinations.

The difference between peripheral and central vertigo is summarized in the table 2 below.

Table 2. Central versus Peripheral.

Differentiation of Central versus Peripheral		
	Peripheral	**Central**
Hallucinations of movement	Definite	Less definite
Onset	Usually, paroxysmal	Seldom paroxysmal
Intensity	Usually, severe	Less severe
Duration	Seconds to hours	Weeks to months
Induced by head position	Frequently	Seldom
Nystagmus	Present	Present or absent
Nystagmus pattern	Uni-directional, increased by loss of fixation	Direction changing, no change with fixation, other forms of nystagmus
Autonomous nervous system symptoms	Definite	Less definite or absent
Tinnitus	Frequently present	Seldom present
Hearing loss	Frequently present	Seldom present
Disturbance of consciousness	Absent	More frequently present
Other neurological signs	Usually, absent	Frequently present

In practice "How do I do it":

The examination starts when the patient enters the room, or the physician sees a patient lying in bed. The general examination and a thorough ENT examination are done with the patient in the chair. During the examination of the throat and larynx I would test the function of the IX and X cranial nerves. Next, I usually test all the cranial nerves with the exception of IX and X. While testing the III, IV and VI nerves I would also do all the other special eye movements tests as mentioned under the vertigo examination. Lastly, I would examination the cerebellar functions that can be examined in the chair namely the past-pointing, dysdiadokokinesis, dysmetria, fast tongue movements and finger-nose test.

Before taking the patient to the examination bed, I would take the BP because the patient has been sitting for quite a while and then instruct the patient to stand up and take the BP again to check for orthostatic hypotension.

The next step would be to take the patient to the examination bed, but before that, with the patient standing I would do the Romberg (and Unterberger tests). On the way to the examination bed, I would instruct the patients to walk in a straight line and turn respectively to the right and then to the left. This will also reveal any forms off central ataxia.

On the examination bed, with the patient lying I usually start off with the rest of the cerebellar tests namely heel-shin. After this I complete my CNS examination by testing the motor, sensory and reflex systems. This I do before examining the vestibular systems, otherwise one tends to skip that part especially in a patient who experienced vertigo and nausea during the vestibular tests.

I would start with the Dix Hallpike test, followed by the lateral canal testing. After this I usually do the head thrust and headshake tests. Lastly, and only when indicated I would do caloric tests, because this usually induces vertigo and nausea in most patients.

Some "HINTS" to differentiate vestibular neuritis from a stroke

Both vestibular neuritis and a posterior fossa stroke present with symptoms known as acute vestibular syndrome (AVS). Patients complain of vertigo, nausea, vomiting, and unsteadiness. This can be extremely unnerving to differentiate a self-limited disorder, vestibular neuritis, from a potential life-threatening stroke. Only half of the patients with a stroke will complain of diplopia, dysarthria, dysphagia, and focal sensory and motor symptoms. On the other hand, hearing loss is not always a sign of ear disease and can be part of an anterior inferior cerebellar artery infarction. Initial MRI can be falsely negative in 12% of cases. This section will focus on the acronym "HINTS" to try and assist you to differentiate between the two conditions on clinical grounds.

"HINTS" stands for **H**ead **I**mpulse Test, **N**ystagmus Pattern, and **T**est of **S**kew Deviation. The HIT tests the vestibular ocular reflex (VOR), which lets you focus on one spot while moving your head (Figure 7).

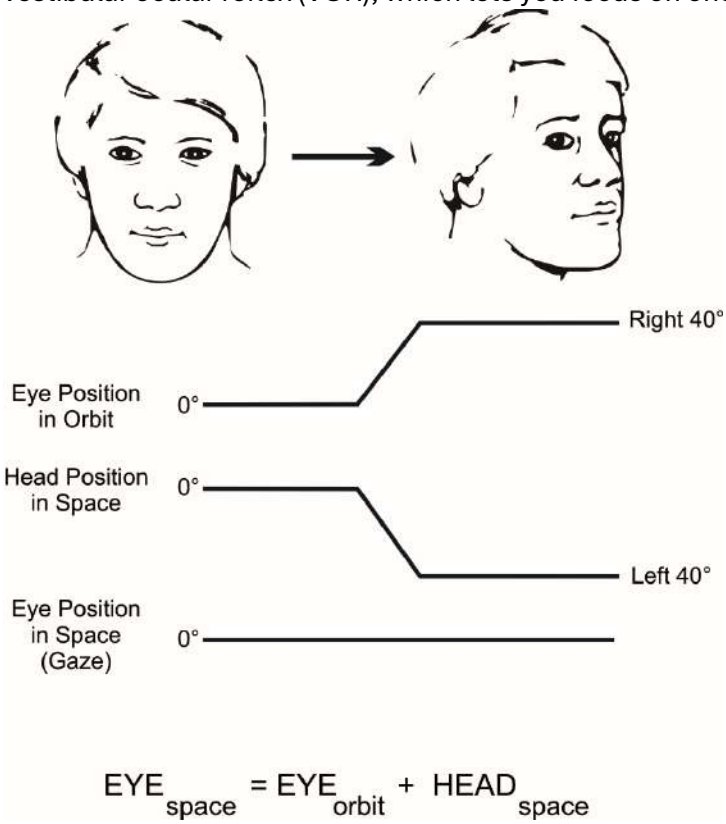
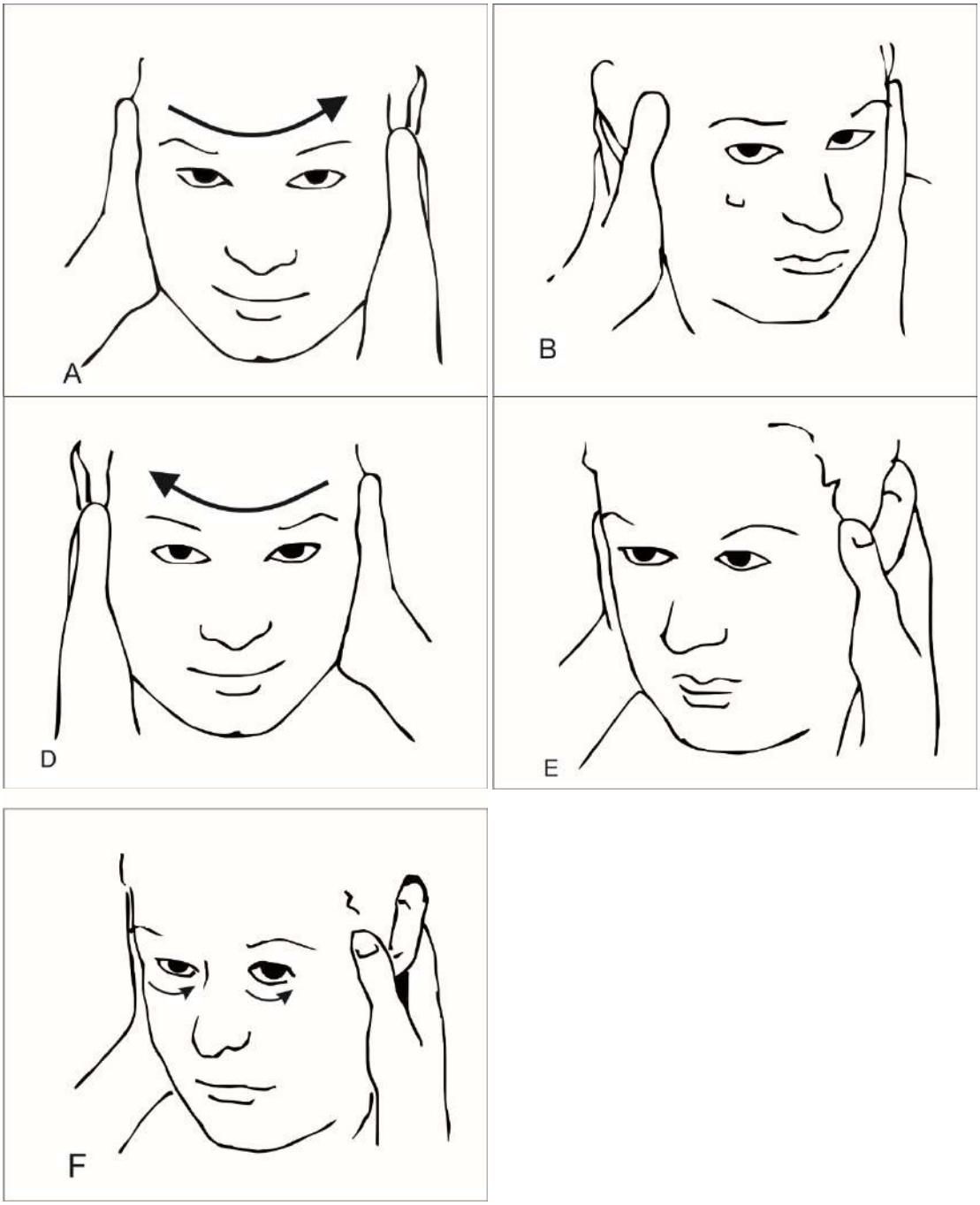


Figure 7. HIT

In the HIT, you need to instruct your patient to focus on your nose, relax the neck, and then briskly turn the head 20-30° away from the midline in the horizontal plane. This needs to be randomly to the left and right. As examiner you need to see if your patients' eyes stay focussed on your nose, or whether there is a corrective eye movement after turning the head (saccade). The pictures A and B demonstrate a normal and D, E and F an abnormal HIT.



Pictures A-F. HIT testing.

A normal HIT in a patient with AVS strongly indicates a central lesion. An abnormal HIT usually indicates a peripheral problem but can be due to lateral pontine lesions.

Retrospective studies confirm that nystagmus is frequently noted, but in most cases completely inaccurate. A comprehensive overview of nystagmus falls outside the scope of this chapter, but I would like to stress a couple of important points. In these patients the nystagmus will be spontaneous as opposed to induced. As examiner you need to differentiate peripheral from central nystagmus patterns as discussed in table 3.

Table 3. Peripheral versus Central nystagmus.

	Peripheral	**Central**
Nystagmus pattern	Uni-directional horizontal nystagmus	Direction changing, vertical, gaze-evoked nystagmus

Vertigo	Will always complain about vertigo	May or may not complain about vertigo (although nystagmus is present)
Direction	Nystagmus increases when looking in the direction of the fast phase	Variable
Fixation	Nystagmus increased by loss of fixation	Variable. May be induced by fixation

In practice ask the patient to look at your finger. The first thing is to figure out the direction of the fast phase of nystagmus. If this is in one horizontal direction (either to the left or right), and is increased by asking the patient to look in the direction of the fast phase, and the direction of the fast phase is not influenced by the position of the eye, then you have a peripheral nystagmus pattern. For all other practical purposes everything else will be central in origin. Remember that in some cases you might only be able to assess the nystagmus with loss of fixation (Frenzel glasses, dark room, video nystagmography).

Skew deviation is vertical misalignment of the eyes. The alternate cover test will identify this. Sometimes it can be accompanied by a head tilt and ocular torsion. Skew deviations strongly point to a central problem.

Therefore, in vestibular neuritis, the HIT will be abnormal towards the affected side, the fast phase of nystagmus towards the healthy side, and no skew deviation. Patients with strokes will have a normal HIT, weird patterns of nystagmus, and skew deviation (sensitivity 98-100%, specificity 85-96%). Table 4 shows the differences between vestibular neuritis and stroke.

Table 4. The differences and also some additional points to remember.

	Vestibular neuritis	**Stroke**
HIT	Abnormal	Normal
Nystagmus	Uni-directional, horizontal	Varies
Skew deviation	Typically, absent	Present
Other symptoms	Hearing loss with labyrinthitis	Diplopia, Dysarthria, Dysphagia
Nausea and vomiting	In proportion to nystagmus	Either greatly increased or minimal in proportion to nystagmus
Walking and ataxia	Usually able to walk	Severe gait or truncal ataxia. Typically, unable to sit unassisted.
Stroke risk factors	Absent	Present (Smoking, hypertension, hyperlipidaemia, diabetes, atrial fibrillation, eclampsia, hypercoagulable state, recent cervical trauma, prior stroke or myocardial infarct)

A differential diagnosis for central forms of nystagmus

- Purely torsional nystagmus usually reflects intrinsic brainstem involvement within the vestibular nuclei and suggests syringomyelia.
- Downbeat nystagmus in primary position usually reflects disease at the craniocervical junction, such as the Arnold-Chiari deformity or degenerative lesions of the cerebellum.

- Upbeat nystagmus in the primary position occurs with lesions at the pontomedullary and pontomesencephalic junction or within the fourth ventricle.
- Periodic alternating nystagmus is a form of central vestibular nystagmus and is usually caused by lesions in the nodulus of the cerebellum.
- Nystagmus on attempted eccentric gaze and with slow phases that show a declining exponential time course results from an unsustained eye position command. This commonly occurs as a side effect of certain medications such as anticonvulsants, hypnotics and tranquilizers. It can also occur in patients with diseases of the vestibulo-cerebellum or its brainstem connections.
- Nystagmus with accelerating slow phases is typical of congenital nystagmus.
- Acquired pendular nystagmus may be a manifestation of multiple sclerosis, tolune intoxication, or a result of brainstem infarction with inferior olivary hypertrophy. Acquired pendular nystagmus frequently is disconjugate and may even be horizontal in the one eye and vertical in the other.
- Convergence-retraction nystagmus usually occurs with midbrain lesions.
- Seesaw nystagmus, when the one eye goes up and the other down, also occurs in midbrain lesions.

Part II – Clinical Diseases

Differential diagnosis

Important concepts to remember are the following. Vertigo does not imply peripheral disease; it can be from a central cause as well. Vertigo does imply some form of hallucination of movement, mostly spinning. All other symptoms are grouped under dizziness.

After history and examination, the clinician needs to decide the following:

- Is it peripheral labyrinthine, eight nerve, central vestibular disease, or diffuse?
    - If labyrinthine, is it unilateral or bilateral?
    - Is this acute isolated, episodic spontaneous, episodic positional, or chronic disease?
- If central, is it brainstem or cerebellar disease?

Three typical forms of peripheral vestibular dysfunction can be identified based on their characteristic symptoms and signs

1. Acute or subacute unilateral vestibular failure, characterised by rotational vertigo, oscillopsia, and a tendency to fall toward the affected ear. Usually acute vestibular neuritis.
2. Bilateral peripheral vestibular failure (bilateral vestibulopathy), characterised by instability of gait and posture, and oscillopsia induced by head movement
3. Paroxysmal peripheral vestibular stimulation or inhibition, characterised by attacks of vertigo and oscillopsia, for instance, in BPPV, Meniere's disease, and vestibular paroxysmia.

A differential diagnosis for the most common causes of vertigo is presented in table 5.

Table 5. Vertigo causes.

Vertigo	
Peripheral	Central
Common	*Common*
• BPPV • Vestibular neuritis • Meniere's disease • Bilateral vestibulopathy	• Phobic postural vertigo • Vestibular migraine • Pathological forms of nystagmus for example ○ Down beat nystagmus

• Vestibular schwannoma / Acoustic neuroma	○ Upbeat nystagmus ○ Gaze nystagmus
Rare	*Rare*
• Superior semi-circular canal dehiscence (SSCD) • Vestibular paroxysmia (vascular loop compression) • Perilymph fistula • Labyrinthitis • Auto-immune inner ear diseases	• Central positioning vertigo • Dizziness syndromes of unclear aetiology / familial • Episodic ataxia type II • Arnold-Chiari malformation • Psychogenic dizziness
Other problems	
Physiological / pathological stimulation	*Central nervous system diseases / causes*
• Motion disease • Caloric stimulation ○ Water exposure ○ Wind exposure • Rotational stimulation ○ Flying ○ Driving • Pressure changes • Changes in specific gravity ○ Alcohol induced vertigo	• Multiple sclerosis • Vascular disease • Tumours • Epilepsy • Infections • Medications

A differential diagnosis for dizziness is presented in table 6.

Table 6. Dizziness causes.

Dizziness	
Condition	Causes
Pre-syncope / syncope	• Arrhythmias • Reduced cardiac output ○ Hypovolaemia ○ Pericarditis • Orthostatic hypotension • Autonomic dysfunction • Vasovagal syncope • Hyperventilation • Hypoglycaemia
Central nervous system	• Normal pressure hydrocephalus • Posterior fossa tumours • Primary orthostatic tremor
Cardiovascular system	• Blood pressure • Vascular diseases
Metabolic	• Glucose metabolism • Thyroid hormone production
Medication	• Blood pressure medication • Antibiotics - aminoglycoside • Chemotherapy • Psychotropic mediation • Tranquilisers
Proprioceptive / Somatosensory	• Neck disease

	• Peripheral neuropathy
Eye	• Poor vision
Psychogenic	

Looking at a clinical correlation, it is easy to follow the schematic description below.

- Is it vertigo?
    - Yes
        - Rotatory
            - Vestibular neuritis
            - BPPV
            - Meniere's disease
            - Vestibular migraine
        - Sensation of boat
            - Bilateral vestibulopathy
    - No
        - Dizziness
- Duration
    - Seconds to minutes
        - BPPV
        - Vestibular paroxysmia
    - Minutes to hours
        - Meniere's disease
        - Vestibular migraine
    - Hours to days
        - Vestibular neuritis
    - Varies
        - Fistula
        - SSCD
- Precipitating / Exacerbating factors
    - Present in rest
        - Vestibular neuritis
    - Worse when walking
        - Bilateral vestibulopathy
    - Precipitated by turning the head to the left and right
        - Vestibular paroxysmia
    - Turning in bed to one side
        - BPPV
    - Coughing / pressing / sounds
        - Fistula
        - SSCD
    - Social or environmental condition
        - 3PD
- Accompanying symptoms
    - Inner ear
        - Tinnitus, hearing loss, aural fullness
    - Central nervous system
        - Diplopia, dysphagia, sensory disturbances, dysarthria, paralysis of arms or legs
    - Headache
        - Vestibular migraine

BPPV

BPPV is the most common cause of vertigo. It affects mainly older patients and has a female predominance. It can also follow after vestibular neuritis, head trauma, prolonged bed rest and Meniere's disease but more than 90% is idiopathic. It is caused by otolith crystals getting stuck in a semi-circular canal. As the name implies this is a benign condition with a spontaneous resolution in weeks, however 30% of cases will persist. As said previously posterior canal BPPV is the most common variant (90%) and will be discussed in more detail.

It is characterized by brief attacks of vertigo after turning in bed, lying down in bed, looking up or bending down. The vertigo usually last less than 1-2 minutes and patients would typically lie still. Some patients confuse the time frame of the vertigo versus feeling unwell that follows the vertigo, but a careful history will clear this up.

It is diagnosed by doing a Dix Hallpike test as described earlier. A corrective turning manoeuvre is done to move the crystals out of the affected canal. There are two manoeuvres namely the Semont or Epley. Both are equally effective with cure rates of more than 95%. The Epley manoeuvre will be describe (see figure 7). This is a turning manoeuvre with 1–2-minute intervals at certain positions. The patient's head is kept in the same position the nystagmus was seen after the Dix Hallpike test for 2 minutes. The head is turned 90^0 towards the opposite side and wait another 2 minutes. The patient is then turned on their side, with head turning a further 90^0 in the same direction. The patient will be facing the ground and typically will experience vertigo again (which is a good sign). After 2 minutes instruct the patient to keep their chin as far as possible on their shoulder and bring them into an upright position.

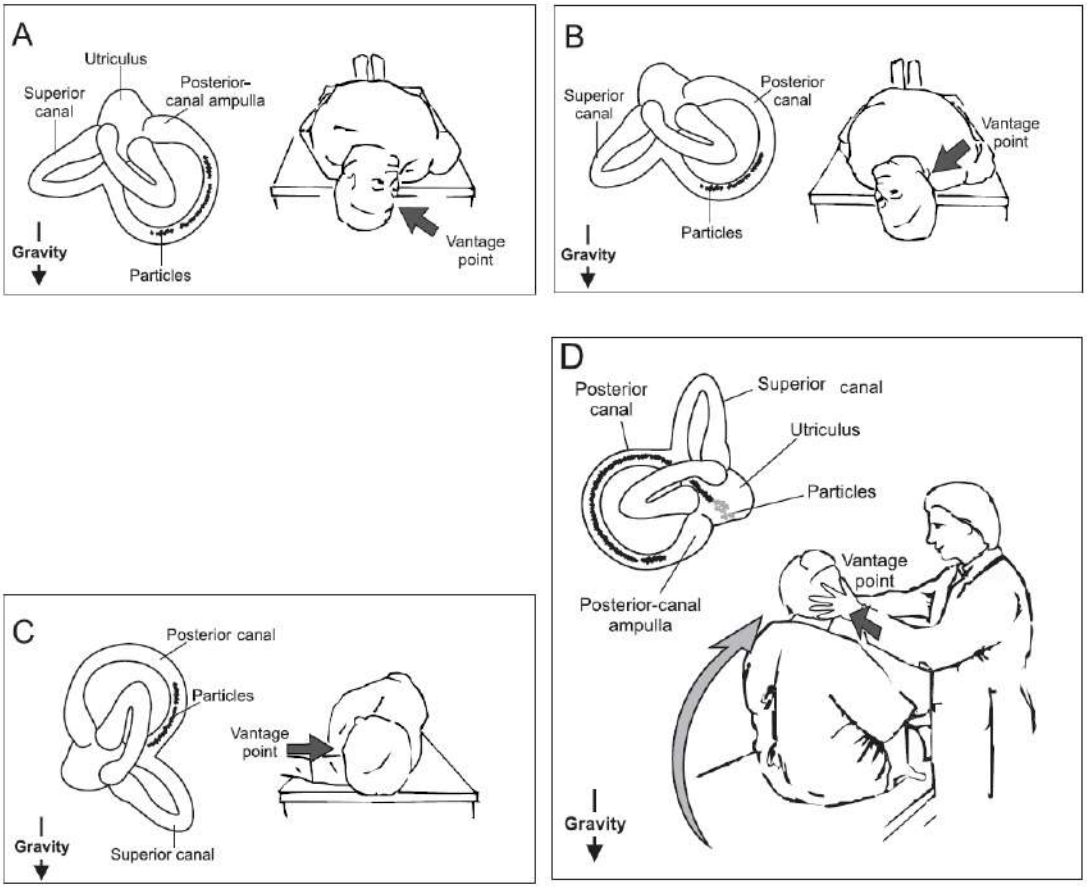


Figure 7. Epley manoeuvre (A-D) for a right sided posterior semi-circular canal BPPV.

Afterwards, I instruct the patients not to do any movements that can trigger the BPPV again such as lying down or any up / down movements for 10 days. I also instruct them not to lie on the affected side or completely flat for

3 days. Some centres instruct their patients to do the manoeuvres at home. Unfortunately, the recurrence rate of BPPV is 15 – 30% per year

Vestibular neuritis

Vestibular neuritis is the second most common cause of peripheral vertigo after BPPV. It is characterized by acute / subacute sustained horizonto-rotatory nystagmus with oscillopsia and imbalance with falls, nausea. The nystagmus is suppressed by visual fixation and there is a pathological HIT. An otherwise normal neurological examination and the ability to stand unassisted without lateropulsion are important in distinguishing vestibular neuritis from a brainstem bleeding or infarction (also see HINTS). There is no hearing loss associated with vestibular neuritis, in contrast to labyrinthitis, which presents the same but is extremely rare. There is sometimes history of upper airway infection and evidence suggests that vestibular neuritis is caused by reactivation of a latent Herpes simplex type I virus.

Although the onset of VN is more predictable, the chronic course of the disease is more variable. In most cases, the initial episode will resolve, and the patient experiences disequilibrium while ambulating and momentary dizziness with rapid head turns lasting up to three months. Clinically this correlates with and impaired HIT. Therefore, as long as a patient has an impaired HIT, he / she will still complain of symptoms of dizziness. Some patients (10%) experience repeated episodes of severe vertigo much like the initial episode. BPPV may follow vestibular neuritis, and the physician should always be on the outlook for this.

Treatment is based on the following:

- Supportive treatment
    - IV fluids
    - Anti-emetic agents
- Vestibular sedatives for 3 days
    - Diazepam or others
- Causative treatment
    - Methylprednisolone 100mg/d and reduced every fourth day by 20 mg
    - Valacyclovir 1gr tds po for 7 days
        - Unfortunately, the administration of antivirals alone or in combination with steroids have no additional benefits
- Improved vestibular compensation
    - There might be some evidence to support the use of high dose betahistidine (Serc®). Unfortunately, the dose of 48 mg tds po is impractical and expensive in South Africa.
- Vestibular exercises
    - Depending on the severity of the patient, this is usually initiated only after 3 days (in mild patients, it can be started earlier)
    - 30 minutes three times per day
    - At first it is a basic VOR exercise
        - Patient focusses on a spot / their own thumb (50cm) and do a horizontal VOR. By implication this means at a slow pace, will keeping focus on the spot / thumb, look left and right.
        - After this, the vertical VOR is introduced on the same principle, and later on any complex head movement while keeping focus.
        - Once the patient is comfortable with this, introduce smooth pursuit, whereby the head is kept still, and the patient tracks his / her own thumb again first horizontally, then vertically, and lastly random patterns.
        - Finally, the fixation suppression test can be done by moving the head and thumbs in the same direction and lastly in opposite directions.

The question always arises whether or not to scan a patient. In a straightforward case there is no need to do a scan, however more often than not one is confronted with an atypical case or a possible cerebellar infarction. These patients should be scanned but also referred to a specialized unit.

Meniere's disease

The diagnosis of Meniere's disease is based on a combination of symptoms namely episodic vertigo lasting minutes to hours, fluctuating low tone hearing loss which becomes permanent, tinnitus, and aural fullness.

The diagnosis can be difficult to make as the classical picture can take years to develop. In contrast to the classical picture, there are also two variants namely:

- Cochlear variant
    - Fluctuating hearing loss, tinnitus and fullness with no vertigo
- Vestibular variant
    - Episodic vertigo with no hearing loss, tinnitus or fullness

There is no single diagnostic test to confirm Meniere's disease, and the diagnosis is made by exclusion. Therefore, an MRI scan is advised on all patients with a possible diagnosis. Meniere's itself can have a variable course, with some patients spontaneously going into remission and other patients developing Meniere's in both ears.

Treatment consists of lifestyle modifications, medical, and surgical options. Lifestyle modifications are best remembered by the acronym **CATS**. This stands for the avoidance of **c**affeine, **a**lcohol, **t**ension and a low **s**alt intake. Usually a maximum of 2 gram/day sodium diet is advised, but in an even distribution. Many other treatments have been published such as allergy and sugar control, diuretics, external ear devices, ventilation tubes, placebo treatments and the psychic of the patient.

An acute attack is treated with sedatives and anti-inflammatory medications, much like vestibular neuritis. Only 10% of patients will require additional treatment to reduce the number of acute attacks. The main factor that needs to be taken into account is the level of hearing loss when deciding on treatment options. Currently the most common would be intra-tympanic steroid injections. Unfortunately, there are minimal treatment options for the hearing loss (except hearing aids) and tinnitus associated with the disease. Because of the difficulty in diagnosis the patients and the various options available, I think it's best to refer these patients.

Bilateral vestibulopathy

As mentioned previously, these patients have minimal vertigo, but presents with postural imbalance, broad based gait, increased gait variability, and oscillopsia. In fact, it is the most common cause of postural imbalance in the elderly.

The symptoms will always worsen when the patient needs to rely on the vestibular organs such as closing the eyes, dark environment or walking on uneven ground (typically when going to the bathroom for the elderly patients). On examination there will be an abnormal HIT and Romberg test with eyes closed.

In ¾ of patients one is unable to establish the aetiology, but known causes are antibiotics, Meniere's disease, meningitis, and encephalitis. The treatment consists of balance training (discussed under vestibular neuritis).

Vestibular schwannoma / Acoustic neuroma

This is a rare tumour of the vestibular nerve. In most cases it is sporadic, but there is a genetic component in 5% of cases. It presents with hearing loss (95%), tinnitus (70%), imbalance (50%), and vertigo (20%). In severe cases it can cause brainstem, and cranial nerve V and VII symptoms. Any patient who presents with all three inner ear symptoms, namely hearing loss, vertigo, and tinnitus should rather be referred to an ear, nose and throat surgeon.

Brun's nystagmus is an unusual type of nystagmus which can occur in large cerebellopontine tumours including vestibular schwannoma. It entails slow, large amplitude nystagmus when looking towards the side of the lesion, and quick, small amplitude nystagmus when looking away.

Superior semicircular canal dehiscence syndrome (SSCD)

As the name implies this syndrome is caused by exposure of the superior semi-circular canal to the cerebro-spinal fluid (CSF) surrounding the brain. SSCD is characterized by vertigo induced by noise / pressure, pulsatile tinnitus, autophony, and unilateral hearing loss / fullness. Sometimes a patient gives as history of chronic disequilibrium following minor head trauma.

Examination and special examinations are complex, and it is best to refer these patients.

Vestibular paroxysmia

This is characterized by spontaneous attacks of vertigo lasting seconds to minutes. Attacks occur mostly spontaneously but can but induced by hyperventilation, exercise, and head turn. It is assumed that vascular compression of the 8th nerve is the cause (as in trigeminal neuralgia, hemifacial spasm and superior oblique myokymia). The condition is treated with carbamazepine.

Persistent postural perceptual dizziness (3PD)

PPV is common, accounting for the second most common diagnosis in a tertiary referral balance unit[5]. The patients complain of swaying vertigo, light headedness, and gait unsteadiness that are continually present but fluctuate in severity. Symptoms are often accompanied by anxiety, fear of falling, but without actually falling. This is usually followed by increasing avoidance behaviour especially large open spaces. Typically, the patients have obsessive compulsive disorder (OCD) personalities.

In general, the symptoms are worse during day, improve by taking moderate amounts of alcohol, and exercise. These patients typically have symptoms when standing or walking, but as the balance task gets more difficult, they improve.

A decoupling hypothesis explains the underlying mechanism. Patients are more aware of normal body movements / sway and interpret this as abnormal. Treatment consists of explanation, desensitization, and selective serotonin re-uptake inhibitor (SSRI) will help in a third of cases. In general, three quarters of patients will improve.

Vestibular migraine

Vestibular migraine is the most common cause of central vertigo[6]. Up to 1/3 of all migraine patients have experienced vertigo. It presents with a combination of vertigo and ataxia of stance of gait. The vertigo usually lasts minutes to hours and rarely days. 60 % of patients have auras during vertigo accompanied or followed by

head pressure, pain, nausea, and vomiting. In some patients there is no correlation with a headache. Treatment consists of regular migraine medications.

Episodic ataxia type II

Episodic ataxia type II is characterized by vertigo attacks lasting hours and ataxia. It is often provoked by stress or exercise. It is an autosomal dominant disorder and treated with acetazolamide.

Down beat nystagmus (DBN)

Down beat nystagmus is the most common form of central nystagmus. The leading symptoms are postural imbalance and oscillopsia usually worse in the morning and improving during the day. On examination the patient presents with fixation nystagmus with an increase in intensity during lateral and downward gaze and when lying prone with the nose down.

DBN is usually due to bilateral dysfunction of the flocculus of the cerebellum. Its three most common causes are cerebellar atrophy, ischemia, and Arnold-Chiari malformation.

Gaze nystagmus

These patients are unable to sustain eccentric gaze. It is probably the most common form of acquired nystagmus. Known causes are a range of drugs such as anticonvulsants, benzodiazepine, alcohol, and midline cerebellar diseases.

Psychogenic forms of vertigo

The patient describes experiencing frequent postural imbalance or a diffuse feeling of dizziness (a feeling of numbness, light headedness, unsteadiness when walking, a feeling of toppling over) or very rarely rotatory vertigo[9].

Depending on the underlying psychiatric illness, the following additional symptoms can be present: disorders of motivation and concentration, decline in performance, restriction of daily and professional activities, vegetative symptoms (accelerated heartbeat, nausea, sweats, apnoea, fear of suffocating, loss of appetite, weight loss), emotional and mood disorders, sleep disturbances and symptoms of anxiety.

These patients are best referred to the appropriate discipline.

Motion Sickness

Introduction:
Motion sickness traditionally refers to symptoms and signs that occurs during travel by car, at sea, or in the air. Nowadays it also includes concepts such as microgravity (space), large visual environments (cinema), and virtual environments (simulators, video games). Classical symptoms and signs include dizziness, nausea, sleepiness, apathy, cold sweat, increased salivation, pallor, and headache.

Incidence and pathogenesis:

The peak incidence is in children between the ages of 2-12 years and rarely occurs after the age of 50 years. It's more common in woman, especially during pregnancy and menstruation. There is a strong association with migraine and a genetic polymorphism as well as otolith organ asymmetry has recently been reported.

Since birth visual, vestibular, and proprioceptive information is processed in the brainstem and cerebellum. A blueprint is formed in the cerebellum based on the input from these three systems. As you are able to perform more complex tasks, such as walking or running, the process becomes automatic, and the cerebellum only matches the input of the three systems to a blueprint. At times the cortex will be consciously aware of your balance and gives the okay. This is the reason why motion sickness rarely presents before two years.

Motion sickness develops if there is a mismatch between the visual, vestibular, and proprioceptive input and the blueprint, or if there is no blueprint for that specific stimulation (space flight). Therefore, anybody can develop symptoms or signs depending on the quality and quantity of stimulation, although there is a huge individual variation. It is therefore a normal response to an abnormal situation.

Classical examples of mismatch include:

- Abnormal visual and vestibular stimuli – watching the waves over the side of a ship, looking at the ground through a binocular out of a helicopter
- Visual stimuli without vestibular input – virtual environments, cinema, space
- Vestibular stimuli without visual input – elevator, reading in a car, closed cabin in a ship

Treatment:
The best treatment remains avoidance of the stimulation. If that is not possible the following options are available:

- Behavioural measures:
    - Make a conscious effort to match visual, vestibular and proprioceptive information, for example by looking at the horizon when at sea, sitting at the window when flying or in a train, looking forward out of the window in a car or rather drive. Keep head upright and avoid unnecessary movements when in motion. Other measures include acupuncture (P6 point), avoid tiredness and alcohol.
- Adaptation:
    - It is well known that repeated or continued exposure to motion results in a declining motion sickness response in most individuals. This can be due to a decreased response of the receptor system (adaptation) or decreased neuronal activity (habituation). A classic example is ice skaters being able to perform severe rotation without developing nystagmus.
- Drug treatment:
    - Three neurotransmitters, histamine, acetylcholine, and noradrenaline, play an important role in the neural process of motion sickness. Medication is most effective when taken before exposure.
    - Hyoscine butylbromide (scopolamine) (Buscopan®), an acetylcholine antagonist, is the most effective anti-motion sickness agent. Unfortunately, it can have moderate to severe anticholinergic side effects and also withdrawal symptoms.
    - First generation antihistamines can be very effective but also has side effects (newer generation antihistamines are ineffective). This group includes diphenhydramine, chlorpheniramine (Allergex®), cinnarizine (Stugeron®), phenothiazine (Vallergan forte®), promethazine (Phenergan®), cyclizine (Valloid®).
    - Studies have shown that rizatriptan (Maxalt®) prevented motion sickness symptoms in patients with migraine and vestibular migraine.
    - Other medications that have positive effects are phenytoin (Epanutin®), flunarizine (Sibelium®), dextroamphetamine (only used in astronauts), and ginger. When prescribing a medication, one should take the patients age and side effects in consideration.

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13) Anatomy of the nose and paranasal sinuses

The nose and paranasal sinuses are complex structures that play crucial roles in respiration, olfaction, and overall health.

Nasal Anatomy

External nasal anatomy
Nasal bones (weaker) and Frontal Process of the Maxilla (FPM) make up the bony pyramid that supports the external nasal framework. They attach to the facial skeleton and provide attachment for the nasal septum and lateral nasal cartilages. The lateral cartilages, include the upper lateral cartilages (form part of the internal nasal valve), lower lateral cartilages and alar cartilages.

The nasal septum is attached to the bony pyramid at the keystone area, and the upper lateral cartilages are attached to the nasal bone and FPM superiorly. The upper and lower lateral cartilages are attached at the scroll area. The scroll area is where the lower border of the ULC and upper border of the LLC meets, and it also forms an important part of the internal nasal valve. The lower lateral cartilages (LLC) have lateral, intermediate and medial portions. The medial aspect of bilateral LLC's joins to form the columella. Posterior to this is the fibrous septum devoid of cartilage (and hopefully the site of nasal piercings). Behind this is the nasal septum itself, the most caudal end.

Internal nasal anatomy
This comprises the lateral nasal wall (with its projections), nasal septum and the ethmoidal labyrinth.

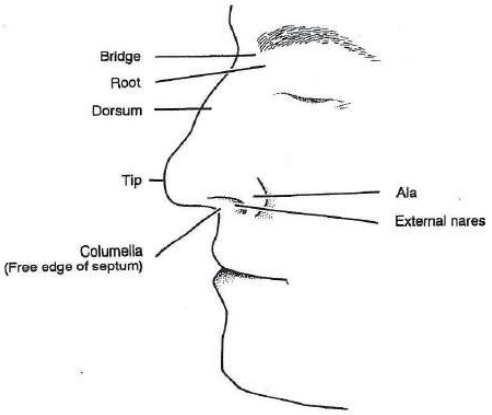
The lateral nasal wall comprises several bones:

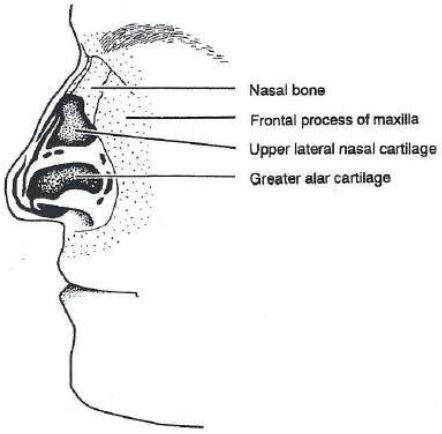
- Maxilla
    - Forms the anterior part of the lateral wall
- Perpendicular plate of the ethmoid bone
    - Contributes to the posterior part
- Lacrimal bone
    - The nasal surface lies posterior to the maxilla and houses the nasolacrimal duct
- Ethmoid bone:
    - Superior and middle turbinate's which project from the lateral nasal wall and increase the surface area for humidification and filtration
    - Uncinate process is a 'sickle' shaped bone that protects the maxillary sinus opening
    - Ethmoidal labyrinth – lateral masses, box-shaped structure between the nasal septum and nasal wall containing the ethmoid sinuses or air cells
    - Lamina papyracea – this forms the medial wall of the orbit separating the eye from the ethmoid sinuses
- Inferior turbinate
    - Is a separate bone projecting from the lateral nasal wall just above the nasal floor
- Medial pterygoid plate
    - Part of the sphenoid bone, part of the posterior part of the lateral nasal wall just anterior to the opening of the Eustachian tube

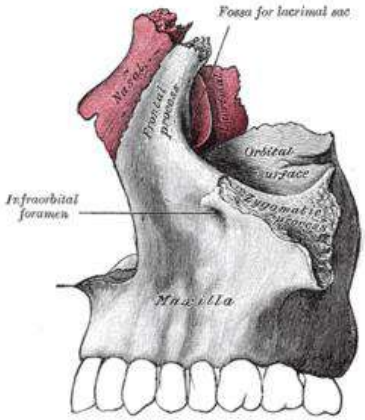
The nasal turbinates project from the lateral nasal wall and create spaces under each of them, namely, inferior meatus, middle meatus and superior meatus into which specific structures drain:

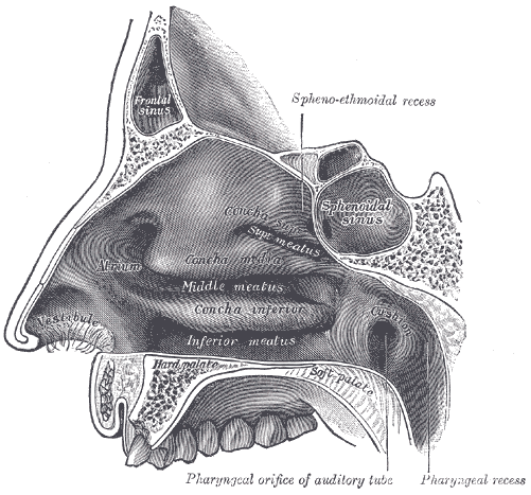
- Lacrimal duct into inferior meatus
- Maxillary sinus (via the hiatus semilunaris), anterior ethmoids and frontal sinus into the middle meatus

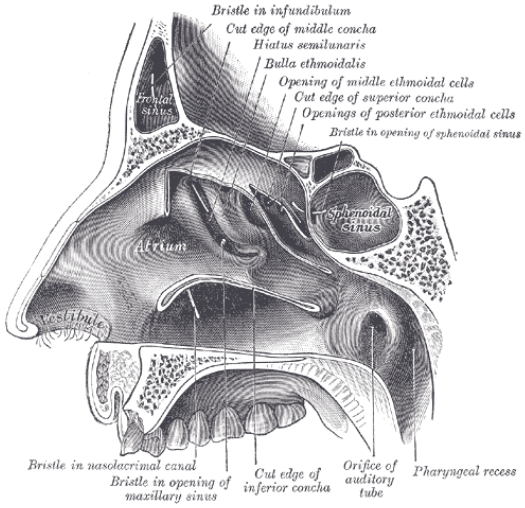
- Sphenoid sinus and posterior ethmoids into the superior meatus

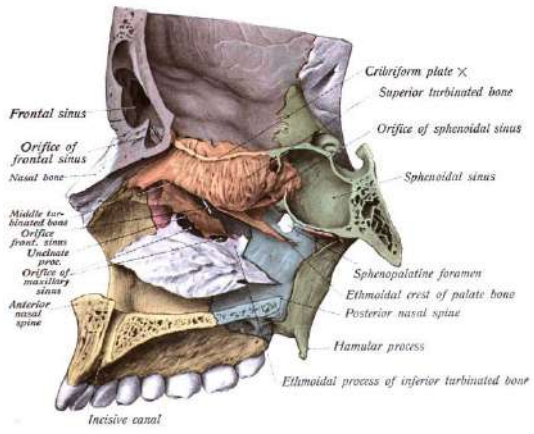


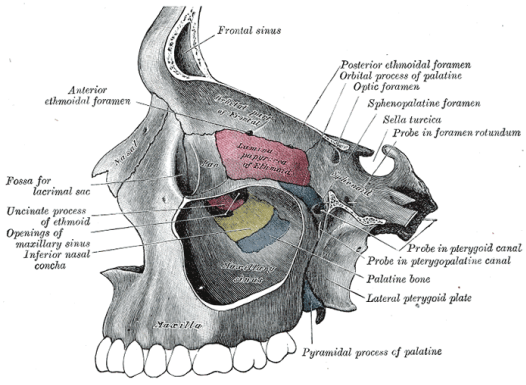












The nasal septal cartilage attaches to the maxillary crest inferiorly and the anterior nasal spine antero-inferiorly. The septum is partly cartilage and partly bone.

Bone:

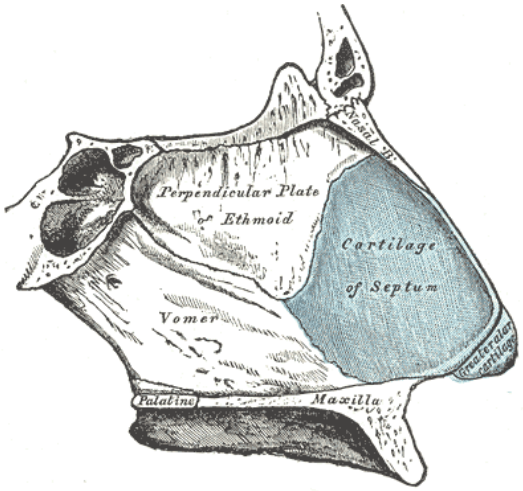
- Perpendicular plate of the ethmoid forms the superior part – attach to sphenoid rostrum posteriorly
- Vomer forms the postero-inferior part - attach to sphenoid rostrum posteriorly

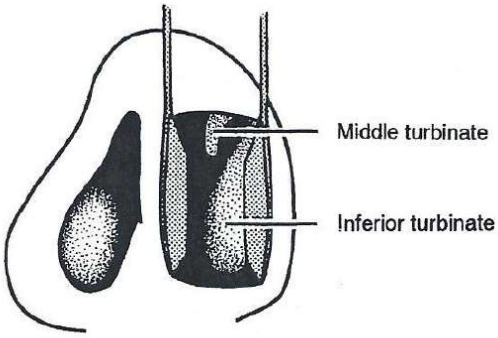
Cartilage:

- Quadrangular cartilage provides support to the anterior septum. It attaches to the bony vomer postero-inferiorly and the bony perpendicular plate of the ethmoid postero-superiorly.

The quadrangular cartilage and the latter two join the sphenoid rostrum posteriorly.

Clinical note:
The septum is derived from two separate embryological elements, the frontonasal process and the palatine process. A dense fibrous layer forms around the septum, namely the perichondrium (over the cartilage) and the periosteum (over the bone). The blood supply to the cartilage runs through this layer and has implications in surgery and trauma. In nasal trauma with septal fracture, vessels may be torn resulting in a **septal haematoma**. If not recognised and managed appropriately the cartilage may undergo ischaemic necrosis (as the blood vessels that supply, it are pushed away) with subsequent "Saddle" nose deformity.





Blood supply
The blood supply to the nose is extensive and includes branches form both the internal carotid and external carotid arteries. The predominant artery involved in primary refractory epistaxis is the sphenopalatine artery, which provides supply to the majority of the lateral nasal wall.

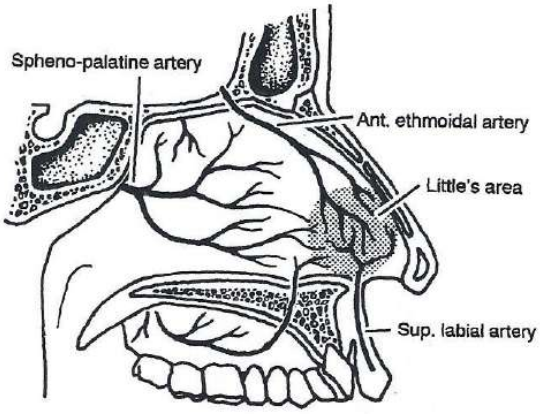
External carotid artery:
Sphenopalatine artery (a branch of the maxillary artery) enters the lateral nasal wall via the sphenopalatine foramen and supplies most of the lateral nasal wall.
Superior labial artery (a branch of facial artery) gives a branch to the nasal septum.
Greater palatine (a branch of descending palatine artery) supplies the nasal septum.

Internal carotid artery:
Anterior and posterior ethmoidal arteries are branches of the ophthalmic artery and supply the superior aspects of the nasal cavity (septum, turbinates, lateral nasal wall).

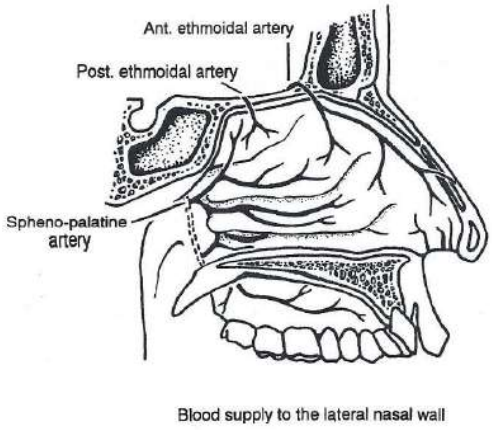
Little's area is an anatomical location over the anterior nasal septum where multiple blood vessels converge at **Kiesselbach's plexus**. Blood vessels that contribute are septal branches of the following:

- Anterior ethmoidal artery
- Sphenopalatine artery
- Superior labial artery

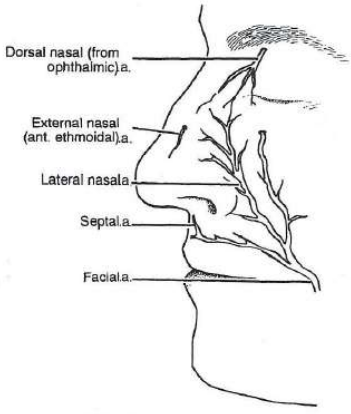
- Greater palatine artery



Blood supply of septum – note that the posterior ethmoid and greater palatine arteries are not annotated.



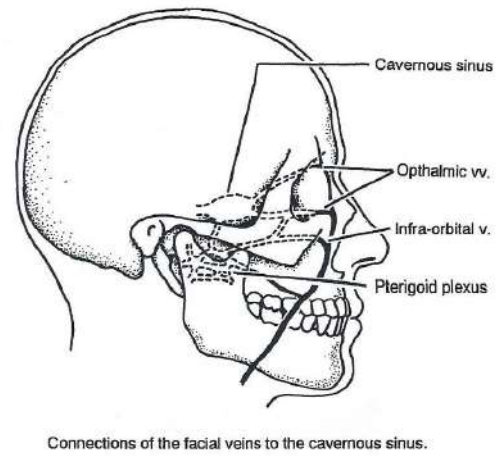
Blood supply to the lateral nasal wall



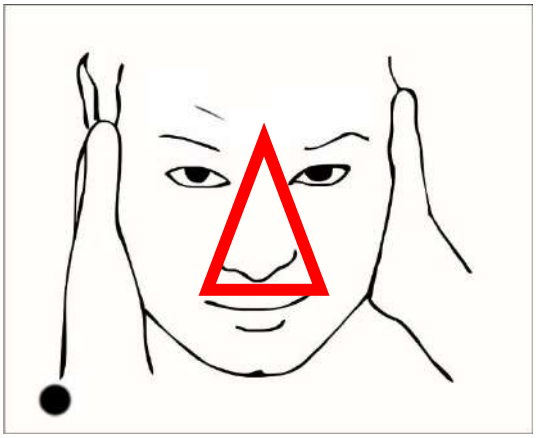
Chief arteries about the external nose

<u>Venous drainage:</u>

Venous drainage can take place to the facial veins or to the intra-cranial cavernous sinus. The reason being the venous connections are devoid of valves. This can have serious implications in infective nasal and sinus conditions, and the area is known as the danger triangle (shown in the picture below as the red triangle).



Connections of the facial veins to the cavernous sinus.



<u>Clinical note:</u>

The anterior ethmoidal artery runs through the roof of the ethmoid sinuses. In cases with significant fronto-nasal trauma that involve the ethmoid sinuses this artery may be injured resulting in significant epistaxis refractory to nasal packing. This requires imaging to identify potential ethmoidal bone and anterior skull base injury and referral as appropriate if epistaxis does not resolve.

Nerve supply

Several nerves provide innervation for the nasal cavity:

Lateral wall:

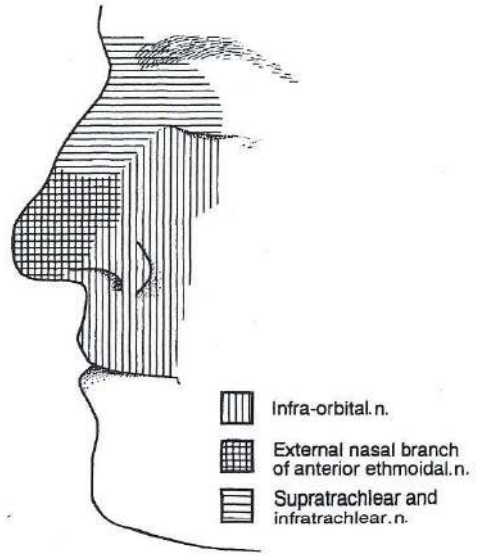
- Anterior – anterior ethmoidal and anterior superior alveolar nerves
- Posterior – lateral branch of the sphenopalatine nerve and branches of the greater palatine nerve

Septum:

- Anterior ethmoidal and nasopalatine nerves

Roof:

- Olfactory nerve passes through the cribriform plate and innervates the olfactory mucosa



Innervation of the skin of the nose

Lymphatic drainage

Lymphatic drainage of the nose and paranasal sinuses can be divided into anterior and posterior groups. Anterior sinuses include the frontal, anterior ethmoids and anterior part of the maxillary sinus which all drain to the submandibular nodes.

Posterior sinuses include the posterior ethmoids, sphenoid and posterior maxillary sinus which drain to the retropharyngeal and superior deep cervical nodes. The lateral pharyngeal and retropharyngeal nodes are the main drainage sites for the posterior nasal structures. Lymphatic vessels run through the parapharyngeal space connecting the nasal cavity to these nodes.

Anatomy of the paranasal sinuses

The paranasal sinuses are paired bilateral cavities in the facial skeleton that develop as a result of pneumatisation into this bony framework.

Maxillary sinus
Largest of the paranasal sinuses and located behind the cheeks in the maxilla. Drains via the maxillary sinus ostium through the semilunar hiatus into the middle meatus.
Important related structures include:

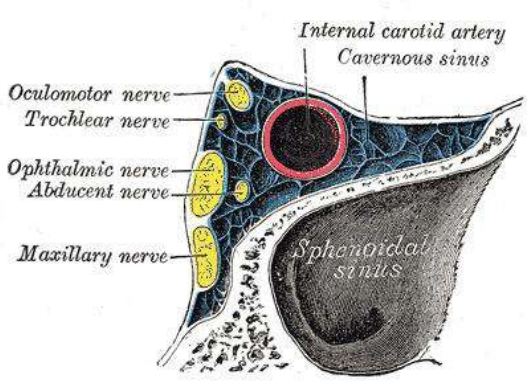
- Orbit and Infraorbital nerve superiorly
- Teeth and oral cavity inferiorly

Frontal sinus
Triangular cavities in the frontal bone which forms the forehead. Drains via the frontal sinus ostium to the frontal recess and then through the ethmoidal infundibulum into the middle meatus.
Important related structures include:

- Frontal lobe of the brain posteriorly
- Orbit inferiorly

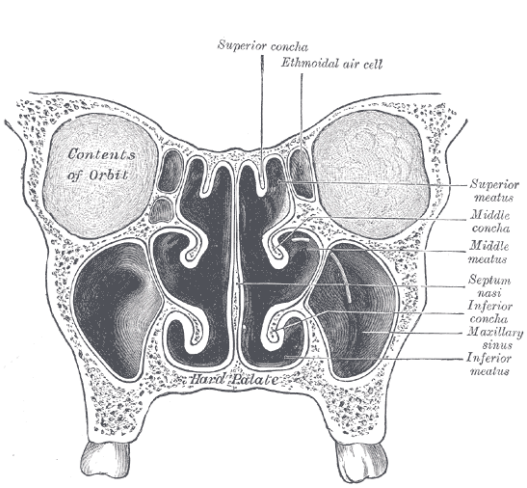
Sphenoid sinus
Most posteriorly located sinus in the sphenoid bone underneath the 'Sella turcica' (Turkish Saddle) in which the pituitary gland lies and the optic chiasm. It drains via the sphenoid sinus ostium into the sphenoethmoidal recess or superior meatus.
Important related structures include:

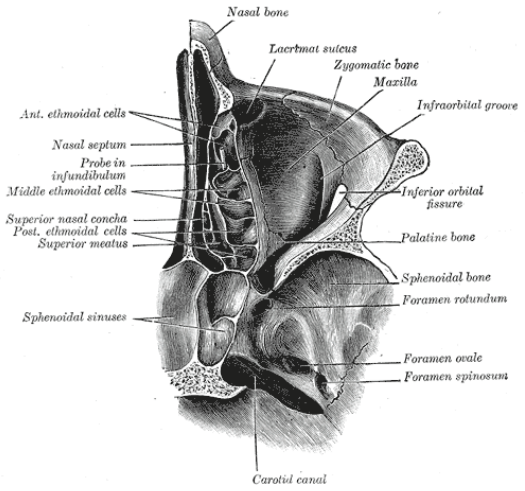
- Internal carotid artery, V2 (maxillary nerve), Cavernous sinus all lateral to it.
- Pituitary fossa above it.



Ethmoid cells (sinus)
A collection of fine air cells located between the orbit and the middle/superior turbinates. Divided into anterior ethmoids drain via the ethmoidal infundibulum into the middle meatus and posterior ethmoids which drain into the superior meatus.
Important related structures include:

- Lamina papyracea (Latin = Paper) and the orbit laterally
- Cribriform plate (anterior skull base) superiorly
- Anterior ethmoidal artery runs through its roof





Physiology of the nose and paranasal sinuses

The nose is a multifunctional organ that plays a crucial role in respiration, air conditioning, olfaction, and vocal resonance. Its functions are essential for maintaining respiratory health and sensory perception.

Respiration
Respiration is an essential function of the nose, especially in newborns who are obligate nasal breathers. The nose serves as the primary pathway for air to enter and exit the lungs. In cases of nasal obstruction, such as choanal atresia, respiratory distress can occur, highlighting the importance of nasal breathing for efficient gas exchange.

The nasal cycle, which involves alternating congestion and decongestion of the nasal mucosa, is influenced by autonomic innervation. Sympathetic stimulation (via superior cervical ganglion) causes vasoconstriction, reducing nasal resistance to increase airflow during active phase. Parasympathetic stimulation (via pterygopalatine ganglion) increases mucous secretion and vasodilation during rest to improve mucosal hydration and defence.

Conditioning of Inspired Air
The nose acts as an air conditioner for the lungs by regulating the temperature, humidity, and cleanliness of the air. This process involves several mechanisms:

- Warming: The nasal mucosa, rich in blood vessels, rapidly warms cold air to near body temperature. This warming process is crucial for maintaining optimal respiratory function.
- Moistening: The mucous membrane in the nasal cavity humidifies the air, ensuring that it is moist and warm by the time it reaches the lungs. This humidification process is essential for preventing dehydration of the respiratory tract.
- Filtration: The nose filters inspired air through several mechanisms:
    - Vestibular Hairs: These small hairs at the entrance of the nose trap large particles, such as dust and pollen.
    - Mucociliary Blanket: A layer of mucus is produced by goblet cells in the pseudostratified epithelium that lines the upper airway. Ciliated cells beat at 700-800 times per minute creating directional flow of mucus towards the nasal cavity and throat. The mucous layer has a gel layer at the bottom and a sol layer on top. This traps and removes finer particles, including pathogens, allergens and debris at a rate of 1-25mm/min (and moves them towards the throat or front of the nose for expulsion). Ciliary beat frequency increases with nitric oxide (NO) or mechanical stimulation and decreases with IL-13 and impaired clearance contributes to rhinosinusitis.

- Lysozymes (Muramidase): Enzymes in nasal secretions help destroy bacteria and viruses, providing additional protection against infections.
- Immunoglobulins (IgA and IgM) assist in mucosal immunity.
- The turbulent airflow in the nose, enhanced by the turbinates, increases contact with the mucosal surfaces, allowing for efficient filtration and conditioning. By the time air reaches the pharynx, its humidity is approximately 75%, and its temperature is around 36°C, with most particulate matter removed.

Olfaction (Smell)

- Olfaction is the sense of smell, mediated by the olfactory epithelium in the nasal cavity. During quiet respiration, about 5-10% of inspired air passes through the olfactory cleft, while during sniffing, this increases to up to 20%.
- Odorants that enter the nose must pass through the olfactory cleft. They must be converted from an air phase to the aqueous phase of the olfactory mucous
- Mucous provides:
  - Moist/protective environment for olfactory neuroepithelium
  - Aids spread (diffusion) of odorants to olfactory receptors.
- The olfactory receptors bind odorant molecules, transmitting signals to the brain for smell perception.
- Retro nasal airflow from the nasopharynx occurs during swallowing which stimulates olfactory receptors adding 'smell' to taste.

Voice resonance

- The sinuses amplify and modulate sound during speech to improve vocal quality

Other

- Lightening of the skull – air-filled spaces rather than solid bone
- Facial growth – influence midfacial development
- Shock absorption – cushioning during facial trauma

Additional information

Please also see endoscopic videos of nose:
https://youtu.be/tSJFTdh_hLk; https://youtu.be/6ttFlcb4Ybo

14) Olfaction and Olfactory Dysfunction

An intact sense of smell enables important environmental and social functions:
Provides information regarding:

- Safety of a substance for example spoiled foodstuff.
- Safety of environment such as leaking liquified petroleum gas (LPG itself has no smell, but mercaptan, an odorant is added to it for safety) or smoke of a fire.
- Aesthetic properties of objects such as a rose or dirty laundry.
- Essential communication cues for example a mother and infant.

It aids digestion of food by triggering normal gastrointestinal secretions.
Loss or dysfunction leads to reduced quality of life, impacting livelihood (e.g. food critic, firefighter, wine merchant), psychological wellbeing and longevity. Mortality risk in non-demented older persons is 2.5 times higher in those with low odour identification test scores vs high odour test scores.

Anatomy & Physiology

- Human nose has 2 independent nasal passages – serve respiratory and olfactory functions.
- Warm / humidify / filter pathogens / pollutants.
- Olfactory neuroepithelium covers the upper septum, middle and superior turbinates.
- Only 10-15% of the nasal airstream reaches this olfactory mucosa.
- Retronasal airflow from the nasopharynx occurs during swallowing → this stimulates olfactory receptors adding 'smell' to taste.
- Odorants enter the nose in a gas phase and must pass through the olfactory cleft.
- Odorants then dissolve in the mucous of the olfactory epithelium.
- Mucous provides:
    - Moist/protective environment for olfactory neuroepithelium
    - Aids spread (diffusion) of odorants to olfactory receptors

Olfactory neuroepithelium:

- Pseudostratified columnar epithelium with highly vascularised lamina propria
- 6 classes of cells
    - Bipolar sensory receptor neuron – extends odorant receptor-containing cilia into the mucous
    - Supporting cell – regulates mucous production, degrades odorants
    - Duct cell of Bowman's glands – secretes most mucous in the olfactory region
    - Microvillar cell – sends tufts of microvilli into nasal mucous
    - Dark basal cells – stem cell
    - Light (globose) basal cells – multipotent basal cell that can give rise to neurons and non-neuronal tissue
- Olfactory receptors reflect the expression of about 1000 genes (1% of all expressed genes, giving an idea of its importance)
- 6 million receptors coalesce into 30-50 fascicles forming olfactory fila
- Olfactory fila cross the cribriform plate and pia mater and synapse with 2nd order neurons in the glomeruli of the olfactory bulb
- Neurotransmitters – Glutamate is excitatory, and dopamine is a modulator
- For the chemical energy of odorant-receptor binding to be transduced into a neural signal (electrical energy) the activation of G-proteins and several 2nd messenger enzymes is required

Olfactory Dysfunction

Definitions:

- Hyposmia – reduced sense of smell
- Anosmia – no sense of smell
- Hyperosmia – heightened response to an odour (not increased ability to smell)
- (e.g. pregnancy, Addison's disease, head trauma, migraine)
- Dyssomnia – altered perception of smell
    - intact olfactory system, at least in part, compared to anosmia
    - E.g. Cacosmia – perception of a normal smell as foul or malodourous, consider cancer or granulomatous nasal disease although most causes of smell disturbance may result in cacosmia

History:

- Onset
    - Acute (viral upper respiratory tract infection is the most common cause for permanent olfactory loss)
    - Sudden (head trauma / prior surgery) – injury to olfactory tract or cortex
    - Chronic (nasal / sinus disease e.g. allergic rhinitis / chronic rhinosinusitis)
    - Progressive (Sinonasal tumours / neurodegenerative diseases – e.g. Alzheimer's / Parkinson's disease / Multiple Sclerosis)

Olfactory dysfunction may be the first sign of Alzheimer's disease (AD) and idiopathic Parkinson's disease (PD) and can be used in diagnosis and prognostication. The University of Pennsylvania Smell Identification Test (UPSIT) score has a better predictive value than any cognitive testing for predicting PD and AD development.

- Sinonasal cause – Look for other symptoms:
    - Nasal obstruction / congestion
    - Rhinorrhoea (watery = cerebrospinal fluid leak [CSF]; mucoid = chronic rhinosinusitis)

- Red flags
    - Cacosmia
    - Pain
    - Cranial nerve fallout / other focal neurology
    - Clear rhinorrhoea (CSF leak)
    - Epistaxis

Examination:

- Features of skull base fracture – raccoon eyes / battle sign / reduced GCS / CSF leak / Cranial nerve deficit / Long tract signs
- Eyes / Nasal framework / skin / V2 changes –? Cancer
- Anterior rhinoscopy – mucopus / crusting / inflamed mucosa / large inferior turbinates / polyps / masses

Investigation:

- Sniffen sticks (pen-like device used to assess odour threshold, discrimination and identification)
- ENT referral for nasal endoscopy (exclude sinonasal disease / mass)
- Imaging if:
    - Suspect skull base injury – CT
    - No cause found for anosmia – MRI (to exclude intracranial lesion)
    - Any other neurological fallout / concern for IC lesion

Prognosis:

It is not clear what predisposes someone to viral-induced smell dysfunction or the mechanism behind it. COVID -19 seems to affect the olfactory supporting cells and not directly infect the neurons so is more often temporary.

Chronic rhinosinusitis (CRS) may cause a conductive and sensorineural (SN) smell loss, thus is not always reversible with surgery. A trial of oral prednisone with restoration of smell in a patient with CRS pre-operatively is a favourable prognostic feature and surgery is likely to be beneficial (mostly a conductive smell loss reversed by surgery).

Management:

Management depends on the cause

- Conductive loss is more amenable to treatment than SN dysfunction

- If a structural / anatomical problem, then referral needed
    - e.g. CRSwNP or anterior cranial fossa lesion → ENT / Neurosurgery
    - Options: Medication / Surgery / Chemo- Radio- therapy
    - Medical – Inhaled nasal corticosteroids +/- systemic steroids
    - Surgery – Endoscopic sinus surgery / Polypectomy
- Acute bacterial rhinosinusitis – systemic antibiotics / decongestant / analgesia / anti-pyretic

- SN Olfactory loss has a worse prognosis
    - Most patients that recover after trauma do so in <12 weeks (may take years)
    - If for other reasons for example post – infection/trauma/toxins/idiopathic then:

Olfactory retraining therapy

- Use 4 scents – rose / lemon / clove / eucalyptus (vanilla / coffee) (1ml onto cotton in 50ml jar) *
- 2x daily, each scent for 20-30 sec for 24 weeks
- Higher concentration and more scents may be better
- Longer duration is better (1 yr vs 16 wks) #
- Post-infectious dysfunction does best e.g. post-COVID – consider steroid with retraining therapy

References:

- *Hummel T, Rissom K, Reden J, Hähner A, Weidenbecher M, Hüttenbrink KB. Effects of olfactory training in patients with olfactory loss. Laryngoscope. 2009 Mar;119(3):496-9.
- #Konstantinidis I, Tsakiropoulou E, Constantinidis J. Long term effects of olfactory training in patients with post-infectious olfactory loss. Rhinology. 2016 Jun;54(2):170-5. [PubMed]

15) An Approach to Nasal Obstruction

Nasal obstruction may vary in severity from a troublesome symptom (e.g. allergic rhinitis) to being life threatening (choanal atresia).

Causes

Congenital

Choanal atresia
Choanal atresia is the obstruction of the nose at the level of the posterior choanae and will present with respiratory distress at birth because neonates are obligate nasal breathers until 2-6 months of age. It may be associated with other abnormalities for example CHARGE syndrome that require further investigation. An oral airway or McGovern nipple is a good temporising measure, failing which other supportive ventilation may be required (CPAP or oral intubation). A diagnosis is confirmed with a CT scan, and the obstruction is surgically opened with or without nasal stent insertion. Unilateral choanal stenosis usually present years later and can be asymptomatic in the young child.

Choanal stenosis
Choanal stenosis is narrowing of the posterior choanae. It may present with stertor, poor feeding or failure to thrive due to the increased work of breathing especially during a concurrent upper respiratory tract infection.

Pyriform aperture and/or midnasal stenosis
This typically would not allow passage of a 5F feeding catheter beyond 1-2cm into the nasal vestibule. In contrast, in choanal atresia the feeding catheter usually won't pass beyond 4 cm.

Masses

- Encephalocoele
- Meningocoele
- Meningoencephalocoele
- Nasal glioma
- Dermoid / Epidermoid

Acquired

The internal nasal valve is the smallest diameter in the nasal cavity and is both a common and accessible site of nasal obstruction. The internal nasal valve is the cross-sectional area at the head of the inferior turbinate, the septum and the caudal end of the upper lateral cartilage. An abnormality in any of these structures, or a nasal mass / foreign body may cause obstruction.

Anatomical

- Septal deviation – this may occur during childbirth or from trauma later in life. May require surgical correction.
- Enlarged inferior turbinates – may occur for several reasons, most commonly in persistent allergic rhinitis during the late phase with infiltration of the turbinate with inflammatory cells. Endoscopic surgical reduction of the inferior turbinates may be required in cases refractory to medical management. It may be confused with a nasal polyp, but the defining features of a turbinate are – mucosa covered, sensate whereas a polyp has an appearance of a pale water balloon and is insensate. Rhinitis medicamentosa is also a common reason for turbinate enlargement, as well as conditions with increased oestrogen (pregnancy) or low testosterone (older males).

- Nasal valve collapse – due to weakening of the lateral nasal cartilages from prior trauma, surgery or from age. May use cartilage grafts to correct this either endoscopically or with an open approach.
- Septal haematoma – following nasal trauma a septal haematoma MUST be actively excluded because if missed may result in an abscess with risk of cavernous sinus thrombosis and loss of cartilage support with resultant saddle nose deformity. Treatment is an emergency and includes incision and drainage of the haematoma and quilt type sutures to prevent re-accumulation.
- Foreign body – young children with foul smelling nasal discharge

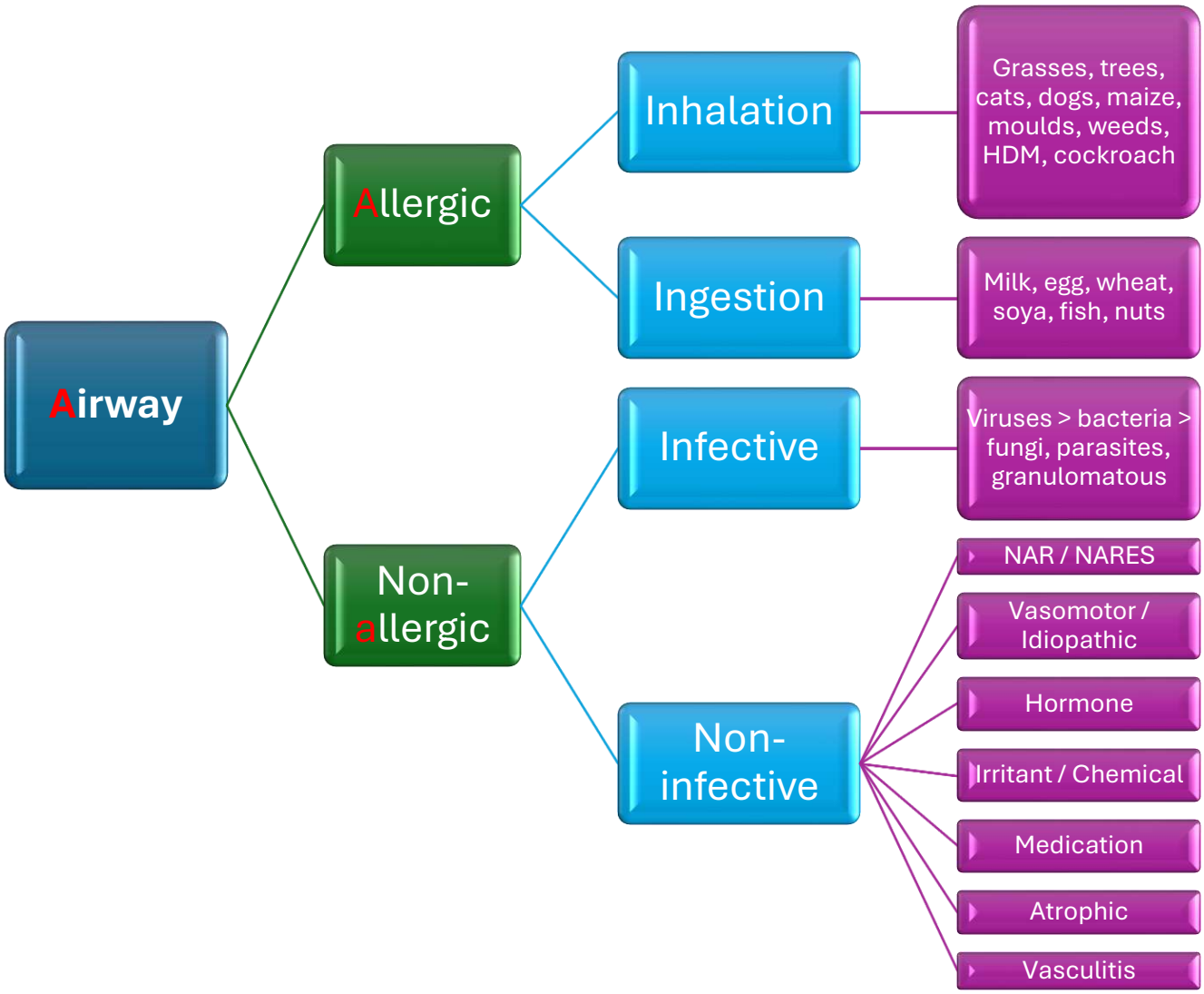
Inflammatory

- Infective – Acute rhinosinusitis. Viral is the 'common cold' or bacterial (see acute bacterial rhinosinusitis)
- Allergic rhinitis – managed primarily with intranasal corticosteroids +/- antihistamines (see allergic rhinitis chapter)
- Chronic rhinosinusitis with or without nasal polyps – medical management and surgery to improve access for medication or to remove polyps. (see chronic sinusitis chapter)
- Non-allergic rhinitis – inflammation and symptoms similar to allergic rhinitis but not caused by an allergen but rather other irritants or triggers such as:
    - Environmental irritants – dust, smoke, fumes.
    - Certain foods – spicy foods or alcohol.
    - Medications – β-blockers, NSAID's, oral contraceptive.
    - Hormonal changes – during pregnancy or puberty.
    - Weather changes – dry or cold weather may worsen symptoms.
    - Rhinitis medicamentosa – overuse of topical nasal decongestants with rebound congestion.
- Enlarged adenoids – most common cause of nasal obstruction in children and becoming more severe and affecting younger children and infants. Lymphoid tissue that forms part of Waldeyers ring and plays an importance role in host immune function. Surgical removal if persistent stertor, even if mild as it affects the normal sleep cycle with neurocognitive and behavioural abnormalities.

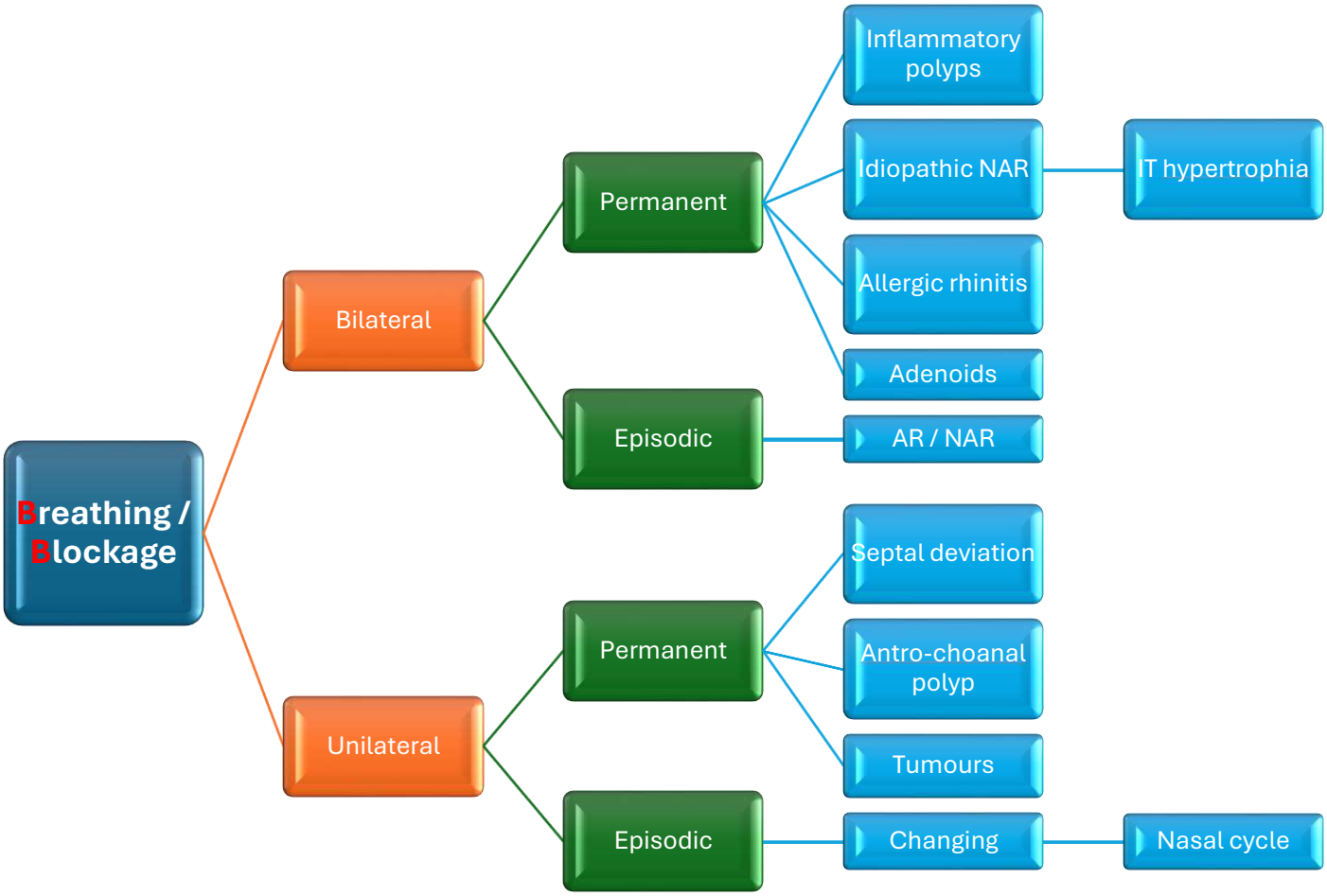
Tumours

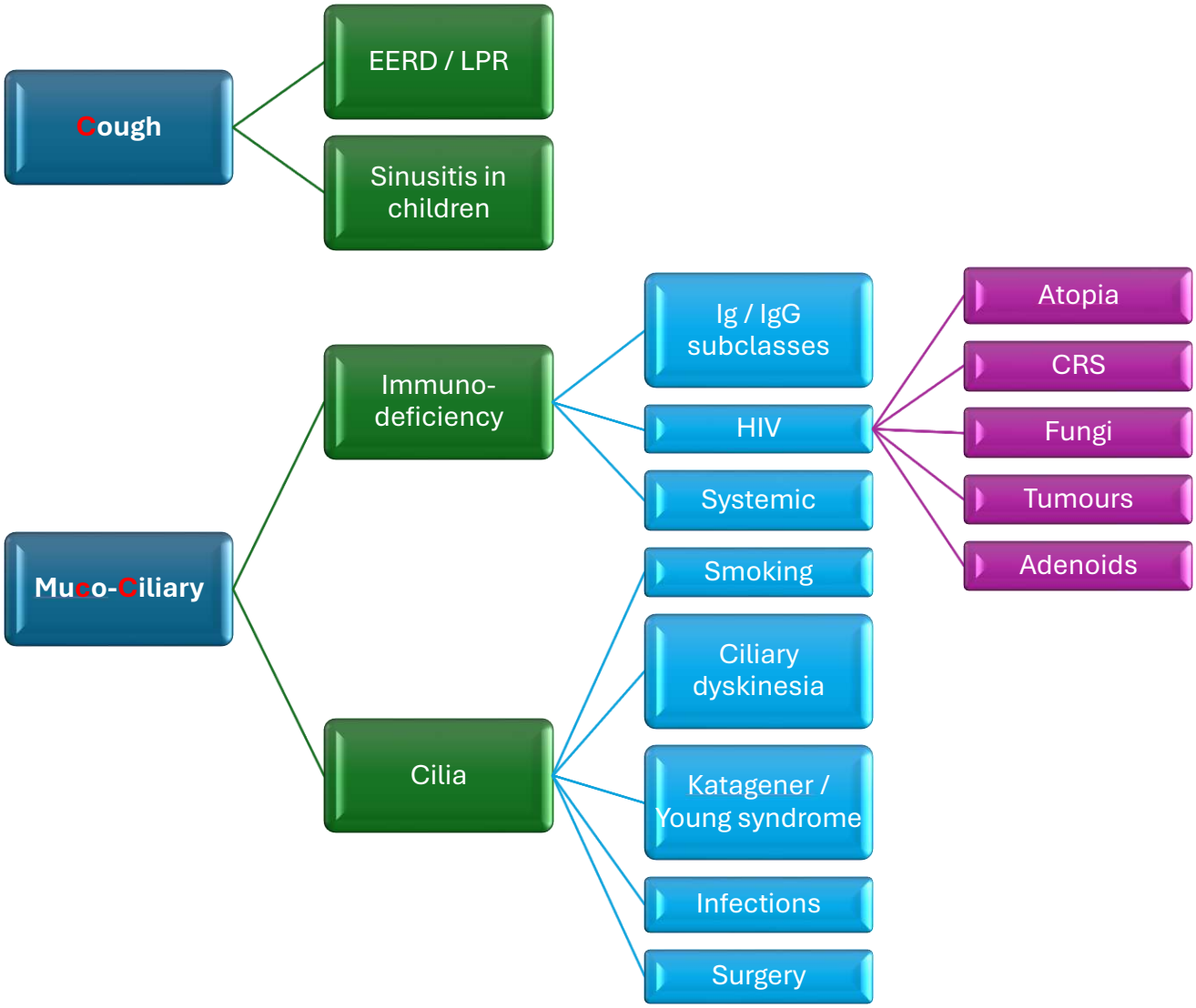
- Inverted papilloma – fleshy, unilateral benign but locally aggressive nasal mass. HPV infection may play a role, and it has local recurrence rate of 10%, malignant transformation potential of 10% and is bilateral in 10% of cases.
- Antrochoanal polyp – unilateral nasal polyp originating from the maxillary sinus and enlarges towards the choana which it may fill causing significant obstructive sleep symptoms.
- Juvenile (nasopharyngeal) angiofibroma – usually young adolescent males, may present with recurrent epistaxis and/or nasal obstructive symptoms.
- Malignant – Multiple different cell lines in sinonasal tract so many types of cancer. Most commonly, squamous cell carcinoma (>70%), others include salivary gland tumours, lymphoma, nasopharyngeal carcinoma, melanoma, intracranial tumours with extension into the nose.
- The primary management of most sinonasal tumours is surgery with the notable exception of lymphoma and nasopharyngeal carcinoma that respond to chemo- and radiotherapy.

Practical approach - ABC



NAR – Non-allergic rhinitis
NARES – Non-allergic rhinitis with eosinophilia





16) Allergic Rhinitis (Hay fever)

Introduction

- Characterised by inflammatory changes in the nasal mucosa caused by exposure to an inhaled allergen to which an individual has become sensitised (type I hypersensitivity reaction)
- Sensitisation phase – allergen exposure and B cells produce allergen-specific IgE
- Early phase – subsequent exposure to allergen results in IgE cross-linking and mast cell degranulation
- Late phase – inflammatory cells infiltrate mucosa with persistent symptoms = **OBSTRUCTION**
- AR forms part of unified allergic airway – allergic asthma / conjunctivitis
- Allergen = antigen (usually protein) that causes allergic diseases
- Hypersensitivity = altered immune response to an antigen that can cause damage
- Atopy = tendency to become sensitised and produce antigen specific AB's (IgE) in response to an ordinary exposure to allergens
- Not all sensitised people are symptomatic
- An Atopic person (40% in the developed world):
    - Usually have high serum total IgE
    - Strong familial tendency
    - Increased susceptibility to allergic diseases (AR / Atopic dermatitis)

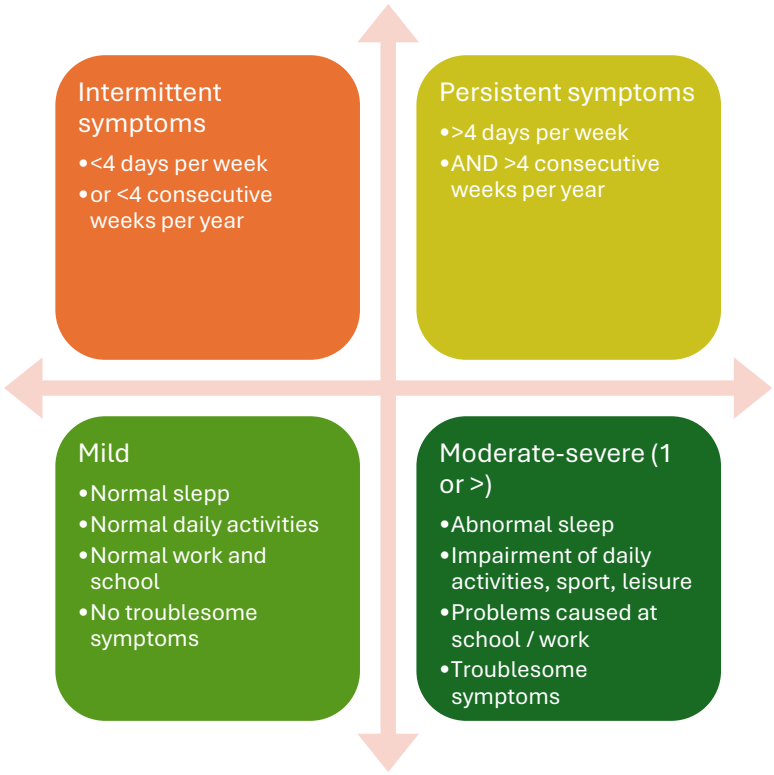
Development of sensitisation

- Mast cells and Basophils are the primary effector cells
- IgE related immediate allergy is associated with early and late responses
- Early phase symptoms are due to preformed mediators
- Late phase due to newly formed mediators and infiltration of other leucocytes
- Involves Th2 cytokines that activate eosinophils and other leukocytes
- Chronic allergic inflammation is associated with tissue destruction and remodelling
- T cells are the only cells capable of recognising an antigen presented by an antigen presenting cell (APC) thus central in the development of allergic disease
- An allergen is presented to the Th2 cell by the Dendritic cell à release of inflammatory mediators with mast cell degranulation and Histamine release (amongst other mediators)

Immune response:

- Initially naïve T helper cells (CD3+/CD4+) may mature in different ways depending on the environment
- TH1 response
    - Th1 cells differentiate in the presence of IFN-ɣ and IL-12 à they produce IFN-ɣ and IL-2
    - High dose antigen exposure as with injectable, favours a Th1 response (how desensitisation works)
- TH2 response
    - Th2 cells differentiate in the presence of IL-4 and produce IL-4 / IL-5 / IL-13
    - Recruitment and activation of Th2 cells with above cytokines is typical in AR / Asthma
    - Low dose antigen exposure via mucosal surface with an inhaled antigen is typical of a Th2 response

Classification



History

Atopic conditions (Pt or family - if parents are atopic then 3-6 x increased risk)

- Childhood eczema / Asthma
- Atopic march = infantile eczema and AR and Asthma
- Intermittent vs Persistent symptoms

Identifiable allergen (house dust mite / pollen / grass /fungus / dander)

- Difficult to modify exposure

Clinical diagnosis with 2 or more of the following symptoms:

- Anterior or posterior rhinorrhoea (watery)
- Sneezing
- Nasal obstruction
- Itching
    - Other symptoms - itchy eyes, pharyngeal itch, reduced smell, cough and sore throat

Any prior treatment and response to it?

Red flags - **NOT** typical of AR and should prompt further investigation

- Unilateral symptoms
- Purulent discharge
- Epistaxis
- Pain

Examination

- Allergic shiners / Dennie-Morgan lines
- Allergic salute
- Enlarged inferior turbinates
- Inflamed mucosa over IT's (dull/blue/purple)

Investigation

Can be correlated with diagnostic tests if allergen not clear from history

- Skin prick tests
- Specific IgE levels in blood
    - Can do individual allergen of bundle tests (phadiotop for inhalent / FX5 for foods)

Both have similar sensitivity and specificity

Management

- Allergen avoidance if possible
    - Animal dander / pollen / grasses / fungus / house dust mite
    - Stop smoking / exposure to 2nd hand smoking
- Symptomatic therapy (pharmacotherapy)
- Desensitisation

Medical treatment

- Systemic +- topical Antihistamine – act on H1 receptors
    - Effective in partial suppression of immediate allergic response
    - Little to no effect in the late response

- Topical Corticosteroids (nasal spray) - **Cornerstone of treatment**
    - Act on intracellular receptors causing anti-inflammatory effect
    - Allergen-induced Th2 proliferation and cytokine release is very sensitive to low concentrations of corticosteroid
    - Highly effective on late response (Th2 related)
    - No effect on the immediate response
    - Do not confuse it with other nasal sprays
        - Iliadin, Otrovin, Drixine, Sterimar, Salex
    - You need to use it everyday
        - Prophylactic treatment
    - 6% incidence of nasal bleeding
        - Technique of spray
        - Other methods of delivery (Ampules / Nasules)

Educate on technique of use of corticosteroid spray

Shake bottle and prime bottle. Bend head forward and direction of spray (left hand for right nose, right hand for left nose and aim for eye on the same side)

As you spray inhale gently and exhale through your mouth

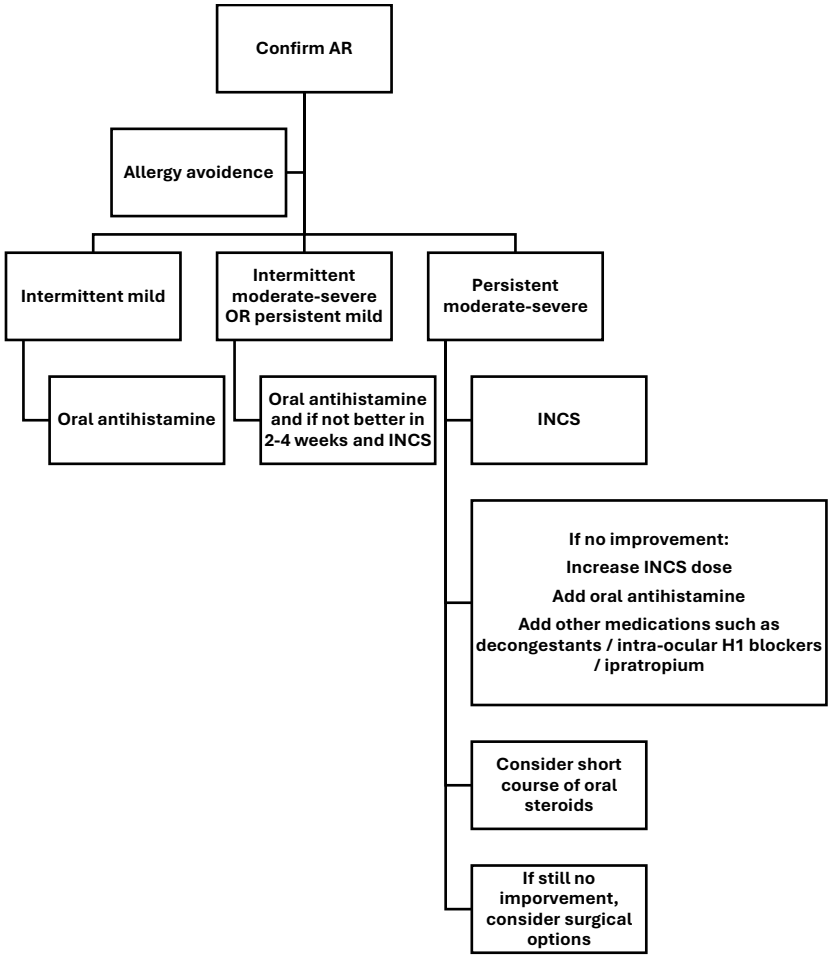
Allergen-specific immunotherapy (Desensitisation)

- Only curative therapy option and highly effective (Cochrane review*)

- Involves giving increasing doses of an allergen to suppress the symptoms when re-exposed to that allergen
- Risk of anaphylaxis thus perform in appropriate setting
- 2 major routes of administration
    - Sublingual immunotherapy (SLIT)
    - Subcutaneous immunotherapy (SCIT)
    - Mechanisms of action
        - Desensitisation of mast cells and basophils
        - Upregulation of blocking antibodies that bind to allergen (reduce antigen presentation)
        - More regulatory T and B cells (Fewer tissue mast cells/eosinophils)

Other treatment

- Sodium cromoglycate as eye drops (mast cell stabiliser) – useful for allergic ocular symptoms
- Topical anticholinergic (ipratropium bromide) – if rhinorrhoea still troublesome with INCS
- Leukotriene receptor antagonist (montelukast) if co-morbid Asthma (especially in children)
- Subcutanous anti-IgE antibodies (Omalizumab)may be effective in severe disease
    - Cost limits its use (R14 000 per/month and up)



Conclusion

- Antihistamine – mild/intermittent disease
- INCS is mainstay

- If rhinorrhoea still troublesome – topical anticholinergic (ipratropium bromide)
- Rarely oral corticosteroid short course (e.g. 20mg prednisone Dly x 5/7)
- Immunotherapy / desensitisation (Treat the cause)
    - Sublingual / subcutaneous
- Consider ENT referral if poor / inadequate response to medication

17) Rhinosinusitis

Introduction

Rhinosinusitis is a common inflammatory condition affecting the mucous membranes of the paranasal sinuses and nasal cavity with or without the involvement of the underlying bone. This condition affects millions of individuals worldwide and constitutes one of the most common reasons for healthcare visits. The pathophysiology typically involves impaired mucociliary clearance and obstruction of the osteo-meatal with subsequent inflammation. This process can be triggered by various factors including viral infections, allergies, anatomical variations or underlying systemic conditions.

Diagnosis

<table>
<tr><th>Adults</th><th>Children</th></tr>
<tr><td colspan="2">Symptoms</td></tr>
<tr><td colspan="2">≥ 2 Nasal Symptoms, of which at least one of:
• Nasal blockage/congestion/obstruction
• Nasal discharge (Anterior - Rhinorrhoea/Posterior – Post nasal drip)</td></tr>
<tr><td colspan="2">Additional Symptoms</td></tr>
<tr><td colspan="2">Facial pain/pressure</td></tr>
<tr><td>Olfactory dysfunction – Anosmia or Hyposmia</td><td>Cough</td></tr>
<tr><td>Endoscopic signs
• Nasal polyps
• Mucopurulent discharge from middle meatus
• Oedema/mucosal obstruction primarily in the middle meatus</td><td>CT changes
Mucosal changes within the osteo-meatal complex and / or sinuses</td></tr>
</table>

OR

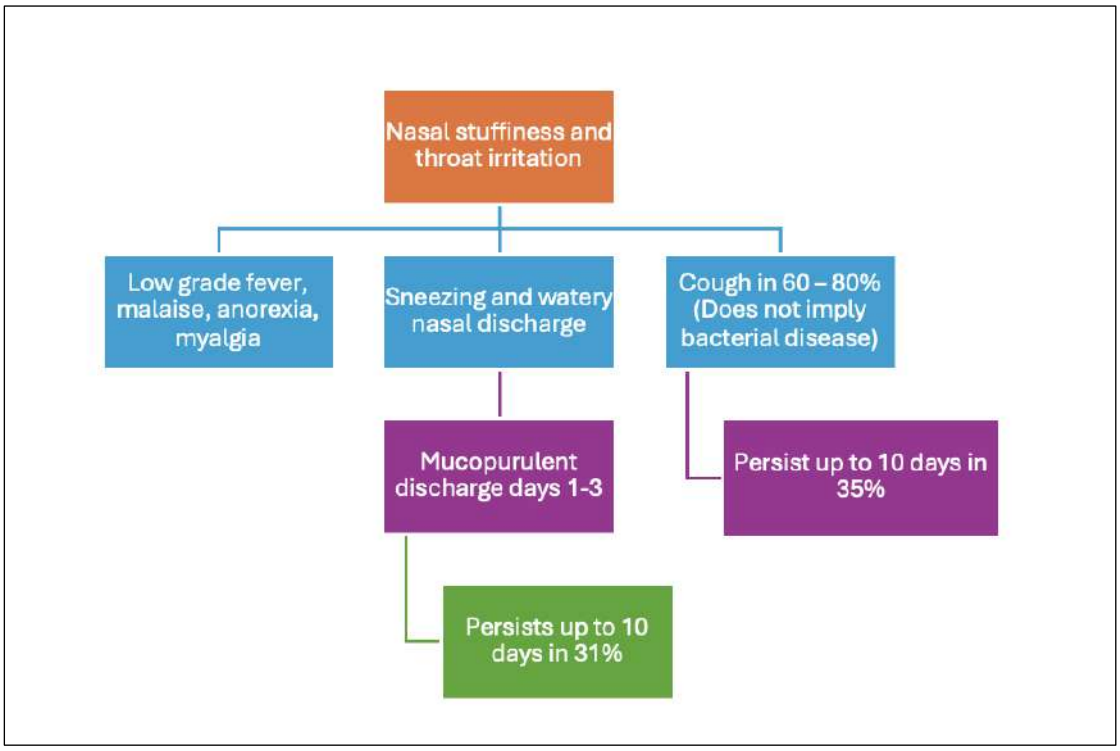
Rhinosinusitis represents a spectrum of disease, ranging from acute to chronic forms:

- **Acute rhinosinusitis (ARS)** lasts less than 12 weeks and is predominantly viral in origin (98%), with bacterial infections accounting for fewer than 2% of cases.
    - **Acute viral rhinosinusitis (AVRS)**, often referred to as the 'common cold', typically lasts less than 10 days and is self-limiting.
    - **Acute Bacterial Rhinosinusitis (ABRS)** often follows AVRS when symptoms persist without improvement for at least 10 days or when symptoms initially improve but then worsen again (double-sickening phenomenon).
- **Recurrent acute rhinosinusitis (RARS)** describes a pattern of four or more episodes of acute rhinosinusitis per year, with symptom-free intervals between episodes.
- In contrast, **chronic rhinosinusitis (CRS)** persists for more than 12 weeks and is associated with ongoing mucosal inflammation.

Thus, relying on symptoms alone is a poor predictor of diagnosis, as the clinical presentation is similar, however the time frame changes. This classification system provides a framework to make a diagnosis and manage appropriately.

Acute (Viral) Rhinosinusitis – AVRS (or) "Common Cold"

Acute viral rhinosinusitis (AVRS) is caused by a range of respiratory viruses, including *Rhinovirus, Respiratory Syncytial virus (RSV), Influenza virus, Corona virus, Parainfluenza virus, Adenovirus and Enterovirus*. On average, adults experience between two to five episodes of AVRS per year, while children may have up to ten episodes annually, with some lasting as long as six weeks in children. Several factors predispose individuals to recurrent acute rhinosinusitis (ARS), including active and passive smoking, odontogenic sources, and anatomical variations, which are more commonly associated with recurrent cases. Ciliary impairment, whether due to smoking, allergic rhinitis, prior surgery, or conditions such as primary ciliary dyskinesia (PCD) or cystic fibrosis (CF) also plays a role. Additionally, the impact of allergies and gastro-oesophageal reflux disease (GORD) on recurrent ARS remains uncertain. Notably, a high prevalence of cough is observed in viral rhinosinusitis, contrasting with the Centor criteria for bacterial pharyngitis, where the absence of cough increases the likelihood of a bacterial cause.



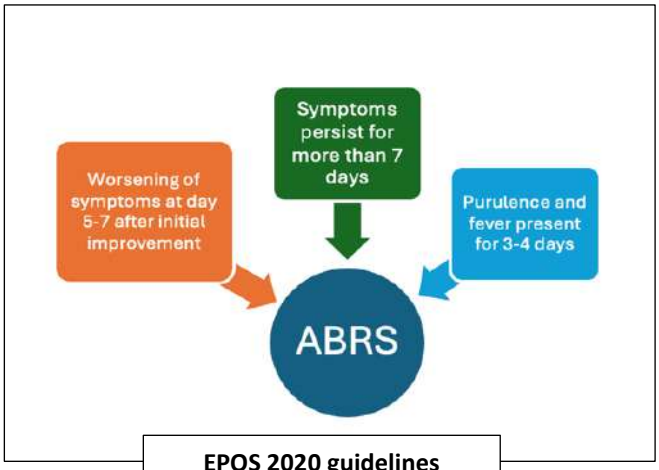
Acute Bacterial Rhinosinusitis (ABRS)

Acute bacterial rhinosinusitis (ABRS) accounts for fewer than 2% of all ARS cases, with the vast majority (98%) being viral in origin. Antibiotics are generally ineffective in viral cases and do not prevent complications. Excessive antibiotic use is harmful to the patient's microbiome and promotes bacterial resistance. Many bacterial infections can also resolve spontaneously (i.e. without antibiotics), with resolution rates of approximately 30% for *Streptococcus pneumoniae*, 60% for *Haemophilus influenzae*, and 80% for *Moraxella catarrhalis*.

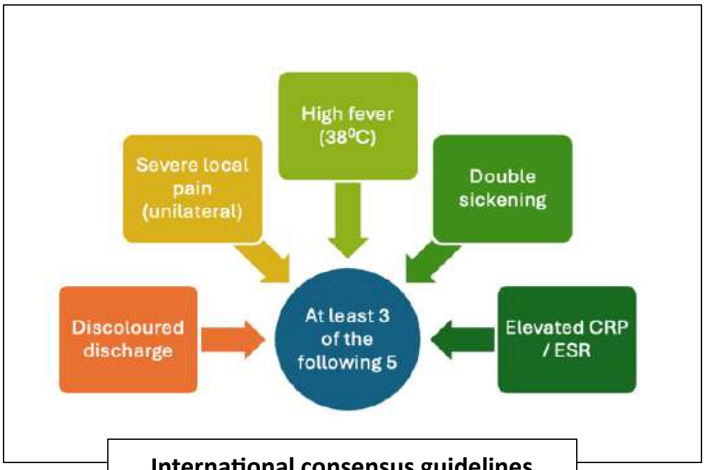
Risk factors predisposing individuals to ABRS				
Dental	**Iatrogenic**	**Immunodeficiency**	**Mechanical**	**Mucosal oedema**
Infections or procedures (Anatomical proximity to maxillary sinus)	• Sinus surgery • Nasogastric tubes • Nasal packing • Mechanical ventilation	• HIV • Immunoglobulin deficiencies (IgA, IgG, IgM) • Cilia problems – Smoking, PCD & CF	• Deviated septum • Nasal polyps • Hypertrophic inferior turbinate • Trauma • Tumour • Foreign bodies • Granulomatosis with polyangiitis (GPA)	• Preceding AVRS • Allergic rhinitis • Vasomotor rhinitis

So, when is ARS bacterial and when are antibiotics indicated?

Determining when ARS is bacterial and when antibiotics are indicated remains a challenge, as there is no universally agreed-upon definition. International consensus guidelines, such as the European Position Paper on Rhinosinusitis (EPOS 2020) and other international consensus statements, provide criteria to help distinguish bacterial infections.



EPOS 2020 guidelines



International consensus guidelines

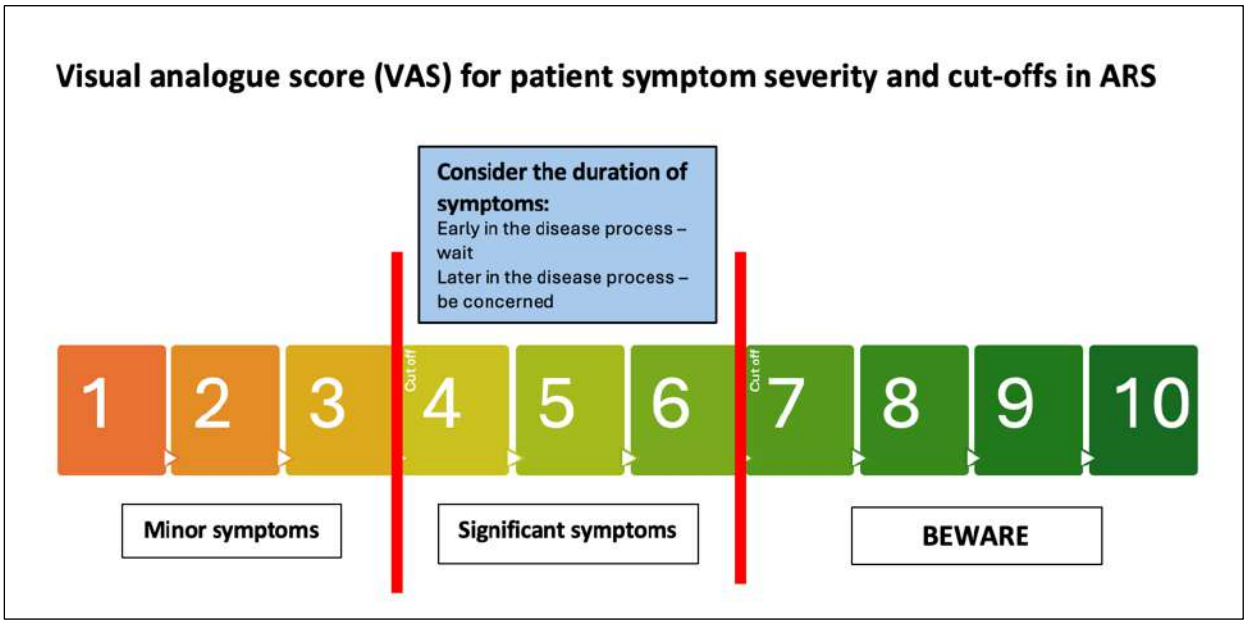
Markers suggestive of bacterial involvement include:

- Acute onset
- A fever exceeding 38°C is strongly associated with *Streptococcus pneumoniae* and *Haemophilus influenzae* and especially in conjunction with more severe symptoms.
- Biomarkers such as C-reactive protein (CRP) and erythrocyte sedimentation rate (ESR) can guide antibiotic use. If low avoid antibiotics, however, if elevated it correlates with bacterial disease on MCS from a sinus puncture and CT scan changes.
- Procalcitonin (PCT) also help guide antibiotic use. If normal levels, antibiotics were avoided without any detrimental outcomes.

Despite variations in classification criteria, antibiotics are generally recommended in the following clinical scenarios:

1. **Complicated sinusitis** – involvement of the eyes, brain or bone.

2. **Double sickening** – an initial illness with partial recovery followed by worsening symptoms, which is the best indicator of secondary bacterial infection.
3. **Stepwise worsening** – progressive symptom deterioration, particularly if the visual analogue scale (VAS) score exceeds 6.
4. **Severe frontal headache** – especially if the VAS score is greater than 6.
5. **Symptoms persisting beyond 10 days** – particularly if significant (VAS >



For example, in a 35-year-old man presenting with nasal obstruction, mucopurulent rhinorrhoea, and fever for 2–3 days with moderate severity (VAS 4–5), conservative treatment with symptomatic management may be sufficient. However, if the same patient presents on day 6–7 with persistent symptoms and poor follow-up, antibiotics may be considered. In contrast, if he has a severe frontal headache with a VAS score of 8–10, antibiotic therapy is more strongly recommended.

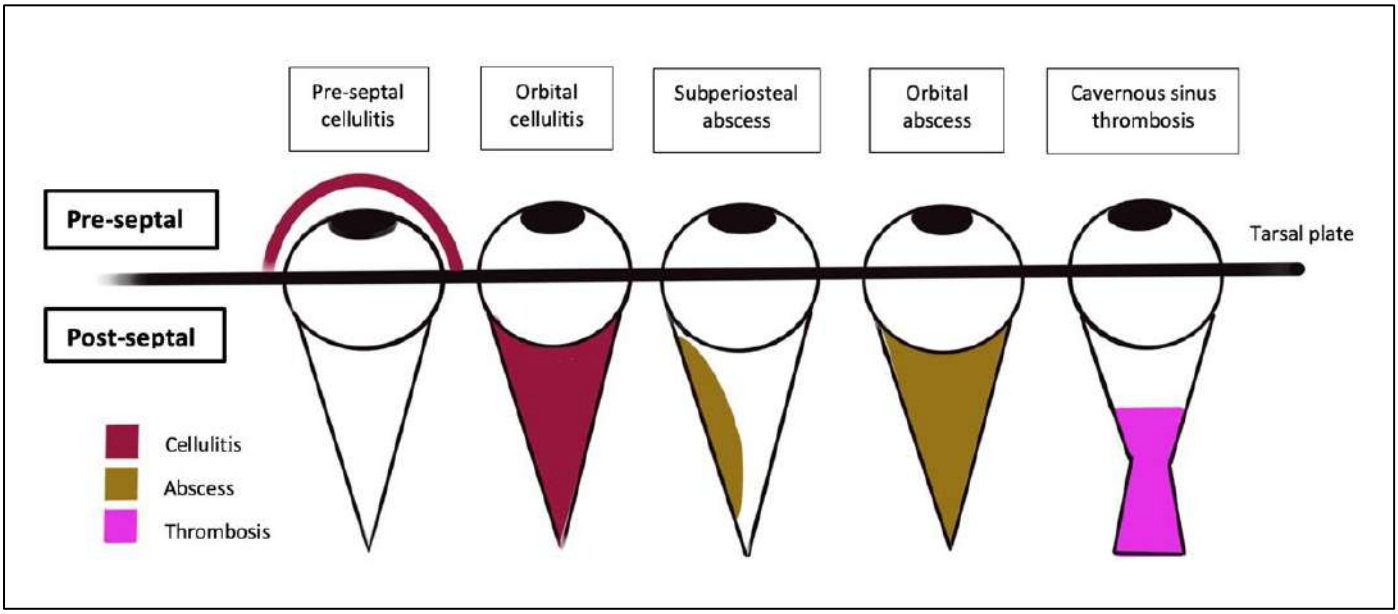
	AVRS	ABRS	Recurrent ARS
Symptoms	Two or more symptoms, of which one should be either: **Blockage/obstruction/congestions** OR **nasal discharge** (anterior/posterior) +/- facial pain/pressure +/- hyposmia/anosmia **(cough in children)**		
Additional symptoms	fever, cough, toothache, halitosis, otalgia, tiredness, pain on bending forward, dysphonia and sore throat		
Time frame	< 10 days No severe fever No lasting purulence No worsening	Can occur early after an AVRS but is rare. Chances of it being secondary bacterial infection correlates with days after onset and severity of symptoms.	≥ 4 / year

Complicated Sinusitis

Complicated sinusitis should be ruled out in every patient with ARS

Eye involvement occurs in 60-80% of cases of complicated sinusitis and is particularly common in children. It is classified according to the Chandler system:

1. **Pre-septal orbital cellulitis** - infection is limited to the soft tissue anterior to the tarsal plate.
2. **Post-septal orbital cellulitis** – infection extends into the orbit
3. Subperiosteal abscess – pus collection between the bony orbital walls and periorbita (usually medially over the lamina papyracea or supero-lateral)
4. **Orbital abscess** – abscess formation within the orbital contents
5. **Cavernous sinus thrombosis** – development of retrograde phlebitis via the ophthalmic veins to the cavernous sinus



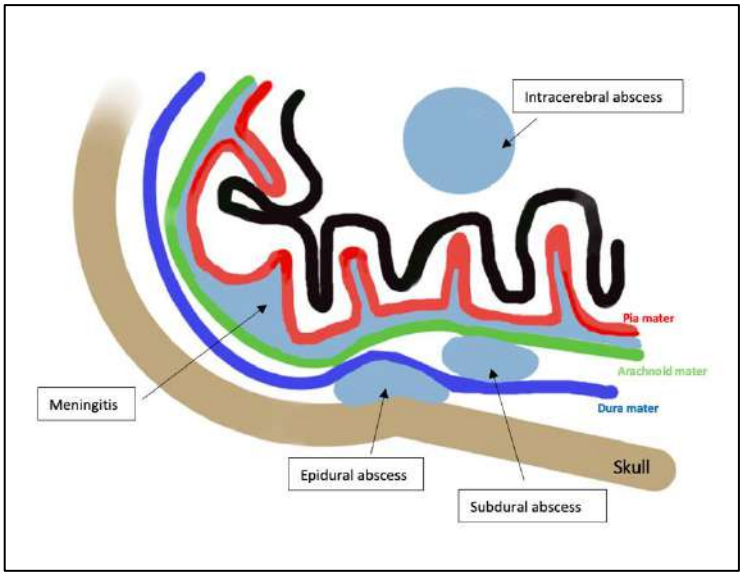
Key signs of orbital involvement include conjunctival chemosis (oedema), erythema, proptosis, limited extraocular eye muscle movements (ophthalmoplegia) and a relative afferent pupillary defect (RAPD), which can be assessed using the swinging flashlight test. Additional indicators such as abnormal colour vision (especially red and green) and visual acuity or field deficits may appear later in the disease process.

Clinical note: Orbital complications are diagnosed clinically. RAPD – reflex arc has afferent (optic) and efferent (oculomotor) nerve supply. First, shine the light into the normal eye causing bilateral pupil constriction because the afferent limb is intact and there is consensual innervation to the efferent limb on the opposite side. Then shine into the affected eye. The optic nerve (afferent limb) is not working as well (due to increased pressure and ischaemia in the orbit), thus the signal is not perceived as well in the affected eye and the pupil dilates. This is a relative afferent pupillary defect (RAPD).

Brain complications are seen in 15-20% of complicated sinusitis cases and is often seen in young adults. Possible intracranial complications include:

1. **Extradural abscess** – localized collection of pus between the dura mater and skull.
2. **Subdural empyema** – pus accumulation between the dura and arachnoid mater.
3. **Bacterial meningitis** – infection of the meninges, often presenting with fever, neck stiffness, photophobia and an altered mental status.
4. **Intracerebral abscess** – localized infection within brain parenchyma, which may lead to neurological deficits.
5. **Cavernous sinus thrombosis** – characterized by bilateral eye swelling due to venous congestion from interconnected veins draining both orbits.

Intracranial complications can present with non-specific symptoms, including fever, headache and subtle behavioural changes. Thus, computerised tomography (CT) imaging is essential to exclude these potentially life-threatening complications.



Bony complications are seen in 5% of cases

1. **Osteomyelitis** – most commonly affects the frontal bone, with maxillary involvement being rare.
2. **Frontal subperiosteal abscess** – previously referred to as Pott's puffy tumour, characterized by a localized swelling over the forehead.

Clinical Note: Any unexplained frontal swelling in children and young adults should raise suspicion for a sinus-related complication. In such scenarios a CT scan is essential, especially to exclude any associated intracranial complications.

Treatment of Acute Rhinosinusitis

AVRS	ABRS

• Education
• Prevention of AVRS: Probiotics & Exercise
• Decongestants < 10 days
• NSAIDS/Paracetamol
• Zinc
• Vitamin C
• Nasal rinses
• Herbal medicine
  ○ BNO1016 / Sinupret®
  ○ Cineole
  ○ Andrographis paniculata
	• As per AVRS
	• Antibiotics as per above clinical features

After diagnosing an ABRS, what antibiotics and what dose?

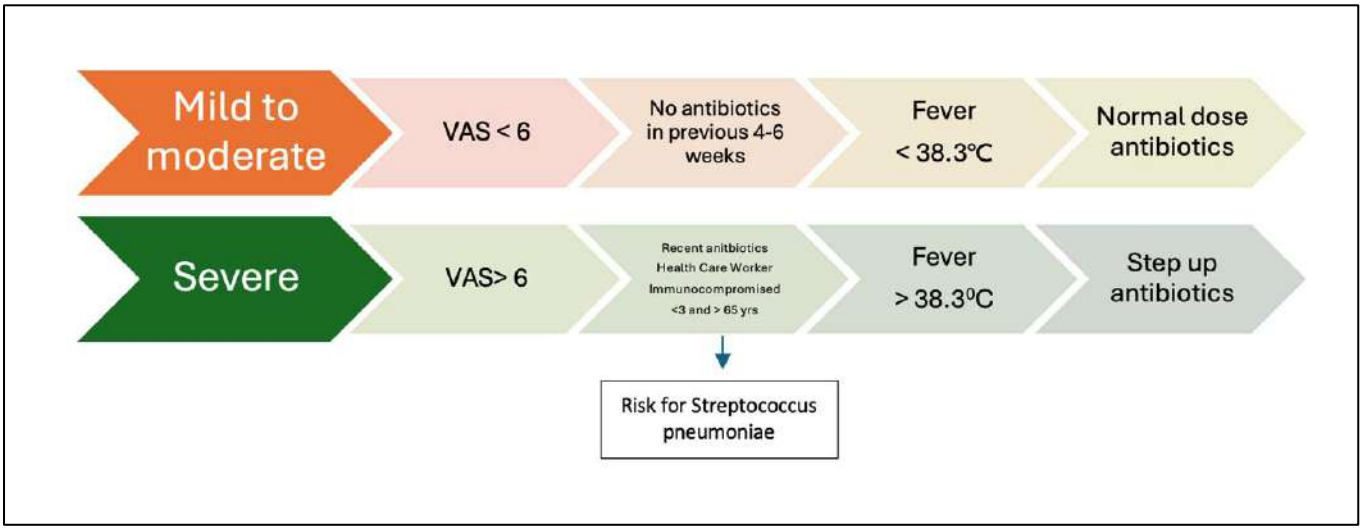
Organisms implicated with spontaneous resolution rates (i.e. without antibiotics)

- Streptococcus pneumonia – 30% → more virulent and may need antibiotics
- Hemophilus influenzae – 60% → Most common strain is non-typeable H. influenza which is resistant to penicillin so need β-lactamase inhibitor (**Co-amoxiclav)**
- Moraxella catarrhalis – 80%

Recommended antibiotic choice:

- Amoxicillin (80-90 mg/kg/d) divided into 2 doses (1g 8hrly) if no prior antibiotic course
- Amoxicillin-Clavulanate (90mg/kg/d – 6.4 mg/kg/d) divided into 2 doses
  - If higher doses of Amoxicillin needed, add Amoxicillin only, no need for extra Clavulanate
- ***β***-Lactam Allergy:
  - Non-type 1:
    - Cefuroxime (30mg/kg/d) divided into 2 doses (1g 12hrly)
    - Cefpodoxime (16mg/kg/d) divided into 2 doses (400mg 12hly)
  - Type 1:
    - Azithromycin (10mg/kg/d) (500mg dly) – 3 days
    - Clarithromycin (15-30mg/kg/d) in divided doses (500mg bd or 500mg modified release daily dose)
    - Erythromycin (40mg/kg/d) in divided doses

- Alternative for Type 1 Allergy:
  - Children: Levofloxacin (20mg/kg/d – daily or divided doses)
  - Adults:
    - Levofloxacin 500mg 12hrly (OR) 750mg dly
    - Moxifloxacin 400mg dly
- Failure of antibiotic treatment after 48-72 hours:
  - Amoxicillin-Clavulanate (90mg/kg/d – 6.4mg/kg/d) divide in 2 doses
  - Ceftriaxone (50mg/kg/d) daily dose (1g dly) – 3 days
  - Alternatives:
    - Clindamycin (90-150mg/kg/d) in 3 divided doses (450mg 8hrly) +/- 2nd/3rd generation Cephalosporin (5-7d)



When to use a higher antibiotic dose:

- Risk factors for *Streptococcus pneumonia (as above)*
- Severely ill, toxic patient
- Complicated disease

18) Cranial Nerves

EXAMINATION

Perspective

As ENT problems are located in the head and neck, which is where the cranial nerves run, they frequently present to the clinician signs of cranial nerve dysfunction (palsies). What is more, the presence of a cranial nerve palsy has a particularly sinister and worrying significance. The importance of a good system of cranial nerve examination should therefore not be underestimated.

Technique

CN I. Olfactory:
Enquire about:

- Hyposmia / Anosmia / Cacosmia
- Onset (slow / sudden / fluctuating)
- Causative factors (trauma / upper airway infections / nasal surgery)

The most common test to confirm if a patient can smell, is to with their eyes closed put some coffee on their tongue. If a patient can smell, they will say they taste coffee. However, in anosmic patients, they only report a very bitter taste. Other classical odours can also be used such as vanilla, lavender, and lemon.

CN II. Optic:
Examine:

- Visual acuity
    - Snellen chart or handheld Snellen chart
- Visual fields
- Colour vision
- Pupillary reflexes
    - RAPD
- Accommodation

CN III, IV, VI:
Examine:

- Eye movements
    - Lateral Rectus CN6, Superior Oblique CN4, Rest CNIII
- Proptosis

CN V. Trigeminal: V1, V2, V3:
Examine:

- Facial sensation
- Corneal reflex
- Muscles of mastication (masseter)

CN VII. Facial:
Examine:

- Motor to muscles of facial expression (see facial palsy)
    - Differentiate upper from lower motor neuron disease
- Stapedius reflex

- Taste anterior two-thirds of tongue

CN VIII. Vestibulocochlear:
Examine:
- Hearing (cochlea)
- Balance (vestibular) - a HINTS exam is useful in differentiating between central and peripheral vertigo

CN IX. Glossopharyngeus & X. Vagus:
Examine:
- Sensation in oropharyngeal region or saying "AH" looking for asymmetries in the soft palate (CN IX)
- Afferent path of gag reflex (CN X)
- For Recurrent Laryngeal branches of X
    - Vocal cord closure (speech, cough, aspiration)
- (Gastric secretion)
- (Pulse etc)

CN XI: Accessory:
Examine:
- Check contraction of sternocleidomastoid's sternal head when patient turns head forcibly against resistance from your hand
- Check for trapezius function by elevating the shoulders
- Check for ability to abduct the arms above shoulder height

CN XII. Hypoglossal:
Examine:
- Tongue movement: straight; left; right
- Check for atrophy, fasciculation (with tongue inside oral cavity), and deviation

19) Congenital conditions of the Head and Neck

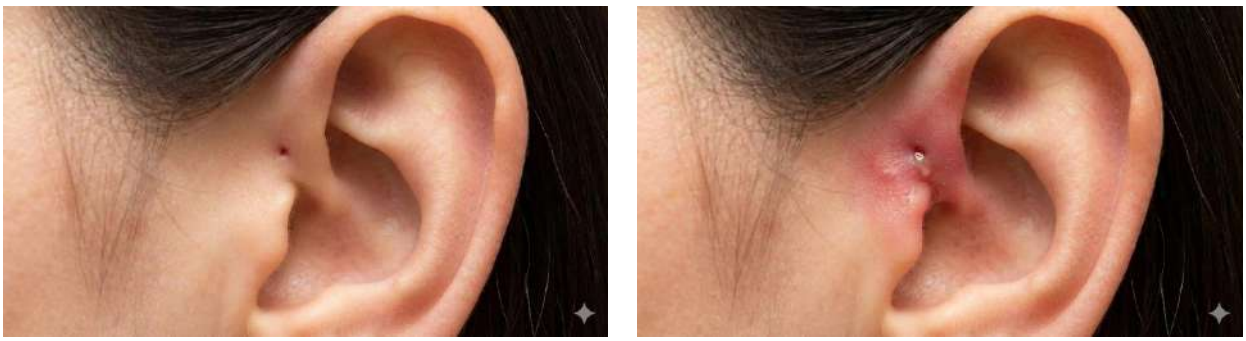
It is impossible to discuss all the congenital conditions of the Head and Neck. The most important will be covered here.

Congenital conditions

Origin	Conditions
Pinna	Pre-auricular pits, sinus and cysts
Branchial apparatus	Branchial clefts and pouch defects
Skin	Dermoid and Epidermoid cysts
Thyroid	Thyroglossal duct cyst
Lymphatic	Macro Micro Mixed
Vascular	Haemangioma Capillary Venous AV fistulas
Larynx	Laryngomalacia Vocal cord palsy Web Subglottic stenosis
Mouth	Ankyloglossia Torus

Pre-auricular

Pre-auricular cysts, also known as pre-auricular pits or sinuses, are benign congenital malformations that occur in the soft tissues in front of the ear. They are characterized by small openings that may lead to a sinus tract under the skin, which can sometimes become infected or form cysts. Most individuals with preauricular cysts are asymptomatic and may not realize they have them until an examination reveals the condition or an infection occurs. Once infected, the treatment of choice is antibiotics and / or aspiration. Try to avoid an incision and drainage, as this will results in a higher recurrence rate after formal surgical removal. Uninfected ones are surgically removed if the patient desires so.



The picture on the left shows a pre-auricular pit / sinus, which is infected on the right. Often, there can be a palpable / infected cyst as well.

Branchial apparatus conditions

Epidemiology

- 19% of paediatric cervical masses
- 2-3% are bilateral
- Tendency to cluster in families
- Manifest mostly in early adulthood
- Peak incidence 3rd decade
- No ethnic or gender predilection

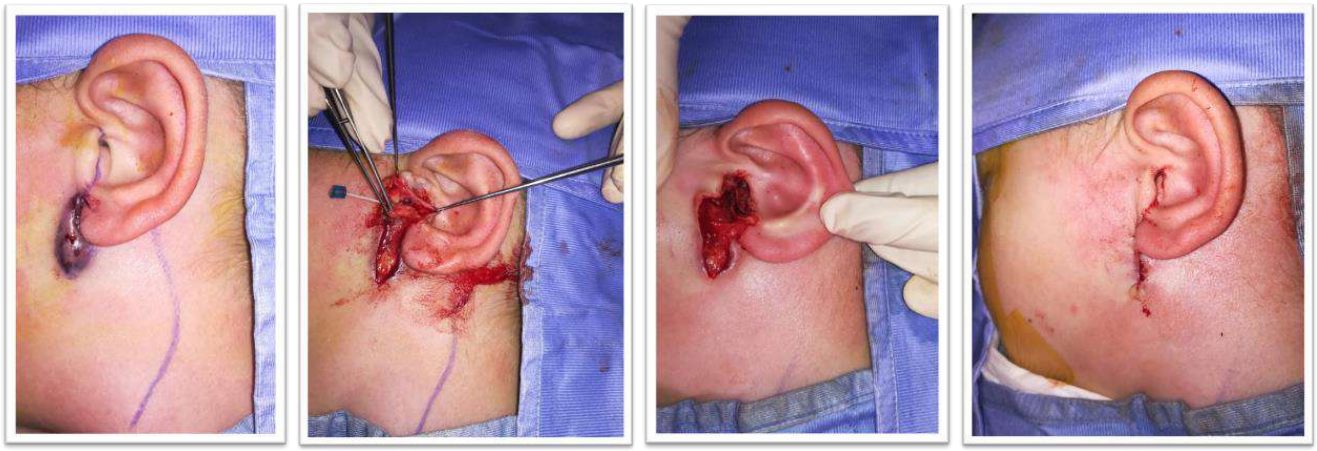
Presentation:

- Mostly asymptomatic lumps or sinuses
- Lateral neck cyst
- Infection or enlargement
- Lateral neck fistula / sinus
- Starts to drain

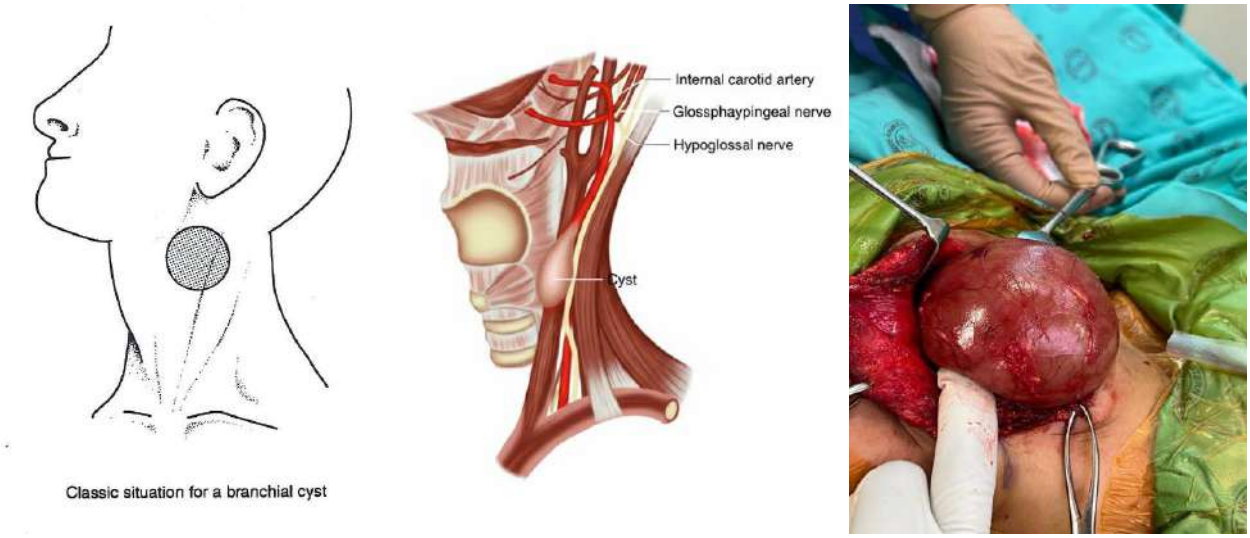
Types:

- First (5-8%)
    - External opening lies on a line between tragus and hyoid bone
        - Can also be post-auricular
        - Often inconspicuous
    - May be a second opening (fistula)
        - Anterior to tragus
        - In EAC at the osseocartilaginous junction
    - Classified into 2 types by Work (1972)
- Second (90-95%)
    - Commonest of the branchial cleft anomalies
    - Sinuses are more common
        - Open low in the neck
        - Anterior to the SCM
        - Leak mucoid or clear fluid
    - Fistulas are rare
        - Not always patent throughout length
        - Communicate with supratonsillar fossa
    - Cysts
        - Inferior to angle of mandible and anterior to SCM
    - Association with branchio-oto-renal syndrome
- Third (rare)
- Fourth (rare)

First branchial cleft operation type I

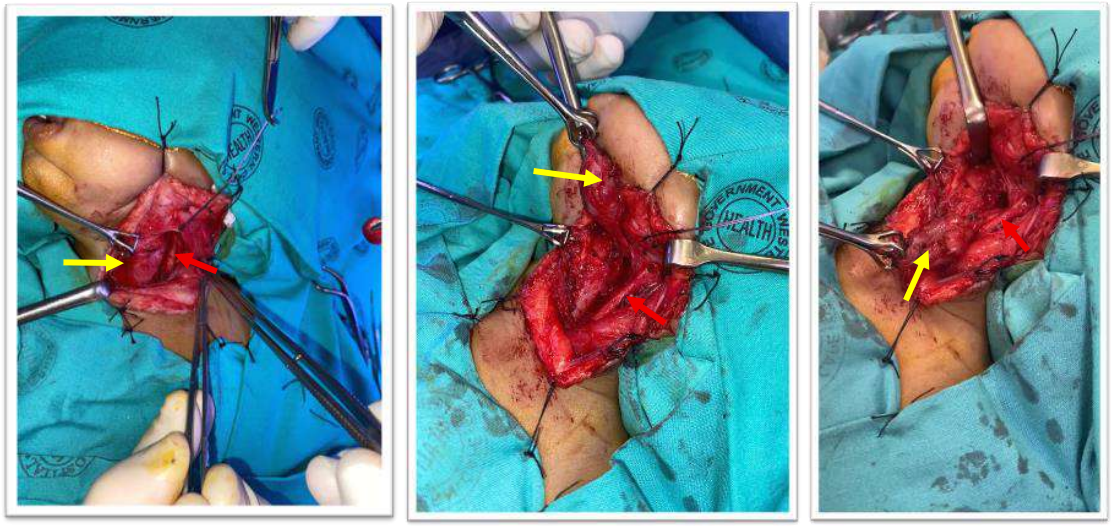


Second branchial cleft classic position and operation



Three pictures showing the classical position on the left side (level IIa), its embryological pathway and tract with a possible opening at the supratonsillar fossa, and an intra-operative picture of a very large second branchial cleft cyst.

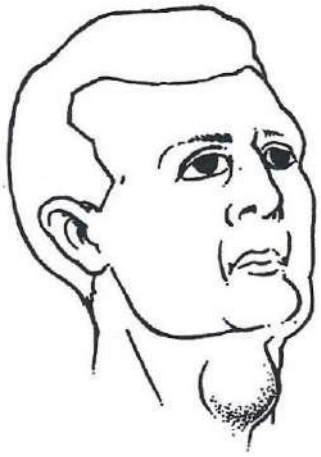
Fourth branchial cleft operation



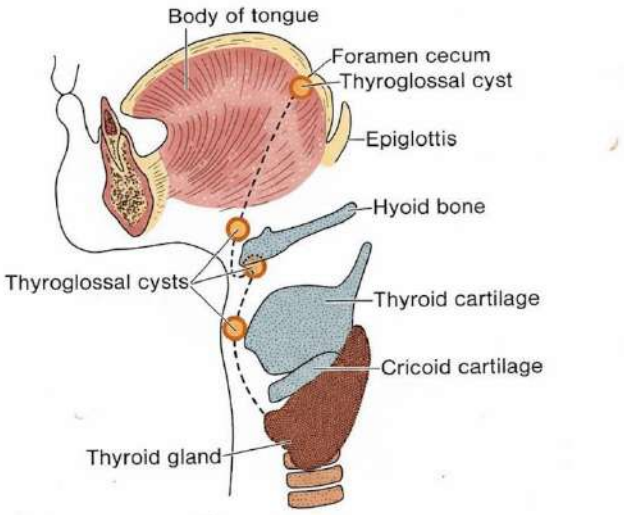
A fourth branchial cleft cyst. The yellow arrow points to the cyst and the red to the carotid artery.

Thyroglossal duct cysts

It is said to compose one third of all congenital neck masses. Typically, in the midline position but can be off to the side in some cases. Rarely occurs in children and typically seen in young adults as the cyst accumulate enough mucous. It can rapidly enlarge when cysts become secondarily infected or when there is haemorrhage. There is a very small chance of developing into a thyroid cancer. It can occur anywhere along the line of development of the thyroid between the base of the tongue and the thyroid itself (see picture below). Occasionally there is an associated fistula, due to the cyst having burst. Surgical excision is the preferred treatment and entails removal of the cyst and its tract, with the body of the hyoid bone (Sistrunk operation). However, before surgery it is important to investigate if the thyroid gland is in its normal position.



Thyroglossal cyst This is the usual site. In addition to moving with swallowing, a thyroglossal cyst also moves upwards when the tongue is protruded.



Embryological tract of the descending thyroid gland, with typical areas where a thyroglossal duct cyst can occur. The most common area is over the hyoid bone.

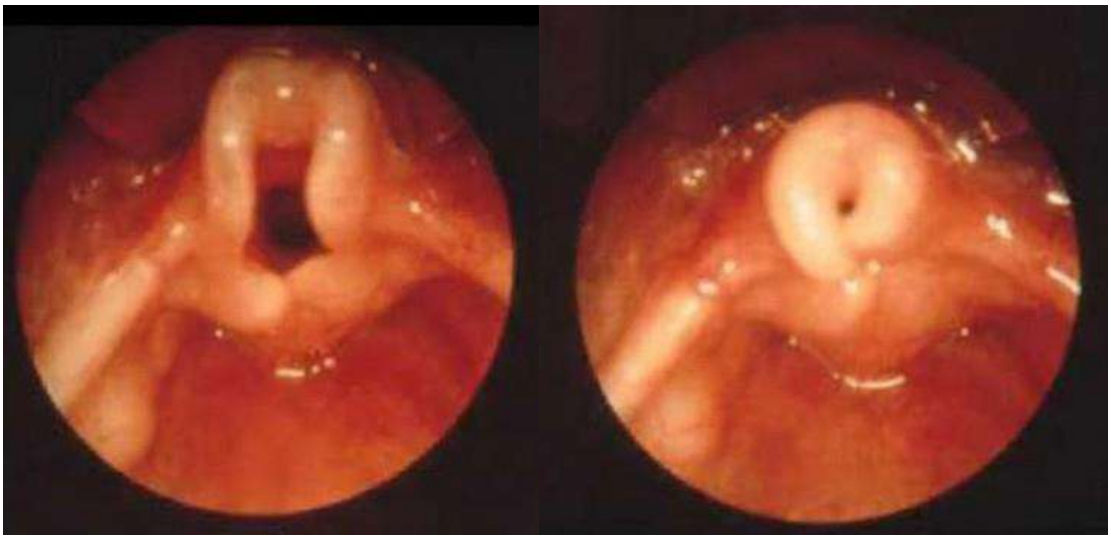
Laryngomalacia

Laryngomalacia is the most common reason for stridor in children. It causes 60% of all laryngeal disease in children. Typically, the children develop an intermittent, low tone, inspiratory stridor within 2 weeks after birth

(the stridor is typically not present at birth). In general, it worsens up and to 9-12 months and 75% will clear up spontaneously at 18 months. Rarely it can last up to 5 years. Signs of a complicated laryngomalacia (and therefore referral to an ENT) include:

- Feeding problems
    - As long as the babies does not need to "come up for air" while breast or bottle feeding, interventions are usually not needed.
- Failure to thrive
- Apnoea
- Cyanosis
- Pectus excavatum

Various theories try to explain the underlying pathology such as a neuro-muscular problems, floppy cartilage, and hypotonia. The most common finding is that of an omega shaped epiglottis, but various others have been described such as short aryepiglottic folds, posterior collapse, mucosa prolapsing into larynx from the arytenoids, and a deep narrow glottic inlet (see pictures below). Mild laryngomalacia will improve with crying, as opposed to severe forms which will worsen with crying. The babies also improve when turned to the prone position. Reflux plays an aggravating role, and babies are routinely put onto PPI. Only 20% will require intervention and it is usually the group with complicated features mentioned above. Therefore, the majority is managed by explaining the disease process to the parents, given advice regarding PPI and positing of the child, and follow up.



Two pictures demonstrating the typical omega shaped epiglottis on the left and near complete closure on inspiration on the right (causing the stridor).

Vocal cord palsy

It is the second most common congenital condition in the larynx after laryngomalacia. It is most commonly due to intracranial, mediastinal, or heart pathologies. This includes Arnold Chiari malformations and various heart defects. The clinical picture and management depend on whether it is unilateral or bilateral.

	Unilateral	**Bilateral**
Presentation	Fairly asymptomatic except for weak cry and aspiration	Can have catastrophic stridor
Spontaneous recovery	70% of non-iatrogenic palsies clears up in 6 months	50% of non-iatrogenic palsies clears up in 24-36 months

Intervention	Rarely interventions	Frequently a tracheostomy is placed for the child to grow to such an age (1-2 years) that a definitive procedure is planned

Congenital subglottic stenosis

Congenital subglottic stenosis is the most common reason for kids under the age of 1 year to require a tracheostomy. It comprises 20% of all congenital laryngeal conditions and frequently occurs with other problems such as Down syndrome and laryngeal clefts. It is due to a narrow cricoid (< 3.5 mm lumen in term babies) and presents with bi-phasic stridor. Sometimes kiddies present with prolonged croup-like symptoms. Any child with upper airway symptoms should be referred to an ENT specialist. Management entails waiting until the child grows and then performing an enlarging procedure either endo-luminal or external. In most cases cartilage grafts are used to achieve this.

Congenital haemangiomas

Is said to be the most common neonatal tumour and occurs in 4-10% of children below the age of one year. It is more common in females and 50% of them occurs in the head and neck area. The most common area affected in the head and neck is the subglottic area. Haemangiomas in trigeminal distribution should raise the suspicion of a subglottic haemangioma. Currently, congenital haemangiomas are divided into rapidly involuting, non-involuting, and partially involuting. Despite these different types, classical teaching involved that they enlarge up until the age of one year. Thereafter, 50% would disappear at the age of five years, 70% at seven years, and 90% and 9 years respectively. Management is complex, and these patients should be referred to ENT paediatric units.

Ankyloglossia

Ankyloglossia is commonly known as “tongue tie”. It is due to failure of tongue to separate from the mouth floor and occurs in varying degrees. The most common presentation is a short frenulum that can impair sucking and later on speech development. There is some debate whether it can cause teeth alignment problems. The prevalence is estimated at 4-11% but the cause is still unknown although genetics may play a role as it tends to occur in families. Sometimes it occurs in conjunction with upper lip tie as well. In general, it will be more severe in the first-time breast-feeding mother.

Important points to ask and examine are the following:

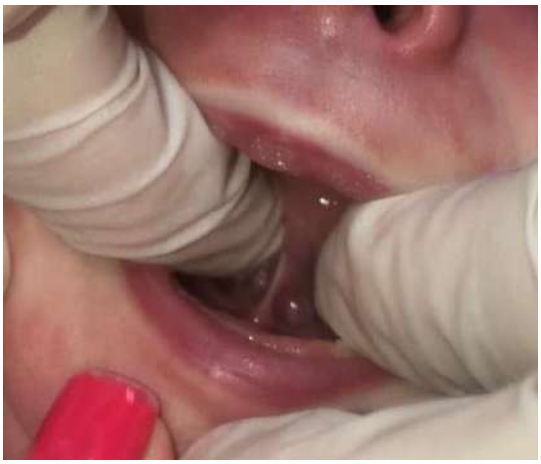
- Baby symptoms
  - Not sufficient weight gain
  - Aerophagia
  - Numerous feeding times required
  - Stop-start feeds
- Maternal symptoms and signs
  - Breast pain
  - Nipple problems
  - Breast does not empty fully with feeding
  - Mastitis

Various classifications exist, but the two most often used are:

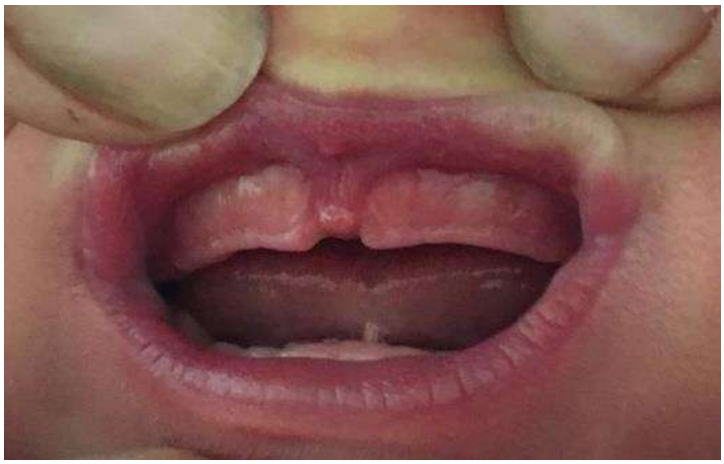
- Coryllos criteria
  - I – Frenulum attached to tip of tongue
  - II – Frenulum 2-4 mm behind tip of tongue

    - III – Frenulum attached to mid tongue
    - IV – Frenulum attached to base of tongue, but thick and inelastic
- Kotlow upper lip
    - I – No significant attachment
    - II – Attachment mostly into the gingival tissue
    - III – Attachment in front of the anterior papilla
    - IV – Attachment into the papilla or extending into the hard palate

The management of the babies remains controversial, mainly because of lack of high-level evidence. However, it is estimated that the USA will save $13 billion if 90% of mothers complied with exclusive breastfeeding for 6 months. In reality, 80% of mothers who initiate breastfeeding but will stop if tongue tie is not corrected. Maternal nipple pain is a sensitive indicator for tongue / lip problems. Furthermore, isolated posterior tongue-tie problems have been neglected / missed in the past as these babies often present with aerophagia and subsequent abdominal cramps and vomiting. The authors advise that patients should be referred to an ENT with a keen interest in the subject.



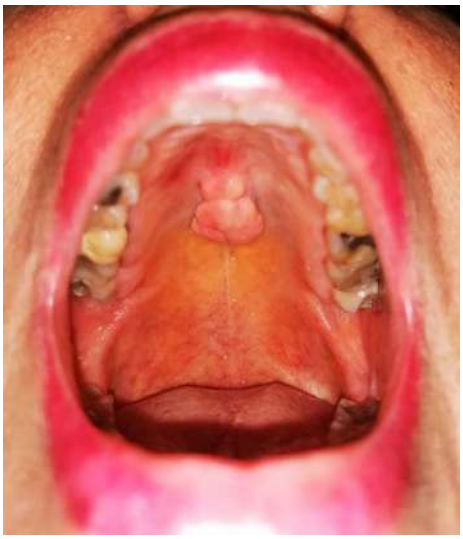
Picture showing a tongue tie (left).



Picture showing a lip and tongue tie (right).

Torus

Torus mandibularis and palatine are benign bony overgrowth. They typically occur on the inner table of the mandible and on the hard palate. The mucosa over them may be injured causing an ulcer. Surgical removal is indicated if they are symptomatic.



Picture showing a torus palatine.

20) Airway obstruction and compromise - a clinical approach to Hoarseness, Stertor, and Stridor.

Managing a patient with acute upper airway obstruction remains one of the most challenging conditions you might face in your career. This is even more so when it is a child. This chapter aims to give you an overview of how to approach it from different angles, but also emphasises that taking a history, stabilising the patient, and deciding on the appropriate intervention happens all at once the more severe the obstruction.

Definitions

Hoarseness
A non-specific, general term used to describe any change in voice quality, perceived as rough, harsh or breathy.

Stertor
Due to vibration of tissue above the level of the larynx. An example is adenotonsillar hypertrophy. Think "English Bulldog" (low pitch, rough, snoring sound).

Stridor
Usually a high pitched, musical, or harsh sound often mistaken for "wheezing". It can be:

- Inspiratory
    - Larynx
- Expiratory
    - Distal trachea and mainstem bronchi
- Biphasic
    - Glottis, subglottis, and upper trachea

Approach

Classical – think in terms of aetiology

- Congenital
- Acquired
    - Infective
    - Inflammatory
    - Trauma
    - Neoplastic
    - Allergic
    - Medications

Time – think in terms of onset and severity

- Acute - Life threatening
    - Anaphylaxis
    - Foreign body in larynx / trachea
- Sub-acute
    - Upper airway infections
        - Laryngotracheobronchitis
        - Epiglottitis / Supraglottitis
    - Juvenile onset respiratory papillomatosis – although this is a late sign, patients frequently present with severe stridor
    - Adenotonsillar hypertrophy

- Chronic
    - Laryngeal cancers – although this is a late sign, patients frequently present with severe stridor
    - Airway stenosis – often misdiagnosed and mismanaged as “asthma”

Position – think in terms of pathology

- Lumen
    - Intra-luminal
    - Luminal
    - Extra-luminal
- Level
    - Nasopharynx
    - Oral cavity / Oropharynx
    - Hypopharynx and Larynx
    - Trachea
    - Bronchial tree
    - Mediastinum

Differential diagnosis

Below follows a broad approach to possible differential diagnosis, and it should not be seen as a comprehensive list of all possible causes.

Pathological conditions – Based on aetiology and level

	Naso-, Oro-, Hypopharynx, Oral cavity	**Larynx, Trachea, Bronchi, Mediastinum**	**Other**
Congenital	• Choanal atresia • Mid nasal stenosis • Skull base dehiscence with meningocele / encephalocele • Skull base anatomical problems with syndromes such as Down’s, Pierre Robin, Treacher Collins	• Laryngomalacia • Webs • Cysts • Vocal cord palsy • Tracheomalacia • Tracheal vascular rings	• Any developmental abnormality with hypoplasia, hyperplasia, atresia, anaplasia

Infective / Inflammatory	• Adenoids
	• Tonsils
	• Diphtheria
	• Cellulitis or abscesses in potential spaces of neck
	  • Retropharynx
	  • Prevertebral
	  • Danger space
	  • Parapharyngeal
		Classical diseases
		• Epiglottitis
		• Croup
		• Bacterial tracheitis	

Trauma	• Inhalation • Thermal • Chemical • Ingestion • Foreign body • Caustic • External		

	• Blunt • Penetrating • Crushing • Iatrogenic • Intubation • Operations		
Neoplastic	• Squamous cell carcinoma (typical) • Nasopharynx cysts	• Squamous cell carcinomas (typical) • Laryngeal papillomatosis • Haemangiomas	• Benign and malignant tumours of any tissue differentiation e.g. Minor salivary gland tumours; sarcoma; lymphoma

Allergic / Medication	• Rare	• Common
		  • Anaphylaxis
		  • Angio-neurotic oedema	

Pathological conditions – Based on Hoarseness

Organic	**Cause**
Inflammatory	Acute laryngitis, Chronic laryngitis
Neoplasia	Papillomatosis, Cancers
Neurological	CVA, Multiple sclerosis, Guillain-Barre, Myasthenia gravis, Carcinoma of the lung / mediastinum, Idiopathic (virus), Spasmodic dysphonia
Iatrogenic	Post surgery – thyroid, neck, carotid, larynx, oesophagus
Systemic	Hypothyroidism, Rheumatoid arthritis

Pathological conditions – Based on Age specific causes of stridor

Age group	**Cause**
Neonatal	Congenital cysts, webs, tumours
	Laryngomalacia
	Subglottic stenosis
	Vocal cord paralysis
Children	Laryngotracheobronchitis
	Supraglottitis (Epiglottitis)
	Acute laryngitis
	Foreign body
	Retropharyngeal abscess
	Respiratory papillomatosis
	Diphtheria
Adults	Laryngeal cancer
	Laryngeal trauma
	Acute laryngitis
	Supraglottitis (Epiglottitis)
	Laryngeal / Tracheal stenosis
	Angioneurotic oedema / Anaphylaxis
	Diphtheria

Management

History

- When did it start?
    - At birth / later in the case of a child
- How did it start?
    - Suddenly / Slowly progressive
- Is it fluctuating or constant?
- Is it stridor or stertor?
- Enquire about
    - Voice / Hoarseness
    - Feeding
    - Aspiration
    - Airway / Breathing
    - Dying spells (cyanotic spells with apnoea)
    - Cough
- The effect of
    - Exercise
    - Infections
    - Position
    - Feeding
    - Previous treatments
- Think about the possible differential diagnoses
    - Aetiological
        - Congenital
        - Acquired
    - Onset and severity
    - Anatomical level
- Look at associated symptoms and signs such as
    - Tachypnoea
    - Tugging
    - Accessory muscle use
    - Apnoea / Dying spell
    - Coughing
    - Signs of acute infections

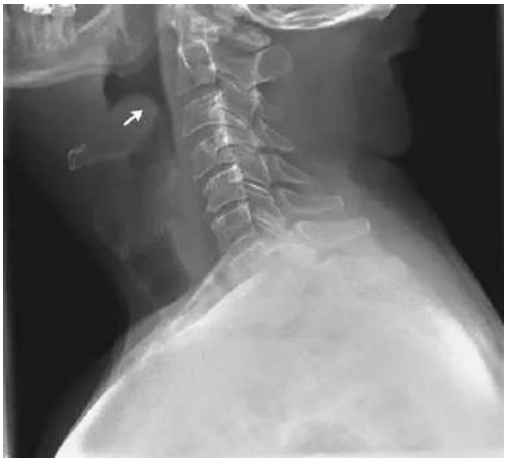
Examination

- The more acute the situation, the more focused the initial approach
    - This means to absolutely focus on securing an airway
    - The management of these patients will be discussed later
- ENT
- General (infection)
- Look specifically for
    - Cyanosis / Pallor
    - Nasal flaring
    - Use of accessory muscles
    - Tracheal plugging
    - Chest wall recession
    - Tachycardia

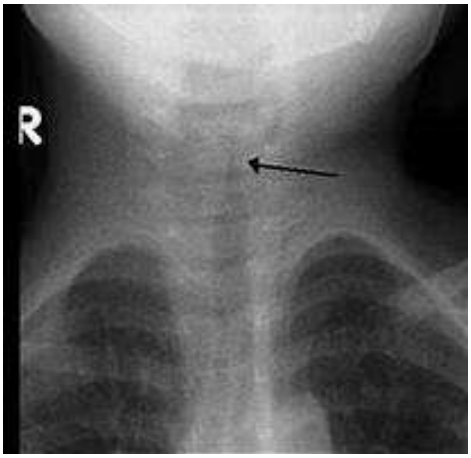
    - Tachypnoea
    - Cough
    - Exhaustion
        - Can paradoxically be quieter

Special investigations

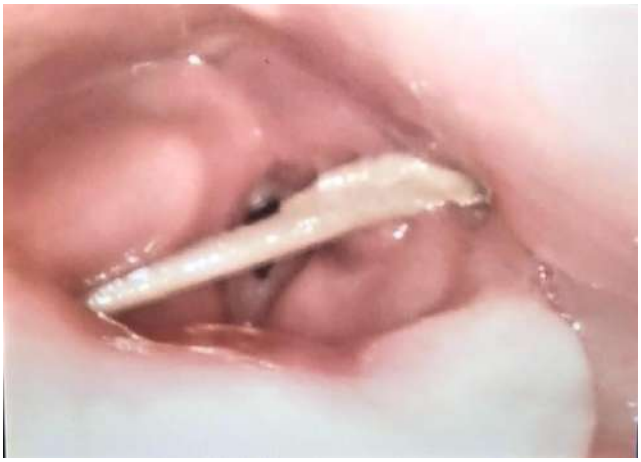
- SaO2
- Arterial blood gas
    - Should not unnecessarily delay or cause more distress in the patient (especially children)
- XR – can be useful in acute setting
    - Thumb sign – points to epiglottitis (lateral neck Xray)
    - Steeple sign – points to laryngotracheobronchitis (AP neck Xray; Not Chest Xray)
    - Foreign body
- Bedside endoscopy – only if patient is stable enough. In general, this will only be in the scope of an ENT specialist
- Examination under anaesthesia in theatre
- CT / MRI
- Bloods
- Other



Thumb sign (arrow demonstrates the swollen epiglottis)



Steeple sign (arrow demonstrates the narrow subglottis)


Foreign body (bone) in supraglottis

Management in general
This will differ depending on your level of experience, but unfortunately also whether you are working alone or in a team setting. Sometimes, you may be confronted with managing a severe airway compromise without any cover.

As a GP, you can do the following after a history and examination:

- Put the patient on oxygen
- If there is any suspicion of and anaphylaxis, use adrenaline immediately
- Steroids
- Nebulize – Adrenaline (1:1 concentration, meaning 1ml amp of adrenaline and 1 ml saline)
- Calm down / Re-assure
- Other
    - SaO_2
    - Blood gas
    - Radiology
        - XR, CT, MRI
- Get help

An ENT will, in general, do the above and:

- The most important step is visualization of the larynx
    - Flexible laryngoscopy at the bedside (only if the condition allows it)
    - Rigid laryngoscopy (direct laryngoscopy) in theatre
- Where should this be done?
    - Rooms / Theatre (OR)
    - The more severe the stridor, signs of acute infection, the younger the age of the patient – rather theatre

Management of the acute airway obstruction - Securing the airway

- If there is any doubt about the severity, rather take the patient to theatre (OR) if that is available to you and it does not cause unnecessary delays. Otherwise, you need to manage it there and then
- Always mobilise the team which can include an ENT, Anaesthesiologist, Paediatrician, Nurse, ICU staff
- Equipment – make sure that all the equipment is available

    - Various endotracheal tracheal tube sizes, laryngoscopes blades, flexible scopes, visualization scopes (C-Mac)
    - Suction and instruments such as a Magill forceps
    - If available, the tracheostomy set should be open
- Stick to ABC principles
    - AIRWAY
        - Avoid unnecessary manipulation of the airway and muscle relaxants
        - Most cases can be intubated, and a tracheostomy is rarely needed, however the most common mistake is to absolutely fixate on intubation and losing track of other means to ventilate
        - Therefore, a stepwise approach to ensuring oxygen supply is
            - Oxygen mask
            - Ambu bag
            - Laryngeal mask
            - Intubation
            - Surgical airway
                - Needle cricothyroidotomy
                - Surgical cricothyroidotomy (avoid in patients under 12 years of age due to small cricothyroid membrane, funnel shaped larynx and increased risk of subglottic stenosis)
                - Quick tracheostomy set
                - Tracheostomy

Surgical airway
Hopefully, very few of you will be confronted with performing a surgical airway. Once you get to this stage, the situation is usually dire. This is often after failed intubation(s). In children the "time" at hand is even less, and they can decompensate extremely quickly. Two acronyms are frequently used namely:

- CICO – can't intubate, can't oxygenate
- FONA – front of neck access

To complicate matters even further, the pathology leading to the airway compromise often alters the neck, hypoxic patients never lie still and is frequently combative, and there are lots of critical structure in the way

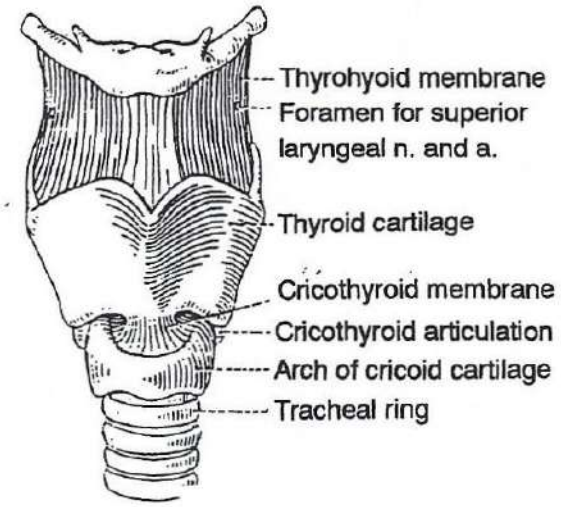
You have two options:

- Cricothyroidotomy
    - Needle
    - Scalpel (surgical)
- Tracheostomy
    - Open
    - Percutaneous

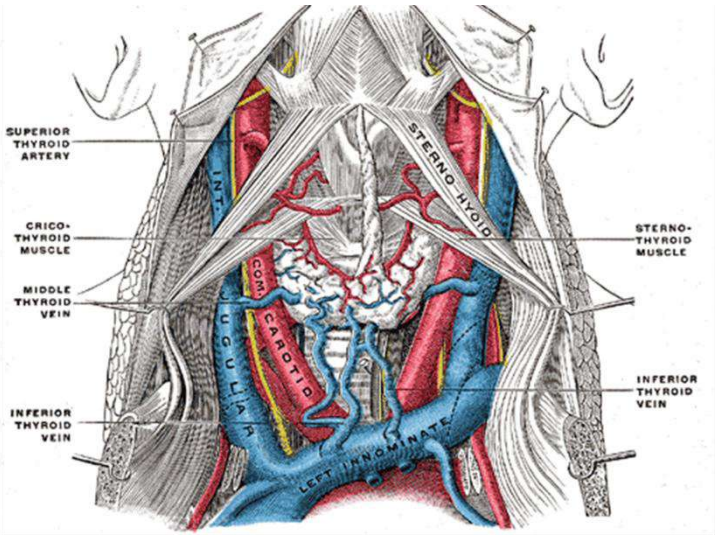
Also consult the following online resources:
https://www.youtube.com/watch?v=B8I1t1HlUac
https://youtu.be/WQOwSLWlHec

Below follows a brief outline of the anatomy and options available to you.

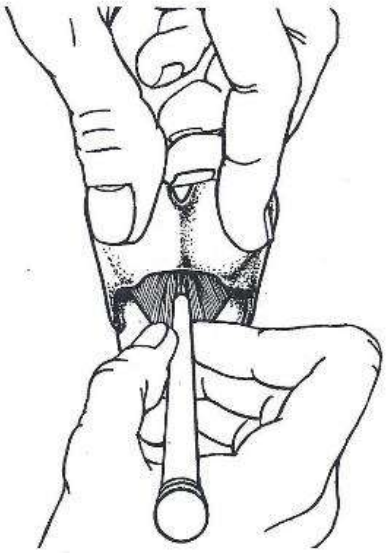
Basic anatomy



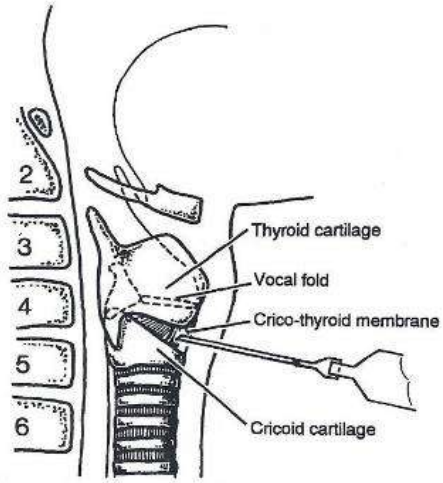
Cartilages of the larynx from the front



Needle cricothyroidotomy is done through the cricothyroid membrane, but it can also be inserted directly into the trachea. Use large bore (14 – 16 gauge) intra-venous needles attached to a syringe (5-10 ml) halve filled with water. Introduce through the skin and apply suction. As soon as you see air, advance cannula inferiorly.

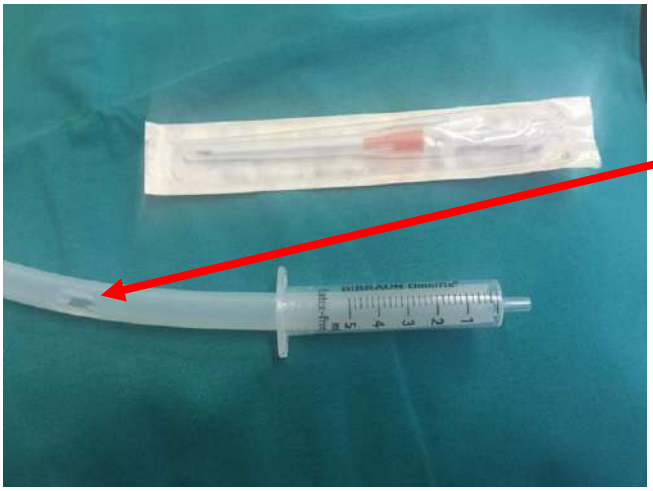


1. Identify cricothyroid cleft. Insert trocar and cannula centrally through cricothyroid ligament.



Crico-thyrotomy
Done through notch between tyroid and cricoid cartilages which can easily be felt with the finger.

To ventilate after inserting / advancing at least 3-4 needles inferiorly, you can attach a suction tube to the oxygen outlet and then onto a 5 ml syringe the fits into the back of the needle you have place. Cut an opening in the tube that acts like a valve, meaning when you close it with your finger, you force oxygen into the trachea. REMEMBER, to place another 3-4 needles in the same airway for air to escape otherwise you risk over inflating the lungs with possible pneumothoraxes. This buys you 15 – 30 minutes to do a more formal surgical airway.



Some units have “quick sets” as shown below. You may attempt to do a tracheostomy or cricothyroidotomy with such a set.

Commercially available quick tracheostomy set by Rüsch Medical (PTY) LTD company.

For a **surgical cricothyroidotomy** you will need a:

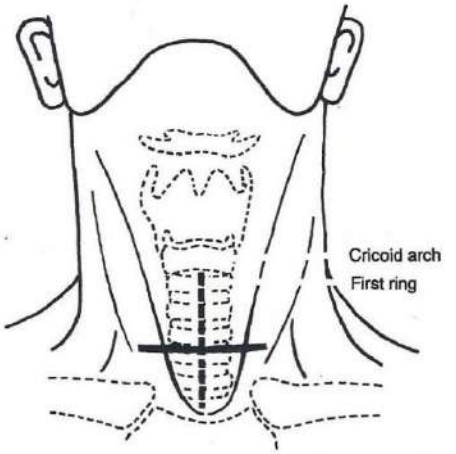
- Blade
- Bougie
- Endotracheal tubes (different sizes)
- Tracheal dilator
- Laryngoscope with small blade

The steps are:

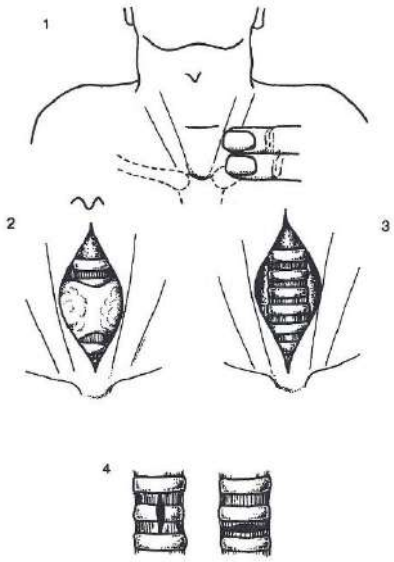
- Position your patient first – chin lift and neck extension and stay in the midline!
- Make a vertical incision of 6-8 cm
    - Remember, it will bleed
- Perform a laryngeal handshake with your non-dominant hand AND use your fingers to pull the tissue apart and palpate for the cricothyroid membrane
    - A laryngeal handshake is shown below, but it means holding the thyroid lamina between your thumb and fingers
- Insert scalpel blade vertically and while in-situ twist through 90^0
- While the scalpel blade is still in-situ, insert bougie and railroad endotracheal or tracheostomy tube over bougie

- Sometimes, a laryngoscopy with a small blade may come in handy. Use the blade to "hook" the tissue downwards, with the tip of the blade as close to or in the trachea. The light at the tip helps to "see" in the depths of the wound.

In the rare event that you attempt a **surgical tracheostomy**, we advise a vertical incision on the skin and in the trachea. In reality, this will only be done by very experienced ENT or Trauma surgeons.



Incisions for tracheostomy. The transverse incision is preferable for elective procedures, but the vertical one is quicker in an emergency. The thyroid isthmus usually overlies the second, third and fourth tracheal rings, and so must be divided for access to the trachea at its correct level. It is absolutely essential that the first ring is kept intact.



How to perform a tracheostomy
1. Midline incision
2.+ 3. Dividing the thyroid isthmus when necessary.
(Usually the latter can be retracted superiorly out of the way)
4. Incising the trachea. NB preserve 1st tracheal ring.

If you are confronted with a surgical airway:

- Try to stay calm
- Try to position the patient as best you can – neck extension / chin lift
- Stay in the midline
- "Search" for the airway with your fingers and a syringe (filled with water) and needle
- It will bleed
    - Compress once airway secured

Golden rules in Stridor

- Acute onset stridor is a late sign, and should alert you to the seriousness of the situation
- Remember the more severe the obstruction the quieter the child / stridor
    - Anxious / restless to semi-comatose
- The smallest mucus plug can cause a complete obstruction
- Take note of the possibility of supraglottitis in adults
    - Especially in the immunocompromised group
- Failure to respond to medications is usually an indication of a severe case, and / or one also needs to consider other pathologies
- Early use of adrenaline is cardinal in the treatment of anaphylaxis (within 30 minutes of onset of symptoms)

Diseases

Some disease will be briefly discussed. Please also refer to other textbooks.

Laryngotracheobronchitis

Also known as croup and is characterized by

- Most common infectious cause of stridor in children
- 3-5% of children have at least one episode
- Parainfluenza viruses (other as well)
- Clinical features
    - Nonspecific viral prodrome
    - Triad of hoarseness, stridor with a distinct expiratory barking cough, and varying degree of upper airway obstruction
    - Usually not toxic
    - 3-7 days
- Specific points / parameters to consider
    - Whether the stridor is inspiratory, expiratory, or biphasic
    - Respiratory rate
    - Chest retractions
    - Air entry into chest (auscultation)
    - Anxiety / restlessness
    - Colour or cyanosis
    - Level of consciousness
    - Oxygen saturation
- 30% admitted, and less than 5% needs intubation

Treatment / Management

- Keeping the child and the parent calm
- Steroids
    - Oral / IV / nebs
    - Oral and IV equally effective
        - Oral prednisolone 1mg/kg
        - Oral dexamethasone 0.15mg/kg
        - IV / IM dexamethasone 0.6mg/kg
    - Low vs high dose studies have been equivocal
- Adrenaline
    - Nebs
    - Remember potential rebound swelling, therefore children should be observed for at least 3-4 hours
- Humidified air
- Securing the airway
    - Intubation vs tracheostomy

Croup should be considered atypical

- If,
    - It occurs in infants younger than 6 months
    - Lasts more than 7 days
    - Is unusually severe
    - Does not respond to the appropriate treatment

- In this group consider
    - Respiratory papillomatosis
    - Supraglottitis / Epiglottitis
    - Foreign bodies
    - Thermal injuries or caustic ingestion
- This group of patients needs a laryngoscopy

Two subcategories

- Recurrent croup
    - 5% of children
    - Consider
        - Gastro oesophageal reflux disease (GERD) / Laryngo-pharyngeal reflux (LPR)
        - Congenital subglottic stenosis or haemangiomas
- Spasmodic croup
    - Nighttime acute episodes of croup-like symptoms without a preceding viral prodrome
    - It is linked to allergy reactivity and GERD / LPR
    - Responds to nebulized adrenaline

Supraglottitis / Epiglottitis

Characterised by

- Rapid progressive, life-threatening airway emergency
- Cellulitis of the supraglottic structures
- Traditionally it affects children below the age of 5 years (median 3), and was caused by *H. Influenza* type B
- After the introduction of the HIB vaccine the median age shifted from 3 years to 6 – 12 years
- In addition, a greater proportion of cases now occur in adults
    - Especially in the immunocompromised group
    - Presents with a much milder form of the classical disease
- The causative organisms have changed, and are now include *Strep pneumonia, Strep pyogenes, Staph aureus,* and other *H. Influenza*

Hallmark features include

- Dysphagia and throat pain
- Drooling
- Respiratory distress that are rapid in onset and progression
- Patients are toxic and anxious, and typically sits in the tripod position
- Shallow breathing
- Stridor and respiratory retractions are late and concerning signs of impending complete obstruction

Treatment / Management

- All patients suspected of having supraglottitis should be taken to theatre (OR), Resuscitation room
    - Except adult cases with very mild symptoms or
    - If the patient is too unstable to transport
- Securing the airway is the most important thing
    - Oedematous, erythematous epiglottis with varying degrees of airway obstruction
    - Frank ulceration, sloughing, and rarely an abscess may be present
    - Classically a cherry red epiglottis, but in the adult population one now sees a pale and swollen epiglottis
- Cephalosporin, steroids, HIB vaccine

Differential diagnosis

- Croup
- Bacterial tracheitis
- Retro- or para-pharyngeal abscess
- Diphtheria
- Foreign body

Retropharyngeal abscess

Characterized by

- Almost exclusively seen in infants and children, due to lymphadenitis in retropharyngeal nodes
    - These nodes undergo atrophy as you get older
- Sudden onset and acutely ill child
- Inspection of oral cavity – huge bulge from posterior pharyngeal wall
- Needs incision and drainage

Differential diagnosis of Upper Airway Infections in Children

	Croup	**Supraglottitis**	**Bacterial tracheitis**	**Retropharyngeal abscess**
Age	6 months – 3 years	1 – 8 years	6 months – 8 years	1 – 5 years
Onset	Slow	Rapid	Rapid	Slow
Prodrome	URI symptoms	None / mild	URI symptoms	URI symptoms
Fever	Variable	High	High	Usually, high
Hoarseness / Barking cough	Yes	No	Yes	No
Dysphagia	No	Yes	Yes	Yes
Toxic	No	Yes	Yes	Variable
Radiographs	Subglottic narrowing – steeple sign (AP neck Xray)	Rounded enlarge epiglottis – thumb sign (lateral neck Xray)	Subglottic narrowing, Diffuse haziness	Widened prevertebral space (lateral neck Xray)

Laryngomalacia

Also see section under congenital conditions

- Most common cause of stridor in infants
- It presents with intermittent inspiratory stridor within the first two weeks of life (typically not present at birth)

- The vast majority will resolve spontaneously over the next 7-9 months (rarely after 18 months)
- It worsens with feeding, and often the infant needs to take breaks while feeding
- Mild forms will improve with crying, as opposed to moderate to severe laryngomalacia which will worsen
- Often the stridor improves when turning the infant prone
- There is a strong association with GERD / LPR
- Rarely a child requires surgery to correct this
- Refer a child if there are any of the following
    - Apnoea
    - Failure to thrive
    - Feeding difficulties
    - Aspiration
    - Cyanosis

Foreign body

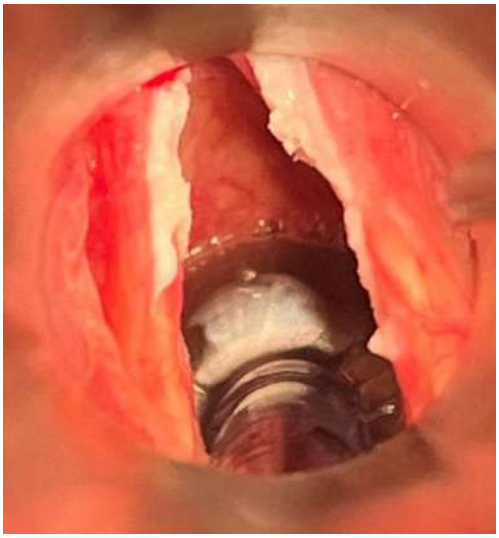
- Clinical picture depends on where the FB got stuck
- The narrowest area is at the level of the cricoid
- If it is stuck at the cricoid
    - Sudden onset of coughing, wheezing and stridor in a previously healthy child
    - Stridor can be severe
    - Needs urgent referral
- If it passes the cricoid and lodge lower down
    - Asthma type picture with repeated chest infections

Respiratory papillomatosis

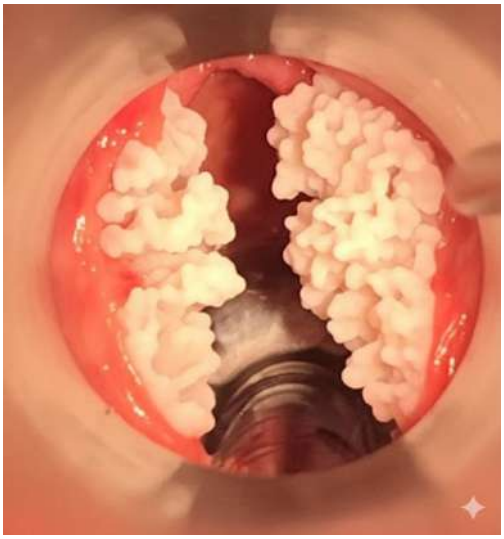
- HPV 6 & 11, 16 & 18
- Exposure
    - In utero for kids
    - Contact for adults
- Presents with dysphonia with or without stridor
- Severe cases present with life threatening stridor
- Far more common in children and more aggressive compared to adults
- Invariably missed diagnosed as “asthma” before the stridor gets severe

Laryngeal cancers

- Refer to Head and Neck cancer chapter
- Squamous cell carcinoma is the most common
- Smoking and alcohol use are the main risk factor
- Any patient with continuous hoarseness for more than 3-4 weeks needs a laryngoscopy
    - If there are any suspicious lesion(s) a biopsy is indicated
- Diseases causing acute hoarseness can lead to airway narrowing / compromise and eventually stridor



Endoscopic picture of leukoplakia of both vocal cords. This patient only had dysplasia on histology.

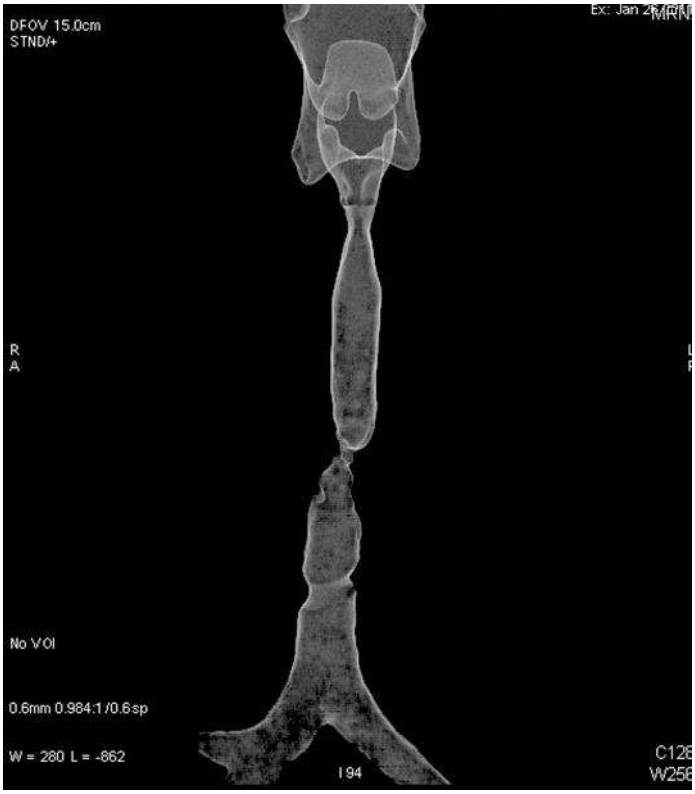


Same picture altered using AI to demonstrate disease progression to laryngeal cancer and airway obstruction.

Tracheal or laryngeal stenosis

- Can be congenital or acquired
- The majority you see will typically be acquired after intubation
- Risk factors associated with intubation includes
    - The time
        - The longer, the worse
    - The quality of intubation
        - Number of attempts
        - “Crash” intubation
        - Pressure of the cuff
        - Movement of the patient
    - Patient factors
        - Inherent tendency to make scar tissue (think keloids)
        - Blood pressure
        - Haemoglobin / Albumin levels
        - Infections

        - Reflux
- The pathogenesis involves necrosis of the mucosa as soon as the cuff pressure exceeds the capillary pressure
- These patients are almost invariably diagnosed as having asthma once discharged. The key is that they do not respond to medications
- Sometimes, patients present with acute stridor on extubating and require immediate airway intervention
- Tracheostomy placement in long term ventilated patients lowers the risk to develop tracheal injuries significantly. In general, the earlier a tracheostomy is done, the better



CAST reformation from a CT scan showing a tracheal stenosis.

This link is of a video of a patient with tracheal stenosis: https://youtu.be/cb0ol-C4B40

Angioneurotic oedema

- Swelling of the face, mouth, tongue, larynx, hands, or feet over a period of minutes to hours
- Can be heredity or acquired
- Triggers can be
    - Allergy to foods
    - Medications
        - ACE inhibitors
    - Specific factors in heredity angioneurotic oedema such as touch, pressure, thermal
- If it leads to airway compromise and stridor, intubation or a tracheostomy may be required

Anaphylaxis

- Causes
    - IgE mediated

        - Antibiotics
            - Penicillin, Cephalosporin, Tetracyclin, Vancomycin, Chlooramphenicol, Bacitracin, Neomycin, Amphotericin B
            - Allergens
                - Bees, Horses
            - Complement
    - Food
        - Milk, Eggs, Nuts, Fish, Legumes, Bananas, Beetroot, Mango, Kiwi fruit
    - Foreign proteins
        - Bees
        - Serum, ACTH, Insulin, PTH
    - Drugs
        - Allergen extracts, Muscle relaxants, Steroids, Vaccines, Streptokinase
    - Immune complex / Complement mediated
        - Blood, Ig, Plasma
        - Methotrexate
    - Arachidonic acid pathway
        - Aspirin
        - NSAIDS
        - Tartrazine
        - Benzoate
    - Direct histamine release
        - Opiates
        - Curare / Alkaloids
        - Dextrose
        - Contras
        - Mannitol
- Risk factors
    - Pre-existing asthma
    - Current asthma attack
    - Food allergies
        - Especially peanuts, tree nuts, and shellfish
    - Reaction to trace amount of foods
    - Use of non-selective β-blockers
- Initially missed diagnosed and treated as asthma
- Early administration of adrenaline is of utmost importance
- Please consult the resuscitation council of South Africa's website at: https://resus.co.za/subpages/RCSA_Information/Resources/Algorithms.html

EMERGENCY MANAGEMENT OF ADULT & CHILD ANAPHYLAXIS

1 RECOGNIZE THE SUDDEN ONSET OF EITHER:

EXPOSURE TO KNOWN OR UNKNOWN ALLERGEN

- **SKIN/MUCOSAL INVOLVEMENT** (rash, swelling) **AND ANY OF:**
- **RESPIRATORY COMPROMISE** (dyspnoea, wheeze), **OR**
- **CARDIOVASCULAR DYSFUNCTION, OR**
- **SEVERE GASTROINTESTINAL SYMPTOMS** (abdominal pain, repetitive vomiting)

AFTER EXPOSURE TO KNOWN ALLERGEN

- **RESPIRATORY DIFFICULTY** (stridor, voice change, wheeze, hypoxaemia, distress)

AND/OR:

- **CARDIOVASCULAR DYSFUNCTION** (shock, hypotension, syncope, collapse)

(No need for skin or mucous membrane involvement)

2 IMMEDIATE TREATMENT:

⊘ REMOVE EXPOSURE
① CALL FOR HELP

ADRENALINE

1mg/ml (1:1000) - 0.01mg/kg IM (Max 0,5ml IM) anterolat aspect of thigh
Repeat every 5-15 minutes if no improvement or use an **auto-injector**
<6yrs - 0,15ml IM; 6-12 yrs - 0,3ml IM; >12 yrs - 0,5ml IM

3 ASSESS VITAL SIGNS: OXYGEN - MONITORS - IV ACCESS

High flow oxygen, maintain patent airway (Intubate/Cricothyrotomy if necessary)
High flow IV line, BP, Sats, ECG monitoring
Lie patient supine with legs elevated if hypotensive

4 ADJUNCTIVE TREATMENT IF NECESSARY

H1 ANTIHISTAMINE
Promethazine
2-6 yrs - 6,25mg IM or slow IV
6-12 yrs - 12,5mg IM or slow IV
>12 yrs - 25mg IM or slow IV
(Avoid if <2yrs old and low BP)

CRYSTALLOID
(e.g. Ringers/Balsol)
Rapid infusion of 20ml/kg (max 1-2 litres)
Repeat IV infusion as necessary
Adrenaline infusion (0,1 - 1 ug/kg/min)
ONLY if unresponsive to IM adrenaline & fluids

NEBULISED BRONCHODILATORS
Every 15-20 mins if severe bronchospasm
Salbutamol 5mg
WITH
Ipratropium 0,5mg

H2 RECEPTOR ANTAGONIST
Cimetidine
IM or Slow IV
5mg/kg (Max - 300mg)
Diluted in 20ml over 2 min

CORTICOSTEROIDS
Hydrocortisone
IM or Slow IV
<1 yr - 25mg; 1-6 yrs - 50mg;
6-12 yrs - 100mg; >12 yrs - 200mg

GLUCAGON
20ug/kg (Max 1-2mg)
IM or slow IV every 5 mins if unresponsive to adrenaline (Look out for vomiting and hyperglycaemia)

RISK REDUCTION STRATEGIES

- Only discharge patient if clinically stable 4-6 hours after resuscitation (may need longer if at risk of biphasic reaction)
- Provide a written anaphylaxis emergency action plan, including how to administer IM adrenaline
- Refer to specialist for investigation and management
- Provide patient eduction (www.allergyfoundation.co.za) and medic-alert bracelet

FAQ's:

When is it appropriate to initiate treatment for Anaphylaxis?
Treat anaphylaxis at diagnosis with IMI adrenaline even if severe respiratory or cardiovascular symptoms are not (yet) present.
Why are Antihistamines considered adjunctive treatment?
H1-antihistamines may relieve itching and urticaria but do not prevent or relieve life-threatening symptoms of anaphylaxis. Antihistamines should not be used alone, or instead of adrenaline, for anaphylaxis.

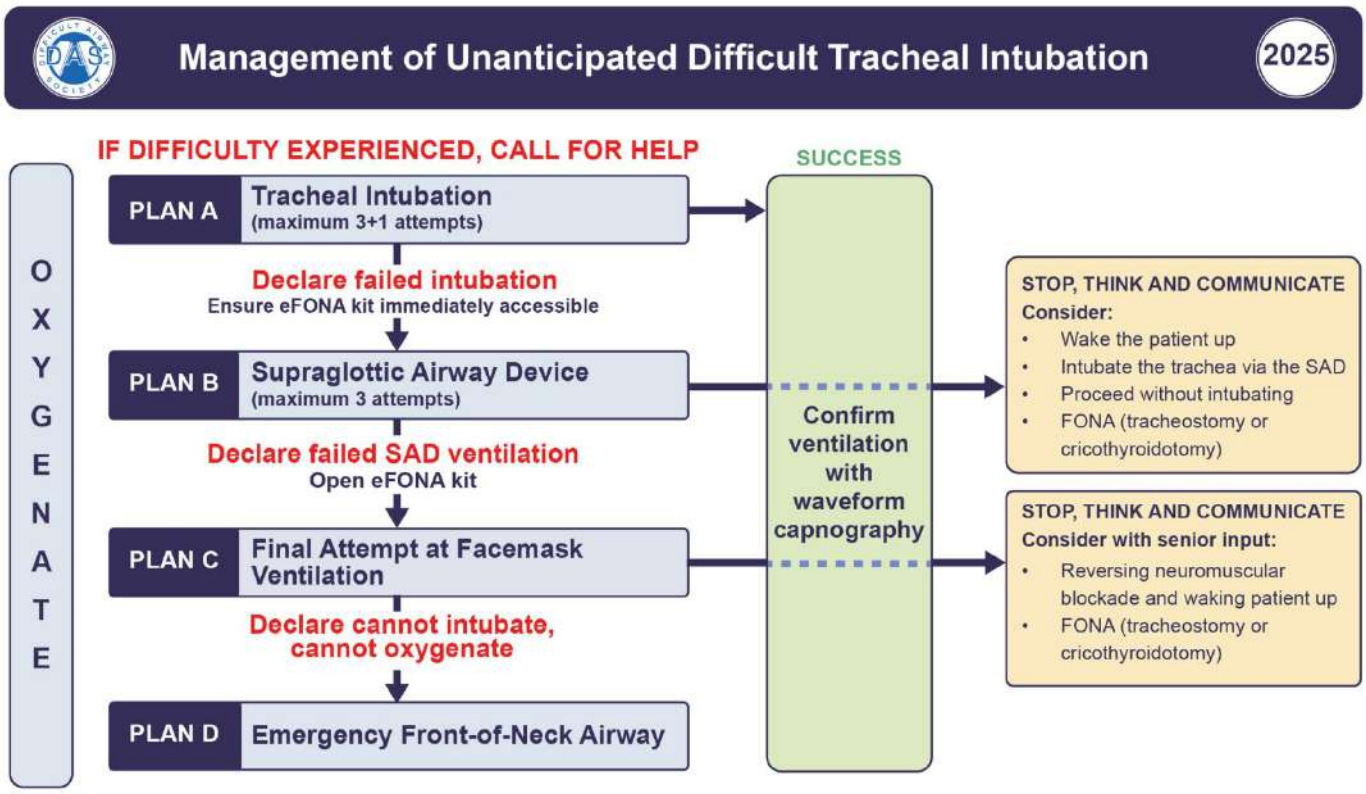
Version May 2024

Ref: Cardona et al. World allergy organization anaphylaxis guidance. WAO;13(10):2020.

Difficult Airway Society

The following article also provides an excellent overview of the approach to a difficult airway more in the context of theatre.

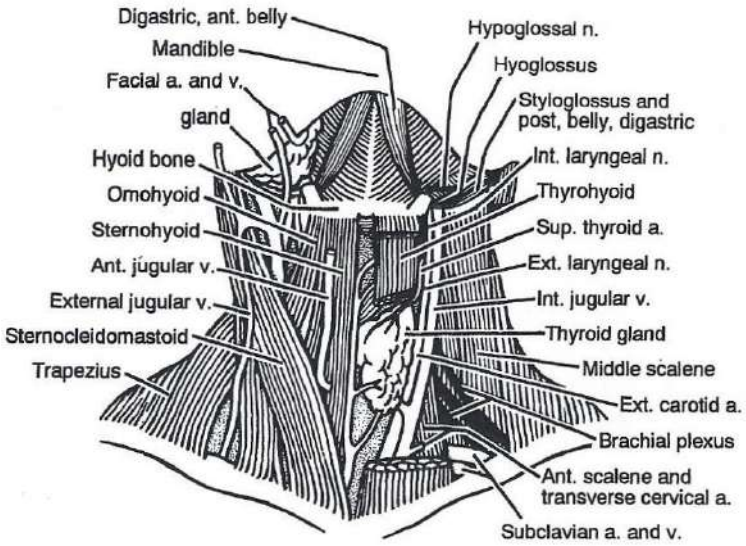
Difficult Airway Society 2025 guidelines for management of unanticipated difficult tracheal intubation in adults.
https://www.sciencedirect.com/science/article/pii/S0007091225006932



21) An approach to a neck mass

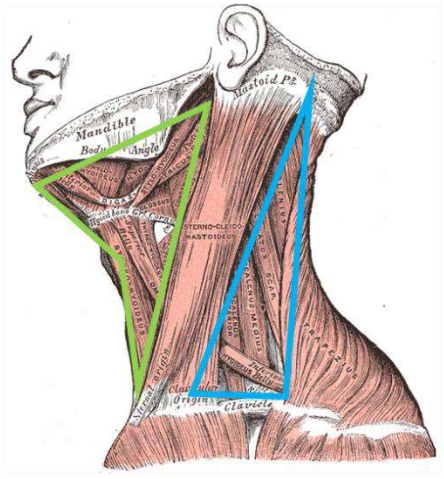
Please also see the section “Head and Neck examination”.

The neck is the “highway” between the head and rest of the body. The anatomy is complex and condensed into a limited space. Basic anatomy is reviewed in the pictures below.

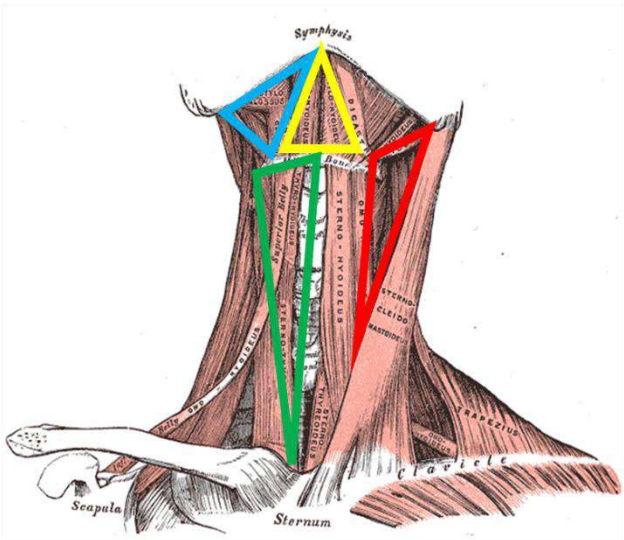


Gland = Submandibular salivary gland

The neck can be divided into levels, zones, or triangles. Levels are used in head and neck cancer surgery, zones in trauma surgery, and triangles as a reference to a certain area. The picture below shows the triangles.

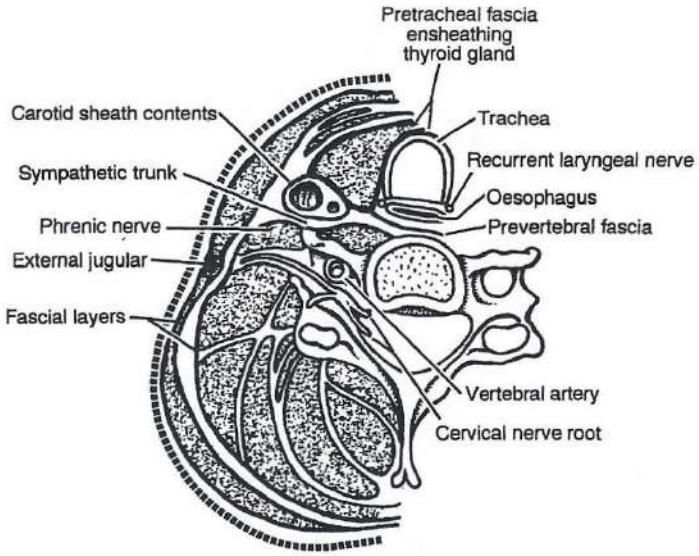


Green = Anterior triangle; Blue = Posterior triangle



Blue = Submandibular triangle; Yellow = Submental triangle; Green = Muscular triangle; Red = Carotid triangle

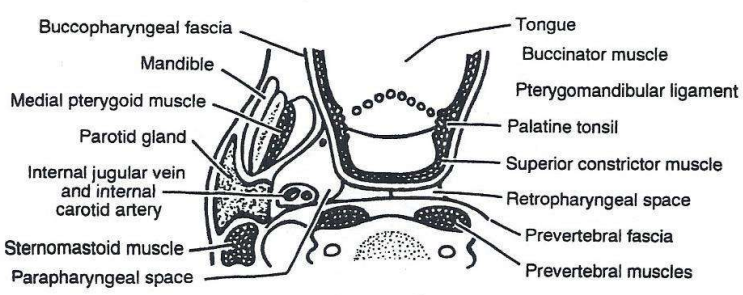
Furthermore, the neck is divided into superficial and deep layers according to the fascial layers as shown in the picture below.



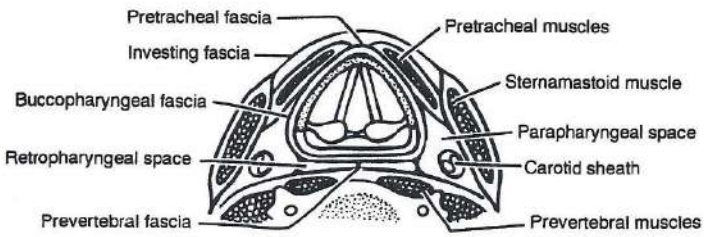
Schematic cross-section of neck

Lastly, the neck contains potential spaces (typically affected in infective processes in the neck) and spaces with structures in it. We will not expect of you to know this in detail, but the most important spaces are:

- Perimandibular space
- Mental space
- Buccal space
- Canine (infra-orbital) space
- Masticator space
- Submandibular space
- Sublingual space
- Submental space
- Submasseteric space
- Parapharyngeal (lateral pharyngeal) space
- Retropharyngeal space
- Prevertebral space
- Danger space
- Peritonsillar space
- Pretracheal space



Fascial compartments of neck at level of C.2



Fascial compartments of neck at level of C.5

Because of your exposure to head and neck cancer surgery with us, neck levels are discussed in reasonable detail under head and neck examination. However, we do not expect of you to have any in depth knowledge of this.

Neck masses / Nodes

As with any diagnostic dilemma, a thorough history and examination of the head and neck will allow the correct diagnosis to be made most of the time. It should be borne in mind, that simply resorting to the attitude that the easiest way to find out what the lump is, is to take it out, can adversely affect the patient's prognosis if the mass happens to be a metastatic lymph node or a vascular tumour! Incidentally, the correct management may often include needle aspiration and cytological examination. **Rarely**, a lymph node excision is required in the case of lymphoma (note that fine needle aspiration flow cytometry often produces a result). Partial removal of a primary neoplasm will also adversely affect the prognosis.

The first step would be to differentiate normal palpable structures in the neck from pathological processes.

Normal palpable structures

Thyroid gland
The thyroid gland has a classic position around the trachea, below the thyroid cartilage. The thyroid is normally attached to the pretracheal fascia and when the trachea and larynx are lifted up by muscle action on the hyoid bone during swallowing, the thyroid gland also move up. This characteristic can make thyroid disease easier to differentiate from other disease. Pre- and para-tracheal lymph nodes may also be attached to the pretracheal fascia and will therefore also move on swallowing. Thyroid diseases resort under General Surgery at Tygerberg Hospital.

Salivary glands
Both parotid and submandibular glands are palpable in almost all patients. Both have classic positions namely at the angle of the mandible and pre-auricular for the parotid and in the submandibular triangle for the submandibular gland respectively. Lymph nodes may also be found in these anatomical areas and therefore may cause confusion.

The parotid gland is much more extensive than commonly realized. The part of the gland that is often forgotten about is below the angle of the jaw, and this can be confused with swellings in the upper cervical region. These include the jugulo-digastric lymph nodes. Parotid enlargement and disease are discussed separately but remember that pathology of the deep lobe will present with a bulge in the oropharynx that will push the tonsil and uvula to the opposite side.

The submandibular gland again has a classic position, below the middle of the jaw. Swellings in this region should always be **bimanually palpated**, with one hand in the submandibular region and a finger of the other hand in the floor of the mouth. This allows the mass to be balloted between the two fingers and allows stones within the duct to be identified. Submandibular salivary gland swellings have to differentiated from

submandibular lymph node swellings. These are relatively rare, except when there is an oral cavity tumour with secondary spread or dental caries.

Bone and cartilage
The inexperienced clinician often mistakes the normal bony or cartilaginous structures in the neck for pathological lumps. The most common structure to be confused, especially in thin necks, is the transverse process of the axis which is deep below the angle of the jaw. The transverse processes are bilateral but one may be more prominent than the other.

Other normal structures such as the hyoid bone, the thyroid and cricoid cartilages should not be difficult to identify. An accessory cervical rib can sometimes be palpable.

Blood vessels
Normally the carotid artery wall is not palpable, although pulsations within it are. Arteriosclerotic thickening of the wall often makes the artery palpable and the pulsations within it less so. A bruit can often be heard by auscultation over an arteriosclerotic narrowed carotid artery. Carotid body tumours are extremely rare and do usually enter into the differential diagnosis of a lump in the neck. They classically present in the region of the carotid bifurcation.

Pathological diseases

Knowing the site / area of the mass already tells the clinician the most likely organ / structure / site involved and thereafter the pathology. The mass can be situated in any tissue from the skin superficially to include all the deeper structures. The table below structures the differential diagnoses according to age groups and position in the neck in relation to the most likely aetiology. It does not cover skin associated masses such as dermoid, epidermoid, and sebaceous cysts.

Differential diagnosis of neck masses

Age groups	**0-15**	**16-40**	**>40**
Order of likelihood	Inflammatory Congenital Malignant Benign	Inflammatory Congenital Benign Malignant	Malignant Benign Inflammatory Congenital
Differential diagnosis according to triangles			
Aetiology	**Midline**	**Anterior triangle**	**Posterior triangle**
Congenital	Thyroglossal cyst Laryngocele Dermoid Lymphangioma	Branchial cyst Salivary glands Lymphangioma	Lymphangioma
Inflammatory	Lymphadenitis (Bacterial / Viral / Granulomatous)	Lymphadenitis Sialadenitis	Lymphadenitis
Tumour	Thyroid Lymphoma	Thyroid Salivary glands Lymphoma Metastatic nodes Paragangliomas Carotid body	Metastatic Lymphoma

Lymph and lymph nodes

Roughly 90% of interstitial fluid is returned to blood and 10% enters the lymphatic pathway. Lymph nodes are part of the lymphatic pathway. They are organized in superficial and deep groups of nodes and eventually drain via the right lymphatic duct and left thoracic duct into the subclavian or internal jugular veins.

Most lymph nodes have and oval or bean shape, with a depression at the hilar region. The hilar region is where the afferent, efferent, vein and artery enter the lymph node. Normal lymph nodes are usually less than 1 cm in size, except for jugulodigastric nodes, which can be up to 1.5 cm in size.

Although lymph nodes perform a filtration effect, their main function are lymphopoiesis and the creation of an immune reaction. The lymph brings antigens to the node and exposes them to antibodies. Furthermore, it activates the B lymphocytes of the humoral immune system, and T-cell and macrophage of cellular immune system respectively. In addition, the phagocytic apparatus of the sinuses filters the lymph, retaining foreign antigens and substances. The passage of the lymph and cells from one chain of lymph nodes to the next is a means by which the immune response is conveyed from the peripheral to the more central lymph nodes. Normally, the lymph node is estimated to recirculate its entire population of lymphocytes within about 12 days. This flow can double or triple when the node is antigenically challenged.

There are several hundred lymph nodes on each side of the neck. About 1/3 of all lymph nodes in the body is in the head and neck area. In adults, lymph nodes are not normally palpable and should be investigated. In children and adolescents, it is normal to palpate some lymph nodes, mainly because they are chronically inflamed due to the repeated upper respiratory and alimentary tract infections in this age group. Another reasons why lymph nodes are easier to palpate in children is the lack of fat deposition in the neck. The lymph nodes, although generously distributed have definite pattern of distribution. The only groups which are inaccessible to palpation being the retropharyngeal node. Their pattern of drainage from the different areas of the head and neck is also relatively constant. In an infective or neoplastic disease not all nodes in the chain draining the affected part are necessarily affected, and one can find “skipped” lymph nodes.

If a lymph node is enlarged it implies pathology in the head and neck region, the only **exception** being the **supraclavicular nodes**. These nodes also drain from the **thorax** and, in addition, on the left there is drainage from the **upper abdomen** because of the relationship to the thoracic duct (Virchow-Trossier node). Lymph node enlargement can only be due to infections, non-infective inflammatory diseases or neoplasms.

Lymphadenopathy

- Infective (known as lymphadenitis)
    - Viral
        - Adenovirus, Rhinovirus, Enteroviruses (coxsackie), Epstein-Barr, HIV
    - Bacterial
        - Suppurative lymphadenopathy
            - Staphylococcus aureus and group A β-haemolytic Streptococcus
        - Mycobacterium
            - Mycobacterium tuberculosis and atypical mycobacterium species
        - Toxoplasmosis / Cat-Scratch disease
    - Fungal
- Non-infections inflammatory diseases
    - Autoimmune diseases (RA, SLE)
    - Sarcoidosis
    - Amyloidosis
    - Storage diseases
    - Kawasaki disease

    - Sinus histiocytosis
    - Drug induced
- Primary and Metastatic tumours
    - Squamous cell carcinomas
    - Any head and neck cancer
    - Lymphomas

Lymph nodes and infections

Lymph node enlargements which are secondary to inflammation are mostly painful. Often several glands are affected and they usually are, or at some stages have been, tender to palpation. The most common sites to be infected are the pharynx including the tonsils, teeth, and nose. Correspondingly, the upper deep cervical (jugulo-digastric) lymph nodes are the ones most commonly affected. As stated earlier, palpable lymph nodes are common in children, who normally have recurrent upper respiratory and oropharyngeal infections. In adolescents, infectious mononucleosis must be considered. Here the lymphadenopathy is usually multiple and bilateral and can involve lymph node groups apart from those of the head and neck. In adults, infective enlargement of a lymph node is uncommon, and **neoplasm** is the more likely possibility.

Primary tuberculosis of the neck nodes is, however, still a possibility following ingestion of the organisms and is not necessarily associated with pulmonary tuberculosis. The nodes are not usually tender. Occasionally, cervical tuberculosis may present as an abscess or as a fistula (especially if it was incised and drained).

Lymph nodes and cancer

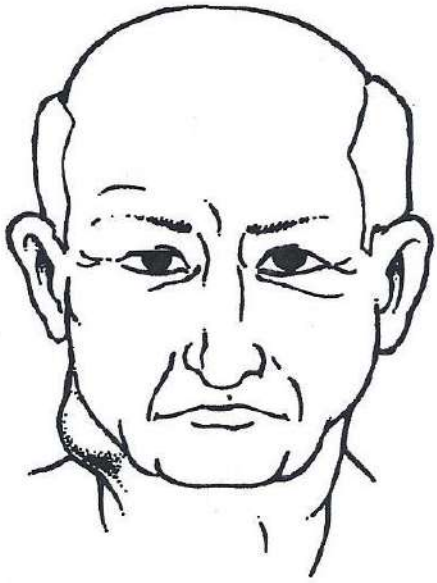
Head and neck cancer typically spread via lymphatic pathways. The reason being that capillaries have tight inter-endothelial cell junctions, there is a basement membrane, and there are surrounding pericytes. If metastatic cells are to enter these vessels, they must actively penetrate these various layers, and this process is known as intravasation. By comparison, the lymphatic capillaries have loose inter-endothelial cell junctions, valve openings, no basement membrane, and no surrounding pericytes. As a result, tumour cell entry into lymphatic vessels is considered a passive process, whereas entry into the blood vessels is considered an active process.

In adults, an enlarged lymph node in the neck must be considered a neoplasm until proven otherwise. In adults under the age of 40, the most likely neoplasm is a lymphoma. In those over 40, it is likely to be a secondary from a primary squamous carcinoma from somewhere in the head and neck. In all patients the first thing to do is to thoroughly examine the head and neck, paying particular attention to other lymphatic tissue in the tonsils, the postnasal space and the base of the tongue. The area which primarily drains to the enlarged node should be examined but not to the exclusion of the other areas. The primacy site for a squamous carcinoma is very often silent, that is without symptoms or signs. This is not surprising as the head and neck have many spaces where a neoplasm has to be fairly big before it causes symptoms. Examples of such spaces are the nasopharynx, the pyriform fossae, the supraglottis, the base of tongue, the retromolar trigone, floor of the mouth (gutter), the tonsil and the oral cavity.

The majority of these sites are not easy to examine, and it should, therefore, be the rule that an otolaryngologist should examine every adult with a neck swelling. Having completed the examination, the otolaryngologist will be faced with one of two situations which are handled in different ways. One is an obvious primary with secondary lymph node involvement and the other no obvious primary with secondary lymph node involvement. We don't expect of you to know what to do, but general principles are important:

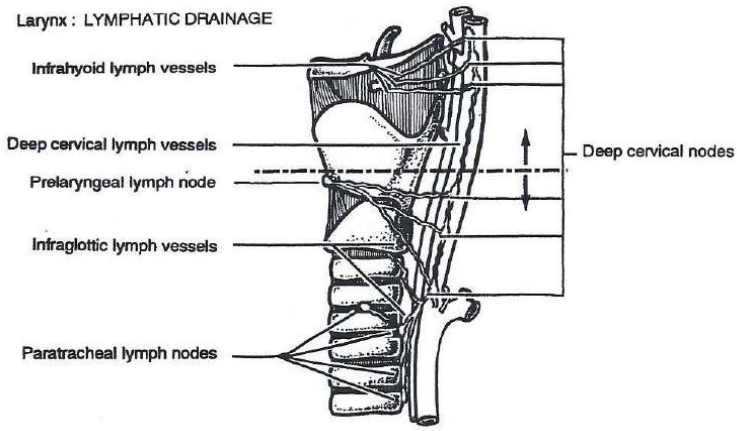
- "Search" for the primary and rather biopsy the primary
    - Remember that lymph nodes, in general, follow an orderly pattern of involvement (see head and neck examination)
- Do not incise or remove a lymph node!

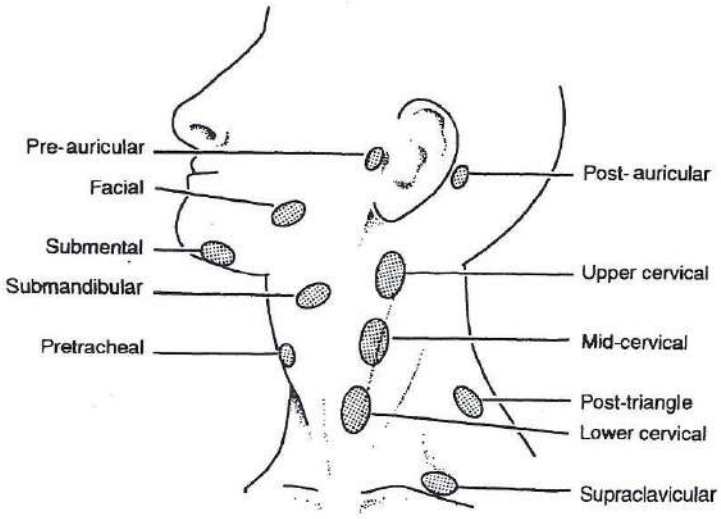
- Rather do a FNAC, with or without a cell block (cell block entails performing a fine-needle aspirate, and sending the aspirate in a specimen container with normal saline for cell block cytology)
- Rarely, you may do a core needle biopsy
- Do general bloods (FBC, U&E, Albumin, HIV)
- REFER



Metastatic mass in the neck. The mass was fixed, and very hard which made the diagnosis of metastatic lymp node most likely. A careful search of the mouth, throat, etc., was negative. A very small primary tumour was found in the scalp.

The normal drainage pathways are shown below





Classic pattern of distribution of neck lymph nodes

Nodes	Areas draining to these nodes
Submental	Lower lip, Floor of Mouth, Lower gum
Facial / Submandibular	Face, Nose, Paranasal sinuses, Oral cavity, Submandibular gland
Pretracheal	Larynx, Thyroid
Pre-auricular	Anterior scalp, Forehead, Parotid
Upper-cervical	Oral cavity, Oropharynx, Nasopharynx, Hypopharynx, Supraglottic larynx
Mid-cervical	Thyroid, Larynx, Hypopharynx, Cervical oesophagus
Lower cervical	Intra-abdominal organs, Breast, Lung, Oesophagus, Thyroid
Post-auricular	Posterior scalp, Posterior ear
Supraclavicular	Nasopharynx, Thyroid, Oesophagus, Lung, Breast

The Hot Neck Node

It is very important to distinguish between what is referred to as a “hot neck node” and a “cold neck node” on clinical grounds. The reason being twofold. Firstly, we don’t want you to incise into “cold” neck nodes, because most of them will be neoplasms and this will increase their regional spread (and result in poorer outcomes). Secondly, you can under the ideal circumstances manage a patient with an obvious (but limited) infective process in the neck. If it is fluctuating, warm to touch and red you can incise it to facilitate the drainage of the puss, break down the loculation, rinse and pack the cavity. The only exception to this rule is secondarily infected congenital neck masses. Rather aspirate them, because once incised and drained they have a higher occurrence rate after definitive surgery. The more severe cases, or if you don’t have the experience or supervision, needs to be referred to a hospital. The table below lists the differences.

	Hot	Cold
Aetiology	Infective process / Lymphadenitis	Either neoplastic or congenital
Signs of infection such as fever, tachycardia, raised inflammatory markers	Yes	No
Signs of an abscess such as warm to touch, fluctuant, erythema of skin	Yes	No
Features of the node	Fluctuant / Soft	Hard / Fixated / Infiltrated
Management	Depending on your skill level and infrastructure	Do not incise and drain!

	• Aspiration • Incision and drainage • Formal exploration in theatre	By far the majority would be metastatic lymph nodes or congenital conditions Fine needle aspiration cytology is allowed Rarely core needle biopsy Remember that most congenital masses in the neck are cystic in nature
Further management	MCS Antibiotics	Refer

Fine needle aspiration cytology (FNAC)

Sometimes it is also referred to as a fine needle aspiration biopsy. This is a technique that you certainly can acquire the skill to do it yourself. FNAC can be done blindly or under ultrasound guidance. In our Head and Neck clinic we employ the blind technique with good results. The reader is referred to an excellent chapter on "BIOPSY OF HEAD & NECK TUMOURS & CERVICAL LYMPH NODES" in the OPEN ACCESS ATLAS OF OTOLARYNGOLOGY, HEAD & NECK OPERATIVE SURGERY at the following webpage Head and neck lymph node and tumour biopsy techniques. It also discusses various other biopsy techniques.

Fine needle aspiration cytology (FNAC) with Cell Block

This is a technique that you certainly can acquire the skill to do it yourself. Following a FNAC, the specimen is placed into a container with sterile normal saline (rather than onto cytology slides) and is sent for "cell block" cytology examination.

22) Head and Neck Cancers

What do you need to know?

- You need to know how pre-malignant mucosal lesions look:
  - Leukoplakia / Erythroplakia
- Correlate this with the macroscopic appearance of carcinomas
  - Ulcerative / Fungating – Exophytic / Infiltrative - Submucosal
- Examine your patients
  - Bi-manual oral and oropharynx examination / Neck
  - Remember that head and neck cancers have a loco-regional spread
- Help to expedite the process if you work in a setting where there is a time delay to refer the patients to a tertiary centre
  - Biopsy of primary / FNA node(s) / Bloods / TB / HIV
- Differentiate "hot" from "cold" neck masses
- Have a differential diagnosis of oral mucosal lesions

Historical context

History of Cancer

- First reported description of cancer
  - Edwin Smith Papyrus 3000 BC of breast cancer
  - Ebers Papyrus 1500 BC describes several types including skin, uterus, stomach, and rectum
  - Associated it with a curse of the gods
- Hippocrates (460-370BC)
  - First to use the word carcinoma
    - Crab and adheres to surroundings with claws
    - Excess of black bile - bloodletting
    - Galen (130-200) used the term oncos (swelling)
  - Wonderful era until ~ 300
    - Fall of Rome 476
    - Church banned autopsies in 1215
    - Nothing happened until the 16th century
- 18th century
  - 1775 Percival Pott noticed that chimney workers develop cancer of the scrotum
  - Ludwig Rehn observed the association between aniline dye and bladder cancer
  - Microscope
    - Virchow stated that cancer is a disease of cells
- 20th century
  - Proven association between chemicals and cancers
  - Expanded to include radiation and viruses (in chickens)
  - 1914 Theodore Boveri postulated chromosome and other genetic abnormalities

History of Cancer Surgery

- Egyptians
  - Various reports on cancer surgery, especially on the breast
- Important surgical dates

- 1809, Ephraim McDowel removed the first ovarian tumour without anaesthesia!!
- 1846 – anaesthesia (ether)
- 1867 – antisepsis
- 1873 – laryngectomy
  - Theodor Billroth
  - Considerable bleeding, coughing and arousal from anaesthetic
  - Patient lived for 7 months
  - He also performed the first esophagectomy (1871) & gastrectomy (1881)
  - 1880 – 50% peri-operative mortality
- 1894, William Halsted introduced the radical mastectomy for breast cancer. He introduced the en bloc resection of all surrounding tissue – even humerus head. This became known as the "cancer operation"
- It wasn't for 74 years before someone questioned this operation!
- Fisher noted that a radical mastectomy was both too much for small tumours, and too little for large tumours
- He also showed that less radical surgery with radiation accomplished the goal with much less morbidity

History of Radiation

- The era of radiation started in 1894
  - First documented success was in 1899, when Thon Stenbeck treated a 49-year-old patient with a basal cell cancer of the nose. She was alive and well 30 years later on
- External beam radiation started in 1922
- In 1928 it was shown that head and neck cancers could be cured by fractionated radiation
- Radiation became very popular between 1920-1950
- By the 1950's it became apparent that no matter how complete the resection or how good the radiation, cure rates had flattened out
  - Only about a third of cancers could be cured

History of Chemotherapy

- The use of drugs to treat cancer began about 70 years ago (1950's). After World War II, Louis Goodman and Alfred Gilman and their colleagues at Yale University noted that people who had been exposed to mustard gas often had bone marrow suppression. They identified the active chemical in the gas, gave it to patients, and saw responses in patients with hematologic cancers. Thus, alkylating agents were developed and became key components of treatments for many forms of cancer.
- Not all useful drugs emerged as a product of accurate hypotheses. Sidney Farber mistakenly thought that leukemic cells looked megaloblastic, and so he gave folate to some children with acute leukaemia. Their disease accelerated, and the children died. This led to the hypothesis that an antifolate agent might be effective, and aminopterin and amethopterin (methotrexate) were born

Treatment of HNC before 1990's

- Surgery first options and if feasible proceed to postoperative RT
- First available drug was methotrexate
- Cisplatin was introduced in 1968 by Dr Rosenberg (it remains one of the mainstay chemotherapeutic agents till today)
- In 1984, Dr Muyi Al-Sarraf used the first combination chemotherapeutic agents' neo-adjuvant
  - Cisplatin and 5-FU
  - Clinically complete response rate in 40%
  - Led to the first trails to test "nonsurgical" treatment programs

- >50 years ago – radical surgery with minimal (no) regards to outcome

Current treatment
See below

Future Treatment Options
- Targets include
  - Modifying tumour suppressor genes
    - P53
    - CDKN2A
  - Oncogenes
    - RAS, RAF/MEK/ERK, PIK/AKT/MTOR, PTEN
  - EGFR, VEGF, PDGF, FGF, TGF-a, TGF-β, IL-8, PD-1, PD-L1
  - Gene silencing
  - Epigenetic targets
- Mechanisms of action
  - Anti-proliferative, pro-apoptotic, anti-angiogenic, CRT-sensitizing properties

Molecular changes

What changes are needed on a molecular level to develop a cancer?
- You need the following
  - Resistance to growth inhibition
  - Evasion of apoptosis
  - Angiogenesis
  - Evasion of the immune system
  - Ability to invade and metastasis
- Final common pathway is genetic alterations
  - Factors playing a role
    - Environmental
    - Tumour suppressors genes
    - Proto-oncogenes
    - Chromosomal abnormalities
    - Protein based alterations

For benign neoplasms you need
- Genetic mutations
- Evasion of apoptosis
- Uncontrolled replication
- Evasion of the immune system

And for malignant neoplasms you need
- All of the benign changes
- Angiogenesis
- Loss of inter-cellular adhesions
  - Metastases

A common feature of all malignant cancers includes genomic instability and a pro-inflammatory state.

Head and Neck Cancers in general

How does a cancer look?
Remember that cells can't "jump" from a normal cell to a malignant cell (the only exception might be oropharyngeal induced HPV cancers). There are steps in between and we refer to these as pre-malignant lesions. It is of vital importance that you need to know how they present, to refer these patients to your ENT specialist for further management. Pre-malignant lesions' macroscopical appearance are:

- Leukoplakia – white lesion that you can't wipe clean
- Erythroplakia – red lesion that you can't wipe clean
- Other
    - Submucosal fibrosis
    - Lichen planus (controversy if it is truly a pre-malignant lesion)

Microscopical they can report:

- Dysplasia (most important)
- Atypia
- Hyperplasia
- (Hyperkeratosis / Parakeratosis / Acanthosis)

Unfortunately, head and neck cancers does not have an orderly progression from dysplasia to carcinoma in situ, to infiltrative carcinoma. If you compare it to cervical HPV induced cancer, which follow a "one-way" street (CIN I→CIN II→CIN III→CIS→CA), head and neck dysplastic can move in both directions. This makes managing patients with severe dysplasia challenging, especially the group with "field cancerisation" where large areas of the mucosa are affected.

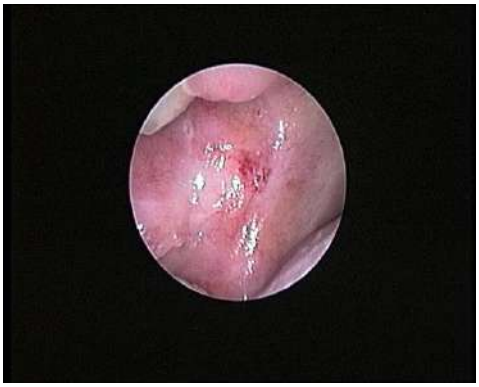
On the other hand, the macroscopical appearance of malignant tumours are

- Ulcerative
- Fungating - exophytic
- Infiltrative – submucosal

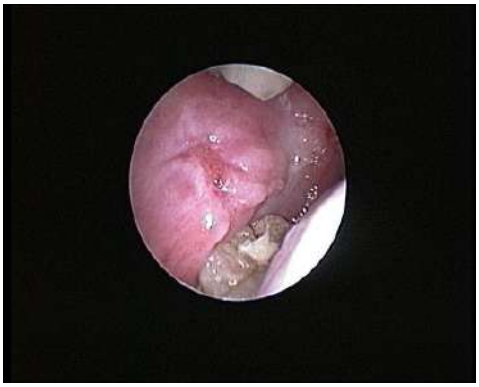
Microscopical they are classified according to their origin cell. Remember, that by far the majority of head and neck cancer you are going to see will be squamous cell carcinomas. The table below shows benign and malignant tumours in relation to their origin.

	Benign	**Malignant**
Epithelial	Papilloma	Squamous Ca
Salivary gland	Adenoma	Adenocarcinoma Mucoepidermoid Adenoid cystic
Bone	Osteoma	Osteosarcoma
Cartilages	Chondroma	Chondrosarcoma
Vascular	Haemangioma Angioma	Angiosarcoma Kaposi
Muscle	Leiomyoma Rhabdomyoma	Leiomyosarcoma Rhabdomyosarcoma
Fibrous tissue	Fibroma	Fibrosarcoma
Fat	Lipoma	Liposarcoma
Haematopoietic	Paraganglioma	Lymphoma

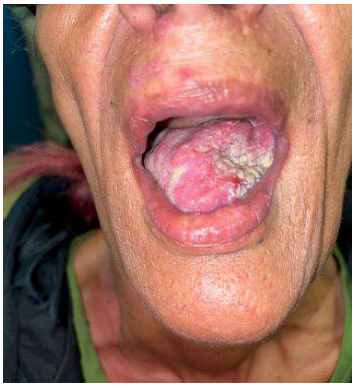
Clinical Pictures



Area of erythroplakia in buccal mucosa.



Area of leukoplakia and erythroplakia in buccal mucosa.



Infiltrative (submucosal), ulcerative lesion in left anterior 2/3 of oral tongue.

Leukoplakia, erythroplakia, possibly ulcerative lesion of upper dento-alveolar ridge.

Cancer statistics are reported by Stats SA. According to them, South Africa had the following number of cancer cases as shown in the table below.

All cancer in South Africa

Number of cases	2008	2019
Females	28 748	43 811
Males	26 538	41 491
Total	55 286	85 302
Total deaths	33 720	43 613

Extracting Head and Neck cancer data from this report shows that it only accounted for 4.4% and 1.9% of all cancers for males and females respectively. In men, the most common Head and Neck cancer was the larynx (26%), followed by the mouth (21.6%), nasopharynx and oropharynx (21.4%), tongue (19.8%), salivary glands (7.4%), lips (2.3%), and lastly gums (1.5%). In females the mouth (27%) was the most common site, followed by the tongue (20%), nasopharynx and oropharynx (19.5%), and salivary glands (14.2%), larynx (12.8%), gums (4.2%), and lips (2.3%).

Head and Neck cancers – background

It is reported to be the fifth most common cancer in the world. A recent article reported 1.5 million new cases and 500 000 death per year (Sung H, Ferlay J, Siegel RL, Laversanne M, Soerjomataram I, Jemal A, et al. Global Cancer Statistics 2020: GLOBOCAN Estimates of Incidence and Mortality Worldwide for 36 Cancers in 185 Countries. CA Cancer J Clin. 2021 May;71(3):209–49.)

By far the majority is squamous cell carcinomas and the combined 5-year survival is unchanged at 50% over last 3 decades. Squamous cell carcinomas will be discussed in more detail. Remember that other benign and malignant tumours can occur in the head and neck such as:

- Salivary gland tumours (see separate chapter)
- Thyroid gland tumours
- Benign and malignant mesodermal tumours – all the sarcomas
- Haematological / Vascular tumours – Glomus, Lymphangioma, Haemangioma, Lymphomas
- Congenital tumours – Teratomas

Head and neck squamous cell carcinomas (HNSCC)

Epidemiology and Aetiology

- In 2020 more than 1.5 million people worldwide were diagnosed with HNSCC
    - With 500 000 deaths
- There are five major mucosal sites namely
    - Nasopharynx, Oropharynx, Hypopharynx, Oral cavity, Larynx
- Remember that skin cancers also spread to the lymphatics of the neck
    - In countries such as Australia, skin SCCs draining to the parotid lymph nodes are the most common reasons for parotid cancers (43% of all parotid tumours)
- The majority of HNSCC are related to tobacco and alcohol use
- However, in North America, Europe, and Australia, there is an epidemic of oropharyngeal SCC caused by infection with Human Papilloma Virus (HPV)
    - This has not been the case in South Africa, for reasons unknown to us currently (this might change)
    - HPV is discussed in more detail below
- Nasopharyngeal SCC is related to Epstein-Barr virus

- The incidence of most HNSCC has decrease globally, and this is strongly linked to the decrease in the rate of smoking
    - The rate of smoking has decreased from 43% in 1965 to 21% in 2004 in the USA
- Alcohol consumption is also decreasing. Adults, on average, consumed 5 litres of alcohol in 2013 compared to 3.9 litres in 2023
- However, the incidence for tongue, tonsils, salivary glands and thyroid cancers are on the increase
    - Only tonsils (oropharyngeal site) associated with HPV can be explained

Risk factors

- Smoking and alcohol are the most important risk factors
    - There is a synergistic interaction between alcohol and smoking, which is multiplicative for the oral cavity, additive for the larynx, and in between for the oesophagus
    - There is also a dose and time dependant relationship for alcohol and tobacco
        - This means the longer and more you smoke and / or drink, the higher your chance is
    - It has been calculated that the risk for smokers and drinkers, to ever develop a HNSCC, is 4-177 higher compared to non-smokers and non-drinkers
- Other risk factors implicated are
    - Previous head and neck cancer
    - Previous radiation
    - Genetic / Immunologic
        - Presents in younger patients and frequently in females
    - HPV
    - Other viruses
        - EBV
            - Nasopharyngeal cancers
            - EBV is classically associated with nasopharyngeal cancers, however more reports indicate that it might play a role in other head and neck cancers. The opposite is also true, in that HPV might play a role in nasopharyngeal cancers
        - HIV
            - Patients present with more advanced HNSCC
            - Associated with skin SCC
            - Kaposi sarcoma
            - Lymphoma
    - Pollutants / Occupational agents
        - Nickel / Mustard
        - Asbestos
        - Sulphuric and hydrochloric acid
        - Previous radiation
    - Betel nut
        - India / Asian countries
        - Many variations
        - Mainly chewed
    - Laryngopharyngeal reflux
    - Possible risk factors
        - Nutritional factors
            - High fruit and vegetables intake associated with a decreased risk
        - Poor oral hygiene
        - Marijuana

- One study reported an overall increased risk of 2.6 compared to non-users, however in most studies the association is inconclusive

Smoking

- Types:
  - Smoking
  - Tobacco chewing
  - Reverse smoking – especially in India – even higher risk to develop HNSCC
  - Pipe / Cigar – increased risk to develop oral cavity SCC
  - Smokeless tobacco – increased risk to develop oral cavity SCC
  - Environmental / second hand / passive smoking also increases the risk to develop HNSCC
- Only became very popular after world war I & II.
- Single most preventable cause of death and cancer in the world.
- 7000 chemical compounds in a burning cigarette
  - More than 20 carcinogens
- Smoking and cancer
  - Causes mutations of P53 and P16 – loss of tumour suppressor genes
  - Chances of developing cancer is 23 times higher in males and 13 times higher in females who smoke
  - Chances of developing lung cancer before the age of 85 is 22% for males, and 12% for females (compared to 1%)
  - Smokers are three times more likely to die compared to non-smokers, and lose on average at least a decade of life
  - Each cigarette reduces your life by 11 minutes
  - At least half of smokers die as a direct result of smoking
  - In the USA alone, it accounts for roughly 500 000 premature death per year
  - Put into contexts this equates to 3 jumbo jet crashes every day without survivors
  - Economic impact is roughly $300 billion/year
  - For every death caused by smoking, 30 smokers will suffer from a chronic disease caused by smoking
- Incidence
  - 19.3% of adults in US smoke (2010)
  - Down from 20.9% (2005)
  - Down from 42% in the 1950's!
  - Smoking peaked in the 1960's in men and 1980's in women
  - Proportion of daily smokers who smoke one to nine per day increased from 16.4% to 21.8%,
  - Whereas the proportion who smoked more than thirty per day decreased from 12.7% to 8.3%
  - In South Africa
    - A study reporting on the age of onset of tobacco smoking in South Africa found that it peaks between the ages 15 to 22 years but varies by province, sex, location, race, and socio-economic status. It also reported that the lifetime prevalence of smoking was 20,5%. (Fagbamigbe AF, Desai R, Sewpaul R, Kandala NB, Sekgala D, Reddy P. Age at the onset of tobacco smoking in South Africa: a discrete-time survival analysis of the prognostic factors. Arch Public Health. 2020 Dec;78(1):128.)
    - A recent household survey of persons aged 15 years or older, the Global Adult Tobacco Survey (GATS) undertaken in 2021 by the South African Medical Research Council (SAMRC) under the auspices of the National Department of Health (NDoH), reported a higher tobacco use of 29,4%. Tobacco use among males was much higher than that

reported among females, at 41,7% and 17,9% respectively. (03-08-002023.pdf [Internet]. [cited2023Dec10]. Available from:https://www.statssa.gov.za/publications/03-08-00/03-08-002023.pdf)
        - An MMED project at Tygerberg Hospital showed that 98% of Head and Neck cancer ever smoked (www.scholar.sun.ac.za - 2024)
- Advertising (USA)
    - Cigarette companies are spending $1million / hour on advertising (2022)
    - Targeting the young
    - 3200 younger than 18 will start every day
- 70% of adults wants to quit
- Upon smoking cessation, the risk to develop a HNSCC is reduced by
    - 30% in the first 1-9 years
    - 50% in those who have stopped > 9 years

Alcohol

- The precise mechanism is unclear as alcohol itself is not a carcinogen
- It may act as a solvent increasing cellular permeability of tobacco carcinogens through the mucosa
- In terms of laryngeal SCC
    - Smokers develop more glottic SCC
    - Heavy drinkers develop more supra-glottis SCC
- It has a synergistic effect with smoking
- France
    - 34 higher chances of developing laryngeal cancer in patients who smoke and consume more than 1.5 L of wine per day
- Alcohol causes 79 000 deaths / year in USA (for any reasons)
- In South Africa
    - The South Africa Demographic and Health Survey of 2016 reported that 61% of men and 26% of women ever consumed alcohol, and 26% of men and 5% of women exhibited risky drinking patterns (SADHS 2016). (03-08-002023.pdf [Internet]. [cited 2023 Dec 10]. Available from: https://www.statssa.gov.za/publications/03-08-00/03-08-002023.pdf)
    - Naidoo K. (2019) reported that the incidence of smoking was 86.3% and of alcohol use was 74.25% in a cohort of 854 patients with Head and Neck cancers, at Tygerberg Hospital. (Naidoo K. Descriptive epidemiological study in head and neck cancers at a single institution in Southern Africa [Internet]. Stellenbosch; Available from: http://www.sholar.sun.ac.za)
- It is thus clear from these data that our population has high exposure to the most significant risk factors causing Head and Neck cancers.

HPV

- Background
    - More than 200 subtypes
    - Most common STD in the USA
    - Two groups
        - Low risk (HPV 6 & 11)
            - Causes genital warts and respiratory papillomatosis
        - High risk (HPV 16 & 18)
        - Causes cancer of the cervix, penis, anus, and oropharynx
    - CDC
        - 90% will be infected with low-risk HPV subtypes, and 80% with high-risk HPV subtypes

        - Most high-risk HPV infections occur without symptoms, and will go away in 1-2 years
- Natural progression
    - HPV is transmitted mainly via sexual contact
    - The risk for oropharyngeal cancer increases with the number of oral sexual partners
    - It is estimated that at any given time, approximately 7% of the population has oral cavity / oropharyngeal HPV infection
    - The life-time oral exposure rate is unknown, but it is estimated that between 65-100% of sexually active adults have been exposed
    - The precise reason why some individuals develop oropharyngeal HNSCC is unclear, but a delayed clearance of the HPV by the immune system might be the reason
    - In 99% of individuals, the HPV is cleared by the immune system after 12-18 months, and they don't develop HNSCC
- HPV related head and neck cancers
    - Subset of tumours caused by HPV 16 and 18
    - At this stage only associated with oropharyngeal SCC – tonsils and base of tongue
        - It may be that other head and neck sites are also associated with HPV but currently there is no data to support this
        - Remember also cervix carcinoma
- Incidence
    - Massive increase oropharyngeal cancer in many countries since the turn of the century
        - From 1988 to 2004, there was a 225% population increase in HPV-positive oropharyngeal SCC in the USA, and a concomitant 50% decrease in HPV-negative oropharyngeal SCC
        - The overall HPV-positive oropharyngeal SCC increased from 16% to 70% over the same period (USA, UK, Australia, Scandinavia)
    - In South Africa
        - Importantly, studies have not been able to demonstrate the same rise in HPV related Head and Neck cancer in Sub-Saharan Africa as compared to the countries listed above.
        - A systematic review of 31 studies and 3,850 patients specific to Sub-Saharan Africa, the overall p16 positivity was 13.6% (41 of 1037 samples tested), with the highest proportion among oropharyngeal cancers (20.3%). The overall HPV polymerase chain reaction positivity was 15.3% (542 of 3,548 samples tested), with the highest proportion among nasopharyngeal cancers (16.5%). (Okerosi S, Mokoh LW, Rubagumya F, Niyibizi BA, Nkya A, Van Loon K, et al. Human Papillomavirus–Associated Head and Neck Malignancies in Sub-Saharan Africa: A Systematic Review. JCO Glob Oncol. 2023 Jan;(9): e2200259.)
            - P16 is a surrogate marker for HPV in the oropharynx
        - In a study from Tygerberg Hospital South Africa, testing P16 and HPV status, the incidence was only 5%. The exact reasons for this are unclear and warrant further investigation. (Dapaah G, Hille J, Faquin WC, Whittaker J, Dittrich CM, Ebrahim AK, et al. The Prevalence of Human Papillomavirus–Positive Oropharyngeal Squamous Cell Carcinoma at One of the Largest Tertiary Care Centers in Sub-Saharan Africa. Arch Pathol Lab Med. 2022 Aug 1;146(8):1018–23.)
        - Furthermore, the burden of cancer is increasing in LMICs, and especially in Sub-Saharan Africa, where the incidence is approximately twice as high as compared to high income countries. (McGinnis GJ, Ning MS, Bvochora-Nsingo M, Chiyapo S, Balang D, Ralefala T, et al. Management of Head and Neck Cancers With or Without Comorbid HIV Infection in Botswana. The Laryngoscope [Internet]. 2021 May [cited 2023 Dec 10];131(5). Available from: https://onlinelibrary.wiley.com/doi/10.1002/lary.29206)

        - According to the World Health Organization, more than 70% of all cancer deaths occur in LMICs, where resources required for prevention, diagnosis, and treatment of cancer are limited or non-existent. (Cancer in developing countries: facing the challenge [Internet]. [cited2023Dec10]. Available from: https://www.who.int/director-general/speeches/detail/cancer-in-developing-countries-facing-the-challenge)
        - Sub-Saharan Africa has the greatest burden of HPV-driven malignancies, especially cervical cancer, and the highest global prevalence of HIV. This region is also home to two thirds of people living with HIV (PLWH) world-wide. (South Africa | UNAIDS [Internet]. [cited2023Dec10]. Available from: https://www.unaids.org/en/regionscountries/countries/southafrica)
        - Dhokotera T. et al. (2019) reported on the association between HIV and cancer in the South African public health sector, and about half of females with cancer, with a known status, were HIV-positive. (Dhokotera T, Bohlius J, Spoerri A, Egger M, Ncayiyana J, Olago V, et al. The burden of cancers associated with HIV in the South African public health sector, 2004–2014: a record linkage study. Infect Agent Cancer. 2019 Dec;14(1):12.)
        - HIV predisposes patients to increased rates of oral HPV infection and the risk of developing head and neck squamous cell carcinomas. (Okerosi S, Mokoh LW, Rubagumya F, Niyibizi BA, Nkya A, Van Loon K, et al. Human Papillomavirus–Associated Head and Neck Malignancies in Sub-Saharan Africa: A Systematic Review. JCO Glob Oncol. 2023 Jan;(9): e2200259.)
        - Oropharyngeal cancer incidence is 2-6 times higher in patients living with HIV. (Beachler DC, D'Souza G. Oral human papillomavirus infection and head and neck cancers in HIV-infected individuals. Curr Opin Oncol. 2013 Sep;25(5):503–10.)
        - Our local incidence remains below 15%
- Clinical presentation
    - Typically
        - Middle-aged white men from a higher socioeconomic status, with a history of multiple sexual partners (especially oral sex partners)
        - Presents with advance cystic metastatic lymphadenopathy with small primary tumours
    - However, newer report shows a more advanced age (60 years) and no sex predilections
    - Can also present like carcinomas with unknown primaries
    - Not related to smoking / alcohol but it can coincide
        - Remember that you might see a patient, where the HPV tests positive, but the cancer is due to smoking and alcohol (the HPV is an innocent bystander).
    - There are no pre-malignant lesions known at this stage
    - No screening method currently as opposed to PAP smears for cervical cancers
- Mechanism of inducing cancer
    - Produces E6 and E7
    - They bind the tumour suppressor genes *p53* and *pRB*
    - This leads to uncontrolled cell proliferation
- Treatment
    - They respond more favourably to chemoradiation, and have a better prognosis than similar stage smoking / OH induced HNSCC
    - Vaccine
        - Definitely pre-exposure for girls and boys
        - Efficacy post-exposure is unknown, however we do advise it

Approach to HNC

Managing your patients
By this stage, you should have a firm understanding of what is takes to make a cancer cell, know that cell first change to pre-malignant lesions and then into cancers cell, and cancer is classified according to its origin cell type. In the management of patients with head and neck cancers, you will always play an important role in firstly recognising cancers earlier (which translates to improved outcomes) but also in the follow up of patients. The more advanced cancers will always have post treatment sequelae, and they see their GPs for that.

The management follows the standard history, examination, special examinations process, but then and important step is to stage the patient. Families and patients are always focussed on 'what stage does a patient have'. From your perspective it is important to refer patients to units that have multi-disciplinary teams (MDT) caring for these patients. It takes a team effort and rigorous discussion before deciding on the final treatment. Unfortunately, some cases don't end up in MDTs, and this negatively affects their outcomes. Only then is treatment offered and these patients are followed up for at least 5 years.

History
- Loco-regional
    - When did it start?
    - How did it start?
    - What initiated it?
    - Why did it start?
    - Progression?
    - Associated symptoms
- Risk factors
- Previous cancer of radiation
- HPV – sexual history
    - Be sensitive!
- Systemic
    - Pulmonary symptoms
    - Performance status
    - Loss of weight

Site specific history
- Lower airways
    - Voice
    - Feeding – LOW, odynophagia, dysphagia
    - Airway
        - Stridor / Stertor / Hot potato voice
- Lesion
    - Pain / bleeding / referred ear pain
- Trismus
- Nose, Oral cavity, Oropharynx, Nasopharynx, Hypopharynx, Larynx, Oesophagus

General examination
- JACCOLD
- Loss of weight / BMI
- General performance
    - WHO / ECOG

- Co-morbidity indexes
    - Charlson

ENT examination

- Ears
    - Ear pain
    - Blocked ears
- Nose
    - Blocked nose
    - Epistaxis
- Throat
    - Think
        - Voice
        - Aspiration
        - Swallowing
        - Airway compromise
    - Inspections
        - Oral cavity
            - Seven areas
            - Parotid duct opening (Stenson's duct)
            - Submandibular gland openings (Wharton's duct)
        - Oropharynx
            - Four areas
    - Palpation
        - Bimanual as well!
- Cranial nerves
- Neck (Think in terms of triangles or levels)
    - Inspections
    - Palpation
        - Midline (constant landmarks)
            - Supra-sternal notch, Trachea, Thyroid, Cricoid cartilage, Thyroid cartilage, Hyoid bone
        - Lateral
            - Sternocleidomastoid and trapezius muscles
        - Submandibular gland
        - Lymph nodes / Other masses
    - (Percussion)
    - Auscultation

Special investigations

- Bedside
    - (Flexible naso-pharyngo-laryngoscopy) – done by ENT
    - Functional evaluation of swallowing
    - Biopsies
    - FNAC / Core needle biopsy
    - Blood tests
- Radiology

    - Ultrasound
        - FNAC
    - CT
        - CT guided biopsy
        - Staging
    - MRI
    - PET-CT
    - Barium swallow
- Swallowing studies

Staging

All HNC patients are staged using the current 8th edition of the UJACC staging manual. It is based on a T-stage (primary), N-stage (neck nodes), and M-stage (metastases). The staging allows for clinical and radiological features to stage a cancer. Below is an example of the T-staging of glottic cancers.

	Supraglottis	Glottis	Subglottis
Tx	Tumour cannot be assessed		
T0	No tumour evident		
Tis	Carcinoma in situ		
T1	Limited to one subsite of supraglottis and mobile cords	T1a - One cord involved and/or anterior / posterior commissure involved, with mobile cords	Limited to subglottis
		T1b - Both cords involved and/or anterior / posterior commissure involved, with mobile cords	
T2	Invades more than one subsite of supraglottis. Invades one subsite of supraglottis and/or one or more subsite(s) of adjacent area such as glottis, BOT, vallecula, medial wall of pyriform fossa. Mobile cords	Involves adjacent subsite of either supra- or sub-glottis, or impaired cord mobility	Onto cords, ± impaired cord mobility
T3	Tumour limited to larynx with fixed cord, and/or invades paraglottic space, pre-epiglottic space, postcricoid area, inner cortex of thyroid cartilage (lateral pyriform fossa wall)	Limited to the larynx with cord fixed, and/or invasion of the paraglottic space, and/or invasion of inner cortex of thyroid cartilage	Limited to endolarynx with cord fixed
T4a	Moderately advanced local disease. Invasion of outer cortex of thyroid cartilage, extra-laryngeal spread such as oropharynx, trachea, cricoid, oesophagus, thyroid gland, neck soft tissue including strap muscles and extrinsic muscles of tongue		
T4b	Very advanced local disease. Invades prevertebral space, encases carotid artery, mediastinal structures		

Neck staging is shown below.

	N staging for cervical lymph node metastases and non-viral CUP	Pathological N staging	N staging for HPV+ oropharyngeal CA	Pathological N staging for HPV+ / P16 + oropharyngeal Ca	N staging for EBV+ CUP
N0	No regional lymph node metastases				
N1	Single ipsilateral lymph node ≤ 3 cm and ENE-		One of more ipsilateral lymph nodes ≤ 6 cm	1-4 lymph node(s)	Unilateral lymph node, and/or uni- or bilateral metastasis to retropharyngeal lymph nodes, and must be ≤ 6 cm and above caudal border of cricoid cartilage
N2a	Single ipsilateral lymph node 3-6 cm and ENE-	Same, AND Single ipsilateral lymph node ≤ 3 cm with pENE+	Contralateral or bilateral lymph nodes ≤ 6 cm	> 5 lymph nodes (N2)	Bilateral lymph nodes, and must be ≤ 6 cm and above caudal border of cricoid cartilage
N2b	Multiple ipsilateral lymph nodes ≤ 6 cm and ENE-			N/A	
N2c	Contralateral / Bilateral lymph node(s) ≤ 6 cm and ENE-			N/A	
N3a	Any lymph node(s) ≥ 6 cm and ENE-		Any lymph node(s) ≥ 6 cm	N/A	Any lymph node(s) > 6 cm, and/or extension below caudal border of cricoid cartilage
N3b	Any lymph node(s) with ENE+ (Clinical and Radiological)	A single lymph node > 3 cm with pENE+, or Any multiple, bilateral, contralateral lymph node(s) with pENE+		N/A	
	Can document ENE minor (<2 mm invasion beyond capsule), or ENE major (>2 mm invasion beyond capsule), for future changes. Tumour deposit within lymphatic drainage without identifiable lymph node would be recorded as pN+ and pENE+				

Thereafter, patients are staged. Patients and family members are always very keen to hear the "stage" of cancer. Be careful not to lose focus of the "patient" in front of you. Stages are more applicable for larger groups of patients with the same tumour. Nonetheless, below shows stages for non-HPV and non-EBV HNC.

Staging for Head and Neck Cancers				
	T1	**T2**	**T3**	**T4**
N0	I	II	III	IV
N1	III	III	III	IV
N2	IV	IV	IV	IV
N3	IV	IV	IV	IV

M1	**IV**	**IV**	**IV**	**IV**

Once patients have confirmatory histology and was staged, treatment is discussed at a multi-disciplinary meeting. At this meeting, we decide whether the goal / intent of treatment is curative, palliative, or end-of-life treatment (non-beneficial). Making these decisions takes years of knowledge and experience, but we take the following into account:

- Patient factors
    - Co-morbidities
    - Previous treatment
    - Wish
    - Follow up
- Tumour factors
    - Size, Extent, Position and other factors that might influence the choice of surgery versus radiation
- Doctor factors
    - Availability of surgical expertise
    - Availability of reconstructive options
    - Availability of radiation facilities
    - Availability of adjuvant therapies
    - Availability of other medical and support staff (anaesthetist, ICU, speech therapy, dieticians, physiotherapy)

We measure these against the absolute ability to either improve quality of life, or significant increased survival, or both. If we cannot achieve either of these, one should seriously consider if curative treatment is indicated. Patients that either has an unacceptable high rate of morbidity or mortality with curative intent options, should receive palliative care.

Treatment of HNC

In general, the principles for treatment are:

Stage I & II:

- Single modality treatment
- Surgery or radiation

Stage III & IV

- Multi-modality treatment
- Classical options are
    - Surgery and post op radiation or chemoradiation
    - Radiation
    - Chemoradiation

Primary site surgery

- In general, surgery has become more functional, without compromising safe oncological principles
- Reconstruction also plays a major role
    - Local, distal and free flaps

Neck Surgery

- We went from doing a radical neck dissection regardless of nodal status, to a selective neck dissection even for N+ neck disease

Radiation

- Good data regarding dose
- Planning

- Intensity modulated radiotherapy
- Altered fractionation
    - Hyper-fractionated
    - Accelerated

Chemotherapy

- Adjuvant
    - After treatment
    - Eradicating residual micro-metastatic disease
    - Rarely used as single option after surgery
- Neo-adjuvant
    - Before treatment (*Induction)*
    - Response to induction chemotherapy correlated with a subsequent response to RT
- Concurrent
    - With treatment
- Organ preservation concept
    - Concept of offering chemoradiation as first line options, keeping surgery for salvage

Targeted therapies / Immunotherapies

- Molecular targets are becoming more prominent in Head and Neck cancers
- Options are
    - Targeting growth factor independence
        - EGFR
        - Tyrosine kinase inhibitors
    - Targeted drugs that act as radiosensitizers
    - Antitumour immune response using monoclonal antibodies (also referred to as immune checkpoint inhibitors (ICI))
        - PD-1 / PD-L1
        - CTLA4
- While ICIs found initial success in trials for patients with recurrent / metastatic HNSCC, several subsequent phase 3 trials have yielded disappointing results of ICIs in the curative setting in combination with concurrent chemoradiotherapy.
- The negative survival signal seen in concomitant ICI therapy with chemoradiation has led to trials evaluating sequential administration of ICI either in the neoadjuvant or adjuvant phase of treatment.
- Based on promising response rates in early-phase trials, several phase 3 trials are currently underway to investigate whether ICI therapy with curative intent can improve survival in the difficult to treat locally advanced HNSCC population.

Cancer prevention

- Smoking
    - Causes 40% of all cancer related deaths
- Vaccines
    - HPV
        - Gardasil 9
    - Hep B
    - It is estimated that 20% of all cancers are caused in some way by viruses
- Chemicals

- Anti-oestrogens – prevent ductal carcinoma
- Finasteride – prevent prostate carcinoma
- Aspirin – prevent colorectal carcinoma

23) Oral mucosal lesions

As a GP, you will be frequently confronted with patients with oral pathology. Below is a brief differential diagnosis list. Not all conditions will be discussed in detail.

Oral mucosal lesions

- Red / White
    - Leuko-oedema
    - Leukoplakia
    - Erythroplakia
    - Oral hairy leukoplakia
    - Oral lichen planus
    - Candida
- Vesico-Bulleus / Ulcerative
    - Pemphigus / pemphigoid
    - Herpes simplex virus
    - Primary / secondary
    - Aphthous stomatitis ulcers
    - Erythema multiform
    - Eosinophilic granuloma / Toxic granuloma
- Pigmented
    - Intrinsic
        - Melanocytes
            - Nevi
            - Macula
            - Melanoma
        - Syndromic
            - Addison's disease, Cushing disease, Neurofibromatosis
        - Inflammatory
    - Extrinsic
        - Trauma
        - Tattoo
        - Amalgam
        - Drugs

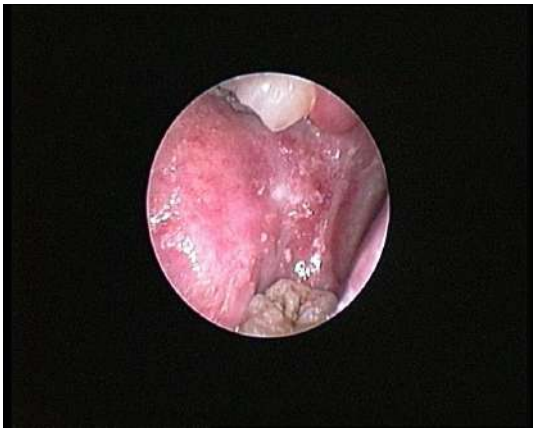
Leukoplakia and Erythroplakia

Leukoplakia and erythroplakia translates to white lesion or red lesion that can't be wiped off respectively. Remember, they can be precursors to head and neck squamous cell carcinomas. As a general rule, 10-15% of these lesions will already harbour squamous cell carcinomas, with erythroplakia having a higher incidence than leukoplakia. They are also discussed in the chapter Head and Neck Cancer.

Their aetiology is the same as the major risk factors known to cause head and neck cancers namely, smoking, tobacco chewing, alcohol, betel nut, and sun exposure on the lower lip (actinic cheilitis). They may have different clinical appearances such as nodular, speckled, and verrucous (see pictures below). Leuko- and erythroplakia

can co exists. On histology they might display from hyperkeratosis, acanthosis, dysplasia, carcinoma in situ, and infiltrative cancer cells. Treatment consists of removing the aetiological factors, active surveillance, incisional biopsies, or excisional biopsies. In general, you should identify these as precursors to head and neck cancers and refer these patients to ENT specialists with an interest in head and neck cancers. Sometimes, one is confronted with patients with extensive erythro-leukoplakic changes. We call this field cancerization or condemned mucosa, whereby multiple mucosal areas, exposed to the same risk factors, are in the process of becoming cancer cells.

The only condition to differentiate between is **leuko-oedema**. This is an extremely common condition and can be seen as a variation of the norm. It occurs mainly in the buccal mucosa and has a more diffuse, opaque whitish appearance compared to leukoplakia. It can also occur in the larynx and vagina. Light pressure applied to the lesion will produce a normal appearing mucosa, that changes to leuko-oedema once pressure is stopped. No specific treatment is needed.



Endoscopic picture showing leuko- and erythroplakia in right retromolar trigone area.

Oral hairy leukoplakia

Very common lesions that present with white streaks on the lateral sides of the oral tongue. It is due to Ebstein Barr virus and has a higher incidence in HIV patients. No definitive treatment is required.

Candida

Extremely common. Remember there are different clinical presentations of Candida in the oral cavity namely:

- Pseudomembranous
- Erythematous
- Atrophic
- Hyperplastic
- Angular cheilitis (fissures in the corners of the mouth)

Remember to exclude local and systemic risk factors such as:

- Local
    - Smoking
    - Foreign bodies such as dentures, nasogastric tubes
    - Radiation

    - Topical steroids
    - Xerostomia
- Systemic
    - Immunosuppression
    - Diabetes
    - Steroid therapy
    - Antibiotics

The clinical picture is dependent on the site and severity of the infection but can include taste abnormalities, odynophagia, and dysphagia. Treatment includes reversing the underlying factors, local, and systemic antifungals.

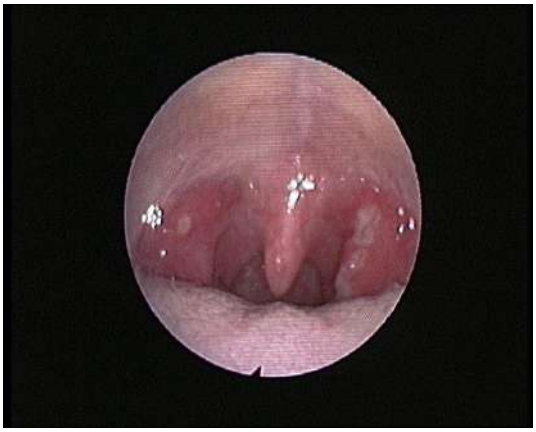
Aphthous ulcers versus Herpes Simplex

Primary herpes simplex virus (HSV) infections affect 60-90% of the world's population. It usually follows 5-7 days after contact with another person, has a prodrome of 48hours, after which the vesicles forms on the lips that lasts 7-14 days. Treatment is systematic with topical steroids and topical antivirals. Some adults will go on to develop repeated HSV infections. Its clinical picture is compared to aphthous ulcers in the table below.

	Aphthous ulcers / stomatitis	**Human Herpesvirus lesions**
Aetiology	Varied, Immune dysfunction	Herpes simplex type I & II
Location	Moveable, non-keratinized mucosa	Keratinized tissue, mucosa
Vesicle phase	No	Yes
Duration	Varies; usually 7-10 days	7-14 days
Management	Topical steroids	Topical steroids Oral antivirals
Prodrome	Uncommon	Often
Triggers	Stress, Light, Foods, Medications	Stress, Trauma
Biopsy findings	Non-specific	Viral cytopathic effect

Aphthous ulcers can be divided into:

- Minor
    - < 10 mm
    - Lasts 7-10 days
    - Heals without scar tissue
- Major
    - > 10 mm
    - Can last up to 6 weeks
    - Very painful
    - Strongly associated with HIV (exclude)
- Herpetiform
    - Multiple <2 mm lesions
    - Rare



Endoscopic picture showing a small ulcer of the right anterior tonsillar pillar and larger ulcers on the left anterior tonsillar pillar.

Tobacco related changes

Tobacco, other than causing cancers, can lead to:

- Gingival retraction
- Dental carries
- Nicotine stomatitis
    - Fissured, cracked leukoplakic changes on hard palate

24) Acute pharyngotonsillitis

Most of the time, acute pharyngotonsillitis is of viral origin. Only 5-10% of sore throats in adults are due to bacterial infections compared to 25-30% in children. Group A β-haemolytic *Streptococcus pyogenes* (GABHS) remains the most common bacterial cause. Because of its ability to cause rheumatic fever, glomerulonephritis, and other septic complications it needs to be differentiated from viral and other aetiologies. Treatment with antibiotics has been shown to prevent the development of rheumatic fever and septic complications, but not the development of glomerulonephritis. Fortunately, there has never been a GABHS isolate that showed resistance to penicillin.

Bacterial tonsillitis

GAHBS

- Humans are the natural reservoir
- Infections are more common in autumn and winter
- Peak incidence between 5-6 years
    - Second peak between 12-13 years
- Several pathogenic mechanisms
    - Patient factors (cytokines)
    - Enzyme production (streptolysin)
    - Cell wall factors (hyaluronic acid)
    - Exotoxins (A&B)

Symptoms of infective pharyngotonsillitis are not specific enough to differentiate between the different infective causes (bacterial versus viral). Symptoms in general include sore throat, fever, dysphagia, and halitosis. Symptoms in favour of acute viral pharyngitis include rhinorrhoea, cough, conjunctivitis, hoarseness, stomatitis, ulcer(s), diarrhoea. Therefore, the question remains how can we diagnose a bacterial pharyngitis and GABHS in particular? We advise using the **modified Centor criteria**. It can only be applied in patients with recent onset (≤3 days) acute pharyngitis. A score of more than 3-4 points correlates with a bacterial infection, and thus antibiotic use.

Modified Centor criteria

Criteria		Points
Age	< 3	0
	3-14	+1
	15-44	0
	> 44	-1
Cough	Present	0
	Absent	+1
Tonsillar exudates / swollen		+1
Temp > 38°C		+1
Anterior cervical lymphadenopathy		+1

Other options to diagnose a possible GABHS include:

- Rapid antigen testing
- Throat swab (PCR as well)
- Nucleic Acid Amplification Techniques (NAATs)

The following link gives a nice overview of subject: https://www.frontiersin.org/journals/cellular-and-infection-microbiology/articles/10.3389/fcimb.2020.563627/full.

Treatment of acute bacterial tonsillitis

- Antibiotics
  - Penicillin / Amoxicillin
    - Pen VK (30 minutes before meals)
      - 250 mg BD po x 10/7 (<27 kg)
      - 500 mg BD po x 10/7 (>27kg)
    - Amoxicillin
      - Children
        - 50 mg/kg OD po x 10/7 (max 1000 mg)
      - Adults
        - 500-1000 mg BD po x 10/7
  - B-lactam allergy
    - Children
      - Azitromycin 10-20 mg/kg/d OD x 5/7
      - Claritrhomycin 15 mg/kg/d in two dosages x 10/7
    - Adolescents and adults
      - Azitromycin 500 mg OD po x 3/7
      - Clarithromycin 500 mg BD x 10/7
- Supportive treatment
  - Adequate fluid intake
  - Pain relief – panado
    - Aspirin is contraindicated because of the risk of Reye's syndrome (encephalopathy and liver failure)
  - Rest

Please refer to the following article on the dosages: https://scielo.org.za/pdf/samj/v105n5/16.pdf

Complications due to infective (bacterial) pharyngotonsillitis includes:

- Infective
  - Peritonsillar abscess
    - Also known as Quincy abscess
    - Between tonsil and lateral pharynx wall
    - Follows after bacterial pharyngitis
    - Pain persists and increases
    - Odynophagia and dysphagia
    - Trismus and drooling
    - Grave prognosis in medical history
      - Due to spread down in neck
      - Not so anymore with treatment
    - Treatment
      - Surgical treatment changed that
        - Aspiration
          - Preferred method
          - You need to be able to do this at GP level
          - See technique at the end of this chapter
        - Surgical drainage with scalpel blade

            - "Hot" tonsillectomy
        - Antibiotics
            - Almost immediate improvement
    - Parapharyngeal abscess
    - Deep neck space abscess
    - Septic shock
- Non-infective
    - Rheumatic fever
    - Glomerulonephritis
    - Paediatric auto-immune neuropsychiatric disorder
        - Sudden and rapid onset of obsessive–compulsive disorder (OCD) and/or tic disorder symptoms
    - Scarlet fever
        - Red rash. The rash looks like a sunburn and feels like sandpaper. It typically begins on the face or neck and spreads to the trunk, arms and legs. Pushing on the reddened skin makes it turn pale.
        - Red lines. The folds of skin around the groin, armpits, elbows, knees and neck usually become a deeper red than the other areas with the rash.
        - Flushed face. The face may appear flushed with a pale ring around the mouth.
        - Strawberry tongue. The tongue generally looks red and bumpy, and it's often covered with a white coating early in the disease.

Other specific entities

Ebstein Barr Virus

- Infective mononucleosis (kissing's disease)
- 90% of adults are seropositive for EBV
- Incubation of 3-7 weeks
- Prodrome of malaise, fever and chills for 1 week
- Followed by sore throat, exudative tonsillitis, lymphadenopathy
- Lymphadenopathy "out of proportion" in relation to the disease
    - Frequently causes acute onset sleep disordered breathing in children
- Hepatosplenomegaly is found in 50% of cases
    - Complicates the diagnosis because of simulating haematological malignancy
- Diagnosis
    - Clinical picture
    - EBV antigens
    - Atypical lymphocytes
- Treatment
    - Symptomatic
    - Penicillin causes a maculopapular rash in 95% of pt
    - Consider 2-3 days of steroids to reduce the size of the lymphoid tissue
- Complications
    - Secondary bacterial infection
    - Acute enlargement of lymphoid tissue with airway obstruction
    - Neurological – Guillain-Barre syndrome, meningitis, encephalitis, cranial neuropathies

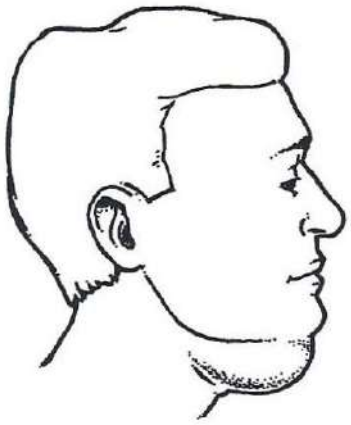
HIV seroconversion

Seroconversion is a short, flu-like illness that occurs in most people after contracting HIV. The symptoms of seroconversion usually appear one to four weeks after infection and can include:

- Sore throat
- Fever
- Rash
- Swollen lymph nodes in the neck, armpits, and groin
- Joint or muscle pain
- Headache
- Mouth ulcers
- Diarrhoea
- Weight loss
- Tiredness

Ludwig's Angina

- Floor of mouth cellulitis usually secondary to odontogenic infections
- Mixed flora
- Cellulitis rather than abscess formation
- Commonly associated with HIV
- Treatment
    - AB
    - Airway management
        - Traditionally a tracheostomy
        - Nowadays high care units with airway monitoring
        - Consider stat dose of steroids
    - Refer urgently to Maxillofacial Surgery

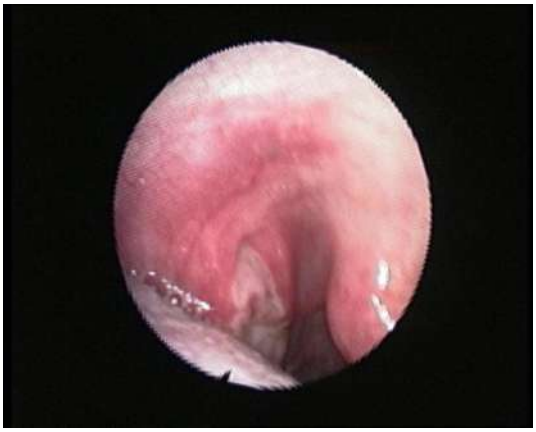


Ludwig's angina. Note the brawny neck swelling, the ill appearance of the patient and the saliva drooling from the mouth.

Differential diagnosis of a membrane in the throat

- GABHS
    - Scarlet fever
- Candidiasis
- Diphtheria
- EBV (infective mononucleosis: dirty appearing tonsils)
- Agranulocytosis

- Vincent angina (necrotizing tonsillitis caused by fusobacteria or spirochete infections)



Endoscopic picture of right tonsil demonstrating the “dirty” appearance in EBV.

Adenotonsillectomy

The tonsil (palatine tonsil), adenoid, tubal tonsil, lingual tonsil, and numerous small lymphoid collections form part of Waldeyer's ring which is part of the mucosa associated lymphoid tissue (MALT). Apart from natural defences (such as skin), these are the first line of defence. The lymphoid tissue consists mainly of B-lymphocytes (65%) and can produce immunoglobulins. T-lymphocytes make up 30% of the lymphoid tissue. The tissue is most active between the ages of 4-10 years. The ability to produce mainly immunoglobulins is also the tonsils' greatest limitation. It does not have the ability to kill more complex organisms, nor can it utilize the rest of the immune system to help. This is precisely the reason why certain patients develop repeated infections.

The first tonsil operations were done around 2000 years ago. It remains one of the most common operations, and in many cases is a patient's first contact with doctors and hospitals. It is estimated that one in eight children in America have their tonsils removed (>500 000/year). Three quarters of the operations are done for obstructive complaints. In South Africa more children are referred for repeated infections, and more focus should be placed especially on identifying children with obstructive symptoms.

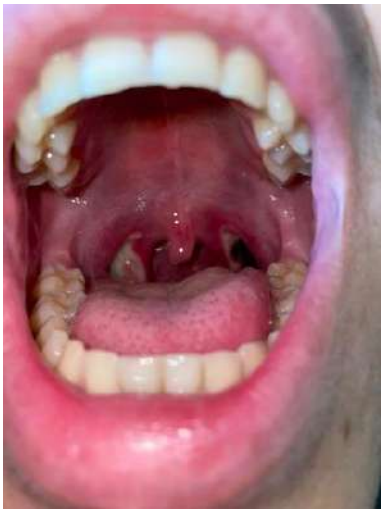
As you will see in the indication for an adenotonsillectomy below, two conditions that are of vital importance to identify are obstructive sleep apnoea syndrome (OSAS) and suspected cancers. Chronic adenotonsillar obstruction leading to OSAS is the most common reason for an adenotonsillectomy in the world. It is mainly caused by exposure to passive smoking and chronic bacterial infection. These patients should be sent for a heart sonar to rule out or confirm pulmonary hypertension / cor pulmonale / congestive right heart failure. They may have facial features of “adenoid faces” such as an open mouth breathing, long faces, retrognathia, high palate, hypoplastic maxilla, small upper lip. These children should be preferably operated but only in hospitals with all the necessary infrastructure such as an PICU, paediatric anaesthetist etc. Suspected cancer speaks for itself. A biopsy can be taken to investigate it further. The other indications are discussed below.

In most cases, there are no serious consequences / complications if it is decided not to remove the tonsils other than the two mentioned above. Complications of tonsillitis can be divided into infectious and non-infectious problems. Before the antibiotic era, dramatic infectious complications were described. These were mainly abscess formations in the throat (peritonsillar) and neck (retropharyngeal and parapharyngeal) but are fortunately very rare these days. Non-infectious complications are rheumatic fever, glomerulonephritis, paediatric auto-immune neuropsychiatric disorder, and scarlet fever. In South Africa we still have a high incidence of rheumatic fever following a GABHS. There are clinical criteria and laboratory investigations to identify a GABHS, and this group of patients needs to be treated with antibiotics to prevent the development of rheumatic fever. An operation is also recommended in patients with already damaged heart valves from rheumatic fever, as well as in children in a community with a high incidence of rheumatic fever. Antibiotics does

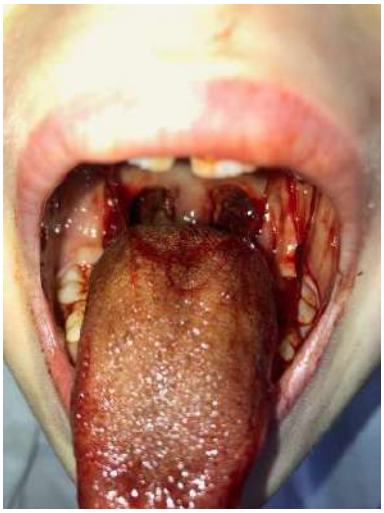
not prevent glomerulonephritis, but an operation is also recommended in the group to limit further damage. Remember that repeated use of antibiotics also has dangers. Antibiotics also destroy your commensal organisms (gut biome) and create the potential for a patient to be colonized with more resistant bacteria. Ironically, this is precisely a risk factor for more infections. There can also be severe gastrointestinal complications with the repeated use of antibiotics.

There are several dangers and complications that can occur post operatively. Patients should be aware of the common dangers, and then also potential serious and life-threatening complications. The global mortality figure is 1/30,000, however, we do not have any accurate figures in South Africa. In a recent study from the USA, it was found that the most common reason for death after surgery is related to medication overdose rather than bleeding (this was due to a genetic alteration in the codeine metabolic pathway and has not been described in South Africa). Parents should be very aware of the potential risk of overdosing children (especially with codeine containing medications). Bleeding from the raw tonsil bed occurs in 1%-10% of cases. This can be either early (<24 hours) or late (typically day 7-10). All post-tonsillectomy bleeds should be referred to a casualty department. As an interim measure you can advise the patient to suck on an ice cube. Remember that the patient will probably need to go to the theatre so instruct them not to eat or drink anything. In a casualty setting, you can use diluted hydrogen peroxide oral rinses (1-3% hydrogen peroxide gargles) or direct pressure with adrenaline-soaked gauze (if the patient is old enough and allows you) to try and stop the bleeding.

Pictures



Post-tonsillectomy picture with the “normal” greyish-white slough in the right tonsillar bed, and a blood clot in the left tonsillar bed.



Active post-tonsillectomy bleeding.

Another immediate complication is the “loss of the airway”. You need to be able, with your anaesthetist, to manage an acute airway compromise in theatre / recovery room / in the ward. This is typically in the setting of and OSAS child with post-operative pulmonary oedema.

In rare cases, there can be velo-pharyngeal incompetence post-surgery. This causes a weak / funny voice and sometimes regurgitation of food / water through the nose while eating (this is why an adenoidectomy is a contra-indication in cleft palate patients). Fortunately, in most cases this will clear up spontaneously with time.

Chronic pain in the throat can rarely occur after an operation. Many more complications are described in social media, but these are the important ones.

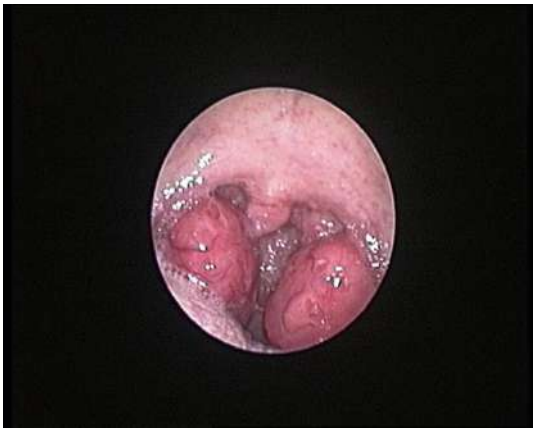
Yes, tonsils can grow back after complete removal (see techniques below – sometimes only a portion of the tonsil is removed). Although the tonsil is in a capsule, the lower part is sometimes continuous with the lymphoid tissue on the base of the tongue (lingual tonsil). There is also widespread lymphoid tissue all over the mucosal surfaces in the mouth and throat. In certain circumstances, this lymphoid tissue begins to enlarge and then fills the space where the tonsil was taken out, giving the appearance that the tonsil has grown back. Remember this also applies to the adenoid which does not have a true capsule to begin with. In these cases, the reason why this happened must be looked for, and in rare cases, the patients are operated on again.

Also remember that the normal incidence of pharyngitis (throat infections) is between 2-4 per year. So even if tonsils are removed, patients can get a sore throat after the operation. Furthermore, 90% of tonsil and/or throat infections in children are viral and not bacterial. Remember we use repeated bacterial infection (and not viral) to decide to take out tonsils.

Indications for adenotonsillectomy

- Absolute
    - Suspected cancer
    - Obstructive sleep apnoea syndrome in children
        - Don’t forget about adults, but a lesser role compared to children
- High priority
    - Repeated acute **bacterial** infections (Modified Paradise Criteria)
        - > 7 infections in 1 year
        - 5/year for 2 years running
        - 3/year for 3 years running
    - Repeated peri-tonsillar abscess
- Moderate
    - Repeated infections with
        - Multiple antibiotic allergies
        - Febrile seizures
    - Adults with OSAS
    - Halitosis (especially in adults)
        - Tonsilloliths (tonsil stones)
            - The tonsil surface has small slits / pouches. These can become blocked by food and tonsil secretions. In certain cases, this results in yellow-gray cheesy hard granules that can be squeezed out. This gives rise to especially bad breath (halitosis) and chronic sore throat. Most patients decide in favour of an operation.
    - Children with sleep disordered breathing and severe snoring, and possible enuresis / learning difficulties / emotional problems
    - Severe adenotonsillar hypertrophy with

        - Speech problems
        - Malocclusion
        - Swallowing problems – dysphagia to solids
- Maybe (Case-by-case individualised decision)
    - Infections with
        - GABHS carrier status
        - Rheumatic fever
        - Periodic fever, aphthous ulcers, pharyngitis, cervical adenopathy (PFAPA syndrome)
        - Chronic tonsillitis
    - Children with repeated AOM / OME
        - Adenoids can be removed separately in this group (see otology lectures)


Endoscopic picture of tonsillar hypertrophy.

Contra indications

- Bleeding tendencies
- Cleft palate
    - Occult cleft palate
- Age – relative. Be careful to operate the very young.

Technique

There are two main types of surgery, namely partial or complete tonsillectomy. A partial tonsillectomy has become more popular in European countries after a number of post-tonsillectomy deaths. Some of the newer instruments describe below also lend themselves better to partial techniques (guillotine tonsillectomies are still performed in some countries). Another way to describe it is extra-capsular versus intra-capsular techniques. Most intra-capsular techniques also aim to remove as much (near total) of the tonsil as possible.

In an extracapsular tonsillectomy the first step of the operation is to identify the capsule, which is the deep surface of the tonsil. Now there are several techniques for physically dissecting tissue, and this is where the differences come in. The choices include cold steel instruments, electric current instruments (bipolar and monopolar), plasma field energy instruments (coblation), CO2 laser, ultrasonic instruments (hormonic scalpel), radio frequency instruments and "powered" instruments (shavers). Worldwide, cold steel and bipolar forceps technique are the most common. Of course, there are a lot of personal preferences involved and also a lot of myths about what is best. There may also be additional techniques such as injecting local anaesthetist and/or other fluids before or after the time. Stitches can also be inserted, just at the base or the entire length of the tonsil bed. The adenoid is usually removed through the mouth with curved instruments by scraping it out or "coblation" instruments. I prefer to do this under vision, by looking down the nose with a small rigid endoscope but many

ENTs does this either blindly or with a mirror. Haemostasis is achieved by using different packs and/or the instruments as mentioned above.

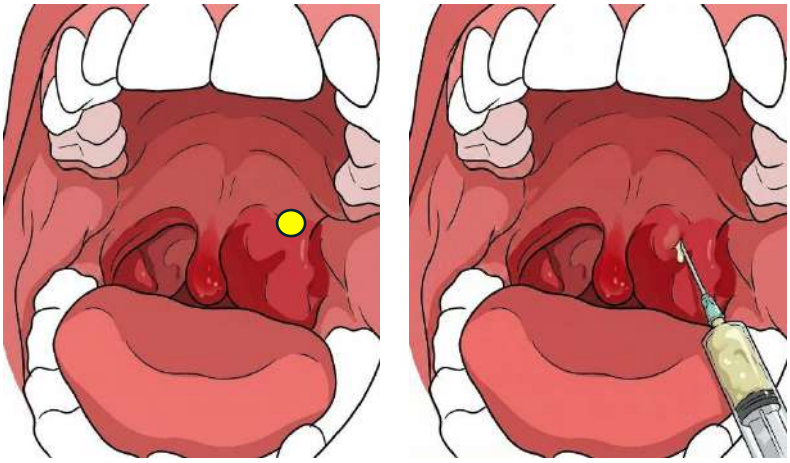
An intracapsular tonsillectomy can only be done with the newer type of instruments (plasma field instruments, ultra-frequency instruments). The core argument in favour of using intracapsular techniques rest on the principal that the diameter of blood vessels that enter the tonsil, after penetrating the capsule, halves in diameter. Therefore, it is mostly a bloodless dissection, requiring less haemostasis and therefore have less post-operative pain. The downside is the potential higher incidence of tonsillar regrowth and re-operations later.

A differential diagnosis for a "sore throat" includes

- Infective
    - Viral most common
        - Adenovirus, Coxsackie A virus, Influenza virus, Parainfluenza virus, Epstein Barr virus
    - Bacteria (5-30%)
        - Group A β-haemolytic *Streptococcus pyogenes* (GABHS)
        - Other
    - Fungi
        - Candida
    - Granulomatous
- Other
    - Irritant
    - Reflux
    - Tumours
    - Auto-immune
    - Trauma
    - Neuralgias

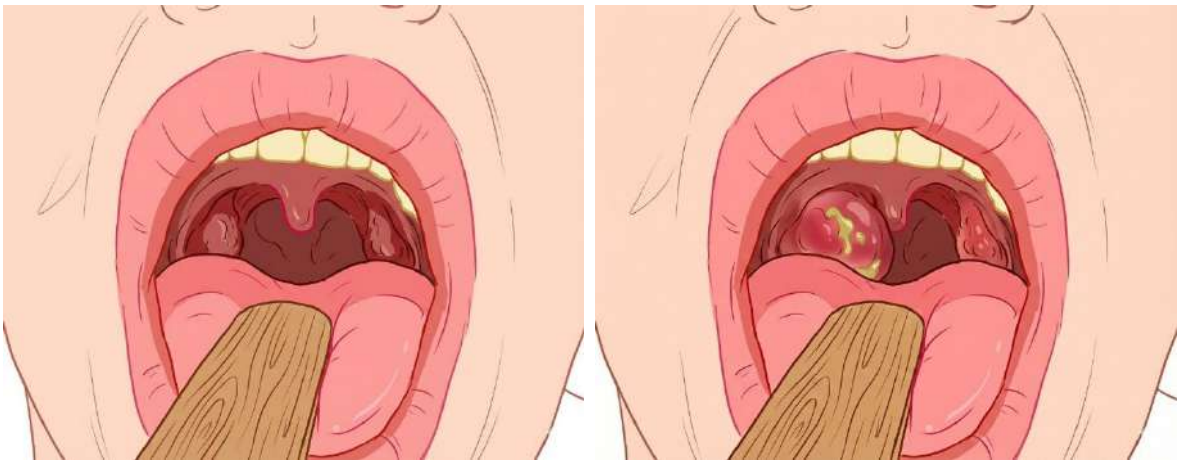
Aspiration of a peri-tonsillar abscess

Fortunately, it is uncommon in children, and the majority of patients are adults. Have the patient sit up and breath through the mouth. Trismus is common and can impair visualising the oropharynx. Inspect the oral cavity / oropharynx while using a headlamp and having both hands free. A peri-tonsillar abscess will produce varying degrees of swelling supero-lateral to the normal tonsil, pushing the uvula to the contra-lateral side. It is not unusual to have lymphadenopathy in the neck, but in severe cases the neck might have a generalised swelling due to deep neck space infection. Having accessed how wide the patient can open their mouth, decide on an appropriately sized syringe (if possible, at least 10 ml) and attach a tick bore needle. If you are afraid of injuring vascular structures (internal carotid) deep to the abscess, a trick is to wind Sellotape / plaster 1.5 – 2 cm from the tip of your needle as a gauge not to insert the needle deeper. Aim for the area of maximum swelling (usually halve way between the uvula and ramus of the mandible. As you insert the needle, apply suction on the syringe. As soon as you aspirate pus, stop advancing and aspirate till no more pus comes out. Remember to send a MCS sample of your pus. Sometimes this needs to be repeated at intervals (12-24 hours). On average you will get less than 10ml of puss, but we have had patients with 70ml! Rarely, one sees a patient having all the features of a peri-tonsillar abscess, but you get no puss on aspiration. It is advisable that if you have any concerns to re-aspirate in 12-24 hours. The pictures below demonstrate the procedure.



The left picture demostrates a left sided peri-tonsillar abscess with the yellow spot indicating where you will aim. The right picture shows the pus after aspiration.

Sometime, an asymetrical tonsillar enlargement might fool you into thinking it is a peri-tonsillar abscess as shown below.



The picture on the left shows a normal throat with the picture on the right showing a massively enlarge (infected) unilateral tonsil. This is very suspicious for lymphomas and should be referred to an ENT.

25) Speech and Swallowing

Speech / Voice

Broadly speaking, hoarseness is due to an alteration in the voice character resulting from an abnormality within the larynx. REMEMBER, any patient with hoarseness for longer than one month must be considered to have a malignancy until this is excluded. Being able to examine the larynx can simplify the diagnostic dilemma considerably. As a student you are often afforded the opportunity to learn the technique of indirect laryngoscopy.

Functions of the vocal cords

- Protect the lower airways
    - Closing
- Voice production
- Valsalva
    - Lifting heavy objects
    - Cough
- Control of ventilation
    - Opening on inspiration
    - PEEP on expiration

Definitions

- ***Speech*** is the expression of, or the ability to express thoughts and feelings by articulate sounds
- ***Voice*** is often used to refer to speech as a whole. However, when used in the context of voice evaluation, it is generally restricted to the acoustic output resulting from the interaction of vocal fold vibration with the vocal tract in vowel production
- ***Phonation*** is a term used to describe the physical and physiological processes of vocal fold vibration in the production of speech sounds
- Impaired voice production due to abnormal vocal fold vibration is known as ***dysphonia***, while no voice or whispery voice associated with no vocal fold vibration is termed ***aphonia***
- ***Hoarseness*** is a non-specific, general term used to describe any change in voice quality, perceived as rough, harsh or breathy
- ***Dysarthria*** is difficulty in articulating words, caused by impairment of the muscles used in speech (think CVA!)
- ***Dysarthrophonia*** is dysphonia in conjunction with dysarthria, for example after a cerebrovascular accident, head injury or part of a degenerative neurological condition, such as motor neuron disease
- ***Dysphasia*** is impairment of the comprehension of spoken or written language (**sensory dysphasia**) or impairment of the expression by speech or writing (**expressive dysphasia**), especially when associated with brain injury
- ***Odynophonia*** is pain when talking
- ***Psychogenic dysphonia*** is marked by loss of vocal control associated with ‘disturbed psychological processes’ (such as stressful life events, anxiety or depression and actual conversion)

A generally accepted and pragmatic definition of a normal voice is one described as having the following characteristics:

- It is audible, clear or stable in a wide range of acoustic settings
- It is appropriate for the gender and age of the speaker
- It is capable of fulfilling its linguistic and paralinguistic functions
- It does not fatigue easily

- It is not associated with discomfort and pain on phonation

Normal voice production requires three essential elements:

- A **pressure gradient** across the vocal folds created by the flow of expired air from the lungs against the partly close vocal folds
- **Vocal folds** of appropriate **structure, mass and elasticity** that approximate with appropriate tension to allow them to vibrate at a range of frequencies
- A **resonating chamber**, the vocal tract, whose size and shape can be changed to modulate the acoustic properties of sound generated by the vocal folds
- This creates a fundamental frequency, harmonics add "colour", and the end result is a unique sound produced – "laryngeal imprint"

Pathological voice production

- Abnormalities in the vocal folds, dimensions or structure of the vocal tract and inadequate control or amount of subglottic pressure can all contribute to a pathological voice
- Abnormalities in the mass, elasticity and tensioning of the vocal folds can have two main effects: on the **frequency** rate and on the regularity of **vibration**
    - Alterations in **frequency** may lead to the voice being perceived as being too high or too low in pitch for the speaker's age and gender
    - Irregular **vibration** of the vocal folds, caused by the abnormalities described above, will affect voice quality by producing a less clear fundamental frequency and harmonic structure. This irregularity is perceived as hoarseness and roughness
- If there is a gap between the vocal folds during phonation, air will escape, reducing the relative amount of energy in the harmonic components and increasing the energy in the subharmonic components. This is perceived as both hoarseness and breathiness or a voice **lacking in power (asthenia**)
- Alterations in the relative size, shape and length of the vocal tract, for example from a mass, increased pharyngeal muscle tension or reflective properties of the vocal tract, can all influence the energy levels and harmonic structure of the radiated sound causing the voice to sound **strained or effortful**

There are three main **restrictions**

- **Impairment**
    - An alteration in the structure or function of the vocal apparatus (structural abnormality, inflammation, neuromuscular abnormality or muscle tension imbalance) causing symptoms such as hoarseness, a weak voice, pitch change, throat discomfort
- **Limitation in activity**
    - Reduction in vocal range in singing or the voice tiring or becoming hoarse with prolonged use in a noisy environment or if raised
- **Participation restriction**
    - Not being able to work or sing in a choir as a result of the voice problem

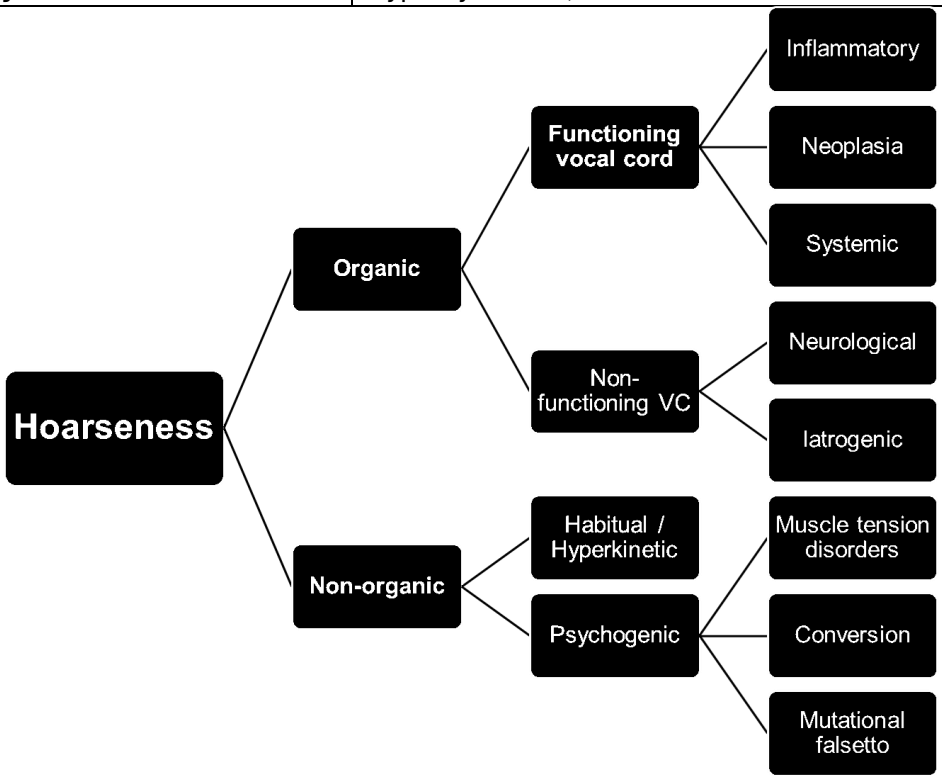
Patients' **complaints** may include

- Changes in voice quality (hoarseness, roughness and breathiness)
- Pitch that is increased or decreased which is not appropriate for their age and sex
- An inability to control their voice as required (pitch breaks, voice cutting out, tremor, glottal tightness)
- An inability to raise the voice or make the voice heard in a noisy environment (reduced loudness)
- An increased effort and/or reduced stamina of the voice or one that tires with use
- Difficulties or restrictions in the use of their voice at different times of the day or related to specific daily, social or work-related tasks

- A reduced ability to communicate effectively
- Difficulty in singing
- Throat related symptoms (soreness, pain, discomfort, aching, dryness, mucus), particularly related to voice use
- The consequent emotional and psychological effects caused by the above

Causes of Voice problems

Organic	Cause
Inflammatory	Acute laryngitis, Chronic laryngitis
Neoplasia	Papillomatosis, Cancers
Neurological	CVA, Multiple sclerosis, Guillain-Barre, Myasthenia gravis, Carcinoma of the thyroid / lung / mediastinum, Idiopathic (virus), Spasmodic dysphonia, Muscle tension disorder, Injury to the nerve after surgery or trauma
Iatrogenic	Post surgery – thyroid, neck, carotid, larynx, oesophagus
Systemic	Hypothyroidism, Rheumatoid arthritis



Approach

History

- The nature and chronology of the voice problem
- Exacerbating and relieving factors
- Lifestyle, dietary, and hydration issues
- Contributing medical conditions or the effects of their treatment
- The patient's voice uses and requirements
- The impact on their quality of life, social, and psychological well-being
- Their expectations for outcome of the consultation and treatment

Examination

You as a GP

- ENT examination
- Head and Neck examination
- Mirror examination of larynx

As an ENT

- Rigid or flexible scope examinations
- Stroboscopic examination
- High speed photography
- Voice analysis
    - Patient scales
    - Perceptual evaluation
        - Auditory
        - Visual
    - Measurements
        - Acoustic
        - Aerodynamic

If the diagnosis is not clear from the initial assessment, the patient may undergo one of the following options

- Further in-depth assessment by a voice therapist: to ascertain more background information including exploration of contributing psychological issues
- A trial of vocal hygiene / lifestyle advice or medical treatment
- Laryngeal electromyography
- Objective voice measurements
- 24-hour pH monitoring ± impedance testing or oesophagoscopy
- Diagnostic micro laryngoscopy
- Referral to another voice disorders team or professional
- CT / MRI

Treatment

If treatment is required, it will usually consist of one or more of the following options, depending on the patient's symptoms, vocal requirements and clinical findings

- Vocal hygiene (see below), lifestyle and dietary advice
- Voice (speech) therapy
- Specialist therapy, e.g. singing therapy
- Medical treatment - PPI
- Phonosurgery
    - Phonomicrolaryngosopy
    - Injection laryngoplasty
    - Laryngeal framework surgery
    - Recurrent laryngeal nerve reinnervation
    - Laryngeal pacing

Vocal hygiene (Important)

- An **explanation** of how the voice works
- **The links** between lifestyle, phonatory and non-phonatory vocal activities and stress on voice disorders

- The potentially traumatic effects to the vocal folds of **'vocally abusive behaviours'**, such as talking or singing too loudly, talking too fast, shouting, throat clearing and harsh coughing
- Communicating effectively **without raising** or straining the voice, e.g. using a whistle in the school playground or using amplification devices where practical and conserving the voice where possible or in extreme situations discussing the possibility of changing jobs
- The importance of **adequate hydration** for vocal fold function, i.e. by drinking water and use of steam inhalation, and avoiding excessive amounts of drinks containing caffeine, i.e. coffee, tea and colas
- **Smoking cessation, reducing alcohol** and social drug consumption (particularly spirits, cannabis and cocaine) and avoiding exposure to fumes, dust and dry air
- **Diet and reflux reduction**, e.g. avoiding eating late at night, large or fatty meals, sleeping upright

Diseases

The most common voice disorders seen in secondary practice in a voice clinic are:

- Muscle tension disorder (MTD)
- Laryngitis / MTD secondary to poor vocal hygiene, dietary and lifestyle issues
- Extraoesophageal reflux (laryngopharyngeal reflux)
- Vocal fold nodules
- Vocal fold polyps
- Vocal fold cysts
- Vocal fold palsy and paresis
- Arytenoid granulomas

Less frequently seen conditions include:

- Sulci and mucosal bridges
- Spasmodic dysphonia
- Papillomatosis
- Microvascular lesions
- Laryngeal trauma, including post-surgical causes
- Other neuromuscular causes
- Hyperkeratosis, dysplasia and carcinoma
- Endocrine causes
- Amyloid
- Other laryngeal tumours

MTD

- MTD is therefore a group of conditions characterized by **an imbalance** of the synergist and antagonist muscles affecting the vocal fold position and tensioning relative to one another and also the position of the larynx relative to the rest of the vocal tract
- There are multiple primary aetiologies of MTD, including:
    - Stress, anxiety and depression
    - Conversion disorders
    - Postural and breathing problems
    - Poor vocal hygiene
    - Talking in poor acoustic environments or above background noise for prolonged periods at work or socially
    - Exposure to excessive environmental dust, smoke or fumes
- Symptoms include

    - Pitch of the voice may be too high or too low and reduced in range
    - A sensation of tightness, constriction or lump in the throat
    - Effortful voice production
    - Discomfort on speaking or singing
    - Vocal fatigue
- Treatment
    - Vocal hygiene, dietary and lifestyle advice
    - Voice therapy targeted at specific muscle groups
    - Laryngeal manipulation
    - Behavioural therapy
    - Medical treatment, e.g. of extraoesophageal reflux

Other neurological problems

- Neuro-muscular disease such as
    - Parkinson's
    - Motor neuron disease
    - Myasthenia gravis
    - Multiple sclerosis (MS)
- Spasmodic dysphonia (Botox)
    - Adductor type – Strained, straggled voice
    - Abductor type – Weak, breathy voice
    - Mixed type
    - Tremor

Inflammatory disorders - Laryngitis

- Inflammation of the larynx can be broadly classified into infective and non-infective causes
- Classification
    - Acute
        - Simple - Viral / Bacterial
        - Specific - LTB, Croup, Epiglottitis
    - Chronic
        - Physical
        - Chemical / Environmental
    - Atrophic
    - Granulomatous
    - Fungi
- Sometimes the aetiological factors are easily identified in the history (e.g. hoarseness associated with an **upper respiratory tract infection**). In many other cases the **cause may be less clear** (e.g. in cases of extraoesophageal reflux), may be **multi-factorial**, may require empirical **treatment** or a **biopsy** and microbiological **culture** (e.g. in the case of tuberculosis) or may **resolve spontaneously** without a cause being identified.
- Patients often complain of:
    - Hoarseness
    - Huskiness
    - Reduced pitch
    - Loss of part of the range of the voice
    - Pitch instability
    - An increased effort to speak
    - Vocal fatigue and pain or discomfort on speaking

- Throat symptoms, such as globus sensation and irritation, dryness, throat clearing or chronic cough
- Laryngitis is simply a descriptive term indicating a **variable degree of erythema, oedema, epithelial change** which may include **ulceration, leukoplakia and stiffness** of the mucosa of the vocal fold. There is often an increased amount of **thick mucus** present, which may be white, grey, yellow or green in colour. There may be **associated inflammation** of the rest of the subglottic, supraglottic and interarytenoid areas.

Vocal fold nodules / Polyps / Cysts

- Nodules
    - Less than 3 mm and mostly bilateral
    - Professional voice users
        - Teachers / Singers / Drill sergeants
    - Treatment consists of voice therapy, vocal hygiene, reflux management and rarely surgery
- Polyps
    - More than 3 mm and usually unilateral
    - May resolve spontaneously
    - Otherwise, voice therapy or removal under surgery
- Cysts
    - May be removed under surgery if not resolving

Reincke's oedema

- Condition where the vocal cords become chronically and irreversibly swollen
- See almost exclusively in smokers
- Complains of
    - Deepening of the pitch of the voice with **women often being mistaken for a man**, particularly on the telephone
    - **Gruffness** of the voice
    - Effortful speaking
    - An inability to raise the pitch of the voice
    - Choking episodes
    - Other symptoms associated with extraoesophageal reflux
- Treatment consists of
    - Vocal hygiene
    - Voice therapy
    - Smoking cessation
    - Surgery

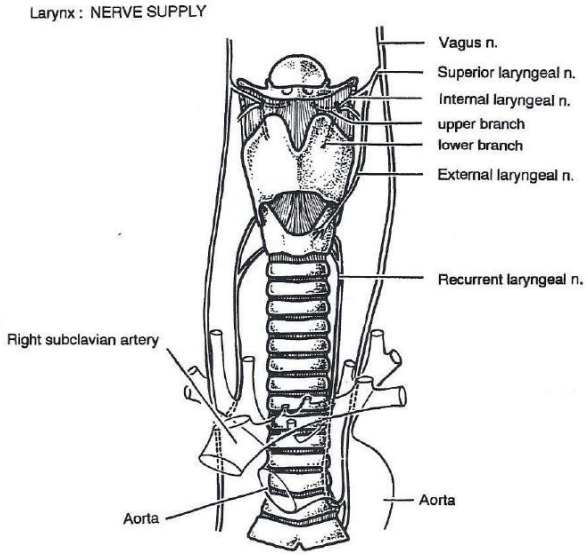
Arytenoid granuloma

- Other terms for them include:
    - Contact ulcer or granuloma / Vocal process granuloma / Intubation granuloma / Contact pachydermia / Peptic granuloma
- These consist of a proliferation of granulation tissue with epithelial hyperplasia. They **result from injury** to the thin mucoperichondrium over the vocal processes from mechanical trauma, either **following intubation** or from repeated high-velocity impact of the vocal processes against each other from **throat clearing, coughing or talking in a habitually low-pitched, creaky, hyperfunctional manner**.
- Men tend to develop granulomas secondary to hyperfunction, while women develop them more commonly as a result of intubation.

- In addition, extraoesophageal reflux is recognized as an important aetiological factor either contributing to the symptoms leading to the mechanical trauma or preventing healing of the damaged mucosa.
- The main treatment principles include reducing the effects of laryngeal irritants, i.e. stopping smoking, improving vocal hygiene, treating any respiratory tract infections, allergies and extraoesophageal reflux
- Voice therapy
- Surgery has a limited role, as does steroids and anti-biotics
- Botox shows promise (injected into the thyro-arytenoid muscle)

Vocal cord paresis / paralysis

Note the anatomical difference between right and left recurrent laryngeal nerves. Vocal cord palsy is more common on the left due to the long intra thoracic course of the left recurrent laryngeal nerve. A vocal cord palsy may result from supranuclear or infranuclear lesions. Infranuclear lesions may involve the trunk of the vagus nerve, the recurrent laryngeal nerves, or the external branch of the superior laryngeal nerves.



Aetiology

- Congenital
- Acquired
    - Vascular
    - Viral
    - Bacterial
    - Neuro-toxic
    - Tumour
    - Trauma
    - Iatrogenic

Pathogenesis

- Thermal
- Stretching
- Direct injury
    - Cut
- Compression
- Vascular

Differential diagnosis

- Classical
    - 1/3 Tumour related
    - 1/3 Post surgery
    - 1/3 Idiopathic
- Detailed
    - Surgery – skull base, thyroid, carotid, neck
    - Trauma – neck, thorax, larynx, CVS
    - Tumour – larynx, thyroid, lung, oesophagus, skull base, mediastinum
    - Mono- and multi-neuropathies – HIV, EBV, CMV, HS, HZ, Drugs, DM
    - CNS – Arnold-Chiari, MS, Myasthenia Gravis, Hydrocephalus, Kernicterus
    - CVS – Cardiomyopathy, Aortic aneurysm
    - Mediastinum / Lung – Mediastinal lymph nodes, Bronchial cancer, Oesophageal cancer

Thyroid surgery

- Hoarseness post thyroid surgery can be due to
    - Recurrent laryngeal nerve injury (RLN) or,
    - External branch of the superior laryngeal nerve (EBSLN) injury
- RLN injury
    - Injury can result in hoarseness of voice (of variable degrees), coughing, micro-aspiration, and several other symptoms that can affect patients' quality of life
    - These changes are especially noticeable in professional speakers and singers; however, all affected patients are susceptible to suffer from voice changes and impaired communication, which can significantly reduce quality of life
    - Rarely an acute bilateral injury will result in severe stridor “on the table” post extubating
    - The incidence of permanent post-operative RLN paralysis is 0.3–3 % and is as high as 2–30 % in revision thyroid surgery
    - Transient paresis occurs in 5-8% of primary thyroid surgeries
        - Transient injuries have different recovery times
        - Usually, it recovers between 4 and 6 weeks but can take up to 12 months
        - Injuries lasting more than 1 year are considered permanent
- EBSLN
    - The reported incidence of EBSLN injury during thyroidectomy varies widely from 0% to 58%, due to the difficulty of assessment
    - Injury to EBSLN increases the risk of aspiration and affects CTM motility, altering the voice quality mainly due to the inability of producing high-pitched sounds and produces a monotonous low tone voice

Symptoms depend on

- Whether the injury is unilateral or bilateral
- Position of vocal cord
    - Median, paramedian, lateral
- If the injury is
    - Temporary vs Permanent
        - Can take up to 12-18 months to recover

Differentiation and treatment

	Lateral	**Median**
Voice	Weak, Hoarseness Reduced number of words per breath	Good Strained
Aspiration	Huge problem, even more so with fluids	Usually not a problem

Airway	Good	Problematic. Can have airway compromise with stridor and reduced exercise capacity
Misdiagnoses	Laryngitis	Asthma type picture
Treatment	Medialise the vocal cord	Create an alternative airway of lateralise the vocal cord
Treatment options	Temporarily – inject vocal with either fat or synthetic material Permanent – External thyroplasty	Cordotomy Lateralisation of vocal cord Tracheostomy

Hoarseness and Vocal Cord paralysis in the Child

In general, it is better to refer these patients to an ENT specialist. Also see the chapter "Congenital conditions in the Head and Neck".

Differential diagnosis

- Congenital vocal cord paralysis
    - Second most common congenital condition of the larynx after laryngomalacia
    - Rarely in isolation and is seen with
        - CNS problems
            - Arnold Chiari malformations
            - Hydrocephalus
        - CVS problems
        - Pulmonary problems
        - Other laryngeal abnormalities
- Congenital laryngeal and tracheal deformities such as
    - Webs
    - Cysts
    - Clefts
    - Stenosis
    - Haemangioma
- Habitual
    - More in boys
        - Due to "screaming" at each other

Swallowing

Physiology

Deglutition is a brainstem reflex controlled by a centre in the medulla. The reflex occurs in three stages, only the first of which is voluntary.

- **Oral stage**
    - The bolus is propelled back into the oropharynx by the contraction of mylohyoid which raises the floor of the mouth
- **Pharyngeal stage**
    - On reaching the posterior pharyngeal wall, the bolus triggers off
        - Closure of the postnasal space by the soft palate
        - Closure of the oral cavity by the faucial pillars and tongue
        - Closure of the laryngeal inlet and cessation of respiration

- **Oesophageal stage**
    - This consists of consecutive pressure changes in three zones
        - Relaxation of the pharyngo-oesophageal junction
        - Generation of a peristaltic wave of contraction down the oesophagus.
        - Relaxation of the oesophago-gastric junction

Definitions

- Dysphagia – difficulty in swallowing
- Odynophagia – painful swallowing
- Odynodysphagia – difficult and painful swallowing
- Aspiration – entrance of any material past the true vocal cords

Background

Dysphagia

- Dysphagia is common: 22% of individuals over 50 are affected
- Aspiration pneumonia is the highest cause of mortality associated with hospital admission
- Stroke is the commonest neurological cause of dysphagia and aspiration
- 76% of HNC patients have been shown to aspirate. This is higher than those with medical, neurological or GI problems
- Dysphagia is a term used to describe difficulty with swallowing. It implies impairment of one or more of the phases of swallowing namely
    - Oral, Pharyngeal, and / or Oesophageal
- It can be divided into oropharyngeal (high) dysphagia and oesophageal (low) dysphagia
- Dysphagia usually arises as a complication of another health condition
- Also classified according to food type
    - Dysphagia towards solids
    - Dysphagia towards liquids
    - Dysphagia to both

Aspiration

- Aspiration is the entry of food or liquid into the airway below the true vocal folds
- It may be due to incompetent or inadequate airway protection, ill-timed, uncoordinated events before, during or after the swallow has triggered
- Silent aspiration is defined as foreign material entering the trachea or lungs without an outward sign of coughing or attempts at expulsion

Causes of Dysphagia and Aspiration

Congenital	Neurological	Infective	Inflammatory	Auto-immune	Trauma	Neoplastic	Motility	Miscellaneous
Cleft lip or palate Cerebral palsy Vascular rings Atresia / Clefts / Fistulas Vocal cord palsies	CVA Parkinson's Multiple sclerosis Motor neuron disease Myasthenia Gravis Vocal cord palsy	Infection of oral cavity, pharynx, larynx, and neck	GERD / LPR Pattersen-Brown-Kelly Sd Eosinophilic oesophagitis	Scleroderma SLE Sjogren's syndrome	Foreign body Food bolus Bruns Caustic ingestion	Benign tumours Malignant tumours	Achalasia Oesophageal spasm Presby-oesophagus	Post HN cancer treatment Medications Globus pharyngeus Pharyngeal pouch

Approach

Patients' complaints may include

- ***Dysphagia***
- ***Aspiration***
- ***Regurgitation***, which can be immediate or delayed, can give an indication as to the level of the problem. Delayed regurgitation of undigested food is typically seen in patients with a pharyngeal pouch
- Symptoms of ***retrosternal discomfort, belching*** and early ***satiety*** indicate gastro-oesophageal reflux disease (GORD)
- ***Odynophagia*** (pain on swallowing), is associated with infection, neoplasia or GORD
- ***Hoarseness*** may indicate laryngeal fixation due to tumour or vocal cord palsy
- ***Choking or coughing***, during or after eating, or frequent chest infections may suggest aspiration
- ***Referred otalgia*** via the IX and X cranial nerves is usually secondary to a head and neck tumour and a poor prognostic sign
- ***Associated neurological symptoms*** such as bulbar dysfunction, dysarthria, diplopia, limb weakness and fatigability can be seen in motor neuron disease and myasthenia gravis. Tremor, ataxia and unsteady gait are features of Parkinson's disease

Examination

- Examination should exclude any obvious **structural cause**, and assessment should be made for signs of **associated systemic or neurological** dysfunction and for **signs of complications** of dysphagia such as weight loss and malnutrition and pulmonary problems due to aspiration
- Lips and Oral Mucosa
    - The lips themselves may be pale suggesting an anaemia due to iron, folate or vitamin B12 deficiency.
    - It is usually easy to diagnose candidal fungal infections by their covering whitish membrane which when removed will reveal a raw area
    - Carious teeth with chronic infection can predispose towards recurrent pharyngitis
- Tongue and Sulci
    - Carcinoma in the tongue is, unfortunately, often of considerable size before being clinically detected
    - Origin is frequently in silent areas such as the bucco-alveolar and the alveolar-glossal sulci
    - Any suspicious areas should be biopsied, and particular attention should be paid to hyperkeratotic white plaques (leukoplakia) or red plaques (erythroplakia) which often precede a carcinoma
- Oropharynx
    - Recurrent pharyngitis is probably the commonest cause of difficulty in eating
    - In the majority of patients, it is of an episodic nature but in some the symptoms can be chronic
    - In the majority, viral infections are responsible and as viruses affect cell types rather than specific areas the whole of the mucosa of the pharynx is usually involved
    - During an acute attack of pharyngitis, the clinical signs are often minimal
    - In some there will be a slight increase in redness and in the gag reflex but in others the pharynx may appear entirely normal
    - When the symptoms are acute, discomfort is fairly predominant
    - In the chronic situation, this is less so and difficulty in getting food beyond the oropharynx is the major complaint

    - The tonsils (although often appearing to), seldom occlude the oropharynx and correspondingly rarely cause dysphagia in adults. They will, however, cause dysphagia if they become inflamed
    - Adenotonsillar hypertrophy can be a cause for dysphagia in children. They typically develop picky eating patterns and prefer soft foods. They will chew "meat" and spit it out
    - Another population where adenotonsillar hypertrophy can cause dysphagia is in the HIV positive group
    - In the oropharynx the tonsils are the most frequent site for carcinoma.
        - As only one tonsil is usually involved, any degree of asymmetry on tonsil size in someone who complains of something there, or food sticking is an indication for biopsy
- Hypopharynx
    - There are three relatively common pathologies that affect the hypopharynx
        - Pharyngeal Pouch
            - Pharyngeal pouches are relatively rare and are thought to be due to secondary swallow in a megapharynx along with a congenital weakness in the pharyngeal muscle layers through which the mucosa herniates
            - The hernia gradually extends into the neck and causes a relative obstruction because of external pressure on the pharyngeal wall from food and retained debris within the pouch
            - As the inlet to the pouch cannot usually be seen either by direct or indirect examination the diagnosis rests on radiological visualization of swallowed barium within the pouch
            - Pouches are usually surgically excised via the neck
        - Muscular incoordination
            - Muscular incoordination is an increasingly recognized entity and can be simply local muscular incoordination or part of generalized neurological disease
            - When part of general disease, such as motor neuron disease or pseudobulbar palsy, there is often overflow into the laryngeal inlet during eating with coughing and aspiration
            - Local muscular incoordination is akin to inability to squeeze a tube of toothpaste consistently along its length to produce a flow
            - Muscular incoordination is diagnosed by seeing an abnormal peristaltic pattern on a video tape recording of a barium swallow, and is difficult to manage, there being no specific therapy
        - Generalized neurological disease such as motor neuron disease or pseudobulbar palsy is usually much more severe and usually fatal.
            - Indirect laryngoscopy often reveals a lax, immobile pharynx with pooling of saliva in the hypopharynx
            - Overflow into the larynx and lungs readily occurs with coughing and aspiration pneumonia
            - Management is extremely disappointing, myotomy and feeding gastrostomy being palliative rather than curative procedures
- **The lower cranial nerves** are assessed for loss of tongue movement, wasting and fasciculation, loss of gag and cough reflexes, loss of pharyngeal and laryngeal sensation and loss of vocal cord mobility
- **The neck** is examined for lymphadenopathy and other neck masses, thyroid enlargement, loss of laryngeal crepitus and the integrity of the laryngeal cartilages
- **General physical and neurology examinations** should look for evidence of malnutrition, weight loss, chest disease, epigastric tenderness and abdominal swellings, loss of coordination, fasciculation, and tremor

Special tests

- Bedside
    - Water swallow test
    - Blue dye test
- Visual documentation
    - Endoscopy / Functional endoscopic evaluation of swallowing (FEES)
    - Video fluoroscopy
- Gastroscopy
- Barium / Gastrografin swallow
- MRI
- Manometry and pH-study

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26) Salivary glands

Physiology

Salivary glands secrete saliva of which the functions are:

- Lubrication
- Digestion
- Antibacterial
- Antiviral
- Act as a buffer
- Teeth protection
- Tissue coating
- Mineralisation

Salivary glands are divided into major and minor glands. Major salivary glands in the Head and Neck are the parotid, submandibular and sublingual glands, found bilaterally. Minor salivary glands are scattered through the mucosal lining of the upper airways and is most commonly found on the hard and soft palate, followed by the buccal mucosa, retro-molar trigone, and lips. The parotid glands are serous, submandibular gland mixed, and sublingual and minor salivary glands mucous in nature respectively. The parotid produces roughly 0.7 ml/min/gland of saliva and the submandibular 0.6 ml/min/gland respectively.

Various diseases affect the salivary glands and only some of the more common diseases are discussed.

Salivary gland pathologies

Inflammatory	Acute	Bacterial	Sialadenitis	Common
				Neonatal
				Recurrent parotitis of childhood
			Abscess	
			Lymphadenitis	
		Viral	Mumps	
			HIV	
			Other	
	Chronic	Obstructive	Sialectasis	
			Sialolithiasis	
			Mucocele or cyst	Acquired
				Retention cyst
		Granulomatous	TB / NTB	
			Actinomycosis	
			Cat-scratch	
			Sarcoid	
			Toxoplasmosis	
			Fungi	
		Necrotising sialometaplasia		
Neoplastic	Tissue type	Epithelial (majority)	Benign	Pleomorphic adenoma
				Warthin's tumour
				Others
			Malignant	SCC
				Adenoid cystic

				Mucoepidermoid carcinoma
				Acinic cell carcinoma
				Adenocarcinoma
				Salivary ductal carcinoma
				Others
		Mesenchymal (more common in children)	Benign	Myomas
			Malignant	Sarcomas
		Haematolymphoid (more common in HIV positive)	Lymphoma	
			Lymphangioma	
			Vascular malformations	
Auto-immune	Sjogren's disease			
	Benign lymphoepithelial cysts			
Trauma	Penetrating			
	Blunt			
	Radiation			
Congenital	Branchial cleft and pouch	First	Work's type I and II	
		Second	In tail	
	Dermoid cyst			
	Ductal cyst			
	Agenesis			
Pseudohypertrophy	Masseter hypertrophy			
	Old age			
	Mass in parapharyngeal space			
	Teeth pathology			
Metabolic diseases	Obesity			
	Diabetes			
	Thyroid			
	Cushing's disease			
	Bulimia			
	Uraemia			
Drugs	Isoprenaline			
	Thio-uracil			
Allergies				

Acute sialadenitis

Acute bacterial sialadenitis

Acute bacterial sialadenitis occurs most commonly in the parotid gland. It typically occurs in the setting of elderly, sick patients, post-operative, and dehydration. It is mostly an ascending bacterial infection due to *Staph Aureus, Streptococcus, Haemophiles Influenza, Pepto streptococci, Bacteroides,* and *Fusobacterium species*. It is acutely painful and even more so when eating. The parotid gland is enlarged with skin signs of inflammation such as erythema and warm to touch. Bimanual examination reveals puss draining from Stenson's duct in up to 75% of patients. Bilateral involvement occurs in 25%. Usually and ultrasound is needed to confirm or exclude abscess formation. Treatment consists of pain relief, rehydration, antibiotics, local applications of heat, massage, aspiration, and draining of an abscess if present.

Acute viral sialadenitis

Mumps

Mumps is a n acute non-suppurative parotitis caused by *paramyxovirus and other viruses*. More than 85% are younger than 15 years of age. There is no natural reservoir, and infection spreads with new cases. Incubation period is 2-3 weeks which allows for this, and the infection period is considered to be 2 days before to 5 days after the onset of parotitis.

There is typically a viral prodrome which is followed by parotitis, otalgia, dysphagia, and rarely trismus. The parotitis starts unilaterally but will affect the other parotid gland in 75% of causes within 5 days. The swelling usually peaks at day 1 – 3. The diagnosis can be made with a PCR on fluids or IgM on serum. Treatment consists of rest, oral hygiene, and rehydration. Complications, although rare, can include deafness, orchitis, meningitis, pancreatitis, and nephritis.

There is a vaccine for mumps, and the disease was basically eradicated in the USA. However, since 2006 there has been a steady decrease in vaccine uptake with a coinciding rinse in the number of mumps cases. A useful link from the CDC is: Mumps.

Chronic sialadenitis

Chronic sialadenitis follows an inciting incident, usually after acute sialadenitis, and leads to sialolithiasis and sialectasis. It occurs most commonly in the parotid glands and presents with episodic pain usually with eating and inflammation. Upon examination little saliva can be milked from the gland. If conservative measures fail, surgery is indicated to remove the gland. Complication of chronic sialadenitis includes lymphoepithelial lesions (formally Mikulicz disease), Kuttner's tumours, and duct carcinoma.

Sialolithiasis

Stones are far more common in the submandibular glands compared to the others. The reasons include:

- High mucin content
- Alkaline pH
- Higher concentration of calcium and phosphate
- Long length of duct
- Dependant position of duct and that it needs to pump against gravitation

Once stones start forming, it leads to duct destruction (sialectasis), which in itself leads to a higher incidence of sialadenitis. This leads to a self-perpetuating cycle of repeated stones and infection, and then chronic sialadenitis.

Stones can be managed with:

- Non-surgical interventions
    - Sialagogues
    - Heat applications
    - Massaging of the gland
    - Rehydration
    - Antibiotics
- Surgery
    - Removal of stone
    - Removal of gland
    - Lithotripsy
    - Sialoendoscopy

HIV associated salivary gland diseases

Parotid pathology is common in the HIV positive patient. The most common diseases include:

- Bilateral enlargement especially early in the disease
- Xerostomia
- Benign lymphoepithelial cysts
- Lymphomas
- Kaposi sarcomas

Benign lymphoepithelial cyst (BLEC) associated with HIV can be difficult to distinguish from other cystic lesions of which some will be neoplasms. In general, HIV associated BLEC will be bilateral, multiple, and show response on ARV medications. BLEC can also be manage with sclerotherapy, such as 90% alcohol.

Salivary gland tumours

Major salivary gland tumours are rare and account for 3-10% of head and neck tumours. Minor salivary gland tumours (MSGT) are even more uncommon and only comprise 9-23% of all salivary gland tumours. According to Globocan 2020 data, which reports on estimates of cancer incidence and mortality produced by the International Agency for Research on Cancer, salivary glands tumours were responsible for 55 583 out of 19.3 million cancers (0.3%) (Global Cancer Observatory). This places them at 28th out of 36 tumours included in the report. Put into perspective, the incidence is very similar to anal carcinomas, but vastly smaller than female breast and lung carcinomas which have more than 2.2 million cases per year respectively. According to Stats South Africa 135 males and 117 females had salivary gland tumours in 2019.

Broadly speaking, salivary gland tumours are divided into epithelial, mesenchymal, and haematolymphoid tumours. The most common being epithelial tumours (>80%). Currently the 5th edition of the World Health Organization classification of salivary gland tumours is used. It includes newly established pathological entities due to the diverse nature of salivary gland tumours. Furthermore, it states that molecular testing of salivary gland tumours for differential diagnostic accuracy and appropriate clinical management is becoming routine.

Classic teaching stipulates that the chance of a salivary gland tumour being malignant increases from major to minor salivary glands. Dictums such as 80% of salivary gland tumours occur in the parotid glands, 80% of parotid gland tumours are benign, and 80% of benign parotid gland tumours are pleomorphic adenomas are commonly taught. The inverse is taught with regards to MSGT, with malignancy rates varying from 50-80%. Two leading textbooks report the malignant incidence as follows:

Table 1. Incidence of salivary gland tumours being malignant.

	Scott Brown*	**Cummings****
Gland	**Malignant**	**Malignant**
Parotid	15-32%	25%
Submandibular	41-45%	43%
Sublingual	70-90%	
Minor salivary glands	80%	85%

* van der Poorten V, Bradley P. Scott-Brown's Otorhinolaryngology and Head and Neck Surgery. Eight. Vol. 3. Boca Raton; 2018.

** Flint PW, Francis HW, Haughey BH, Lesperance MM, Lund VJ, Robbins KT, et al., editors. Cummings otolaryngology: head and neck surgery. Seventh edition. Philadelphia, PA: Elsevier; 2021

However, our data and published articles from South Africa and other countries does not support above mentioned dictums. In general, it clearly confirms that in countries with increased sun exposure, the most common malignant parotid tumours are metastatic squamous cell carcinomas from skin primaries. Furthermore, it is interesting to note that the studies from Cape Town and Australia both had a very high prevalence of metastatic melanomas. Another difference is the incidence of malignancy which were 8.95% in the Nottingham study from the UK (1), 27% in the Cape Town study (2), 28.73% in our study, and a staggering 43% in the Australian study (3). Other important findings in the Australian study were increasing age ($p < .001$), fewer parotidectomies for inflammatory lesions ($p < .001$), reduced incidence of mucoepidermoid carcinoma ($p = .048$), increasing incidence of parotidectomy for cutaneous malignancies ($p < .001$), and reduced facial nerve sacrifice ($p = .034$). Over the four studies, the prevalence of mucoepidermoid carcinomas decreased from 28.57% in the Nottingham study, to 16.85% in Cape Town, 13.46% for our study, and 4.54% in the Australian study. A possible explanation for the reduced incidence of mucoepidermoid carcinomas is improved immunohistochemistry and that a sizeable portion of these were reclassified as salivary duct carcinoma or poorly differentiated metastatic squamous cell carcinoma. However, once skin metastases such as squamous cell carcinomas and melanomas are excluded, it was the most common primary epithelial malignancy in all four studies. Our study also has a more than double the prevalence of carcinoma ex pleomorphic adenomas (11.54%) compared to the next highest of 5.19% (Nottingham study).

With regards to MSGT we did not see the high malignant incidence that is frequently reported in the literature. Benign and malignant minor salivary gland tumours' incidence was almost equal at 46.46% and 53.54% respectively. Pleomorphic adenomas remain the most common minor salivary gland tumour (38.38% of all tumours and 82.61% of benign MSGT) and occurs most commonly in the palate. In general, most minor salivary gland tumours and benign salivary gland tumours occurred in the palate. We did show an unusually high incidence of polymorphous (low-grade) adenocarcinoma which was also the most common malignant minor salivary gland tumour. Interestingly, this was reported in another study from South Africa. Primary sites other than palate and lips should raise the suspicion of malignant minor salivary gland tumour.

In general, the following symptoms and signs will point to a malignant tumour:

- Fast growth
- Pain
- Nerve involvement
- Lymphadenopathy
- Infiltration of skin, muscles, bone

It is best to refer patients to an ENT specialist if you suspect the patient having a salivary gland neoplasia.

A retrospective descriptive study by us, and all pooled data from South Africa showed the following. Our study showed that 55.25% of all tumours occurred in the parotid gland, 14.19% in the submandibular gland, and 30.56% in the minor salivary glands. The incidence of benign tumours was 71.02% in the parotid, 78.95% in the submandibular gland, and 46.46% in minor salivary gland. Pleomorphic adenomas were the most common tumour, accounting for 53.60%, 78.95%, and 38.38% of all parotids, submandibular, and minor salivary gland tumours respectively. All tumours occurred more commonly in females and in general, benign tumours occurred statistically significantly earlier compared to malignant tumours. Warthin's tumour was the second most common parotid tumour (18.75%) followed by squamous cell carcinoma (5.68%). Our series demonstrated a high incidence of polymorphous adenocarcinomas (15.15%) in minor salivary gland tumours followed by acinic cell carcinomas (14.14%)

Our results combined with published studies from South Africa showed that 68.49% of all salivary gland tumours are benign and 31.51% malignant. The distribution between salivary glands showed that 58.49% occurred in the parotid gland, 19.95% in the submandibular gland and 21.56% in minor salivary glands. In parotid gland tumours (n=583) 71.18% were benign and 28.82% malignant, for submandibular glands (n=125) 79.2% were benign and 20.8% malignant, and minor salivary glands (n=1037) 62.2% were benign and 38.8% malignant. In this study, no cases of sublingual gland tumours were found.

Among parotid gland tumours, 50.43% were pleomorphic adenomas, 8.92% Warthin's tumours, 5.66% mucoepidermoid carcinomas, 4.29% squamous cell carcinomas, and 3.60% acinic cell carcinomas. Seventy-five percent of all submandibular tumours were pleomorphic adenomas. Of the minor salivary gland tumours, 48.31% where pleomorphic adenomas, 11.09% adenoid cystic carcinomas, 8.29% mucoepidermoid carcinomas, and 7.52% polymorphous adenocarcinomas.

It is clear from the data that salivary gland tumours (SGT) in SA does not follow the 80% rule especially with regards to the incidence of pleomorphic adenomas in parotid glands (only 50% in SA) and the incidence of malignant MSGT (only 40% in SA). Consolidating all the data from SA, it showed that 70% of all SGT are benign and 30% malignant. 60% of tumours occurred in the parotid gland, 20% in the SMG and 20% in MSG. The proportion of benign versus malignant for parotid glands were 70% versus 30%, for SMG 80% versus 20%, and for MSG 60% versus 40% respectively.

Special investigations

Various special investigations can be conducted, of which the most important are:

- Radiology
    - Sialography
    - CT
    - MRI (Preferred to CT for imaging the salivary glands)
    - Ultrasound
- Histology / Cytology
    - FNAC
    - Core needle biopsies
- Sialoendoscopy

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