CLINIC OF OTO-RHINO-LARYNGOLOGY AND HEAD & NECK SURGERY
MEDICAL AND HEALTH SCIENCE CENTER
UNIVERSITY OF DEBRECEN

ISTVÁN SZIKLAI

OTO-RHINO-LARYNGOLOGY

DU press

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Lecture notes

Reprint of edition of 2010

Debreceni Egyetemi Kiadó
Debrecen University Press
2011

ISBN 978 963 318 001 3

Kiadta a Debreceni Egyetemi Kiadó Debrecen University Press
Felelős kiadó: Dr. Virágos Márta
Készült a DE sokszorosítóüzemében, 2011-ben
11-347

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Preface

This book with the lectures and practice talks is the basics for the final examination in ENT. The activity area of ENT practice, however, is greater than this. New advances in endocrine surgery, skull base and head and neck surgery also pertain to our field, although these are out of the scope of these notes, which is attempted to be as brief as possible, and consequently, only the most important entities, diagnostic procedures and therapeutic maneouvers are collected. These notes, in order, aspire only for the title of introduction to Oto-Rhino-Laryngology.

A pleasant obligation is to call the reader's attention to several Hungarian ENT practitioners and scientists who's contribution to Oto-Rhino-Laryngology were determining. Professor János Czermák (natively Chech) was the head of the Department. of Physiology at the Semmelweis University Medical School in Budapest when enjoyed great reputation for indirect laryngoscopic investigations, in the late XIX. Century. Another great name is Adam Politzer, natively Hungarian, the member of the School in Vienna who is regarded to be the father of otology.

Interestingly, Nobel Prize winners in the ENT field, were only Hungarians, although non of them worked at the time of the nomination in Hungary. Professor Róbert Bárány received the Nobel Prize (1914) for research on the physiology of the peripheral vestibular apparatus. György Békésy discovered the traveling wave as the elementary process in the presentation of the acoustic signals in the inner ear and won the Nobel Prize in 1961.

1. Ear

Even organ. Includes the hearing and vestibular end-organs **(Fig. 1.)**. Behaves like a paranasal cavity since through the Eustachian tube the middle ear functions as an extention of the nasal cavity proper. Behind its connections to the nasopharynx through the Eustachian tube, another communication exists through which the middle ear cavity communicates with the aereted cells of the mastoid process: this is the antrum.

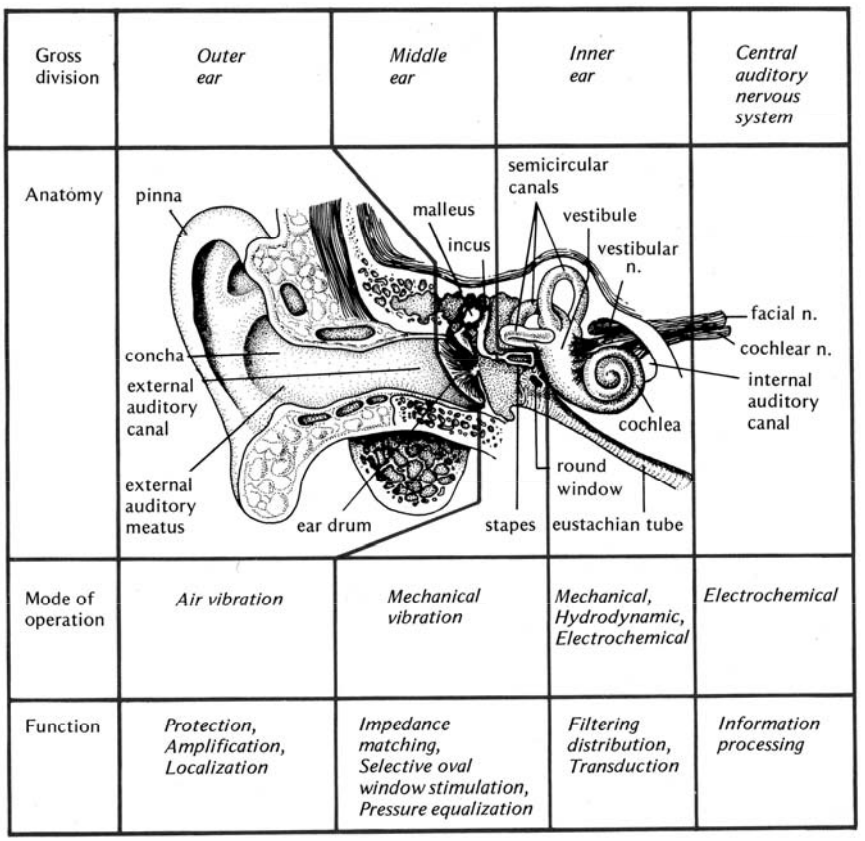


Figure 1. Cross section of human ear, showing divisions into outer, middle and inner ears. Below are listed the predominant mode of operation of each division and its suggested function (adapted from Dallos, 1973).

1.1. Anatomy

1.1.1.Anatomy of the external ear.

Consists of two parts: 1. The auricle (pinna) - that has a framework of cartilage. The skin is closely adherent to the perichondrium. 2. External auditory meatus. This is about 2.5 cm in length from the concha to the tympanic membrane. That is subdivided into:
1. cartilaginous portion; the cartilage itself is deficient superiorly, and this deficiency is continuous with the space between the tragus and the crus of the helix. The skin produces wax from pilosebaceous and ceruminous glands which binds with desquamated keratin.
2. bony portion; the inner two-thirds of the meatus. Lined by thin hairless and gland-free skin, that is closely adherent to the sutures between the tympanic membrane and the squama.

Vascular supply of the external ear: 1. Auriculotemporal branch of superficial temporal artery, anteriorly; 2. Branches of postauricular division of external carotid artery, posteriorly.

Lymphatic drainage: 1. parotid lymph nodes, in front; 2. Posterior lymph nodes, behind; 3. external jugular lymph nodes, below.

Sensory nerve supply: 1. Posteriorly the concha is innervated by the great auricular nerve (from Cervical 2, 3) and by the lesser occipital nerve (Cervical 2).
2. Anteriorly the lower one third is innervated by the great auricular nerve and by the auriculotemporal nerve (from Vth cranial) while the skin of the auditory meatus receives fibres from the Xth cranial nerve. The cavum conchae is innervated by the facial nerve and the vagus in different proportions, the significance of which appears in the herpes zooster of the ggl. geniculi of the VIIth nerve when the herpetic eruptions develop in the innervated area.

1.1.2. Anatomy of the middle ear

1. **Eustachian (pharyngotympanic) tube**. In the adult this 3.7 cm tube runs laterally and posterosuperiorly, to open in the anterior wall of the tympanic cavity. The lateral third is bony, while the lower superior and medial parts are cartilaginous and the remainder is membranous. The tube is closed at rest but is

opened on yawning or swallowing by the combined actions of the sphincter of the nasopharyngeal isthmus and the tensor palati and salpingopharyngeal muscles, which are attached to the cartilaginous medial wall of the tube. The tube is more horizontal and relatively wider and shorter in the infant than in the adult.

2. **Tympanic cavity (Middle ear cavity)**

This biconcave disc-shaped cavity measures 13 mm antero-posteriorly, 15 mm in heights and 2 mm at the narrowest point in the center. The lateral wall is formed mainly by the tympanic membrane. The cavity is artificially divided into 3 parts:

Mesotympanum, lying medial to the membrane

Epitympanum (attic), lying medial to the bone of the horizontal part of the squama (outer attic wall) above the membrane

Hypotympanum, below the drumhead, medial to the tympanic plate

The **tympanic membrane** consists of 3 layers:

- An outer epithelial layer, continuous with the epithelium of the external auditory canal
- A middle fibrous layer (lamina propria), containing inner circular and outer radial fibres, and the handle of the malleus
- An inner mucosal layer

The pars tensa is thickened peripherally into a fibrocartilaginous annulus, which fits into the groove of the tympanic sulcus of the temporal bone. The fibrous layer is absent above the malleolar folds and this part of the tympanic membrane is called pars flaccida (Shrapnell's membrane).

The medial wall has several features:

1. the promontory (the basal coil of the cochlea)
2. the fenestra ovale is occupied by the stapedial footplate
3. facial nerve canal
4. horizontal semicircular canal
5. fenestra rotunda

The anterior wall has 4 openings:

1. Canal of Huguier, through which the chorda tympani escapes from the middle ear
2. canal for tensor tympani muscles
3. tympanic orifice of the Eustachian tube
4. petrotympanic suture, containing tympanic artery

The posterior wall presents an opening (aditus ad antrum) which leads backwards from the epitympanum to the mastoid antrum. Below this is the

pyramid, through which the tendon of the stapedius muscle passes and is inserted into the neck of the stapes.

The floor of the middle ear cavity is separated by a thin plate of bone from the jugular bulb. A dehiscence may be present.

Roof (tegmen tympani) separates the cavity from the middle cranial fossa and it is continuous with the tegmen antri.

Ossicles (**malleus, incus, stapes**) transmit sound from the tympanic membrane to the oval window, and hence to the cochlear fluids.

3. Aditus ad antrum

Leads posteriorly from the epitympanum to the mastoid antrum. The bony prominence of the *horizontal semicircular canal* lies between its medial wall and floor. The tip of the short process of the incus has a ligamentous attachment to its floor.

4. Mastoid antrum

Situated in the posterior portion of the petrous bone. Its anterior wall receives the opening of aditus. Deep to the medial wall lie the posterior and horizontal semicircular canals. The roof separates it from the middle cranial fossa. Postero-inferiorly it communicates by several openings with the mastoid air cells. Although the mastoid air cells can vary considerably in size, number and distribution, the antrum is always present.

Vascular supply of middle ear

1. Superior petrosal and superior tympanic arteries (branches of the middle meningeal artery) to the superior region.
2. Anterior tympanic artery (branch of the internal maxillary artery) to the anterior region (less significant is the ramus tympanici from the internal carotid artery)
3. Inferior tympanic artery (from ascending pharyngeal artery) to the inferior region
4. Posterior tympanic artery (from the mastoid branch of the stylomastoid artery, that derives from the postauricular artery)

Nerve supply of middle ear

1. Sensory: IXth cranial nerve through the tympanic plexus which receives a twig from the VII. nerve.
2. Motor: mandibular branch of the Vth nerve to the tensor tympani muscle
stapedial branch of the VIIth nerve to the stapedius muscle

1.1.3. Inner ear

It is called the labyrinth and consists of the *osseus* and *membranous labyrinth*. The osseus labyrinth's main parts are the vestibule, the bony semicircular canals and the cochlea. The osseus labyrinth contains perilymph in which the membranous labyrinth is situated. The composition of the perilymph is the same like of any other extracellular fluids.

The membranous labyrinth consists of the saccule and utricle, the membranous semicircular ducts and the cochlear duct (scala media). It contains endolymph that is a unique extracellular fluid in the body resembling rather more to the intracellular fluids in its ionic composition (high potassium, low sodium concentration). The saccule and the utricle contain maculae, the sensory end-organs of linear acceleration. The semicircular canals contain cristae ampullaris that are the sensors of angular acceleration.

The ductus cochlearis (**Fig. 2)** is bordered by the Reissner's membrane, the stria vascularis and by the osseus spiral lamina and basilar membrane unit, and ends blindly at the helicotrema. This contains the hearing end-organ, the *organ of Corti*. The scala vestibuli and the scala tympani lie above and below the cochlear duct (scala media). They communicate with each other at the helicotrema in the apex of the cochlea. The scala vestibuli communicates functionally with the middle ear through the oval window, the scala tympani through the round window. The scala tympani is connected with the subarachnoid space through the bony cochlear aqueduct.

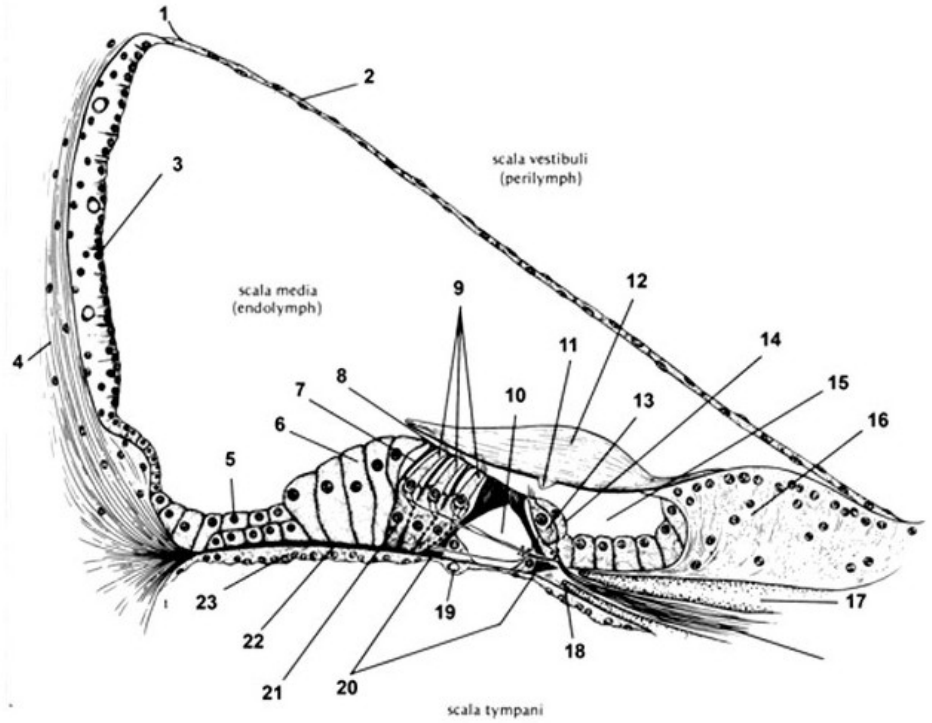


Figure 2. Cross section of the cochlear duct (scala media) housing the organ of Corti. 1, 2: Reissner's menbran ; 3: stria vascularis ;4: spiral ligament ,6: Hansen's cell; 9: outer hair cells; 10: tunnel of Corti; 11: Hansen's stripe; 12: dectorial menbran; 13: inner haor cell; 16: spiral ganglion; 17: spiral limbus; 19: Basilar membrane; 20: pillar cells;21: Deiter's cells

Organ of Corti

Arranged along the inner edge of the basilar membrane (**Fig. 3. b)**. A tunnel, composed of the inner and outer pillar cell's rods, and forming a triangle with the basilar membrane, divides the organ into inner and outer portions. The inner side contains one row of hair cells (inner hair cells, **IHC**). These are the real receptor cells of hearing. On the outer side there are three rows of hair cells (outer hair cells, **OHC**) (**Fig 3. c,d**). The function of these cells are rather more to regulate the stimulus input to the IHCs than functioning as receptor cells. OHCs serve as cellular motors being capable of length changes in a broad frequency range: up to 100 kHz. These length changes are in the nanometer scale and follow the receptor potential evoked by sound. These cells are responsible for the high sensitivity and sharp frequency discrimination in hearing. The tectorial membrane overhangs the organ of Corti and results in a functional unit with the hair cells and the basilar membrane.

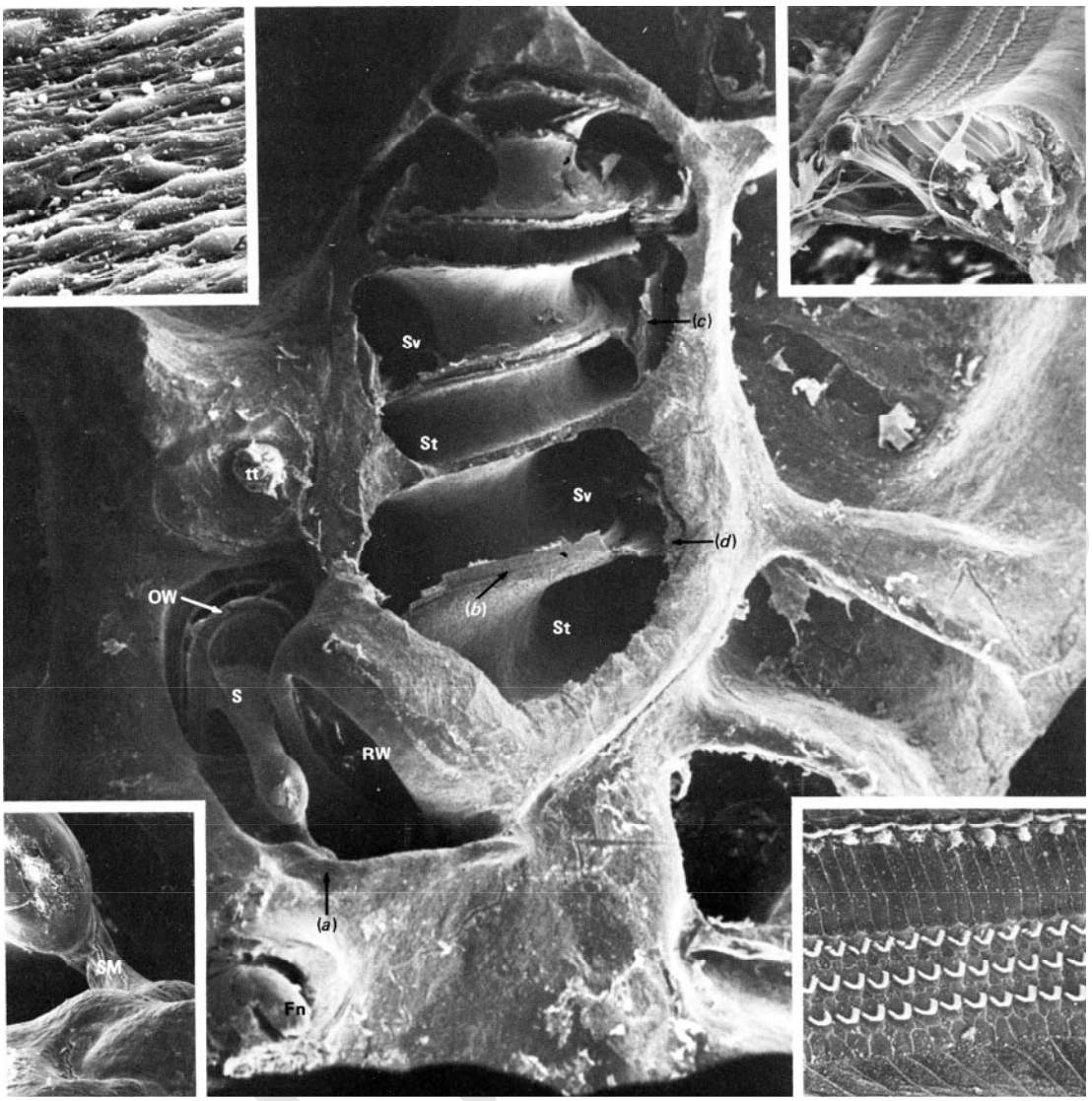


Figure 3. Lateral view of an anatomical preparation of the human cochlea showing the stapes (**S**), the oval window (**OW**), the round window (**RW)** , the scala vestibuli (**Sv**), scala tympani (**St**), facial nerve (**Fn**). Inserted pictures clockwize from lower-left corner with their place of origin depicted as (a), (b), (c), and (d) are the head of the stapes and the stapedial muscle (**SM**) (a), scanning electronmicroscopic views of the basilar membrane (b), the organ of Corti (c) and the cuticular plate with the 3 rows of outer hair cell stereociliary bundles and 1 row of inner hair cell stereociliary bundle (d).

Cochlear nerve.

The terminal fibres end in contact with the hair cells. These fibres are of two types. Type I fibres are the primary afferents and end on the IHCs. Type II fibres are the efferents and end on OHCs, directly. The fibres pass to the sipral ganglion in the modiolus to become the auditory branch of the VIIIth cranial nerve.

Blood supply of the labyrinth.

It derives principally from the internal auditory artery, which arises from the anterior inferior cerebellar artery. The internal auditory artery divides into two branches in the internal auditory meatus: anterior vestibular and common cochlear, the latter soon subdividing into the vestibulocochlear and cochlear branches. In the cochlea, the cochlear artery runs a serpentine course around the modiolus, as the spiral modiolar artery, from which arterioles run centrifugally to radiate over the scala vestibuli and the osseus spiral ligament.

Internal auditory canal.

Nearly 1 cm in length. It transmits the VIIth and VIIIth cranial nerves and the internal auditory artery and vein. It consists of the facial nerve canal and superior vestibular nerve canal superiorly, the cochlear nerve canal and the inferior vestibular nerve canal inferiorly.

1.1.4. Central connections.

After leaving the internal auditory canal, the cochlear nerve enters the brainstem. The nerve bifurcates immediately after entering (**Fig. 4**), and terminates in the dorsal and ventral cochlear nuclei. Thus most of the fibres decussate via the corpus trapezoideum to the lateral lemniscus of the opposite side and then pass to the medial geniculate body and inferior corpus quadrigeminum. Other fibres reach the homolateral centre. Then they pass to the higher auditory centres in the superior temporal gyrus of the cerebral cortex. Apart from the ascending afferent fibres distributed mainly to the IHCs, there are descending efferent fibres coursing from the superior olivary nuclei and ending on the OHCs.

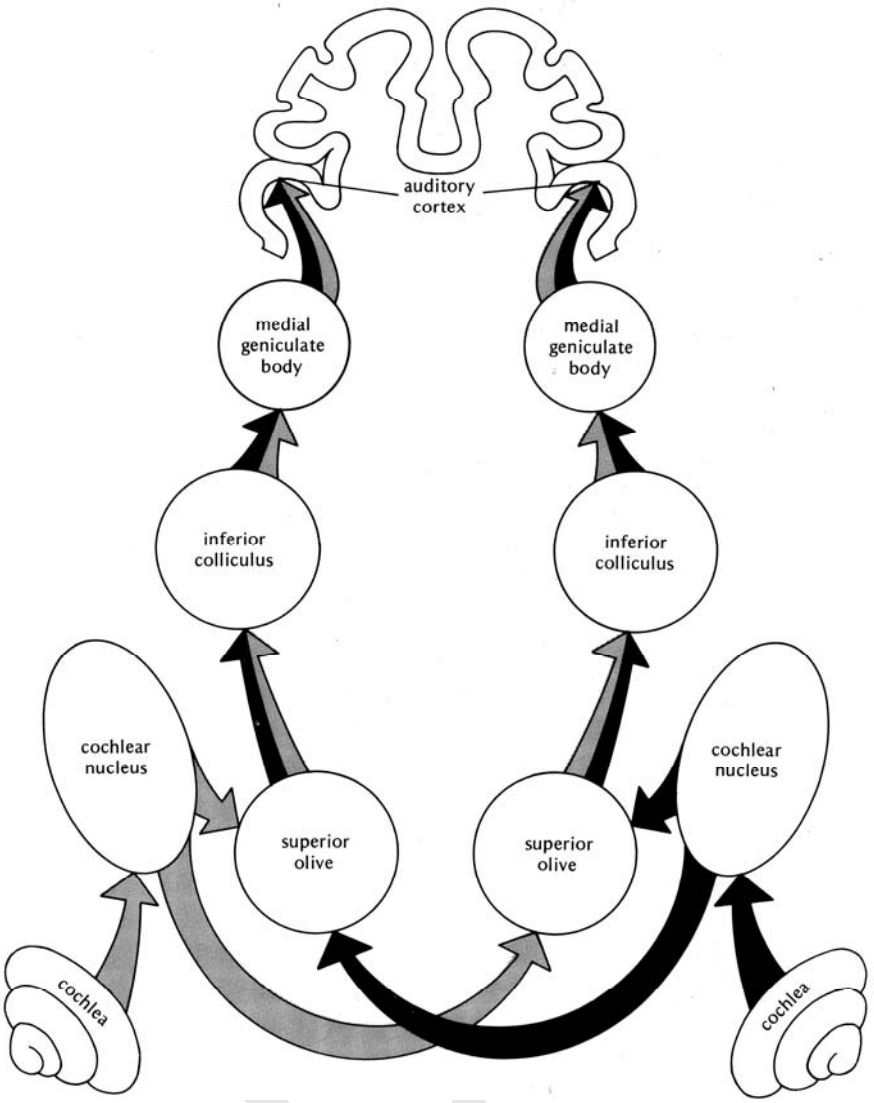


Figure 4. Central connections and bilateral representations of the cochlea.

1.2. Physical examination of the ear

External ear. Inspection may reveal swelling, tumors, ulceration, malformations, fistula, discoloration. Palpation on the mastoid process show us pressure sensitivity. The auricle is examined for pain by pressing the tragus. The regional lymph nodes are located pre-, and retroauricularly and in the upper deep cervical region. Otoscopy might be performed by head mirror indirect illumination or by electric otoscope (a combination of ear speculum, magnifying lens and electric light source). The auricle should be pulled upwards and backwards in an adult, whereas backwards and downwards in children to obtain nearly straight external auditory meatus. When discharge (cerumen) occluded the ear canal, suction or syringing by water at body temperature may help to obtain good acces to the tympanic membrane. Syringing must not be performed in cases where tympanic membrane perforation may be suspected. As seen through an aural speculum, the **tympanic membrane** is grey, lustrous and translucent. The handle of the malleus is normally yellowish in color, passes downwards and slightly backwards. The **light reflex** passes downwards and forwards from the **umbo** (**Fig. 5**), the lowest point of the handle of the malleus.

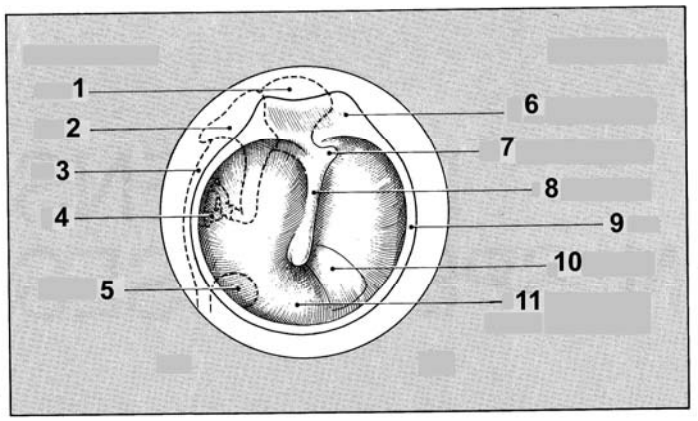


Figure 5. Schematic otoscopic picture of the drumhead and structures that are hidden by the membrane in the middle ear cavity. 1: head of the malleus, 2: body of the incus, 3: facial nerve, 4: stapes, 5: round window, 6: Shrapnell's membrane, 7: short process of the malleous, 8: handle of the malleus, 9: fibrocartilaginous frame of the drum, 10: light reflex, 11: pars tenso

The anterior and posterior malleolar folds going forwards and backwards from the short process of the malleus mark the upper end of the fi brous layer of the tympanic membrane and separate it into two parts: **pars tensa** below and **pars flaccida (Shrapnell's membrane)** above (**Fig. 6**). A perforation in this portion, like any other marginal perforations in the pars tensa indicates disease in the attic. Changes in the middle ear cleft are deduced clinically from changes in the tympanic membrane. Siegle's pneumatic speculum allows magnification and mobility of the drumhead to determin by alternate compression and release of the bulb. Fine details are best observed with the binocular operating microscope.

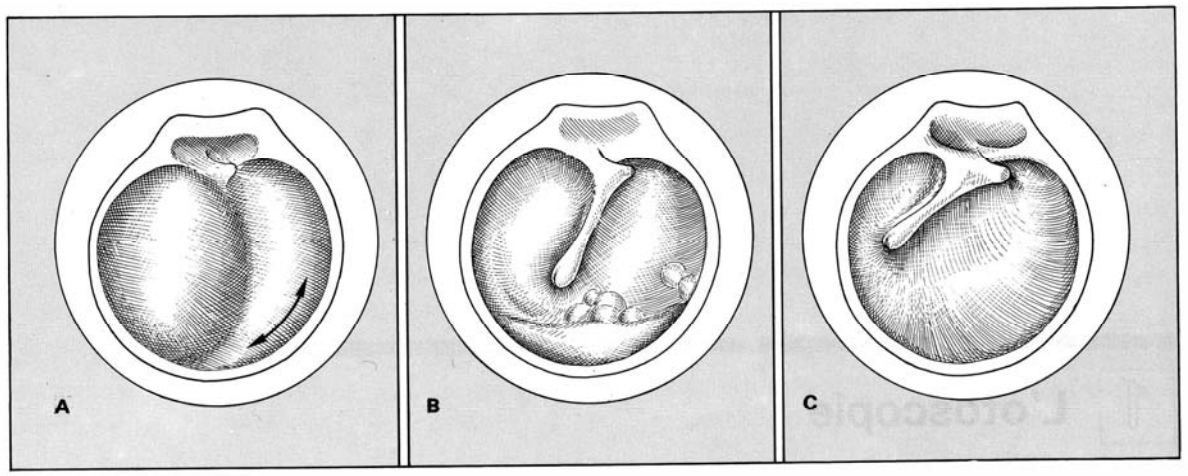


Figure 6. Otoscopic views of different tympanic membrane (rigth side) pathologies. (A) intact tympanic membrane with imbulgement of the posterior quadrants: acute suppurative othitis media; arrow shows appropriate place for myringotomy **(B)** othitis with effusion: fluid and bubbles are seen through the tympanic membrane **(C) negative pressure in the middle ear cavity with elevation and medialization of the handle of the molleous**

Eustachian tube. Its lower end can be inspected by postnasal mirror or directly by nasal endoscope. The patency of the tube can be determined by several ways. The **Valsalva's method** is when the examiner views the tympanic membrane while the patient closes the mouth holds the nose and exhales forcibly into it. The membrane moves outwards if the tube is patent and the membrane is intact. The **Toynbee's** test is when the patient swallows with the nostrils closed and so sucks air out of the middle ear with simultaneous sensation of fullness and hearing loss as signs of a patent Eustachian tube. Another maneuver is the **politzerization**. The nozzle of a Politzer bag is inserted into one nostril. Both nostrils are compressed and the bag is squeezed as the patient swallows. Air entering the ears can be felt if the tubes open. Two other possibilities are the Eustachian tube catheterization or indirect measurement of middle ear pressure by acoustic impedance determination (tympanometry).

1.3. Radiographic examination of the temporal bone.

The value of the X-ray pictures of the temporal bone is: 1. to judge its pneumatization (cellular:aerated; sclerotic: reduced pneumatization); 2. the sigmoid sinus is backwardly or forwardly placed; 3. the dura mater is upwardly or downwardly placed.

Most frequently used view is the lateral oblique (Schüller's) view showing the groove and plate of the sigmoid sinus, the antrum and the pneumatized petrous bone.

High resolution CT (computerized tomography) scan gives very precise imaging of the temporal bone including the ossicular chain, middle ear contents, internal auditory meatus, patency of cochlear duct. Magnetic resonance imaging (**MRI**) gives excellent imaging contrasts about soft tissues in all planes of the human body. MRI is contraindicated in patients with cardiac pacemakers, aneurysm clips and cochlear implants. It does not mean, however, apparent danger of stapedectomy prosthesis being displaced when subjected to the electromagnetic field. MRI is particularly useful to diagnose small acoustic neuromas.

1.4. Audiology

1.4.1. Physical properties of sound.

Sound travels from its source (tuning fork, piano etc.) to the ear by waves consisting of alternating compression and rarefaction of molecules in the air or other medium through which it is transmitted. Sound has certain objective properties which are related to subjective sensations perceived by the listener. The *frequency* or the subjective pitch is determined by the frequency of vibration of the sound source. If a tuning fork vibrates 256 times in 1 second (256 Hz) the hearer perceives the note of middle C in the musical scale. Doubling the frequency to 512 Hz, produces a note one octave higher.

The sensation of loudness is related to *intensity* of applied sound energy. The *decibel* is a logarithmic unit and indicates the ratio between two different intensities. If the two intensities are I_1 and I_2 then the ratio of the intensities in dB equals 10 times the logarithm of the simple ratio to the base 10, or:

$$n\ dB = 10 \log_{10} I_1/I_2$$

For example if one intensity is twice as great as another, then the difference in the intensities in dB is:

$$10 \times \log_{10} 2 = 10 \times 0.301 = 3.01\ dB$$

Again, a tenfold increase in intensity is equivalent to a change of 10 dB, hundredfold increase to a change of 20 dB. In clinical work, the threshold of normal hearing is defined as 0 dB; whisper has an intensity of 30 dB; normal conversation is 60 dB; shout is 90 dB; discomfort level is at 120 dB.

1.4.2. Physiology of hearing.

The ear can be divided into two parts: 1. conducting apparatus (external ear, tympanic membrane, chain of ossicles, Eustachian tube, labyrinthin fluids) 2. sensorineural apparatus (organ of Corti, cochlear nerve and central connections).

Conduction of sounds. Sounds can be transmitted to the inner ear in one of the three ways: 1. By the ossicular chain, from the vibrating tympanic membrane to the oval window; 2. Directly across the middle ear, when waves fall on the round window membrane. This may occure, when there is a large perforation on the tympanic membrane; 3. By bone conduction, when sound energy is taken up and transmitted to the inner ear through the bones of the skull.

The transformer mechanism of the middle ear. Acoustic energy collected by the tympanic membrane is applied through the ossicles to the stapes footplate. The effective ratio of these areas are 17:1. The ossicles constitute a lever mechanism

which has a mechanical advantage of 1.3:1. The combination of the above two is about 22:l, which represents the transformer ratio of the whole mechanism.
Impedance matching. The tympanic membrane-ossicular chain unit matches the impedance (acoustic resistance) of the air (airborn sound) and fluid (the organ of Corti is housed in endolymph). Without impedance matching 99.9% of the energy of sounds were reflected on the air-fluid interface causing 30 dB hearing loss. Proper functioning of the impedance matching requires appropriate pressure equalization of the middle ear cavity through the Eustachian tube.
Disorders of the transformer mechanism. 1. Non-marginal perforation of the tympanic membrane with intact ossicular chain: 10-30 dB hearing loss; 2. Posterosuperior marginal perforation with disruption of the ossicular chain: 40-60 dB hearing loss; 3. Total or subtotal perforation of the tympanic membrane with disruption of the ossicular chain: 40-60 dB hearing loss; 4. Attic perforation with cholesteatoma: 10-60 dB hearing loss; 5. Increased stiffness due to adhesive otitis media or tympanosclerosis: 30-50 dB; 6. traumatic disconnection of the ossicular chain behind an intact tympanic membrane: 40-60 dB hearing loss; 7. Reduced compliance due to middle ear fluid: 20-40 dB hearing loss.

1.4.3.Functional examination of hearing.

1.4.3.1.Tuning fork tests.

Weber-test. (**Fig. 7)** A bone conduction test, useful in unilateral deafness, or when marked difference exists between the two ears. The base of the vibrating C (128 Hz) tuning fork is placed on the forehead. Normal subjects and patients with bilateral equal deafness hear the sound equally in both ears. In conductive deafness the sound is heard in the deafened ear. In sensorineaural deafness the sound is heard in the better hearing ear.
Rinne-test. (**Fig. 7**) Should be performed by the C_3 (1024 Hz) tuning fork. The patient is asked to say whether the vibrating tuning fork sound is louder by air conduction, close to the external auditory meatus, or by bone conduction with the base of tuning fork on the mastoid bone. More correctly the test is done by requiring the subject to indicate when the fork becomes inaudible by bone conduction and then quickly transferring it to the external auditory meatus. It is then audible again for a normal hearing listener (Rinne positive). Conductive deafness produces better bone conduction hearing than air conduction (Rinne negative). Sensorineural deafness gives similar result as to normal hearing listener (Rinne postive).
Schwabach-test. The comparison of the bone conduction of a normal listener and the suspected sensorineural deaf patient. The base of the vibrating tuning fork is transferred to the normal hearing listener's mastoid when the vibration

quits producing sensation on the patients mastoid bone. Normal hearing listener at that moment still hears the fork..

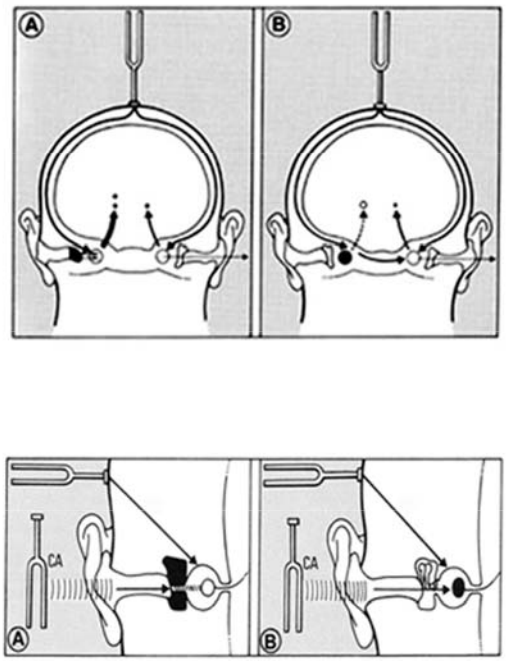


Figure 7. Tuning fork tests. **Upper panels:** the Weber's test reveals lateralization of the sound to the affected side of a conductive hearing loss (a) due to the occlusion phenomenon or to the non-affected side of a sensorineural hearing loss (b). 1 + shows normal loudness. **Lower panels**: the Rinne's test shows better hearing by bone conduction in conductive hearing loss, this is the Rinne's negative finding (a), whereas a better hearing by air conduction in sensorineural hearing loss (b), which is the Rinne's positive or normal finding.

1.4.3.2. Audiometry

Subjective audiometry. Measures hearing which relies upon the patients responding voluntarily.

1. **Pure-tone audiometry.**

This determines the subject's threshold of hearing for pure tones of several seconds of duration. Pure tones are delivered to the ear under test through a suitable earphone (air conduction,**AC**) or by a vibrator applied to the mastoid (bone conduction, **BC**). The frequencies tested usually range from 125 Hz to 8000 Hz, in octave steps, at intensities from -10 dB to 120 dB, in 5 dB steps. Calibration is adjusted so that each frequency 0 dB is the average threshold of a sample of normally-hearing, healthy ears (**Fig. 8**). Masking in pure tone audiometry is very important to obtain true threshold when there is a difference in AC sensitivity between the two ears of more than 40 dB and in all bone-conduction measurements. In the latter case the masking is applied to the non-test ear to prevent any sensation arising from the vibration being transmitted through the skull. Narrow-band filtered white noise centred on the test frequency is the most useful masking noise.

Recruitment. The loudness recruitment phenomenon is an aspect of certain forms of deafness, wherein the increase in sensation of loudness of sound of increasing intensity is greater than in the normal ears. This phenomenon is characteristic of a cochlear sensory hearing loss and helps in differential diagnosis to distinguish cochlear and retrocochlear sensorineural hearing losses. Clinical tests for recruitment are as follows:

a/ **Fowler′s test** (alternate binaural loudness balance test). This compares the stimulus intensities which give equal loudness in the normal and the deafened ear in patients with unilateral sensorineural deafness. A frequency of 1000 Hz is the commonest used.

b/ ***S*hort *I*ncrement *S*ensitivity *I*ndex** (SISI). A continuous pure tone at 20 dB above threshold is increased in intensity by 1 dB for 0.3 s every 5 s. This cycle occurs 20 times. Results are recorded as the number of increments heard, expressed as percentage. Score under 20% is normal, over 60% is typical of cochlear deafness (e.g Meniere's disease).

c/ **Stapedius reflex threshold.** see below among objective audiometry measures

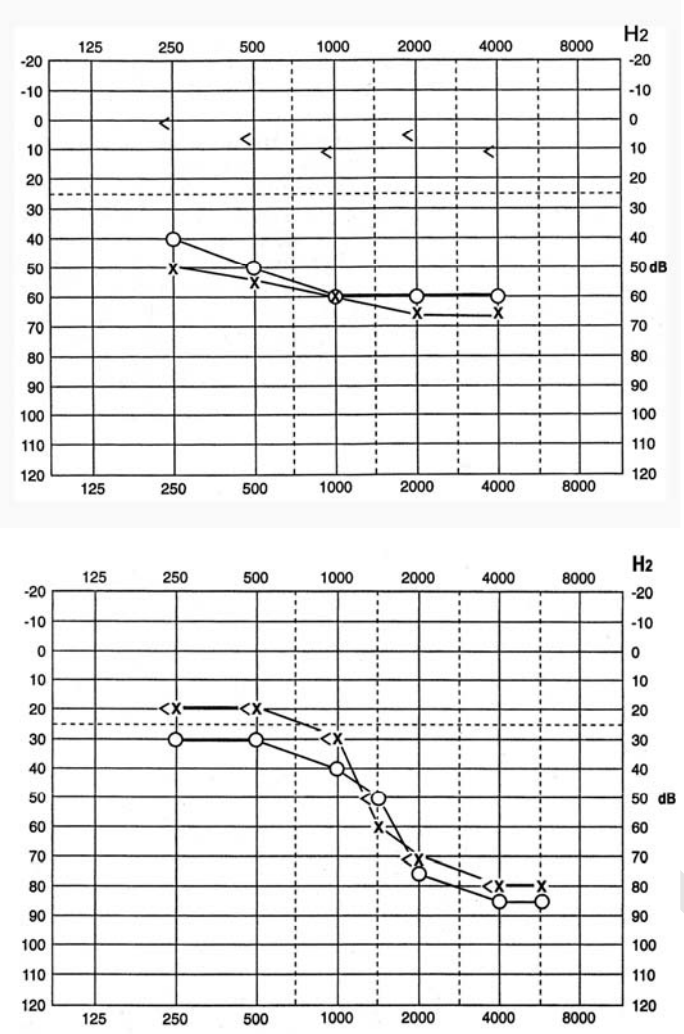


Figure 8. Conventional pure-tone threshold audiograms. **Upper panel:** Conductive hearing loss featured by the air-bone gap; **Lower panel:** sensorineural hearing loss showing a high frequency-slope threshold curve

2. **Békésy's self-recording audiometry.** Sweep frequency testing. Signal frequency is swept from low to high, the traverse requiring about 10 minutes. Intensity is switched, so as to increase or diminish, by the patient himself. The patient is instructed to reduce the volume as soon as he can hear the tone and to increase the volume when the tone becomes inaudible. Pulsed or continuous tones are used. The intensity, as determined by the patient, is automatically plotted on a graph against the frequency. The resulting curve indicates the threshold. The amplitude of the intensity variations represents the difference limen (**DL**) for pure tones. Small amplitudes indicate recruitment.

3. **Speech audiometry (Fig. 9).** Used to analyse the percentage of words heard correctly by the subject. Standardized, pre-recorded word lists are used. Usually groups of 25 words are used at each intensity. Every correctly repeated word scores 1 and the total score when multiplied by a factor of 4 is the percentage, or when used a groups of 10 words should be multuplied by 10 to give the percentage. Normal ears: 100% discrimination score is achieved by 60 dB intensity levels; conductive deafness: 100% discrimination score can be obtained but at higher intensity; Sensory deafness: patients are unable to obtain 100% scores (it is 50-70% usually) before discrimination deteriorates; Neural deafness: very poor discrimination scores are obtained.

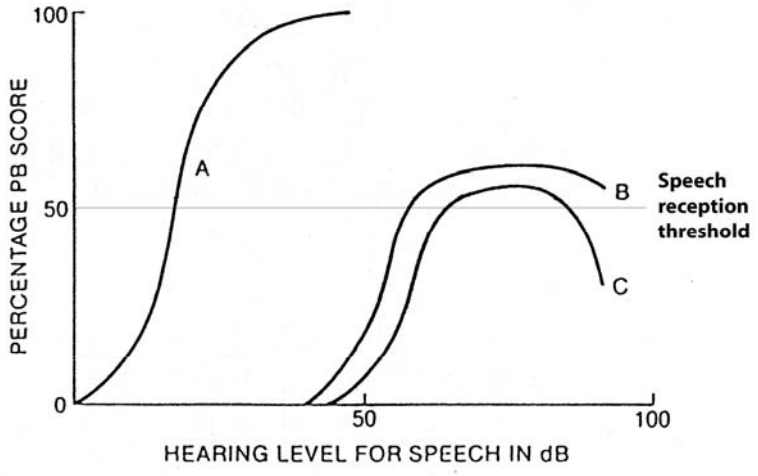


Figure 9. Speech reception threshold. (A) normal curve; (B) cochlear pathology (recruitment); (C) retrocochlear pathology : "rollover".

Objective audiometry.

Methods which do not require the active cooperation of the patient.

1. Impedance audiometry. A low frequency signal or probe tone is introduced into the external ear. If the tympanic membrane is stiff, more of the sound is impeded than when the tympanic membrane is mobile and compliant. The amount of sound reflected is monitored by a sound pressure level meter. **Tympanometry (Fig 10).** The tympanic membrane may be made artificially stiffer by changing the pressure in the ear canal from -200 mm H_2O to +200 mm H_2O by using small air pump attached to a manometer. Changes of acoustic impedance are shown by the meter or plotted as a graph against the pressure changes. The compliance is maximal when the air pressure in the external meatus equals that whithin the middle ear. The graph shows characteristic changes in different conditions of the middle ear.

a/ **Normal ear**: A symmetrical graph with maximum compliance at 0 mm H_2O (Type A tympanogram). b/ **Otitis with effusion**: The graph shows negative middle ear pressure or a flat curve indicating a marked decrease in compliance (Type B tympanogram). c/ **Otosclerosis**: Normally shaped graph with decreased compliance. If the maximum compliance is less than 0.5 ml, usually a highly immobile stapes is found during surgery. Similar result is obtained in other ossicular fixations like malleus or incus fixation. d/ **Ossicular disconnection**: Increased compliance (above 1.5 ml) is usual. Negative pressure in the middle ear is depicted by shift of the compliance peak towards the negative pressure values (c and d are Type C tympanograms).

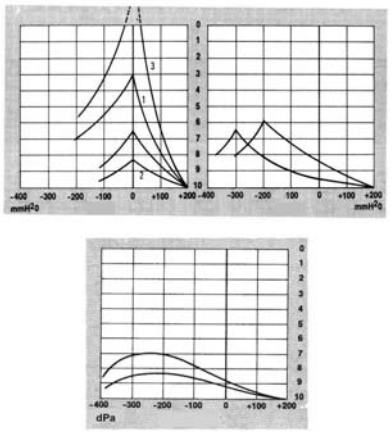


Figure 10. Tympanograms. Typical tympanogram of normal middle ear (Type A tympanogram):A/1; otosclerosis A/2; ossicular disconnection A/3. Pathologic tympanograms: Type B tympanogram: otitis with effusion (C panel); Type C tympanogram: negative pressure in the middle ear (acute catarrhal otitis media) (B panel).

Intra-aural reflex measurements. **Stapedius reflex**. In man, sounds of more than 70 dB in intensity cause the stapedius muscle to contract bilaterally (acoustico-facial reflex arch), unless the sound is sufficient intensity to cause a general startle reaction. Frequencies between 500 Hz and 2700 Hz are best used. The minimum intensity required to evoke the reflex is called the stapedius reflex threshold (SRT). In normal ears, SRT is between 70 and 100 dB for pure tones (0.5 -4 KHz). In conductive deafness, the reflex may be obtainable owing to the middle ear pathology (e.g. fixation of the stapes in otosclerosis). The SRT is usually between 70 and 110 dB above the subjective threshold. In recruiting deafness, the SRT usually occurs at less than 70 dB above the subjective threshold. In neural deafness, the SRT occurs at levels in excess of 70 dB above subjective threshold.

2. Evoked response audiometry

Clinically this involves the use of averaging equipment which adds together individual responses to sound stimuli so that the sum becomes visible and the background noise is diminished.

Electrocochleography (ECochG). Measurement of the electrical output of the cochlea by inserting a needle electrode into the promontory wall through the tympanic membrane Adults are tested under local anaesthesia, children are under general anaesthesia. Component potentials of the human electrocochleogram are the action potential from the VIIIth nerve, the cochlear microphonics (produced by the outer hair cells and the summating potential (field potential in the inner ear). This gives a clear indication of cochlear threshold which is a good measure of the hearing threshold. ECochG predicts the hearing status of young children, however, today it is rarely applied to that purpose due to its invasivity. It also helps in otoneurological diagnosis, like in Meniere's disease. A clear measure of recruitment can also be made. The currently most common clinicl applications of ECochG are:

1. Monitoring the Meniere′s disease
2. Enhancement of wave I. and identification of the I-V interwave interval of the auditory brainstem response (ABR) in the presence of hearing loss
3. Monitoring cochlear nerve function during surgical procedures

Brainstem responses (ABR:auditory brainstem response) (Fig. 11). Electrodes are placed on the ear lobe or mastoid and the vertex. Using acoustic clicks a serious of waves is obtained. The first wave derives from the cochlear nerve, the third from the superior olivary nucleus, the fourth and fifth from the lateral lemniscus and inferior colliculus. Delay between I and V occurs with acoustic neuroma.

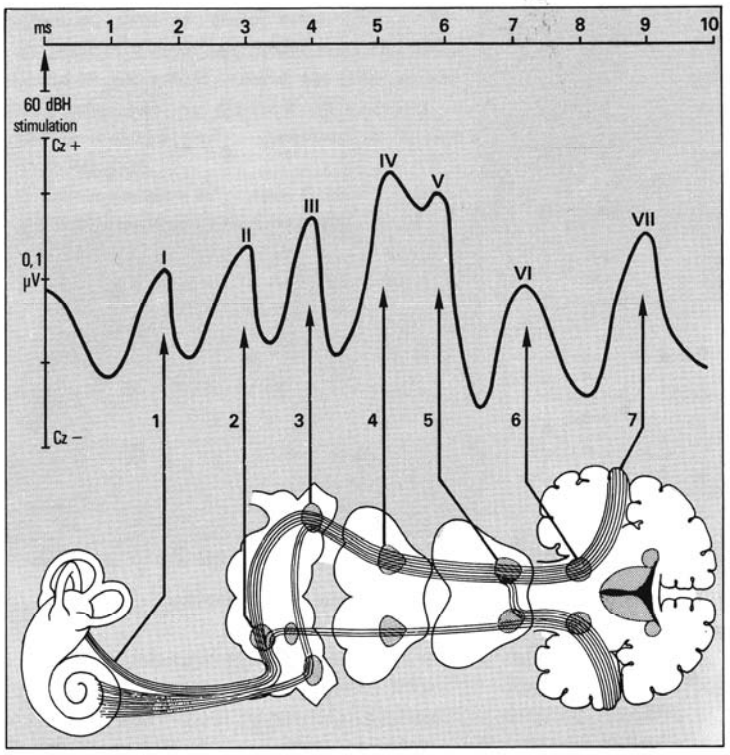


Figure 11. Brainstem potentials and generator sites. 1. auditory nerve, 2. cochlear nuclei, 3. superior olivary complex, 4. lateral lemniscus, 5. inferior colliculus. In 10 msec after stimuli another 2 waves are shown: 6. medial geniculate body, 7. thalamo-cortical fibers. These 2 are not considered brainstem in orgin.

Otoacoustic emission. Otoacoustic emission means that sound is produced in the inner ear and can be detected in the external auditory canal. Up today this is the best proof nearby an active mechanical activity in the organ of Corti. Candidates for these active vibrations in the inner ear are the outer hair cells, which are known to have almost exclusively efferent innervation and are capable of performing axial somatic contractions and elongations in the nanometer range driven by receptor potential elicited membrane potential oscillation. This so called electromotile activity of the outer hair cells follows the receptor potential frequency in the audio range (up to 70 kHz) and assumed to be regulating the stimulus input in the inner hair cells. Doing so, the outer hair cells are responsible for the sharp hearing (the frequency selectivity) and sensititvity (low threshold) of the human ear. The mechanical activity of the outer hair cells produce spontaneous otoacoustic emission in some individuals. An evoked otoacoustic emission (EOAE), however, can be elicited in all individuals who have functioning outer hair cells. This practically means normal hearing, since all cochlear noxious effects impair first the outer hair cells causing consequently a hearing loss of maximally about 50 dB. The EOAE was discovered by Kemp in 1978, and it reflects the cochlea's active response to sound stimulation. It is held at this time that the sound induced mechanical movements of the outer hair cells leave the cochlea, traverse the middle ear in a reverse direction and are detected in the external auditory canal as sound. Today the emission findings can be interpreted in conjunction with routine audiometry. Of the various types of OAEs, the two mainly used are the transiently (tone pips and clicks) evoked OAEs (TEOAE) when the spectra of the emission corrrespond to the spectra of the evoking stimuli and the distortion-product OAE (DPOAE). In this latter case two pure tones with appropriate frequency relations ($f_1 < f_2$; $1.05 < f_2/f_1 < 1.5$) are applied simultaneously and emissions can be recorded at imtermodulation distortion product frequencies such as $2f_1 - f_2$.

The evoked emissions are helpful in screening the cochlear function in infants and young children in the diagnosis of hearing loss (in the absence of external or middle ear abnormalities) and in the monitoring of outer hair cell activity in noise exposed industrial workers and in ototoxic drug-exposed patients.

1.5. Equilibrium.

1.5.1. Physiology of the vestibular system

The vestibular system has two major functions: maintenance of the body equilibrium and hence prevention of injury and maintenance of the position of the eyes in order to obtain maximum resolution of any object that is being observed. The sensory side of the system consists of the vestibular labyrinth, eyes and the somatosensors in muscle, joints and skin. These organs connect with the brainstem, the cerebellum and cerebrum. The receptor organs in the membranous vestibular labyrinth are concerned with the reflex adjustments of posture and subjective sensations. **Utricle**. Utricular maculae are situated in the horizontal plane and quiescent as long as the head is horizontal and stationary. They respond to tilt of the head and linear acceleration. Such a movement also results in compensatory ocular reflexes whereby the visual axis is fixed when the head is deviated from its previous position. **Semicircular canals**. Respond to angular acceleration of the head: the horizontal pair to rotation about a vertical axis, the posterior and superior pairs to tipping displacements about a horizontal axis. Movement of endolymph within the ducts stimulate the cristae ampullares. Stimulation causes reflex nystagmus, in which the eyes rotate slowly in one direction and then, by a sudden flick in the opposite direction. The slow component is labyrinthine, the quick one is a cerebral compensatory movement. In clinical practice, nystagmus is named after the direction of the quick component. **Saccule.** The macula of the saccule is at right angles to the macula of the utricle. It senses linear (up and down) acceleration.

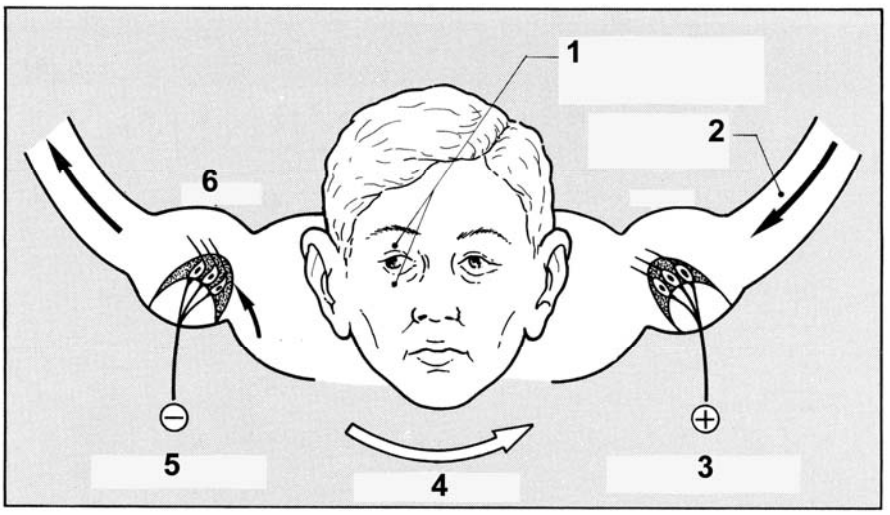


Figure 12. Functioning of the vestibular labyrinth. Rotation of the head to the left side stimulates the left semi circular cannal and inhibits the right side inducing nystagmus to the right side.

1.5.2. Functional examination of the labyrinth.

The function of the vestibular labyrinth can be assessed by stimulating it artificially to produce nystagmus. **Nystagmus** is a coordinated conjugated eye movement around an axis. The movement consists of rhythmic alternating slow and fast beating phases. The direction of the fast component determines the laterality of the nystagmus. Observation with and without Frenzel's glasses is used for diagnosis of spontaneous nystagmus. This is a +15 diopter lenses which completely suppresses visual fixation and subsequently the visual fixation derived suppression of vestibulat nystagmus is abolished.

Nystagmography can be used to record and measure frequency, amplitude, eye speed and duration of nystagmus, either photographically or electrically. In **electronystagmography (ENG)** electrodes are attached to the skin close to each eye. Changes in the corneoretinal potentials as recorded at the electrode sites are proportional to angle of rotation of the eyes from the straight-ahead position. These changes of electrical potential therefore follow the nystagmus faithfully and can be recorded on a moving paper chart. **Photoelectronystagmography**. The eye movements are recorded by a photoelectric cell instead of the corneoretinal potential. The change in the intensity of the light reflected from the surface of the eye is measured.

Caloric tests. If water at 30^{o} or $44^{o}C$ (7^{o} below and above the body temperature) is syringed into the external ear, nystagmus is evoked in normal subject with healthy labyrinth. In a recumbent subject, with the head inclined forwards at 30^{o}, the horizontal semicircular canal is vertical. Hot water heats the fluid in the duct and consequently dilates (its volume increases) and moves the cupula of the crista ampullaris toward the ampulla (a centrifugal displacement). This causes nystagmus with fast beating toward the stimulated side. When cold water is used, the endolymph volume decreases and moves away from the ampulla and produces nystagmus in the opposite direction.

Hallpike caloric nystagmus. The patient lies in a supine position with the head-rest tilted at 30^{o} to the horizontal plane. A continuous stream of cold ($30^{o}C$) or hot ($44^{o}C$) water is directed against the tympanic membrane for 40 sec. Nystagmus is usually results if the labyrinth is normal and commonly lasts for about 2 minutes from the beginning of stimulation. Each ear is tested separately -this is the advantage of this test- an interval of l0 minutes being allowed between each separate stimulation. The duration of nystagmus in each case -cold and hot stimulation- is recorded graphically. **Canal paresis** is present if the duration of nystagmus is reduced equally for both hot and cold tests. The condition may be unilateral or bilateral. **Directional preponderance** is present if the nystagmus responses towards one side are of shorter duration than those towards the other. A combination of canal paresis with directional prepondarence towards the opposite side is commonly found.

Cold tap water test. Should the minimal temperatures above fail to produce nystagmus in either ear, cold tap-water at a temperature of approximately $10^{o}C$ may be used under the same conditions as those described. Failure to elicite nystagmus from one ear indicates severe depression of its function.

Rotation test. The patient is seated in a Bárány chair with the head erect, eyes closed. The chair is rotated ten complete cycles in 20 s, then suddenly stopped. Nystagmus results if the labyrinths are active and is always opposite in direction to that of rotation. The rotation test has the disadvantage of always stimulating both side together. It can, however, be very valuable in determining the presence or absence of labyrinthin function in patients with bilateral total or subtotal sensorineural deafness, particularly in young children on whom caloric tests may be difficult to do satisfactorily.

Fistula test. A fistula in the bony wall of the horizontal semicircular canal may result from erosion by cholesteatoma or from the fenestration operation. In either case, compression of air in the external auditory canal will produce movement of endolymph in the duct, hence vertigo and nystagmus, as long as

the vestibular labyrinth is still functioning. If the labyrinth has been destroyed, the test may be negative even though a fistula is present. Air in the canal can be compressed mechanically either by pushing in the tragus with a finger or by compression of the bulb of Siegle's speculum.

Vestibulospinal reflex tests.

Romberg's test. With incompletely compensated or unilateral lesions, or with bilateral vestibular damage, eye closure results in a dramatic loss of balance, even if proprioceptive sensory function is intact (tabes dorsalis). The patient's body tilts in the direction of the vestibular lesion side.

Finger-nose pointing test. The index finger of the extended arm brought against the tip of the nose with eyes closed. Coordination disorders or ataxia indicate ipsilateral cerebellar lesion.

Craniocorpography objectivates the Romberg′s test result by continuous monitoriung the deviating position of the body.

Posturography is an objective, computer driven examination of the deviation of body in Romberg's position.

1.5.3. Peripheral vestibular disorders

The leading symptome of peripheral vestibular lesions is vertigo. Vetigo can be characterized with a direction of the body imbalance (lateropulsion, retropulsion, rotation, lifting etc.) versus the central vestibular disorders when a discrepancy develeops between visual, vestibular and proprioceptive sensations. The resulted balance disorder, called "dizziness", can not be determined with specific direction.

Vertigo is most frequently due to non-vestibular disorders. The check-up examination of vertiginous patient should clarify the history of hypertonia, diabetes, hypercholesterinaemia, presence of cervical spondylosis and use of contraceptive pills. All these may precipitate vertigo. Any central nervous system disorders or ear diseases can accompany with vestibular lesion.

A **neurotologic** examination starts with audiometric tests (pure tone audiometry, acoustic reflex threshold and decay, ABR) and vestibular tests follow (spontaneous nystagmus, statokinetic tests, positional nystagmus, optokinetic nystagmus, caloric nystagmus, postrotatory nystgamus).

Vestibular neuronitis. Caused probably by a virus infection. Symptomes onset is sudden. Rotatory vertigo lasts few days with severe vegetative symptomes. Vertigo does not associate with hearing loss, tinnitus or other neurologic focal sign.

Ictus cochlearis (minor stroke) is characterized by sudden onset of deafness and rotatory vertigo. The labirinthine sensory epithel stops functioning (canal paresis) due to vascular disorder in the labyrinthine artery.

Labyrinthitis. This is an inflammation of the inner ear due most frequently to purulent otitis media. The **serous labyrinthitis** is characterized by mild vertigo, nystagmus towards the affected side (excitatory symptome) and conductive hearing loss (Weber tuning fork test: the sound is lateralized to the affected - discharging - ear). **Purulent labyrinthitis** is featured by severe vertigo, nystagmus to the non-affected side and total deafness (Weber is lateralized to the non-affected side).

Benign paroxismal positional vertigo (BPPV). Also called cupulolithiasis or epidemic vertigo. It is caused by otolith crystal deposition on the sensory cells. Typically precipitated by head rotation. The onsetting rotatory vertigo lasts few seconds and accompanies with rotatory positional nystagmus.

Méniere's disease (see 1.9.8. section). Should not be mistaken with **Méniere's syndrome**, which is not a specific diagnosis. All that means is that the patient suffers from sudden rotatory vertigo, nystagmus and tinnitus. May be precipitated by any central or peripheral vestibular disorder of sudden onset.

1.5.4. Central vestibular disorders

Vast majority of cases belong to the **vertebrobasilar insufficiency** group. This may also be caused by cervical spondylosis as noxious agent in vertebrobasilar blood supply failure. Predisposing conditions are high blood pressure, high serum cholesterine level, heart disease. The paroxysms are featured by rotatory vertigo, bilateral hearing loss, tinnitus, transient loss of vision (amaurosis) or double vision. Lasts for few minutes and can be provoked by head position changes. Typically the paroxysms present several times a day. Sometimes drop attacks, consciousless associate.

Vetiginous migraine is usually unilateral and causes pulsating headache for a day or so accompanied by nausea or even vomitus. Headache is preceded by rotatory vertigo. Hearing is normal in the attacks. Exploration of case history may record **motion sickness** earlierly.

Multiple sclerosis is a white matter disease in the brain. Otoneurological manifestations are characterized by vertigo lasting for week without hearing loss. Balance disorders disappear in attack-free periods. Suspicion of central vestibular lesion arises when gaze-nystagmus, ataxcia or directional preponderance is present.

Acoustic neuroma may be presented by unsteadiness as first symptome although this is rare and true vertigo is almost never accounted (see section **1.11.**)

1.5.5. Differential diagnosis of central and peripheral vestibular lesions

When the body deviation in the Romberg's position is opposite to the fast component of the spontaneous nystagmus the patient suffers from a peripheral vestibular lesion. This is called “harmonic syndrome”. The direction of the body-tilting to the same side than direction of nystagmus is strongly indicative of a central vestibular disorder and belongs to the care of neurologist. This is called “dysharmonic syndrome”.

1.6. Diseases of the external ear.

1.6.1. Congenital malformations.

These may be associated with others in the middle and inner ear. Development of the face and lower jaw may also be defective.
Types: 1. Complete or partial absence of auricle; 2. Preauricular fistula or cyst; 3. Accessory auricles; 4. Atresia of external auditory meatus; 5. Abnormalities in size or shape of auricle. Including the protruding ear ("bat ear").
Treatment. **Absence of the auricle**: plastic operation (glass, costal cartilage) or auricle-prosthesis implantation (silicone) which is removable and attached to the skull via a titanium screw. A **preauricular fistula or cyst** should be excised completely (especially when infected). **Bony atresia of the external auditory meatus** is best treated for hearing restoration by *b*one *a*nchored *h*earing *a*id (BAHA).

1.6.2. Injuries.

Hematoma auris. Due to rupture of vessels in the perichondrium. This leads to effusion of blood. Haematoma or serous effusion may sometimes arise sponaneously in the elderly. Failure to treat results in fibrosis of the clot, with permanent thickening of the auricle ("cauliflower ear").
Treatment. Incision, drainage and tight dressing for 1 week. It may be necessary to secure the position of the elevated perichondrium by shirt buttons or a moulded splint sutured by monofilament astride haematoma.

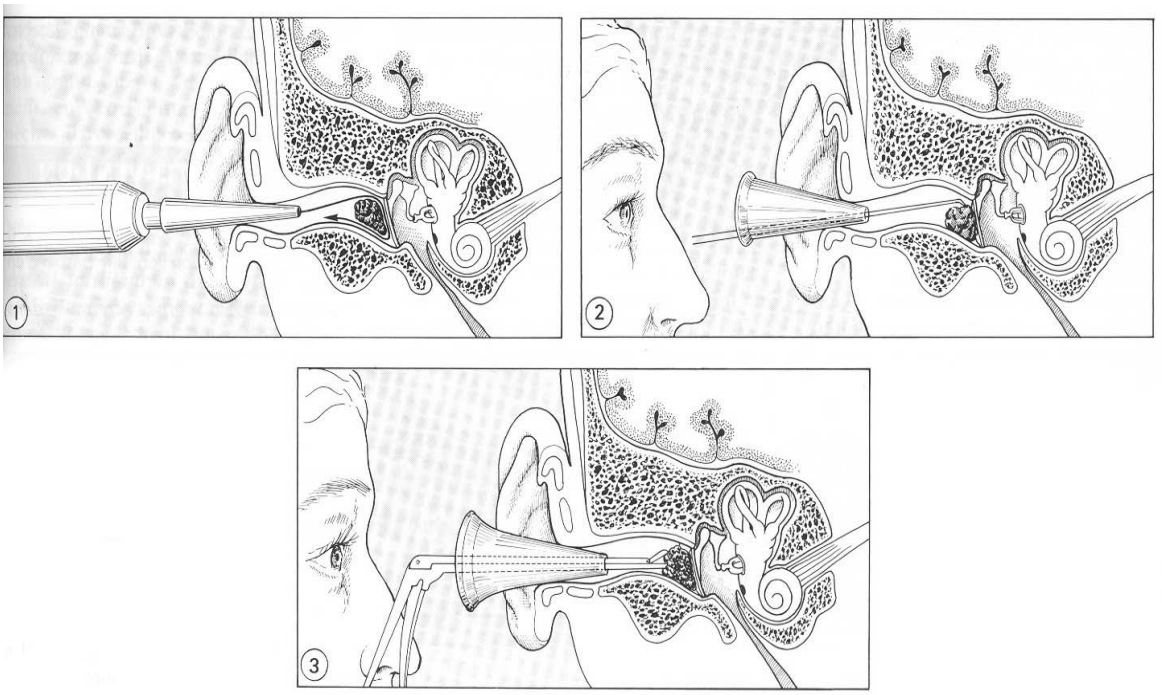


Figure 13. Removal of foreign bodies from the external auditory canal, by syringing (1), by hook (2) and by crocodile forceps (3)

Foreign body. Hard or soft objects, some are hygroscopic, get into the ear canal. Live insects may enter to cause irritation and noise. When the canal is blocked, conductive deafness, tinnitus, and pain develop. There may be reflex cough.

Treatment. Smooth, round-shaped foreign body may be removed by a hook if it can not be removed by syringing. Forceps must not be used as they only push the foreign body further in. Use operating microscope. Other foreign bodies can be removed by: Crocodile forceps, syringing, suction (**Fig. 13)**.

Swelling of canal walls may result from irritation and may cause unsuccessful trial of removal. A postaricular incision must be made, to allow extraction of the foreign body via the posterior wall of the canal.

1.6.3. Otitis externa.

An acute or chronic reaction of the skin of the external ear from local or general causes. May be local appearance of a general disease, like dermatosis (psoriasis) or infections (impetigo, syphilis etc.).

Etiology. 1. Local: due to bacterial or fungal infection. The cerumen in the meatus has acidic pH. This is bacteriostatic and prevents colonization of bacteria. Water entering the auditory canal elevates pH towards neutral values and allows bacteria normally present to colonize.

2. General: Allergic or seborrhoeic states or systemic infection by bacteria or viruses. These can be combined and develop allergic response, eczema or seborrhoeic state.

Clinical forms: 1. Furuncle of the ear canal. 2. Diffuse otitis externa 3. Otomycosis 4. Eczematous otitis externa 5. Seborrhoeic otitis externa 6. Granular myringitis 7. Myringitis bullosa haemorrhagica 8. Herpetic lesions of the external ear 9. Otitis externa malignans (skullbase osteomyelitis).

Furuncle of external ear. A staphylococcal infection of a hair follicle in the cartilaginous portion of the meatus. Usually is single. Spontaneous evacuation occurs in a few days. *Symptomes.* Irritation is the first symptome. Pain follows. Trismus may occur when the boil is anterior. Deafness, which is conductive in type if the canal is occluded by swelling. Regional lymphadenitis is often present. *Differential diagnosis.* May be difficult from acute mastoiditis. Mastoiditis, however, features the following: Preceding history of otitis media; Deafness is present; Tympanic membrane shows signs of middle ear infection; pain or pressure over the mastoid process; Fistula into canal forms in bony protion (protrusion of the posterosuperior canal wall); Radiographic changes in mastoid (opacity over the mastoid air cells).

Herpetic lesions may cause difficulty after rupture of the vesicles. An exostosis should not be mistaken with furuncle.

Treatment. Antibiotic and steroid containing cream often all is required. Analgesics may be used. Incision is seldom required.

Diffuse otitis externa. An infective dermatitis starts near the auditory canal and involves the whole auricle. *Etiology.* Predesposing causes are maceration, scratching and clumsy instrumentation. Much less often occurs due to discharge of an otitis media than expected. *Pathology.* Usually a streptococcal infection but staphylococci, Pseudomonas aeruginosa, Bacillus Proteus, Escherichia coli are not infrequent. *Symptomes.* Itching is the first symptome. Pain follows. Swelling of the canal skin. Infiltration, discharge is present in various degree. In severe acute cases fever and lymphadenitis may be present.
Treatment. General: Systemic antibiotic treatment is necessary in intractable cases. Local: Gauze wicks soaked with a mild adstringent (aluminum acetate, 8%); Aural toilette to remove debris, pus; Antibiotics if locally used should be combined with steroids, they can equally be ointments or ear drops. In recurring cases concho-meatoplasty may be recommended to widen the aperture of the meatus.

Otomycosis. It is becoming more common with use of topical antibiotics. Resembles to the desquamative form of diffuse otitis externa. The commonest fungi are the Aspergillus niger and Candida albicans. *Symptomes.* Discharging ear having a musty odour. Sometimes the mycelium, hyphae and spores can be seen under microscope. Pain is unusual and the whole disease is otherwise symptomless (itching).
Treatment. Nystatin cream is effective in Candida infection. Clotrimazole as 1% cream. Amphotericine cream. Gentian violet is effective for all types but has the disadvantage of staining the skin.

Eczematous otitis externa. An allergic dermatitis. The most common form appears as a contact dermatitis against e.g. spectacle's frames, local application of antibiotics. *Symptomes.* Irritation, oedema, vesication, crust formation which sometimes spreads to the neck. Secondary infection is common and may mask the true nature of the condition. Scaling and fissuring occure at the canal entrance in the chronic case.
Treatment. Hydrocortisone containing ointment, systemically antihistamine drugs. Locally as an adjuvant therapy 8% aluminum acetate drops may be given. In chronic stage silver nitrate 10% may be applied to the fissures. Dilatation of the ear canal should be attempted by polythen tubes of increasing sizes when the canal is stenosed. Plastic operation is sometimes necessary (Concho-meatoplasty).

Granular myringitis. Granulations are seen on the eardrum as a localised form of otitis externa or in association with influenza. Aluminum acetate 8% aften brings the condition under control. If granulations are slow to heal they may be cauterized with silver nitrate. Many cases prove, however, intractable.

Myringitis bullosa haemorrhagica. Usually seen as complication of influenza. *Symptomes.* Haemorrhagic blebs form on the eardrum. They rupture quickly. Pain is prominent symptome. Deafness is conductive in type.
Treatment. Pain relieve, prevention of secondary infections and treatment of otitis media if present.

Herpetic lesions of the external ear. Caused by neurotropic virus infection. There are two varieties: Herpes simplex and Herpes zoster oticus. Pain is usually severe and may be persistent (postherpetic neuralgia). Vesication occurs but disappears early and the collapsed vesicles leave white plaques on the surface of the auricle and sometimes on the eardrum. Cranial nerve lesions sometimes accompany the other symptomes: 1. Deafness, sensorineural in type; 2. Vertigo; 3. Facial nerve palsies. The **Ramsay-Hunt syndrome** includes all the above features.
Treatment. Oral and topical acyclovir and analgesics. Cranial nerve palsies have poor prognosis, so the patient should be admitted to hospital for intravenous acyclovir treatment. Steroid in high dose in antibiotic cover.

Otitis externa malignans (skullbase osteomyelitis). A psuedomonas infection, not infrequently fatal, occuring in elderly diabetic or immunosuppressed patients. *Symptomes.* Pain in the ear. Discharge. Cranial nerve paralysis may affect the VIIth, IXth, Xth, XIth, XIIth nerves. Rarely **Gradenigo's syndrome** (see the section about special forms of acut otitis) may result from involvement of the Vth and VIth cranial nerves. *Diagnosis*: bone scintigraphy visualizes lateral skullbase involvement. Bacterial culture of the discharge is necessary.
Treatment. Medical: Control of the diabetes. Antibiotics systemically and locally according to the sensitivity of the Pseudomonas. Surgical: Removal of granulations and radical mastoidectomy. Treatment may have to be continued for months.

1.6.4. Neoplasms of the external ear.

Benign tumors are ceruminoma that may become malignant so need wide excision. Exostosis which is a new bone formation projecting into the lumen. Can be sessile or pedunculated. Since it can cause conductive deafness, should be surgically removed. If approached from the mastoid and removed with chisel, the facial nerve is easily damaged.

The **rodent ulcer (basal cell carcinoma)** occurs more commonly on the auricle. It grows slowly and does not give distant metastasis. Its treatment is surgical excision. When surgery was not ablastic, the tumor recurrs and may distroy surrounding anatomical region and can extend towards the orbital cavity.

Squamous cell carcinoma is the most common. Also malignant tumors are the **adenocarcinomas.** *Clinical features*. Bleeding polypi. Ulcerates the meatal wall. Serosanguineous discharge. All aural polyp should be examined histologically.

Treatment. Wide excision and postoperative irradiation. The outcome is poor.

1.7. Diseases of the middle ear cleft

1.7.1. Congenital malformations

Various degrees of developmental failure of the tympanic cavity and ossicles can occure, causing deafness. Severe abnormality of the gill-cleft structures is usually associated with deformities of the external ear. The inner ear, being of different origin, may not always be involved. 1. **Abnormal ossicles.** Most commonly the malleus and the incus is deformed as their processes are not present. The ossicles may be fused together. The stapes may be absent or deformed. 2. **Crouzon's syndrome** (craniofacial disostosis):Atresia of the external ear canal with middle ear abnormalities causing conductive deafness. 3. **Treacher-Collins syndrome**: Hereditary malformations of the lower face (mandibulofacial disostosis) in which the mandible and maxilla are hypoplastic, together with varying degrees of developmenta failure of the external and middle ear (atresia, absence of ossicles). **Treatment**. The surgical objectives are to provide : 1. Functioning ossicular chain; 2. A tympanic membrane; 3. A wide meatus.

1.7.2. Injuries.

1.7.2.1. Traumatic rupture of the tympanic membrane.

Etiology. Perforation by foreign body or unskilled instrumentation. Sudden air compression as hand slap, blast, boxing. Sudden fluid compression by a blow, on the ear when the canal is filled with water as in water polo. Inflation of the eustachian tube. *Symptomes.* Pain, deafness, tinnitus and vertigo, perforation and bleeding. **Treatment**. Foreign material should be removed. Infection should be prevented by antibiotics. The ruptured membrane should be reduced as possible. The external auditory meatus is closed by application of sterile gelfoam. Myringoplasty must be considered if the perforation has failed to heal after 3 months.

1.7.2.2.Traumatic disconnection of the ossicular chain.

May be caused by head injury, foreign bodies indenting or perforating the tympanic membrane, not correctly performed myringotomy (paracentezis). The commonest place of the disconnection is the incudostapedial joint. *Diagnosis.* Conductive deafness with normal eardrum, 40-60 dB air-bone gap. Tympanometry shows very high compliance (>1.5 ml) with the absence of the stapedius reflex.
Treatment. Surgery: Short columella between the stapes head and the malleus (may be interpositioned: incus body, cortical bone, bioceramics).

1.7.2.3. Barotraumatic otitis media.

A non-infective inflammatory reaction produced in the middle ear cleft when the air pressure within is considerably below that of the surrounding atmosphere. Its development is due to non-properly functioning eustachian tube. If the tubal lining is oedematous or contains excessive lymphoid tissue, locking occurs at a smaller difference of pressure than normal. *Pathology.* The tympanic membrane retracts inwards. Vascular engorgement occurs throughout the cleft lining. Oedema, ecchymosis and transudation of serum follow. *Clinical features.* Discomfort and pain which disappear in few hours. Deafness may remain for several days. Tinnitus is common, vertigo is infrequent. Otoscopy shows different pictures from the mildest reddening over the malleus handle until the most severe perforation of the tympanic membrane.

Treatment. Decongestant nasal drops ("unlocking the eustachian tube"). Myringotomy if fluid is present in the middle ear cavity. Systemic antibiotics are necessary to prevent infection.

1.7.3. Otitis media.

1.7.3.1. Acute suppurative otitis media.

An acute infection can spread very rapidly over the whole lining of the middle ear cleft, but the symptomes form an ordered progression suggesting successive infection of separate sites. The type of the inflammatory reaction and its progress depends on the virulence of the infecting organism, the resistance and age of the patient, and also on the drainage and therapy. *Etiology.* The cleft becomes infected by 1. extension of nasopharyngitis 2. direct spread via an infected surface exsudate. The common precursors, mostly virus infections, are: 1. Rhinitis; 2. Sinusitis; 3. Nasopharyngitis; 4. Pharyngitis and tonsillitis; 5. Influenza; 6. Nasopharyngeal tumors. *Bacteriology.* The commonest organisms are: Haemolytic streptococcus; Streptococcus pneumoniae; Staphylococcus aureus; Haemophilus influenzae. *Pathology.* Tubal occlusion occurs quickly and then oedema of the cleft lining which is followed by exsudation. The fluid forms in the middle ear and in the mastoid cells may be : 1. serous first, 2. Mucopurulent later. Bulging of tympanic membrane proceeds to the point of rupture. Rupture is due to pressure necrosis, and the fluid escapes as an otorrhoea. *Symptomes.* In the phase of an acute tubal occlusion (**First phase, incipient otitis**): Fullnes in the ear is felt; Deafness, which is conductive in type with autophony; Retraction of tympanic membrane may be seen (**Fig. 6. C**). In a **second phase** there is an acute infection of tympanic cavity. Before perforation: deafness, bubbling sounds, discomfort. The leading symptome is pain. The picture of the tympanic membrane: dilated blood vessels around the handle of malleus and imbulgement of the drum towards the external auditory canal (**Fig. 6. A**). After perforation (Fig 6. A **Third phase**): otorrhoea, and pain relief are significant. When pus retents in the mastoid, pain develops; tenderness can be elicited above the mastoid antrum. Oedema is characteristic at the posterosuperior wall of the deep external ear

Treatment is dependent on the stage of the otitis characterized by the otoscopic picture of the drum. Retracted and vascularized tympanic membrane is sign of early inflammation (and negative intratympanic pressure) and may be treated conservatively by antibiotics (preferably cephalosporin or amoxycillin plus clavulonic acid), nasal decongestant drops and antihistamin. Imbulging tympanic membrane towards the external auditory meatus means exsudation in

the middle ear cavity and requires myringotomy under short-term general anaesthesia. Additionally, those medicines are required to administer as in the early cases. In discharging ear, after spontaneous rupture of the drum, patients need aural toilette plus nasal drops and antihistamin. When recovery is slower than one week, systemic antibiotics are given.

Sequelae. The healing may be comlete with return of hearing to normal. Open perforations may remain. Their position is non-marginal. An ear discharing longer than one month should be operated on. Surgery is mastoidectomy and when needed is completed with myringoplasty.

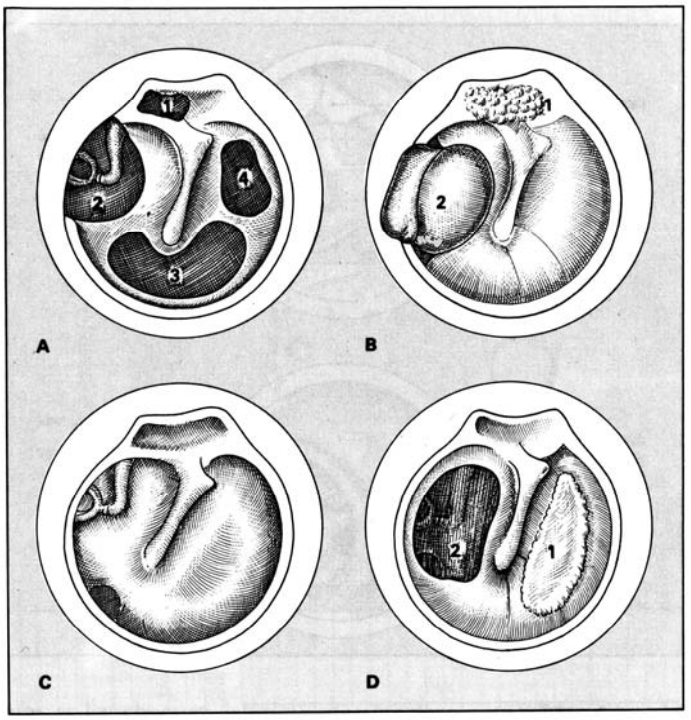


Figure 14. Otoscopic viewes of different tympanic membrane pathologies. (A) perforations: 1: Shrapnelle's membrane perforation; 2: poserosupperior marginal perforation: 3,4: tipical perforations for mesotympanic chronic suppurative otitis media; these locations may be presented as fused big, central, kidney shaped perforations. (B) the high risk type perforations; 1: cholestelotoma in the attic presenting a Shrapnelle's membrane perforation; 2: polyp in the posterior quadrante is also suspect for cholesteotoma. (C) adhesive otitis media is due to middle ear atelectasis; the drum is retracted and fixed to the promontory wall. (D/1) tympanosclerosis, when choke like substance appear in the drum or in the middle ear mucosa.

1.7.3.2. Infection of the pneumatized petrous apex (petrositis)

Suppuration in the petrous bone is usually accompanied by a serous meningitis generally in the middle cranial fossa. Later the intrapetrous abscess perforates and froms an extradural abscess. Septic meningitis follows. *Clinical features. The* ***GRADENIGO'S SYNDROME****:* 1. deep temporal and retro-orbital pain (excitation of the ganglion trigemini) ; 2. otorrhoea; 3. diplopia (paralysis of lateral rectus muscle).
Treatment. Mastoidectomy, systemic antibiotics, drainage.

1.7.3.3. Acute catarrhal otitis media

The disease most often associates to inflammations in the upper airways (rhinitis, nasopharyngitis). The presenting symptomes are fullness sensation in one or both ears, moderate hearing loss of conductive type, autophony and scratching sensation. No pain or perforation is present. Usually the general condition of the patients are dependent upon the upper airway inflammatory disease. Tympanometry shows negative pressure in the middle ear. Treatment is nasal decongestant drops, antihistamines.

1.7.3.4. Chronic non-suppurative otitis media (Synonyms: glue ear, otitis with effusion, secretory otitis media, catarrhal otitis media, serous otitis media)

This term is applied to the clinical conditions characterized by the presence of a non-purulent fluid in the middle ear cleft. Conductive deafness is the principal feature. Acute and chronic forms can sometimes be distinguished according to the mode of onset or by their duration, but distinctions may not be clear and the condition is often recurrent. The disease is typically of preschool-aged children. *Etiology.* 1. Occlusion of the Eustachian tube. May result from: adenoids ; tubal infection as an extention of a bacterial infection in the upper respiratory tract; Thick mucoid fluid plugging the tubal isthmus; paralysis of the palatal muscles and nasopharyngeal tumors. 2. Unresolved acute otitis media. Infection which is inactivated but not entirely resolved, either from failure of natural immunity or from inadequate antibiotic therapy. 3. Viral infections of the middle ear cleft lining mucosa. The adenoviruses, rhinoviruses, RSV causing nasopharyngitis and rhinitis are considered likely to be responsible for many cases of non-suppurative otitis media. 4. Allergy. 5. Cleft palate (even if the pterygoid hamulae are fractured, cause poor Eustachian tube function. *Clinical features. Deafness*. Conductive in type. Frequently both ears are affected in children. There are 3 types of audiometric curves: 1. Retraction of tympanic membrane causes increased stiffness leading to hearing loss at low frequencies and negative intratympanic pressure by tympanometry. 2. Subsequent effusion adds mass to the tympanic membrane causing flattened type conductive hearing loss (air-bone gap at all frequencies). 3. Equalization of the negative intratympanic pressure occurs when the effusion increases so that there is increased mass but without increased stiffness. There is a high tone loss only. *Tinnitus.* Buzzing and whistling. Vertigo and pain are uncommon. *Otoscopy*. The tympanic membrane is usually retracted, the malleus short process is prominent, the handle is foreshortened and more horizontal. When the

middle ear cavity is filled with fluid the tympanic membrane may not be distorted. Partial filling produces a meniscus which appears as a horizontally disposed crescentic hairline. Bubbles, even to the extent of foam formation, may be seen in the fluid. (Fig. 6. B)

The color of the tympanic membrane depends on that of the effusion behind it and is usually pale yellow. On release of suction applied by use of Siegle's pneumatic speculum there is a characteristic snap back of the tympanic membrane owing to the surface tension of the fluid. **Diagnosis**. The condition is suspected in all children suffering from varieties of the "tonsils and adenoid syndrome" and in adults when conductive deafness influenza or a head cold. A meniscus, bubbles or fluid found on myringotomy confirm it. The diagnosis is audiological. Best measure is tympanometry. The tympanogram is Type B. **Treatment**. Etiological or predisposing factors should be eliminated first (adenotomy). Surgical measures are necessary if the effusion persists. *Myringotomy* and evacuation of fluid, under general anaesthesia, using operating microscope. Adenotomy and myringotomy are usually enough if the fluid was serous (the less viscous type). In glue ear, indwelling teflon tubes or grommets are commonly inserted **(Fig. 15)** through the eardrum and left in position until rejected spontaneously, usually after 4-8 months. Mastoidectomy is indicated rarely. Recurrencies occur in about 30% of cases.

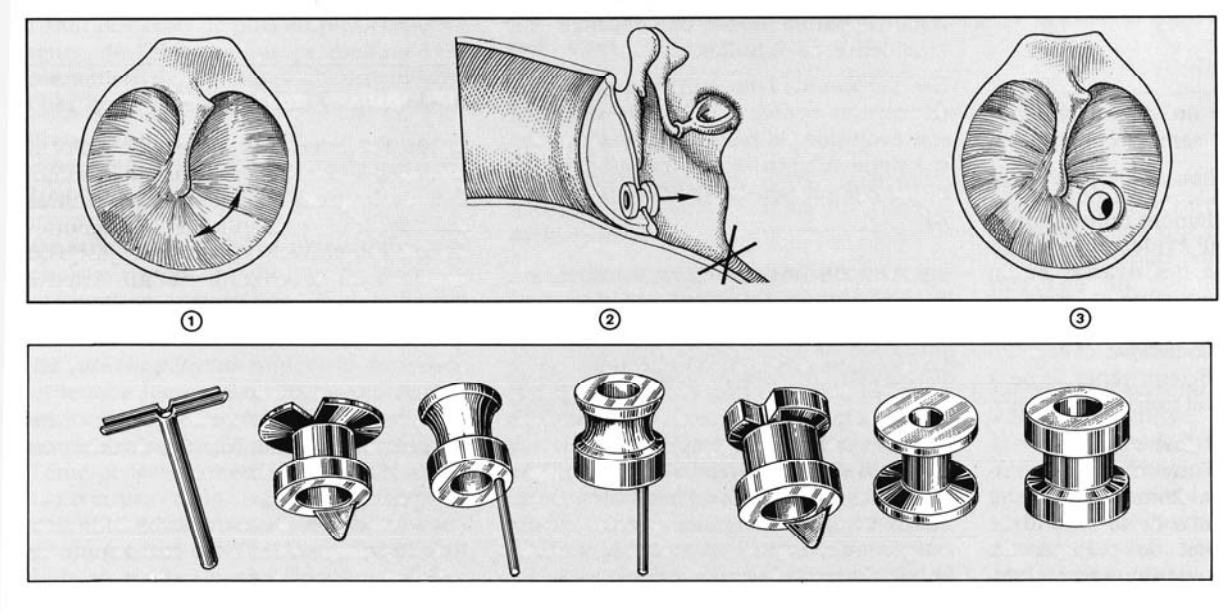


Figure 15. Insertion of ventilation tube (gromet) in the anterior-inferior qudrant of the eardrum. Lower panel shows different types of gromets. The gromets are made from silicone, titanium, gold, stainless steel, polyethylene.

1.7.3.5. Chronic suppurative otitis media

Clinical types.

1. **Mesotympanic chronic suppurative otitis media (Tubotympanal chronic otitis media)**
2. **Epitympanic chronic suppurative otitis media** or **chronic suppurative cholesteatomatous otitis media (attico-antral chronic otitis media)**

Mesotympanic chronic suppurative otitis media (Tubotympanal chronic otitis media)

Regarded as a safe, mucosal inflammation. *Etiology.* This is the residue of recurrent acute suppurative infections which is usually acquired in infancy or early childhood. Lack of attention to re-infections either from the nasopharynx (in adenoids or cleft palate) or through the perforation allow active infecion to persist or to recur. In most cases disorders in the Eustachian tube function can

be found. *Pathology.* The perforation of the tympanic membrane is situated in the pars tensa sparing the fibrous annulus, frequently antero-inferiorly. The ossicular chain often remains intact. The mucosa of the middle ear is pink and may be oedematous and so can appear in the external ear canal as polyp. Areas of ulceration and cholesterol granulation tissue may occure, although rarely. Areas of normal pavement cell epithelium may be replaced by columnar secreting (goblet) cells either by metaplasia or by extension of the mucosa from the eustachian tube. The mastoid is usually acellular. The mucosa of the mastoid cells may show some changes similar to those of the middle ear and the cells may act as a reservoir for secretions. **Symptomes. Central permanent perforation of the tympanic membrane (Fig. 14). Aural discharge** may be present or not. If present it is often mucoid or scanty and intermittent and may become purulent. **Deafness**. Conductive in type. Usually slight when the tympanic membrane perforation is small and anterior. Sometimes hearing is better when the ear is moist than when dry owing to a favourable shielding of the labyrinthine windows by mucus. Pain is unusual but can be present in the ear. *Diagnosis* is established by otoscopic examination and by Schüller's view x-ray (showing non-pneumatized mastoid). Pure tone threshold audiometry (PTA) clarifies the involvement of the ossicular chain. The greater the air-bone gap than 30-40 dB the higher the probability of the incus erosion.

Treatment. Topical antibiotics are given when the discharge is active. Aural toilette should be performed meticulously. Elimination of adjecent foci of infections if present in tonsils, adenoids and sinuses. Mastoid operation is required in those cases when disharge continues inspite of medical treatment or recurrs intermittently. **Mastoidectomy** should be done to eradicate the reservoire of infection in the cells in these cases. This is the operation for acute and chronic mastoiditis (sclerotized mastoid process). It aims at wide exenteration of the entire cellular system of the mastoid. After exposure of the antrum, cells are exenterated from the antral region, root of zygoma, mastoid tip and sinodural angle. The posterior bony wall of the external auditory meatus is left intact (**Fig. 16A)**.

Myringoplasty alone may be peformed for a persistent dry perforation. **Ossiculoplasty** can be performed at the same time.

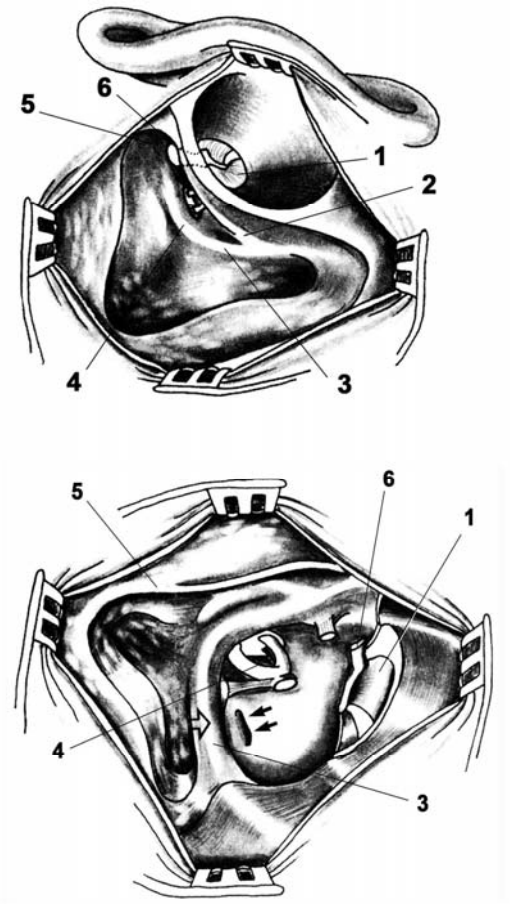


Figure 16. Upper panel Mastoidectomy with canal wall-up technique. The stapedial niche is exposed by drilling away the posterior bony canal-wall above the facial nerve-canal; **Lower panel** Mastoidectomy of the canal wall-down technique (radical mastoidectomy). Open arrow shows the facial nerve canal, dark arrow heads show the round window, here the stapedial niche is fully exposed by making a common cavity from the middle ear, the external auditory canal and the mastoid cavity. 1: drum; 2: chorda tympany; 3: facial nerve; 4: stapes, 5: tegmen antry; 6: mallouse

Epitympanic chronic suppurative otitis media or **chronic suppurative cholesteatomatous otitis media (attico-antral chronic otitis media)**

Associated with cholesteatoma and regarded as dangerous because of bone destruction and osteitis versus the mesotympanic chronic otitis, which is a mucosal disease. Granulation tissue (polyp) may be present. This disease has a very high recurrence rate and although it is not a real tumor it grows expansively distroys surrounding bony structures and causes very often complications. Some of them are life-thretening.

Definitions. **Cholesteatoma:** this is an epidermoid cyst containing layers of keratin. **Cholesterol granuloma:** a non-specific granulation tissue. Cholesterol crystals, foreing-body giant cells, and blood pigments (haemosiderin) are present.

Theories of the pathogenesis of cholesteatoma.

1. Congenital (primary, or genuine) cholesteatoma (Fig. 17). Arises from embryonic epithelial tissue. The diagnoses are confirmed by CT scan and surgery. Genuine cholesteatoma occurs without perforation of the tympanic membrane and without previous history of discharge, myringotomy or middle ear trauma.

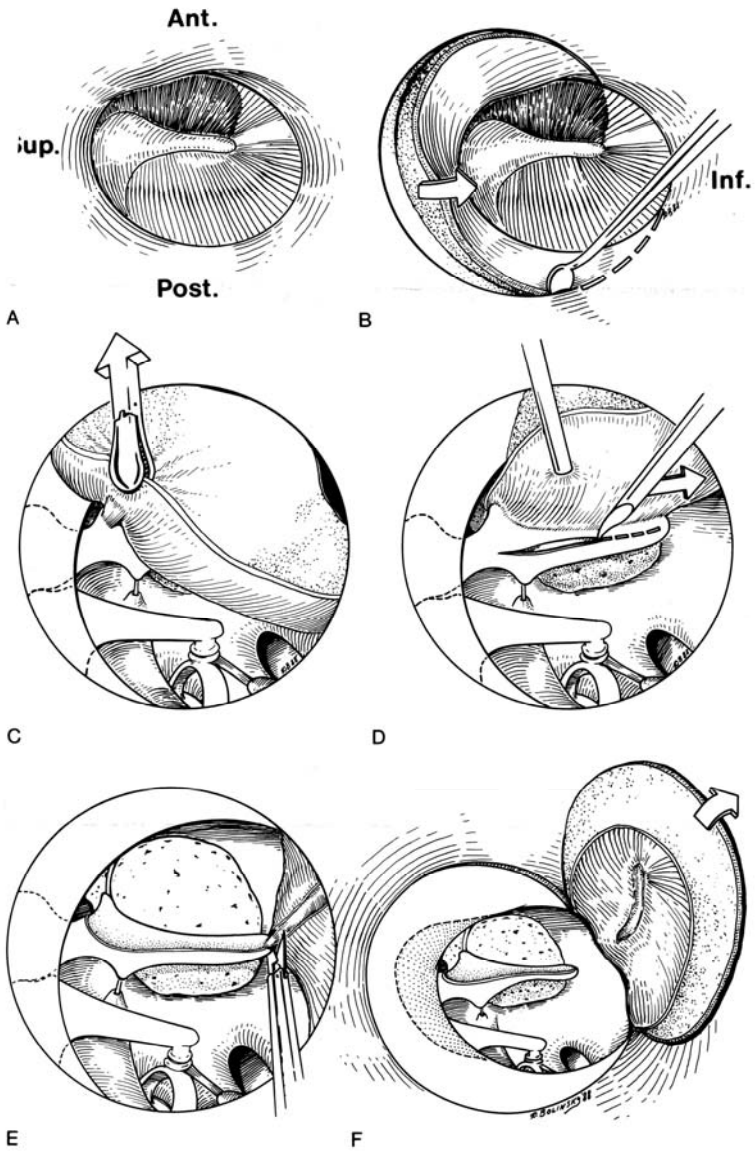


Figure 17. Genuin cholesteatoma anterior and medial to the malleus handle (E-F) presenting no perforation of the drum. Elevation of the drum (A-D) gets it in view. Right ear as the patient is in supine position. The pictures mimick the position of the ear in surgery. 1: malleus,2: incus, 3: stapes, 4: promontory wall, 5: genuine cholesteatoma

2. Acquired (secondary) cholestetatoma. In most cases it arises from retraction pockets in the pars tensa of the tympanic membrane or invaginations in the Shrapnell membrane (this is the flaccid part of the tympanic membrane, which borders the attic laterally).

Theories upon the development of the acquired cholesteatoma.

1. **Implantation** theory.
Rare but a well defined cause of cholesteatoma is the traumatic implantation of squamous epithelium of the external auditory canal into the middle ear cavity. Also the dermal grafts used to apply for myringoplasty surgery in the past induced cholesteatoma. A relatively unsual complication of gromet insertion is cholesteatoma in the same way.
2. **Migration** theory.
The outer layer of the tympanic membrane (squamous epithelium) covering the pars flaccida and the malleus handle show a migration activity in posterosuperior direction and it has a very high proliferation activity. Cholesteatoma is also produced most frequently in the same area may be due to a migration activity in the middle ear cavity direction by an unknown etiology.
3. **Metaplasia** theory.
It is believed that the cubic cells of the normal middle ear cavity lining somehow changes to squamous epithelium by metaplasia similarly to other locations in the human body (nasal cavity, paranasal sinuses, bronchi) where chronic irritation and infections induce a metaplastic change of the mucosa to squamous epithelium. This theory assumes that a papillary invagination may be the origine of cholesteatoma without perforation of the tympanic membrane.
4. **Invagination** theory.
This is the most pupolar theory. Eustachian tube malfunctioning and retraction pocket is addressed as basic mechanism in the development of cholesteatoma.
Pathology. The encysted and concentrically laminated keratine which forms the cholesteatoma is at first limited to the attic. The capsule or matrix of the cyst is covered by tympanic mucosa at any surface projecting freely within the lumen of the cleft. Increase in the amount of accumulated keratin may lead to the following pathological processes:
a/ extrusion of small attic or antral cholesteatoma into the external ear;
b/ Protrusion of finger-like processes of the cyst into the tympanic cavity; c/ filling of the tympanic cavity by the sac;
d/ Encroachment into the mastoid structure as the sac progresses from the antral region;
e/ Provision of pathways facilitating the spread of infection.

Clinical features. **Deafness** which is conductive in type. **Malodorous otorrhoea**. **Perforation**. Situtated in Shrapnell's membrane or marginally in the posterosuperior quadrant of the pars tensa with destruction of the fibrous annulus. **Cholesteatoma** may be visible through the perforation as a greyish paperlike substance or as the typical pearly sheets of keratin which are easily recognized. **Headache, earache, vertigo and facial paralysis** are signs of complications. Suction-clearance under operating microscope is desirable for proper viewing in doubtful cases. *Radiography* shows an acellular mastoid. CT scanning of the temporal bone visualizes the cholesteatoma matrix.

Treatment. 1. Conservative: not to be done

2. Surgical: **Radical mastoidectomy** (Fig. 16) also called “canal wall-down” or “open technique” has to be done in those cases where the cholesteatoma matrix is found in the aditus of antrum and in the stapedial niche. The functional result of the surgery is a serious conductive hearing loss of about 50-60 dB which is sometimes greater than the hearing loss of the patient was preoperatively (in these cases the cholesteatoma matrix itself behaves as an interpositum between the stapedial footplate and the remnant of the drum). During surgery, after exposure of the antrum, the outer wall of the attic is taken down and the posterior canal wall is reduced to create the "facial ridge" overlying the nerve. Finally the bridge, formed by the outer wall of the aditus is removed. Sometimes this is referred to as the "canal wall down" technique. Any remains of the ossicles and tympanic membrane are picked out. A kidney-shaped cavity results (comprising the mastoid cavity, the middle ear cavity and the external auditory canal) which is then partially lined by a flap of skin derived from the external auditory meatus. This surgical procedure aims to provide with an externally (otoscopy) controllable common cavity from those cavities and recesses where cholesteatoma may recurre. Since the conductive apparatus in the middle ear is sacrificed this surgery results in about 60 dB conductive hearing loss. However, when aural discharge and possibility of cholesteatoma recurrence was passed, a reconstructive tympanoplasty can be performed and the hearing of the patients can be given back. The minimum period of aural discharge-free time after radical mastoidectomy to consider the hearing conservation surgery (tympanoplasty) is 1 year.

A **conservative radical mastoidectomy** means that the ossicular chain is saved and it is the modality of choice in cases of small cholesteatomas. The **canal wall-up technique,** also called Jansen’s operation, saves the bony bridge between the posterior external auditory canal and the tegmen antri and tympani. This is not recommended however because the recurrence rate increases and the postoperative situation does not allow direct visualization of the surgical cavity (this procedure is also called as “close technique”). As a **second step surgery** tympanoplasty can be considered if the ear was non-discharging for 1 year. This

is a plastic reconstruction of the tympanic apparatus consists of reconstruction of the deficient tympanic membrane (myringoplasty) plus reconstruction of the sound conducting ossicular chain (ossiculoplasty) (**Figure 18 -19**).

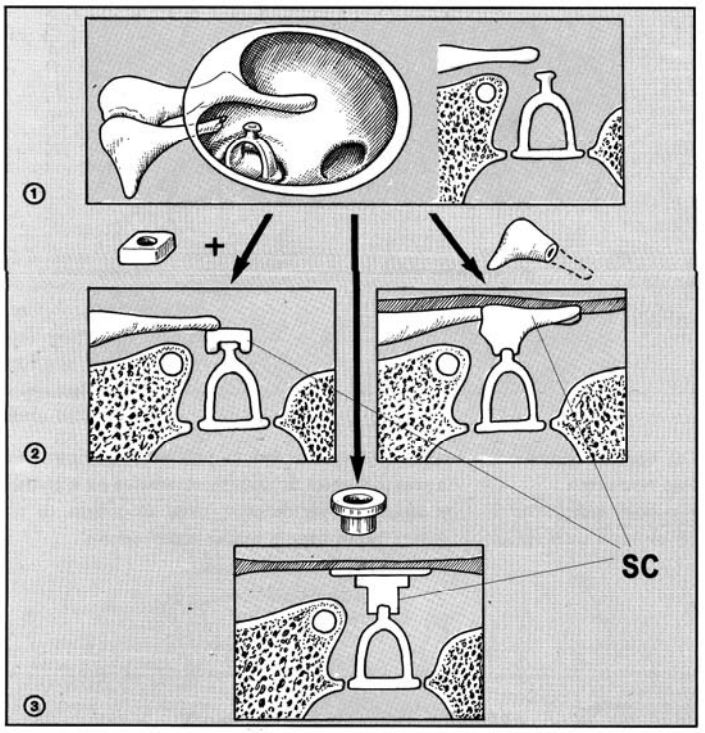


Figure 18. Tympanoplasty (short columella) 1. the long process of the incus is missing. 2. A short interpositum (columella) is placed between the head of the stapes and the malleus. 3.A ventilation tube may also be inserted at the same time in atelectatic middle ear **SC**: short columella

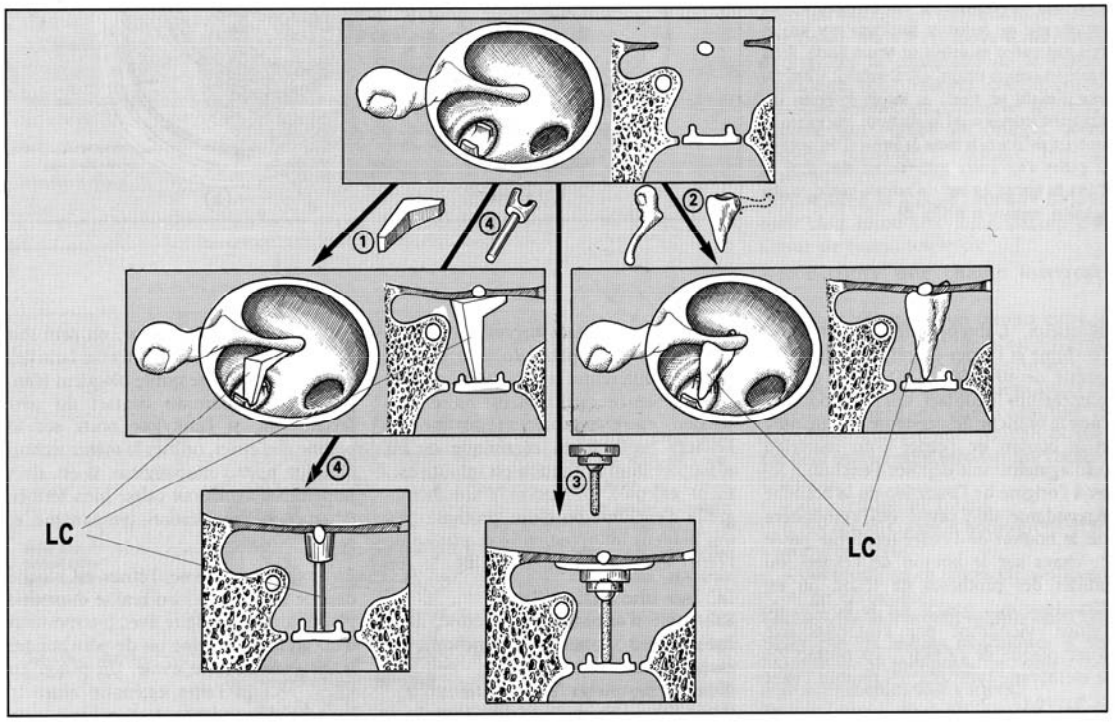


Figure 19. Tympanoplasty (long columella). The stapes superstructure and the incus are missing. A long interpositum (bone, bioceramics) is placed between the stapes footplate and the malleus**. LC**: long columella It can be made from costical bone (1) commercialized bioceramic or titanium (3,4) or from autologous malleus or incus (2).

1.7.3.6. Complications of suppurative chronic otitis media.

Spread of infection beyond the bony walls of the cleft may occur during acute or chronic otitis media. Serious intracranial complications are most frequently seen during acute exacerbations of chronic suppurative otitis with cholesteatoma. General rule in the treatment of complicated chronic supuurative otitis media is that it should be surgically treated. The surgery is radical mastoidectomy and evacuation of pus from the temporal bone and from the middle and posterior cranial fossas.

Mode of spread of infection. 1.Direct spread through bone caused by osteitis, rarely osteomyelitis, caries and erosion by cholesteatoma. 2. *Venous spread.*Venous channels convey the septic process by retrograde thrombophlebitis. 3.*Spread via labyrinth.* Since the labyrinth itself becomes infected first through the windows or a fistula, the infections can spread further towards the internal auditory meatus, the vestibular aqueduct, fracture lines.

Types of complications.

1. **Mastoiditis** (subperiosteal abscess). This is commonest type, especially in children. The auricle is displaced outwards and forwards. Pus has often tracked outwards through minute vascular channels in the suprameatal (Macewen's) triangle. In acute case: fever, tenderness above the mastoid process, and opacity in Schüller's view x-ray above the petrous bone are characteristic. Otoscopic view will demonstrate a prolapse of the postero-superior ear canal wall as a sign of penetrating osteomyelitis from antrum.
2. **von Bezold's abscess**. Perforation of the tip or inner surface of the mastoid may give rise to an abscess in the sternomastoid muscle or in the digastric (Citelli's) triangle.
3. **Epidural abscess** is usually caused by a direct extension of infection from adjacent mastoiditis. It occures with both acute and chronic infections of the middle ear cleft. The collection of pus is usually well encapsulated but on occasion it may spread widely. The symptomes are sometimes indeterminate and the finding of such an abscess is usually made by chance at operation. However, may exactly mimic a brain abscess. A CT scan will clearly differentiate an intracerebral abscess from an extradural collection of pus, however, will less readily distinguish extradural from subdural abscess.
4. **Subdural abscess.** Collections of pus in this situation may occasionally travel widely over the surface of the brain and give little evidence of their presence as cause of intractable headache, fever and progressively severe malaise. The diagnosis may be one of the most difficult in neurology and is often made by chance in the investigation of severe headache without brain damage and fever of unknown cause. CT scan may be crucial for diagnosis.

Treatment is similar to that of epidural abscess. Evacuation of pus through a burr hole or a craniotomy is necessary

5. **Labyrinthitis**. The symptomes are: fever, sensorineural hearing loss, headache, balance disturbances, somnolence, aural discharge. For description in detail, see chapter 1.9.7.

6. **Meningitis** is acute inflammation of the leptomeninges (arachnoid and pia mater). Infection may reach the meninges in direct way (by contiguous spread, such as osteitis, by communicating blood vessels, by the labyrinth), or via the bloodstream. High fever is usually present with headache. Neck stiffness may increase to neck retraction. Rigidity is demonstrated, by first establishing no resistance in side-to-side neck movements, and then attempting a gentle bimanual flexion of the head, when a spasm of the neck extensors may be felt. Kernig's sign: extension at the knee, with the hip joint flexed at a right angle, is painful and limited in both sides. Altered state of consciousness is frequently present. Photophobia, vomiting, cranial nerve palsies, papilloedema, scaphoid abdomen, and the cerebrospinal fluid chemistry (pleocytosis, reduced sugar, increased protein) are diagnostic for the disease. Should be considered very seriously because it can be lethal as today. **Treatment**: large dosis of Penicilline intravenously as continuous infusion or intrathecally. Analgesics, sedatives and ample fluid intake are necessary. Ear surgery in suspected otogenic cases is necessary.

7. **Brain abscess**. **Temporal lobe** abscess results in speech diorders (exclusively sensory aphasia), visual and acoustic hallucinations, central hearing deafness, disorders in smell, gaze paresis and hemianopsia, crossed lesions of the pyramidal tract.

Cerebellar abscess. Includes disorders of the oculomotor and postural system. There is usually a coarse spontaneous nystagmus to the side of the lesion. Even gaze-directional or gaze-paretic nystagmus to the side of the lesion can occur due to pons damage. Further symptomes are dysdiadochokinesis, ataxia, dysmetria, intention tremor, paralysis of the IIIrd, IVth, VIIth, IXth and Xth cranial nerves.

8. **Sigmoid sinus thrombosis**. Infections due to mastoiditis or cholesteatoma destroys bone in continuity of the sinus so that it can break into the perisinus space. This forms a periphlebitis followed by a sigmoid sinus phlebitis. The thrombus initially is mural and later occludes the lumen and extends towards the transverse and sagittal sinuses superiorly, and towards the internal jugular vein inferiorly. Since the thrombus undergoes thrombolysis, blood-borne infected emboli can metastatize in parenchymal organs (kidney, lung, liver). Symptomes are : spiking temperature chart, nausea, vomiting, neck stiffness, somnolence, dyspnea. Diagnosis may be made in the knowledge of high fever, swelling and sensitivity above the mastoid. The classic **Queckenstedt-test** [compression

applied to the internal jugular vein, cerebrospinal fluid (CSF) pressure increases in normal cases or in the non-affected side, versus, on the affected side, the sigmoideus thrombus blocks this effect; the test should be done with neurologist who provide the lumbar puncture and CSF manometry]. Circulation block in the sinus today can be imaged by color laser Doppler and MR.

1.7.3.7.Tympanosclerosis.

A white snow-like layered thickening produced by calcification of the fibrous tissue of the lining mucosa of the middle ear cleft which has undergone hyaline degeneration, initiated by infection. A calcareous plaque of chalk patch in the tympanic membrane is also the result of this process. Surgical removal of tympanosclerosis from the region of the oval window is not infrequently followed by a sensorineural hearing loss.

1.7.3.8. Chronic adhesive otitis media.

The formation of adhesions in the middle ear (**Fig. 14. C)** may follow either suppurative otitis media or non-suppurative otitis. However, fluid is known to have existed in the tympanic cavity for years without the formation of adhesions. The tympanic membrane may show areas of both atrophy and of thickening and is frequently adherent to the promontorium. This condition is often seen after repeated insertion of grommet. The otoscopic picture may mimic perforation. **Treatment**. The recurrence of adhesions after division may sometimes be prevented by the use of Silastic or other plastic sheeting. Myringoplasty with cartilage slice gives the best results. A hearing aid may improve the patients hearing in unsuccesful cases.

1.7.3.9. Tuberculous otitis media.

This increasingly rare disease is secondary to severe pulmonary tuberculosis. It is generally carried up the Eustachian tube from the nasopharynx. *Pathology.* Tubercle formation and caseation occure in the submucosa of the middle ear cleft. *Multiple perforations* develop quietly and without pain. Quiet caries of the mastoid and ossicles. This may lead to *facial paralysis*. A mild chronic type of *labyrinthitis* may be a further complication. *Symptomes.* Conductive deafness and otorrhoea are charcteristically so insidious that the complications may first call attention to the condition. **Diagnosis**. A painless conductive deafness and otorrhoea, a multiply perforated tympanic membrane, detection of Koch's bacilli in the aural discharge are signs of tuberculous otitis. **Treatment**. Systemic antituberculous chemotherapy which is supported by: aural toilet when

otorrhoea is present and mastoidectomy when sequestration or other complications are present.

1.7.3.10. Syphilitic otitis media.

The usual aural manifestation of syphilis is a meningoneurolabyrinthitis, but occasionally the middle ear cleft is involved by a gummatous osteoperiostitis. The combination of middle and inner ear infection is known as an *otolabyrinthitis* (see more in **1.9.7**). In these cases the vestibular organs are always "dead" before an otorrhoea is evident. The gummatous change leads to extensive destruction of the mastoid. **Diagnosis**. Foul, painless otorrhoea, in the presence of sensorineural deafness is suspicious. Congenital stigmata or positive serological tests are confirmatory. **Treatment**. Antisyphilitic is required. Surgery may be needed for removal of sequestra demonstrated radiologically.

1.7.4. Neoplasms of the middle ear cleft

All are realtively uncommon. Squamous-cell carcinoma is the most frequent and glomus tumor is the next.

Glomus tumor (chemodectoma). This is a very slowly growing tumor arising from non-chromaffin paraganglionic (chemoreceptor) tissue situated in, or close to the middle ear. Metastasis are rare. *Pathology.* The tumor may originate from the tympanic branch of the IXth cranial nerve on the promontory (glomus tympanicum) or from the glomus bodies of the dome of the jugular bulb (glomus jugulare), or from the paraganglion juxtavagale of the Xth cranial nerve. The tumor consists of non-myelinated nerve fibres and vessels vary in size from capillaries to cavernous spaces. *Clinical features.* Tympanic tumors give aural symptomes first, jugular tumors present the syndrome of IXth, Xth, XIth and perhaps XIIth cranial nerve paralysis. Deafness is the earliest symptome in tympanic tumors, it is conductive in type. Tinnitus is also an early sign and is swishing in type. Pain and vertigo sometimes present but are mild. Red flush with or without bulging of the tympanic membrane is the first sign. Myringotomy decides the question of whether it is a cholesterol granulation or glomus tumor. The latter may be confirmed by pulsation under otoscope and microscope. **Diagnosis**. High definition CT scan shows erosion of the temporal bone. Since biopsy may produce severe haemorrhage, the otoscopic picture and the CT examination are usually considered to be diagnostic to perform surgery. Cases with neurological signs or with involvement of the internal carotid artery are best treated by telecobalt irradiation.

Carcinoma. Usually a squamous-cell carcinoma, rarely adenocarcinoma. *Clinical features.* Bloodstained otorrhoea is usually present. Granulations or

polypi bleed readily on touch. This should always arouse suspicion of malignancy. Pain may be absent first and intense later from meningeal involvement. Deafness is conductive in type. Facial palsy is of lower motor neurone type. **Diagnosis**. Biopsy is taken and histological examination distinguishes it from inflammatory conditions. Radiographic examinations may show extention of the bony erosion. **Treatment**. *Radiotherapy* and surgery which usually is a radical mastoidectomy.

1.8. Diaseases of the otic capsule

1.8.1. Otosclerosis.

A localized disease of the cochlear bony capsule in which a newly formed sclerotic bone causes ankylosis of the footplate of the stapes. Otospongiosis is more descriptive for the initial period of the disease. Well known race differences are higher incidence in white peaple and practical absence in mongolian race. *Etiology.* Sometimes there is a family history. The sex incidence is about four times in female than in the male. Clinical manifestations begin between 20 and 40 years of age. It rarely starts before 10 years which are rather congenital anomalies. Pregnancy may accelerate the process but never causes it. Persistent **measles** virus infection is assumed to induce otosclerosis. Unknown is, however, why only in the stapes footplate and the otic capsule are targets of the infection. *Pathology.* The commonest site of induction of the otosclerotic focus is on the tympanic promontory, anterior to the oval window (fissula antefenestram). Cartilaginous remnants are demonstrated there. Its proximity to the stapedial footplate explains the ankylosis develops later. Spongy otosclerotic bone is formed in stages. *Clinical features.* Conductive deafness is the prominent symptom and is usually bilateral. It may be limited to one ear at first. Sensorineural high tone deafness develops later due to spreading of the otosclerotic disease to the cochlea. Rarely, a sensorineural deafness may be the first manifestation when the osteoid bone invades the cochlea rather than the footplate. The deafness becomes an appreciable handicap when loss of hearing by air conduction exceeds 30 dB in the better hearing ear. *Paracusis (Willisii)* is often experienced (the patient hears better in noise). Tinnitus is nearly always present and low tone in character. The tympanic membrane is normal looking, but occasionally a "pink" tinge (Schwartze's sign) may be seen through the membrane, due to hypervascularization of the promontory. The Eustachian tube is patent. *Gelle's test* is a characteristic tuning fork test in otosclerosis. Should be performed by the C_3 tuning fork. The otosclerotic patient does not hear the vibrating tuning fork less loud when the air pressure in the external ear canal is increased. *Pure-tone audiometry* shows an upward slope to the right (low-frequency hearing loss), but with cochlear involvement,

there is a downward slope to the right (high frequencies). The bone conduction curve shows a dip at 2 kHz (Carhart's notch). *Stapedius reflex* is absent. *Tympanometry* shows reduced compliance and normal middle ear pressure. *Radiography*. High definition CT may show bone resorption foci around the bony cochlea.

Differential diagnosis. Ossicular disconnection or fixation. May be congenital, traumatic or inflammatory. Sensorineural deafness in young adults may be difficult to distinguish from otosclerosis involving the cochlea. **Treatment.** In pure conductive cases or in combined conductive and sensorineural deafness cases dominated by conductive deafness, the surgery is the good selection: stapedectomy or stapedotomy **(Fig. 20)**. Sensorineural deafness component of otosclerotic deafness applies medical treatment: sodium fluoride, Ipriflavone, Vitamin D + calcium. *Operative technique.* When the stapes is totally removed, the oval window should be covered by fascia or other connective tissue and a prosthesis placed between the incus and the sealed oval window. This can be substituted by the gel-foam-wire prosthesis (Schuknecht's prosthesis). When preferably, only a small hole drilled in the footplate, the prosthesis is a Teflon-wire, stainless-steel wire, gold or titanium piston . The surgery is usually performed under local anaesthesia. *Sequelae of operation.* Sensorineural hearing loss; vertigo (lasts for several days and disappears spontaneously); Damage to facial nerve (rare but if the facial nerve bony canal is uncovered around the oval window area the risk is high); Perilymph fistula; Infective labyrinthitis; Inexplicable failure to gain improvement in hearing; Meningitis.

1.8.2. Miscellaneous conditions of the otic capsule

Osteogenesis imperfecta is an autosomal dominant disease where collagen fails to mature into strong bone. Clinically fragile bones, blue sclerotics and deafness are characteristic. It presents as an otosclerotic deafness with immobile stapes.

Paget's disease (osteitis deformans) has aural manifestations. Deafness is a frequent symptome but is due to the disease itself and not to otosclerosis. Osteitis begins in the periosteal layer of the otic capsule. The deafness may be conductive possibly due to calcium deposition in the vestibulostapedial joint and consequently increased bulk of the ossicles, or may be sensorineural.

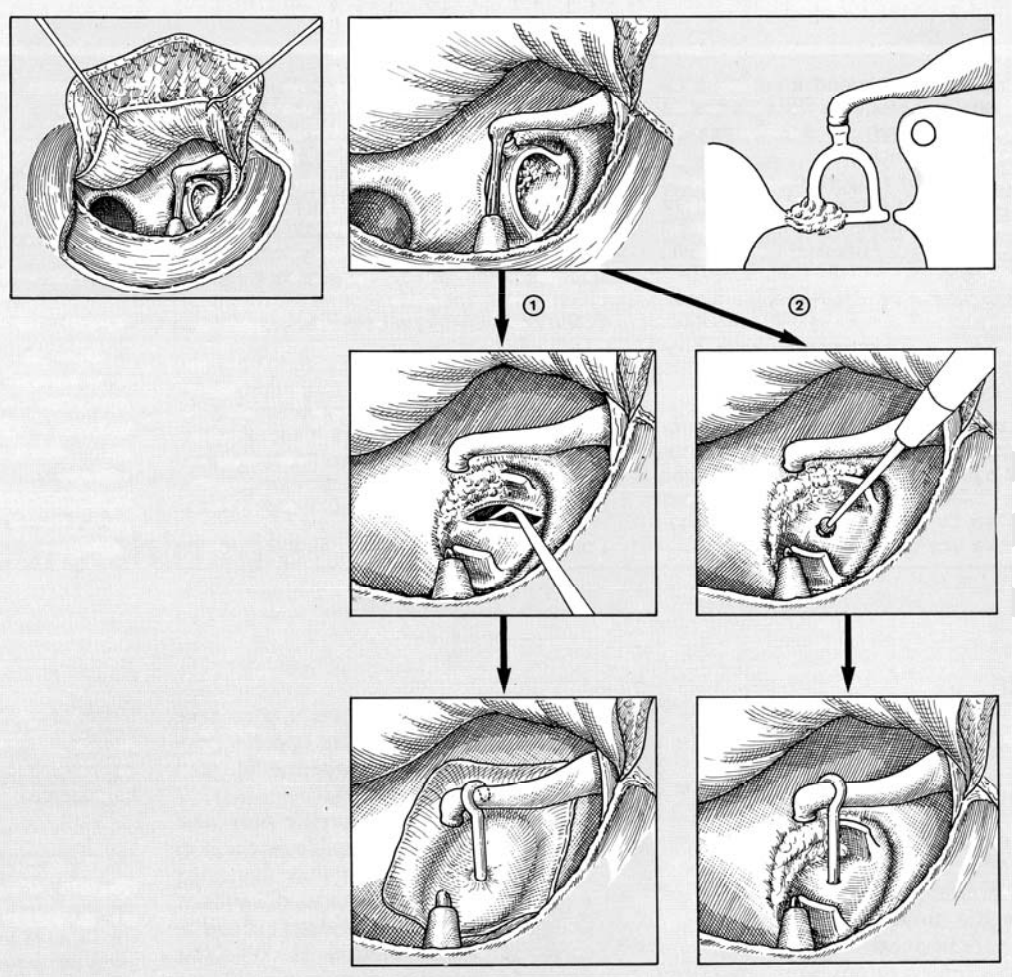


Figure 20. Surgery of otosclerosis. Upper pictures show microscopic and schematic view of an otosclerotic lesion. Middle panels show how to make a hole (1) in the stapes footplate by a hook or drill (2). Lower panels show the hole covered with fascia and a piston positioned on it and fixed to the long process of the incus.

1.9. Diseases of the inner ear.

1.9.1. Congenital malformations and deafness

The bony or membranous part of the cochlea may be absent or show rudimentary development. Several anatomical types are;
1. *Scheibe dysplasia*. The stria vascularis shows hypoplasia, the Reissner's membrane is collapsed with rudimentary organ of Corti. The cochlear neurons are spared and these patients apply for cochlear implantation.
2. *Mondini dysplasia.* The cochlear duct may be reduced to one turn; the organ of Corti may be absent or reduced. This is detectable by CT scanning.
3. *Bing-Siebenmann dysplasia.* There is a normal bony labyrinth but an underdeveloped membranous labyrinth, both cochlear and vestibular. It is often associated with retinitis pigmentosa (*Usher's syndrome*).
4. *Michel dysplasia*. Total absence of both labyrinths

1.9.2. Syndromes of hereditary deafness

Some hereditary, prenatal, natal and neonatal diseases can lead to sensorineural or conductive deafness. Due to maternal illness in the first 3 or 4 months of pregnancy can cause developmental changes in the cochlea of the fetus. The commonest cause are rubella, infuenza, drugs (aminoglycosides, thalidomide), congenital syphilis and cytomegalovirus. Among the neonatal forms **Kernicterus** from Rhesus incompatibility between infant (if Rh-positive) and mother (if Rh-negative) may lead to deafness, characteristically for high tones. There is usually a history of severe jaundice at or soon after birth. **Diagnosis.** May be very difficult but in severe cases should be possible by screeming infants of "risk groups" before the age of half a year. Most helpful is objective audiometry: a combination of measuring otoacoustic emissions and auditory evoked brainstem responses (ABR) with tympanometry and stapedius reflex threshold. **Treatment.** Severe or total deafness leads to deaf-mutism unless hearing is used to the uttermost with bilateral high powered hearing aids and special training is given. In the case of total deafness, cochlear implantation may help transmitting recognizable samples of speech sounds across a range of frequencies. The importance of the early training of a child born deaf is becoming increasingly recognized. The principles of special training include: auditory training, lip-reading, sign-language, speech-training, family guidance. The special training should be given by a trained Teacher of the Deaf.

1.9.3. Congenital hearing loss of genetic origin

These are congential deafness or sensorineural hearing loss cases. Two main types are syndromic and nonsyndromic hearing losses. **Syndromic hearing loss** appears with other abnormalities. The 2 classic types are the Waardenburg-, and Usher syndromes. Former is presented as hearing loss, hyperthelorism, heterochromic iris and albinism. Usher syndrome is association of hearing loss and pigmentous retinitis with or without oligophrenia. **Nonsyndromic hearing loss** is the more frequently presenting type. Several dominantly inherited and about 35 recessive hearing loss forms are known present time. Estimated putative number of recessive hearing losses caused by mutations in a single gene encoding specific proteins in the organ of Corti is approximately 160. GJB2 gene (gap junction protein – connexin- encoding gene) mutations are most common in the Caucasian race. One of these mutations, the point mutation 35delG, is responsible for about 40% of all nonsyndromic recessive hearing loss cases. Its frequency is 1‰ in Europe. A newborn hearing screening program is developed in the EC countries, which includes a transient otoacoustic emission (TOAE) measurement in postnatal time. No measurable TOAE requires repeated TOAE determination in 3 month which is followed by ABR measurement if again no response was obtained. Early screening of hearing loss or deafness provides with early hearing rehabilitation of infants, which is essential for normal mental development. These are hearing aids, cochlear and auditory brainstem implantations.

1.9.4. Fractures of the temporal bone

They are of two main types

Type 1. Longitudinal fracture. The fracture line is along the long axis of the petrous bone and involves the middle ear, the tympanic membrane and the ear canal. *Clinical features.* Conductive deafness. Bleeding from the ear canal or through a perforated tympanic membrane from the middle ear. If the fracture does not reach the tympanic membrane, the blood is retained in the middle ear cavity (haematotympanum) and shows as a black or blue drumhead. Facial paralysis may occur. Rarely cerebrospinal fluid otorrhoea is present.

Type 2. Transverse fracture (less common type). The fracture line runs at right angles to the long axis of the petrous bone involving the labyrinth and the internal auditory meatus. *Clinical features.* Sensorineural deafness. Vertigo and nystagmus until the loss of the homolateral vestibular function is compensated. Haematotympanum may be seen. Facial paralysis is not uncommon.

Treatment. The first instance is the treatment of the head injury. The ruptured tympanic membrane is at first strict non-interference. Failure of conductive

deafness to recover after temporal bone fracture suggests ossicular chain disconnection. In cases of immediate and total facial nerve paralysis surgical exploration must be considered. Prophylactic antibiotics are obligatory. When the fracture involves an ear which is the site of an active chronic suppurative otitis media, systemic antibiotic should be given and mastoidectomy is compulsory.
Complications. Haematotympanum. Tearing of the dura mater. Damage to facial nerve. **Treatment.** Antibiotics should be given systemically.

1.9.5. Noise-induced hearing loss.

This is an injury to the inner ear caused by prolonged exposure to loud noise in certain industrial occupations. Tinnitus is usually a feature. *Etiology.* Continued exposure to sounds above a sound pressure level of 85 dB is unsafe. Degree of deafness is proportionate to duration of exposure, though there is marked variation in individual susceptibility. Examples are to be found in many occupations (e.g. mining, drop forging, pile driving, chipping etc.). Noise induced damages in the inner ear are multiple. First the outer hair cells are injured. *Clinical features.* Initially after exposure to loud noise, there is a threshold elevation in the higher frequency range. After a period away from noise the hearing returns to normal. This is called noise induced **temporary threshold shift**. After some time a **permanent threshold shift** can be observed. Characteristically the hearing loss is greatest at 4 kHz (called "noise notch" on the audiogram). The reason of its development at this specific frequency is that the stapedius reflex doesn't protect above 2.7 kHz; the resonant frequency of the inner ear soft tissues are in the frequency range of 3-4 kHz. **Treatment.** Most importantly this is preventive: screening of personnel in noisy occupations, use of efficient earplugs or muffs, sound isolation in factories, resting and rehabilitation of personnel complaining of early symptomes. No therapeutic means exists, just arrest of progression is available by rest and avoidance of casual factors. **Noise and the law.** The law related to compensation for noise-induced hearing loss varies from country to country. The plaintiff may have a source of redress through compensation from the employer and/or social security provisions provided by the state. The American Academy of Otolaryngology-Head and Neck Surgery recommends that at 25 dB average of 0.5, 1, 2, and 4 kHz should be recognized as hearing handicap.

1.9.6. Caisson disease.

Follows too rapid decompression in diving and submarine work. Bubbles of nitrogen are released suddenly as emboli, leading to disruption with haemorrhages and subsequent necrosis of the organ of Corti. Sudden severe deafness, tinnitus and vertigo complained of, and are accompanied by vomiting. The middle ear is involved as well. Recovery is possible, but percentage of cures is uncertain.

1.9.7. Inflammatory conditions of the inner ear.

Otitic labyrinthitis. The inner ear is infected by extension of acute or chronic suppurative otitis media. *Etiology.* In acute ear disease, labyrinthitis either complicates acute suppurative otitis media or accompanies a myringitis bullosa haemorrhagica of virus origin. In chronic ear disease, this condition remains one of the commonest complications of chronic suppurative otitis media, in spite of antibiotics. Infection may result from: 1. Bony erosion (commonly from lateral semicircular canal; 2. Petrositis; 3. Trauma; 4. Direct spread (through the fenestra ovale or rotunda; this may occur in acute infections). *Pathology.* 1. **Circumscribed labyrinthitis**. This is a localized perilabyrinthine inflammatory process. Results from erosion of the bony wall of the horizontal semicircular canal. 2. Diffuse **serous labyrinthitis**. With only a few round cells in the perilymphatic space. In the above two types, there is no organisms in the perilymph. 3. Manifest diffuse **purulent labyrinthitis**, with massive purulent infection of the peri-, and endolymphatic spaces. 4. "**Dead labyrinth**", This is a later stage of the above, with obliteration by granulation or fibrous tissue later replaced by bone. *Clinical features.* **Vestibular irritation**: 1. Circumscribed labyrinthitis: vertigo, nystagmus (horizontal beats to the affected side), hearing loss, positive fistula test. 2. Diffuse serous labyrinthitis: vertigo, nausea, nystagmus (it is spontaneous, and increased by looking to the affected side), deafness.**Loss of cochlear and vestibular functions**: 1. Manifest diffuse purulent labyrinthitis. Vertigo, nausea and vomiting are marked. Toxaemia is present. Nystagmus is towards the non-affected side, absolute deafness, deep-seated temporal headache is common. 2. '**Dead' labyrinth**: total loss of cochlear and vestibular functions. The vestibular function is gradually compensated by the central nervous system.

Treatment. In acute infection: antibiotic therapy (large doses - 30-50 million unit -of penicilline intravenously) and sedation . In chronic infection, mastoid surgery also is necessary, for removal of cholesteatoma and exposure of fistula. Exploration of the petrous bone may be indicated.

Syphilitic labyrinthitis. Syphilitic deafness is rare except in the congenital and neurosyphilitic types of the disease. Approximately 1 in 3 patients with congenital syphilis develops otological manifestations. *Pathology*. 1.

Macroscopic changes:meningo-neuro-labyrinthitis occurs, sometimes associated with a gummatous petrositis. When the middle ear is involved, it is called otolabyrinthitis. 2. Microscopic changes: Endolymphatic hydrops, obliterative endarteritis, gummatous formations and extended bone resorption in the otic capsule. *Clinical features*. Deafness of sudden onset. **Congenital syphilis:** Deafness involves both ears and occurs in first two years of life. **Acquired form.** Deafness, vertigo, tinnitus. Hennebert's sign (pseudofistula sign) is present in many cases of the late congenital form: There is a fistula sign without a fistula, due to an abnormally mobile footplate of stapes. It is invariably transient and occurs when the hearing loss is minimal. *Diagnosis.* The most reliable serological test is the fluorescent treponemal antibody absorbed test (FTA-ABS) which is positive in 98% of congenital cases and 100% in acquired cases. **Treatment**. Antisyphilitic treatment.

1.9.8. Méniere's disease.

A disorder of the endolymphatic labyrinth characterized by attacks of vertigo, deafness and tinnitus. *Etiology.* Unknown. Autoimmunity against endolymphatic sac proteins are under consideration. *Pathology.* Prospere Méniere reported first, that the disease characterized by deafness, vertigo and tinnitus resides in the inner ear. Hallpike and Cairns demonstrated that a common pathological substrate in Meniere's disease is the distention of the membranous labyrinth: at the beginning in the cochlea, by distention of the Reissner's membrane later the saccule and the utricle. A hydrostatic pressure increase inside the cochlear duct is responsible for the first symptomes, which is, however, compensated by the elasticity of the inner ear boundary epithelium. The low-tone hearing loss derives from this hydrostatic pressure increase. When elastic compensation by Reissner's membrane was exhausted, the morphological phenomenon of hydrops starts to develop and functionally characterized by hearing loss at all frequencies. Degeneration of strial marginal cells, Reissner's membrane mesothelial cells can be seen in post mortem sections.
*Clinical features. A*ttacks may be described as the acute phase, with vertigo as the predominant feature. Remissions with variable intervals constitute the chronic phase, where increasing deafness may become more noticeable. **Vertigo**. Occurs in attacks. The vertiginous feeling is one of rotation, eiher of the patient himself or of objects about him. The duration is variable from minutes to 24 hours, and disappears spontaneously. Horizontal or horizonto-rotary nystagmus is always present during the attack. Partial recovery of vestibular function occurs after each attack, but gradually to a lesser degree until complete loss of function. **Deafness.** Sensorineural in type and first appears at low frequencies. With each attack deafness tends to progress. Recruitment (and sometimes over-recruitment) is present. Hearing aids may not be tolerated.

Tinnitus. May be very troublesome, and is usually high pitched. **Diagnosis**. At the beginning the glycerol test is useful: intravenous glycerol prompt eliminates the symptomes. Later vestibular tests can show canal paresis on the affected side. Audiometry shows low-tone hearing loss or later a flat-type audiogram may be recorded. The only reliable test is electrocochleography, which shows great, positive summating potential. **Differential diagnosis**. 1. Labyrinthitis 2. Cogan's disease (autoimmune collagen-disorder) 3. Vestibular neuronitis 4. Benign paroxysmal positional vertigo 5. Acoustic neuroma 6. Disseminated (multiple) sclerosis 7. Epilepsy, with vertiginous attack 8. Otosclerosis with co-existing endolymphatic hydrops. **Treatment.** *Conservative.* Sedatives (in an acute attack: cinnarizine, perphenazine, promethazine may be given as suppositories or sublingual preparations). Vasodilator drugs (betahistine hydrochloride, pentoxyfilline) Diuretics (Diamoxe). *Surgical*. 1. Insertion of ventilation tube. 2. Chemical labyrinthectomy through the tube with gentomycin. 3. Saccotomy: wide opening of the endolymphatic sac. 4. Selective vestibular neurectomy in intractable cases.

1.9.9. Presbycusis.

Many patients over 55 complain of some degree of "senile" deafness. This is characteristically bilateral and symmetrical sensorineural deafness. *Pathology.* 1. Atrophy of the sensory epthelium at the basal cochlear turns. The number of hair cells is reduced. 2. Atrophy of the spiral ganglion cells. 3. Atrophy of stria vascularis. 4. Loss of elasticity of the basilar membrane. *Diagnosis:* Audiological as to high freaquency hearing loss is measured. *Differential diagnosis:* 1. ototoxicity, 2. noise-induced hearing loss; 3. viral infection; 4. autoimmune hearing loss. **Treatment**: modern hearing aids offer great help to the majority.

1.9.10. Ototoxicity.

By definition this is a damage to the cochlear or vestibular part of the inner ear. *Ototoxic drugs include:* 1. Aminoglycoside antibiotics, including streptomycin, gentamicin; neomycin, kanamycin, vancomycin. 2. Diuretics, including ethacrynic acid, frusemide. 3. Antiprotozoal agents: like quinine and chloroquine. 4. Salycilates 5. Cysplatin 6. Phenytoin. *Pathology.* 1. Degeneration of the stria vascularis. 2. Degeneration of the sensory epithelium. Outer hair cells are affected more than inner hair cells. 3. Degeneration of the ganglion cells. *Clinical features.* Deafness, tinnitus, vertigo. The symptomes of ototoxicity may develop/progress after cessation of administration. They are more severe in young and very old, and in patients with renal or hepatic failure.

Treatment. 1. Preventive: Monitor treatment (regular estimation of the serum levels of drug; Monitor hearing (regular audiometric checks in patients receiving ototoxic drugs); 2. Therapeutic: cochlear implantation.

1.10. Rehabiltation of SNHL

1.10.1. Hearing aids.

Help to patients with a hearing impairment to identify sound, including speech, by selective amplification. Two main types are analog and digital hearing aids.

The hearing aids are composed of:

1. Microphone; 2. Amplifier; 3. Receiver (loudspeaker) of two types: air conduction, bone conduction. Different designes are available, like body-worn, behind-the-ear and canal-inside types. The characteristics of the hearing aids are related to the amplification which is provided. The acoustic gain should be over 25 dB for weak sounds to be heard and can go up to 50 dB. The output has its own limitations. Selective limitation of loud sounds, which would otherwise cause of intelligibility or discomfort, is affected by one of the two methods: a/ *peak clipping:* the strong vowel sounds are clipped more than the weak consonants, and some distortion occurs. b/*automatic gain control:* retain crests of sine waves and also reproduces their rising characteristic, but the amplitude increases more slowly at the beginning than with peak clipping. New generations of conventional hearing aids use digital processing resulting in a high fidelity transmission of sounds obtaining a well-regulated intensity-frequency relationship adjustment. **Bone anchored** (the aid is directly anchored to the skull by a percutaneous titanium implant) **hearing aids** (BAHA) transmit sounds with better efficacy than conventianl hearing aids do. **Middle ear transplantable hearing aids** (**Fig. 21**) work on piezoelectrical or electromagnetic basis and the transducer vibrates e.g. the incus directly. It is recommended in those cases, where conventional hearing aids are not applicable because of recurring inflammation of the external auditory meatus or eczema or do not provide a satisfactory hearing sensation and the patient is not referred to cochlear implantation.

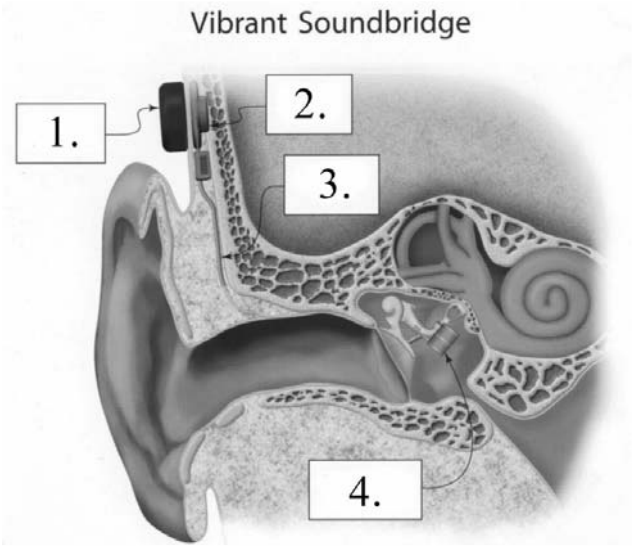


Figure 21. Schematic picture of the middle ear implant (Vibrant). Sound processor: 1; receiver: 2; connecting wire: 3; floating mass transducer on the incus: 4)

1.10.2. Cochlear implants.

Electrical stimulation of residual spiral ganglion neurons restores hearing when all hair cell function is lost and there is no discrimination of speech with a powerful hearing aid bilaterally. The selection criterion is poorer speech reception threshold at 70 dB than 30%. These are the profound hearing loss cases which are classified to prelingual and postlingual deafnesses. Former means those congenital or early childhood onset of hearing loss which made impossible the development of speech. Postlingual deafness develops after development of speech intelligibility and production. Cochlear implantation is suggested to perform in preingula deafness cases before the age of 6 years, because brain plasticity is significantly deteriorated after this age. Recognition of speech (capability to use telephone) without lipreading does not develop after this age limit. Those prelingual deaf children who become cochlear implant user before the age of 2 years can be referred to mainstream school at the age of 6 years. This is obtained by very intensive speech training programs by the teacher of the deaf and speech pathologists and parents. Postlingual deaf people rapidly benefited by the cochlear implant *Etiology* of hearing losses of that severe: 1. Genetic hearing loss, 2. Congential malformations (Scheibe dysplasia, Mondini dysplasia), 3. childhood meningitis, 4. maternal virus infections, 5. ototoxicity, 6. bilateral skullbase fracture.
Multichannel implant. (**Fig. 22)** Sixteen to 24 electrodes in a silicone rubber are passed deep into the scala tympani to reach vicinity of the ganglion cells. It

provides samples of speech at fixed frequencies dictated by effectiveness of nerve electrode interface at fixed sites beneath the basilar membrane. The external part which is attached to the implanted receiver consists of the microphone, the speech processor (which encodes the speech to electrical pulses) and the transmitter, which transmits the pulses via radiowaves. The digital technology makes possible to fit the implants encoding strategy and intensity to the comfort level of the patients. Also, bilateral cochlear implantation is possible improving orientation of hearing and speech intelligibility in noise. Today, about 45 000 cochlear implant users live all over the world. **Hiba!**

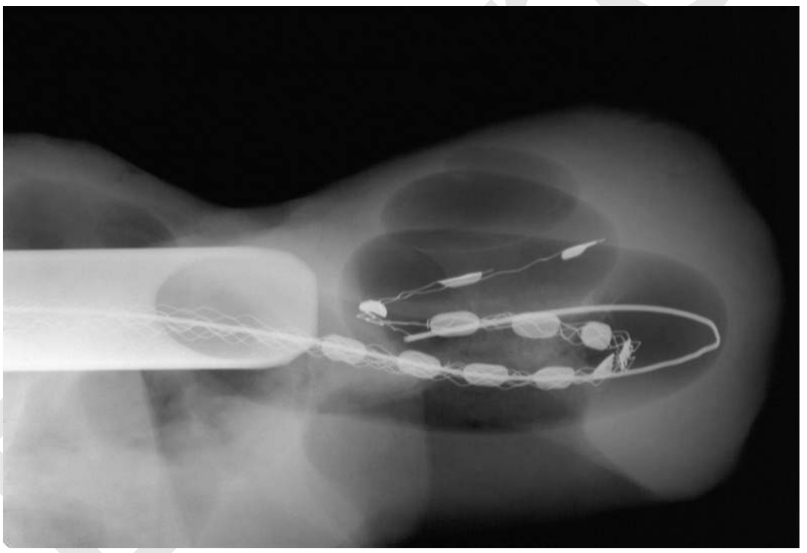


Figure 22. An X-ray microscopic picture of the location of the electrodes pushed forward into the scala tympani (an intracochlear electrode array).

1.10. 3. Auditory brainstem implantation

Bilateral deafness may be due to special tumor of the central and peripheral nervous system. Among these most commonly, neurofibromatosis 2, attacks the vestibular nerve. The disease is a genetic disorder with dominant inheritance. The tumor destroys both vestibular and cochlear nerves. Cochlear implantation is not useful, since the pathology is located central to the spiral ganglion cells. Typical audiological finding is deafness with otoaoustic emission present. The implant is very similar to the cochlear implant except that the electrode array is different. The number of electrodes are similar, however, these are fabricated on a plate with 8x4 mm.

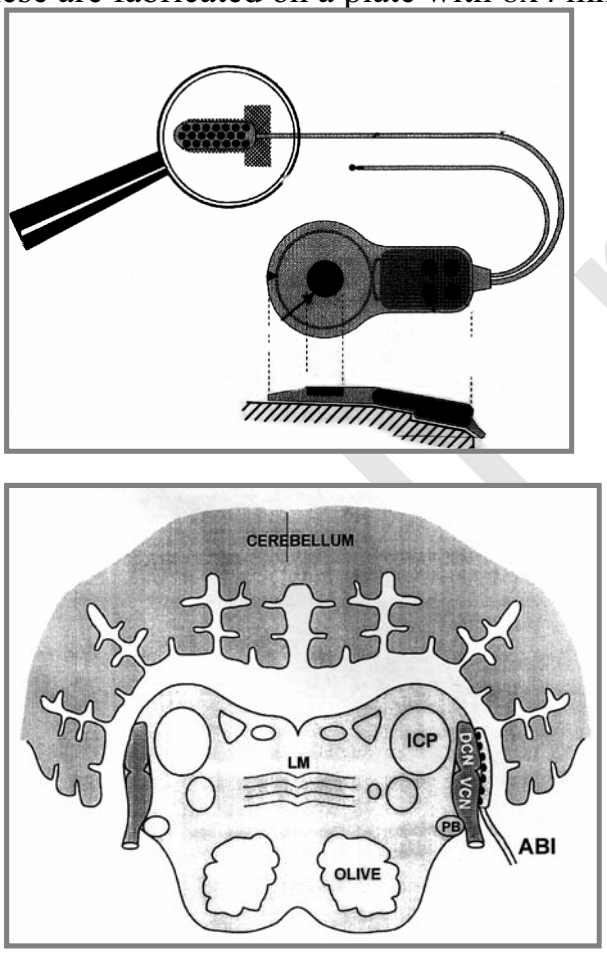


Figure 23. Auditory brainstem implant (upper panel). The electrode array (plate) is under the magnifying lens. Other parts (antenna, demodulators, ground electrode) of the implanted device are same as those of the cochlear implant. Location of the implant on the brainstem (DCN,VCN: dorsal & ventral cochlear nuclei)(lower panel).

The electrode plate is inserted into the foramen Luschka onto the surface of the ventral and cochlear nuclei in the brainstem. The outcome measure is poorer than in cochlear implantation. Brainstem implant users can distinguish the surrounding noise and can communicate with the help of lipreading, however, capability to use telephone does not develop.

1.11. Cerebellopontine angle tumors.

About 90% of these tumors are **acoustic neuromas**. Almost all acoustic neuromas originate within the internal auditory canal, presumably at the junction between the neurilemmal sheath and the neuroglial fibres extending peripherally from the brain stem. Histologically these are Schwannomas. Early tumors do not produce appreciable symptoms, because there is room for them to grow in the subarachnoid space within the internal auditory canal and in the cerebellopontine cystern. Clinical manifestations begin to appear as a tumor impinges on the nerves and vessels. Early symptoms result from impairment of VIIIth cranial nerve function. First presenting symptom may be **tinnitus**. Usually, **unilateral sensorineural hearing loss** is followed by slight dizziness or imbalance and, less frequently, by true vertigo. In some cases, however, the vestibular involvement may precede the auditory findings. The hearing loss is slowly progressive. Seventh cranial nerve dysfunction is rarely an early sign. As the tumor grows outside the internal auditory canal into the cerebellopontine cystern, the adjacent cranial nerves become involved, first of all the Vth cranial nerve, resulting in diminished corneal reflexes and numbness of the face. The diagnosis should be established by acoustic brainstem response (ABR) audiometry that shows an I-V intervawe latency shift and by magnetic resonance imaging (MRI) of the skull. Treatment is surgical removal of the tumor.

1.12. Referred otalgia.

Pain is commonly referred to the ear from lesions of related structures whose nerve supply also sends branches to the ear. Best considered on an anatomical basis.

Via the Vth cranial nerve

1. Lesions of the teeth and jaws: Impaction of the molar teeth; Dental caries; Apical abscess; Malocclusion; Temporomandibular joint arthritis.

2. Lesions of the salivary glands: Acute infection; Calculus.
3. Sphenopalatine neuralgia
4. Lesions of the tongue

Via the IXth and Xth cranial nerves

1. Lesions of the oropharynx: Acute pharyngitis and tonsillitis; Peritonsillar abscess (quinsy); Parapharyngeal and retropharyngeal abscesses; Tonsillectomy; Tuberculous ulceration; Neoplasms.
2. Lesions of the tongue: Ulceration; Neoplasms; Infection.
3. Elongated styloid process
4. Glossopharyngeal neuralgia
5. Lesions of the epiglottis

Via the IInd and IIIrd cervical spinal nerves

1. Cervical disc lesions
2. Arthritis of the cervical spine
3. "Fibrositis" of the upper part of the sternomastoid muscle.

Local referred pain

Temporo-mandibular joint disorders (arthritis, arthrosis)

1.13. Tinnitus

Tinnitus is perception of sound in the absence of external sound source. May be objective and subjective. The sounds heard by the patient are in great variety e.g. pulsatile (vascular), blowing (noise induced hearing loss), wheezing (otosclerosis), high (presbyacusis) or low pitch (Meniere's disease) clicking (palatal myoclonus) etc. Voice or music indicate psychiatric consultation.

The cause of tinnitus can be very simple e.g. ear wax. Therefore otoscopic examination is obligatory as well as pneumotoscopy, which reveals middle ear fluid or mass. Tympanogram show spikes when clicking is heard as a sign of palatal myoclonus which can be further clarified by EMG examination. B-type tympanogram (otitis with effusion) indicates myringotomy and perhaps ventilation tube insertion (glue ear).

Objective tinnitus is rare and means that the sound heard by the patient can be heard by the examiner, also. Beside clicking muscles (e.g. tensor tympani muscle), glomus tumor, middle ear small aneurysms can produce sound of audible intensity. Diagnosis is given by imaging modalities (CT, MR) or angiography. Treatment is surgical removal of the tumor or sacrificing the muscle.

Subjective tinnitus of known causes are peripheral/cochlear. Otosclerosis (conductive hearing loss), inner ear diseases (see above).

Vast majority of the cases belong to idiopathic subjective tinnitus category. A commonly accepted definition for subjective idiopathic tinnitus is that this is a conscious expression of a sound that origines in an involuntary manner in the head of its owner or may appear to him to do so (McFadden, 1982). These are due to reorganization of the auditory central nervous system and mostly are intractable. Brain imaging will exclude known pathologies e.g. multiple sclerosis, tumors, vascular insults. Treatment of idiopathic tinnitus is often difiicult. Suppressors are similar devices like hearing aids. These can be tuned to the frequency spectrum of the tinnitus and serve as noise generator which suppresses tinnitus if applied in same intensity. Not all patients can tolerate it. Psychologic training may help to learn how to tolerate tinnitus.

1.14. Disorders of the facial nerve

The facial nerve **(Fig. 24)** is a mixed nerve with large motor root that arises from a nucleus located in the caudal portion of the pons. The root loops around the nucleus of the VIth cranial nerve before leaving the brainstem. The fibers of this root innervate the muscles of facial expression as well as the stapedius muscle and the auricular muscles, and posterior belly of the digastric, stylohyoid and platysma muscles. The motor root is joined in the brain stem by a smaller portion of the nerves that arises from the superior salivary nucleus, the nervus intermedius, consisting of preganglionic parasympathetic secretomotor fibers for the submaxillary, sublingual, lacrimal and nasal glands. The nervus intermedius also includes special sensory (taste) fibers for the ipsilateral anterior two third of the tongue. The two roots of the facial nerve enter the internal auditory canal. Upon reaching its end then enters into a narrow bony canal (Fallopian canal) in the temporal bone. It passes through the middle ear, the mastoid bone and emerges from the stylomastoid foramen.

Level of lesion. This determines the clinical picture.

1. In the pons. Other cranial nerve nuclei and the long motor and sensory tracts are usually involved. Taste and lacrimation are uneffected. Poliomyelitis, vascular accidents and tumors may act at this level. Forehead muscles are still innervated because of bilateral innervation.
2. Before entering the internal auditory meatus. Meningitis and cerebellopontine angle tumors may involve the Vth and VIIIth cranial nerves with the facial. Taste and lacrimation are uneffected.

3. After entering the internal auditory meatus. The VIIIth nerve and the nervus intermedius may be involved with it, by trauma or by tumor
4. In the Fallopian canal. It suffers alone. Lesions between the union of the motor fibers with the chorda tympani and the geniculate ganglion cause loss of taste. Lesions above the point at which the nerve to the stapedius muscle leaves it may be associated with hyperacusis (phonophobia). Tests for taste (by electrogustometry), stapedius reflex action (acoustic impedance test), salivation and lacrimation (Schirmer's test), if carefully done, permit accuracy in locating the upper extent of a discrete lesion
5. Below the stylomastoid foramen. Motor paralysis only is found; taste, stapedius function and lacrimation are all uneffected.
Pathology. Different degrees of facial injury exist: neuropraxia is a reversible conduction block with good prognosis. Axonotmesis means destruction of the nerve fibres but intact myelin sheath, while neurotmesis is a complete disruption of the whole nerve (denervation) without prospectives to spontaneous recovery of any degree. *Electrodiagnosis.* Essential to determine whether neuropraxia or denervation is present. It may permit conclusions regarding the probability of spontaneous recovery. The minimal nerve excitability test (**NET**) measures threshold of the nerve trunk below the stylomastoid foramen to percutaneous stimulation. Complete loss of excitability denotes severe or total denervation. The test is useful at anytimes after the first 3 days of the paralysis. **Electroneuronography** measures the amplitude of the evoked muscle-action potential and gives a higher degree of accuracy in prognosis.

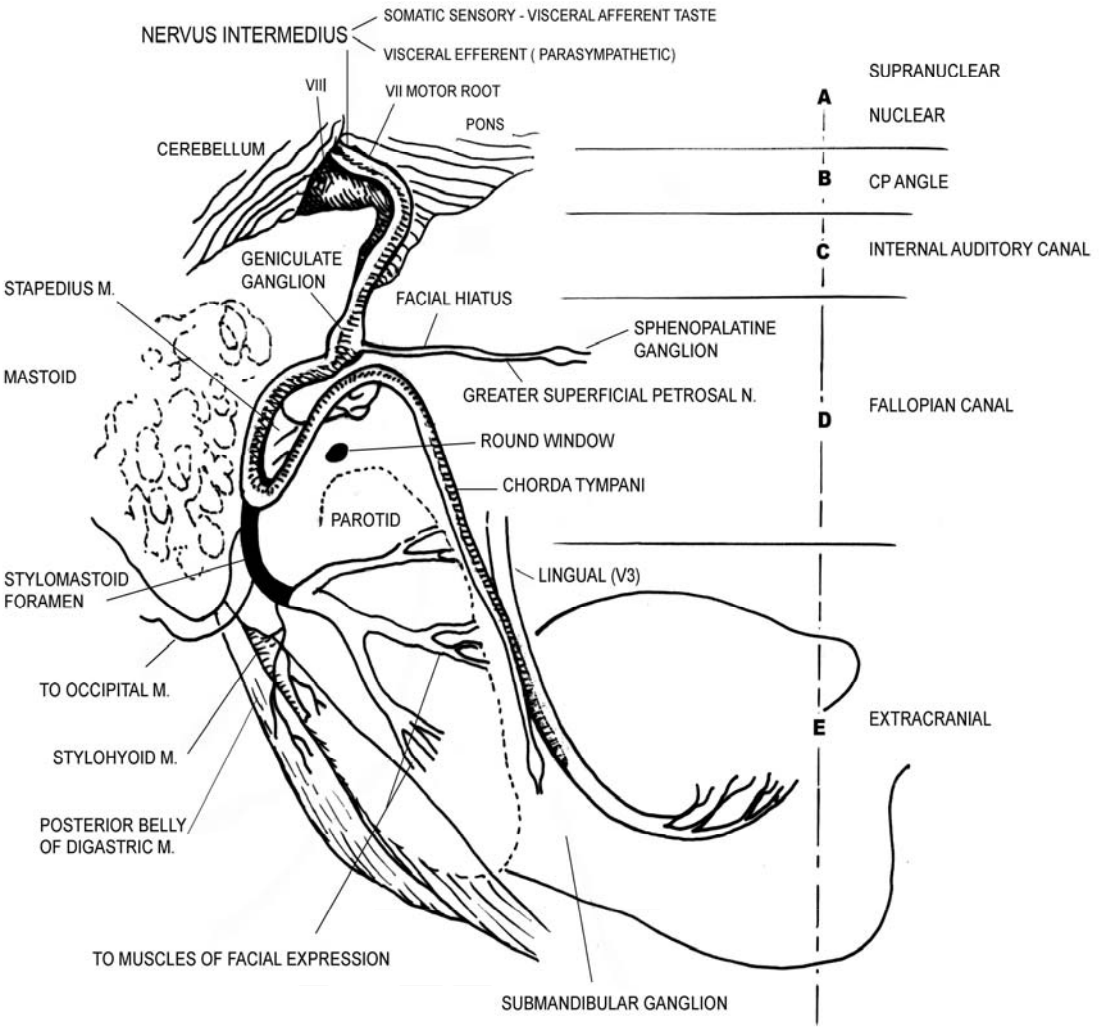


Figure 24. Facial nerve course (CP angle: cerebellopontine angle)

General management of facial paralysis. In neuropraxia cases the spontaneous recovery is likely. In severe or complete paralysis cases care must be taken with eyes to protect them from dust and dry (tarsorrhaphy may be necessary), electrical stimulation of the cheek prevents atrophy of the muscles. When recovery has begun, active excercises are useful. Persistent crocodile tears may be treated by tympanic neurectomy. Surgical reinnervation of the face can be provided by **anastomosing the hypoglossal nerve** (n. XII) with the peripheral trunk of the facial nerve. The revitalization of the facial muscle tone can be obtained by this method. Active intentitional movements are poor.

1.14.1. Bell's palsy

This is an acute facial paralysis of undetermined etiology. Possible causes include viral (herpes simplex) neuropathy with inflammatory swelling and secondary ischemia due to compression in the Fallopian canal. Other possibilities are diabetic and neural ischemias by microvascular disease. There is an 80% of good-to-excellent spontaneous recovery rate. *Symptomes.* Impairment of taste, hyperacusis, paralysis of face muscles. **Treatment**. Prednisolon in large doses: 60-80 mg per day for four days tapering to withdrawal at 9 days, in antibiotic protection. The surgical decompression of the nerve may be considered in cases with poor measures of nerve threshold without tendency of recovery.

1.14.2. Facial paralysis in chronic otitis media.

It occurs if nerve is compressed, later destroyed, by cholesteatoma. **Treatment**. In cases of neuropraxia, immediate operation is mandatory to remove cholesteatoma. In cases of complete denervation operation must include full exposure from stylomastoid foramen to geniculate ganglion. Sheath must be opened and nerve excision and grafting performed if nerve trunk continuity is lost.

2. The nose and paranasal sinuses.

2.1. Anatomy and physiology of the nose.

In discussing the anatomy of the external nose, the following terms are useful for the description of injuries and tumor sites: the *root* is where the nasal and frontal bones articulate: it is divided into the nasion and glabella; the *dorsum* is the ridge line from root to apex; the *apex* is the tip of the nose; the *base* is the triangular portion between the tip and and the lip; the *anterior nares* are divided by the columella and bounded by the alae; the *columella* is the membranous portion of the nasal septum; the *bridge* is the bony structure of the nose the frontal portion of the maxilla plus the nasal bones; the *alae* are the lateral rounded eminencies at the base of the nose, formed by the lower lateral cartilages; the *nasal septum* is made up of the septal cartilage plus the vomer and perpendicular plate of ethmoid.

2.1.1. **Anatomy of the internal nose and paranasal sinuses.**

The lateral wall of the nose has three turbinates **(Fig. 25)**, or conchae; however, the third turbinate is very small, located high and posterior.

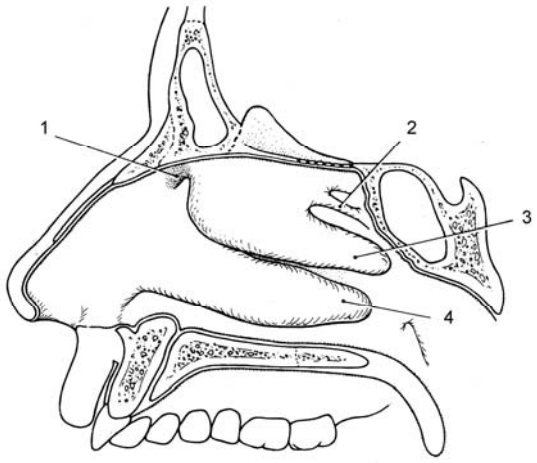


Figure 25. Lateral wall of the nasal cavity proper. 1: agger nasi; 2: superior turbinate ;3: middle turbinate; 4: inferior turbniate

Turbinates have a thin bony framework and are covered by highly vascular tissue that is composed of a dense arterial network and venous plexuses similar to erectile tissue. This covering is particularly evident on the inferior turbinate. The turbinates can swell by means of vascular engorgement to occlude the nose. Beneath each turbinate lies a meatus. The inferior meatus receives drainage from the nasolacrimal duct only. The middle meatus receives drainage from the nasofrontal duct and anterior ethmoid cells anteriorly, from the middle ethmoid cells and maxiallary sinus ostium medially. This is the **ostiomeatal unit**, which is the most important endoscopic anatomical structure in the nasal cavity proper.
The posterior ethmoid cells and the sphenoid sinus open posteriorly into the sphenoethmoid recess..

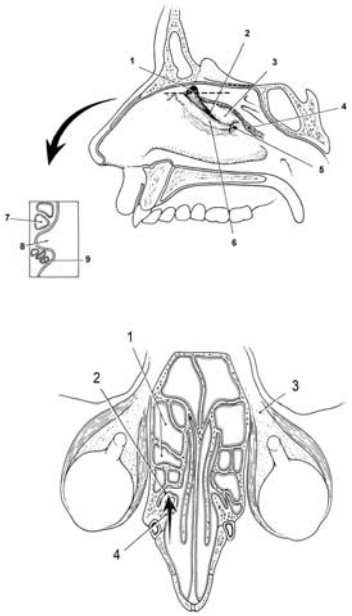


Figure 26. Upper panel: Schematic view of the lateral wall in the nasal cavity showing the ethmoid infundibulum. (1,8) hiatus semionaris (2) sphenoithmoid recess (4-5) uncinate process (6) agger nasi (9) ethmoid cells **Lower panel**: Horizontal section of the ethmoid cells (1) showing the uncinate process.(4) etmoid infundibulum, (2) optic nerve canal (3)

Underneath the middle turbinate (**ostiomeatal unit**) the most prominent structures from anterior to posterior are the **uncinate process**, the **hiatus semilunaris**, and the **bulla ethmoidalis**. **(Fig. 26)** The uncinate process is hook shaped and the gap between it and the bulla ethmoidalis is known as the hiatus (opening) semilunaris. The hiatus semilunaris leads to a groove between the uncinate process and bulla ethmoidalis. The groove is the **ethmoid infundibulum (Fig 26 A)**. The nasofrontal duct opens into the anterior superior aspect of the ethmoid infundibulum and is referred to as the **frontal recess** of the infundibulum. Just anterior to the superior attachment of the middle turbinate and anterior to the frontal recess is the **agger (ridge) nasi.** This prominence on the lateral nasal wall represents the most anterior of the anterior ethmoid cells. Mucopurulent material can often be seen draining from these regions during acute sinus infections, thereby indentifying the site of the infection. The **sphenoethmoidal recess** is a long, narrow space located between the upper portion of the middle and superior turbinate and nasal septum. The posterior termination of the middle turbinate is called **basal lamella**. Behind this are located a few large posterior ethmoid cells and then the **sphenoid rostrum** protrudes into the center of these. The **cribriform plate** forms the roof of this area, and the posterior limit is the anterior face of the sphenoid sinus. More importantly, the olfactory epithelium is located here. The **nasal septum** is formed by cartilage anteriorly and by bone posteriorly. It may grow irregularly or may be traumatized so that it obstructs breathing on one or both sides of the nose. If the deviation is severe enough, it will inhibit proper drainage or will obstruct sinus ostia by putting pressure on turbinates. The **ethmoid sinuses** are a complex of 10 to 18 small sinuses that form a roughly rectangular shape front to back. The poserior length is 4 cm to 5 cm, the height is 2.5 cm to 3 cm, and the width bilaterally between the eyes is 1 cm to 1.5 cm. Some of these sinuses are present at birth, and they are often involved in nasal infections and allergic diseases in children. The **maxillary sinuses** sometimes present at birth, remain small until the development of the permanent teeth. The ostium is located high on the medial wall of the sinus under the middle turbinate. The bony canal encasing the infraorbital nerve travels through the roof and roots of the second premolar and first and second molars often project into the sinus floor. Localized pain and pain in the distribution of the infraorbital nerve are common symptomes of maxillary-sinus infections due to neural irritation. The **sphenoid sinus** begins to develop by the sixth year. They are highly variable in size. The ostium is located high on the anterior wall. The **frontal sinuses** are the last to develop and continue to enlarge during the teen years. Not uncommonly, the frontal sinuses may be absent or, in the other

extreme, may be very large. The *nasal and sinus mucous membrane* is pseudostratified columnar epithelium. Like the skin, the mucous membrane has nociceptors: free, naked nerve endings that respond to chemicals and to changes to temperature, humidity and pressure. These receptors serve to alert and protect the lower airway following exposure of temperature or to pollutants. The nasal part of the *upper respiratory tract* is an irregular, double tubed structure, but even in normal circumstances it is not static. Eighty percent of individuals have a cycle of congestion and decongestion involving first one side of the nose, then the other, known as the nasal cycle. In the normal nose, one nasal passage opens up, accompanied by secretion by the serous and mucous glands, while the opposite side closes down with increasing obstruction and decreasing secretion. There is a shift of autonomic balance, occuring every 1/2 to 4 hours. However , the combined or total airway resistance is unchanged. Patients with cause for a reduced airway secondary to a deviated septum, or with chronically engorged mucous membrane from perennial allergy, will complain that their nose is never completely open and that airway shifts from side to side. The blocked nose creates *hyponasal* speech, recognisable by the substitution of other sounds for the normal nasal consonants of 'm', 'n' and 'ng'. The nose efficiently warms cold air, no matter what the temperature of the air. Warming the air permits humidification up to 95% before the air reaches the trachea. Experience with laryngectomized patients, however, indicates that this process is not vital. The nasal mucosa and the sinus linings are covered by a sticky, mucoid layer. This mucous blanket serves to waterproof the nose, preventing loss of water outwards and drying of the mucosa. The mucus serves to protect the nose from infections.

2.1.2. **Blood supply of nose and paranasal sinuses.**

Supplied by branches of the external and internal carotide arteries **(Fig. 27)**. Derivatives of external carotide artery are: 1. Sphenopalatine artery (branch of maxillary artery): turbinates and most of the septum 2. Greater palatine artery (branch of maxillary artery): lateral nasal wall and the anterior part of the septum 3. Superior labial artery (branch of the facial artery) it sends branches to the Kisselbach's plexus, in Little's area 4. Infraorbital and superior dental arteries (from maxillary artery): maxillary antrum 5. Pharyngeal branch of the maxiallry artery: sphenoid sinus. Derivatives of the internal carotide artery are: *the anterior and posterior ethmoidal arteries* (branches of the ophthalmic artery). They supply the roof of the nose, anterior parts of the nasal septum and the lateral wall of the nose, ethmoidal and frontal sinuses. *Venous drainage.* The veins form a cavernous plexus beneath the mucous membrane. They open to: 1. Sphenopalatine vein and anterior facial veins 2. Ophthalmic veins 3.Veins of the orbital surface of the frontal lobe of the brain 4. Superior sagittal sinus.

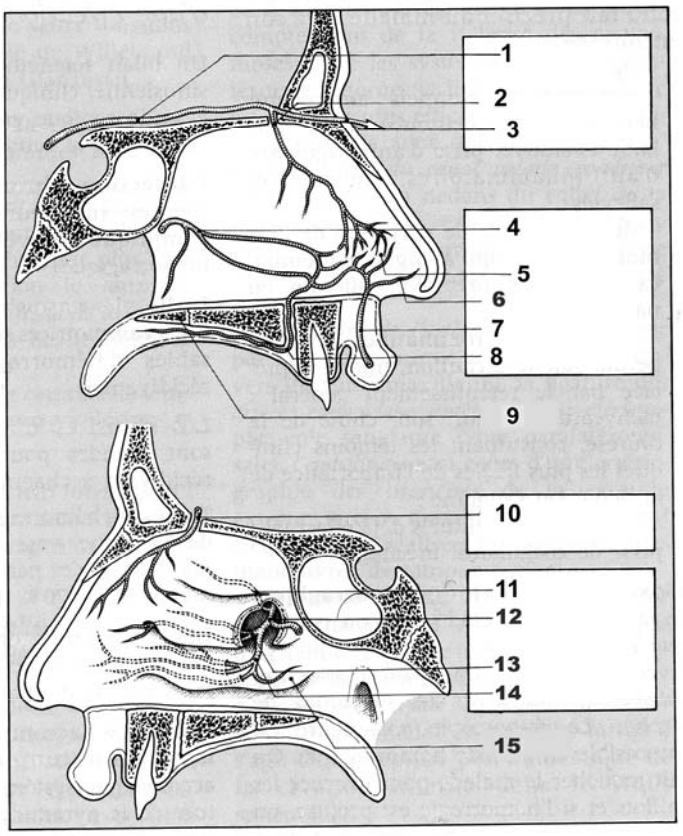


Figure 27. Vascular supply in the nasal cavity. Upper panel shows the medial wall (with the Little's area which receives arteries from both the anterior ethmoid and the sphenopalatine arteries). Lower panel shows the lateral wall. 1,10: ophthalmic artery; 2: etmoid artery; 4, 12: internal maxillary artery; 5,6,7: ascendent palatine artery; 8, alveolar process of the maxilla; 9: hard palate ; 13,14: brancses of the sphinopalatine artery

2.1.3. Nerve supply.

1. Sympathetic fibres. Postganglionic fibres pass from the superior cervical ganglion to the plexus around the internal carotide artery. They then form the deep petrosal nerve which, with the greater superficial petrosal nerve, become the nerve of the pterigoid canal (*Vidian nerve*). They maintain a constant tonic vasoconstrictor action. Section of the Vidian nerve causes vasodilation and a "stuffy nose" is sometimes complained of. 2. Parasympathetic fibres. Preganglionic fibres are derived from the nervus intermedius. 3. Branches of the Vth cranial nerve (anterior ethmoidal nerve: medial branch supplies the anterior part of the septum; lateral branch supplies the anterior part of the lateral wall and the anterior parts of the inferior and middle turbinates). 4. Branches of the sphenopalatine ganglion (a/ greater palatine nerve : inferior turbinate, inferior and middle meatus; b/ short sphenopalatine nerves:superior and middle turbinates; c/ long sphenopalatine nerve: suppplies the remainder of the septum) 5. Olfactory nerves: The sense of smell is supplied by the 1st cranial nerve. Fibres arise from bipolar cells of the olfactory mucosa.

2.1.4. Olfaction

The cribriform plate of the ethmoid transmits the filaments of the olfactory nerve as they descend from the undersurface of the olfactory bulb to their distribution in the mucous membrane covering the uppermost portion of the nasal septum down to and including the cranial surface of the superior turbinate. The brownish psudostratified olfactory epithelium consists of 3 different neural cell types: olfactory cells, basal cells and supporting cells. The supporting cells are firmly attached to each other hence forming a cuticular plate-like terminal web. The lamina propria of the olfactory area contains glands. These produce mucus which covers the olfactory epithelium in a thin layer. Bipolar olfactory nerve cells are distributed among the supporting cells. At their apical portion a modified dendrite extends to the epithelial surface which radiates the olfactory cilia into the mucus blanket covering the epithelium. The cilia lie parallel to the mucosal surface

Seven primary odours, according to the stereochemical theory of olfaction, possess a molecule of unique size and shape and of an electrophilic or nucleophilic nature. The olfactory epithelium then possess appropriate receptors of definite size and shape respectively. The primary odours which excite only the olfactory epithelium are coffee, tea, peppermint, floral odours, parfumes. Other odourous substances stimulate the trigeminal nerve endings e.g. ether, camphor, pungent (ammoniac), putrid.

The **fila olfactoria**, the central axons of the bipolar olfactory cells connect to the mitral cells in the **olfactory bulb** and then leave the bulb to form the **olfactory tract**. The olfactory tract passes along the base of the frontal lobe and enter the pyriform cortex, the anterior comissure, the caudate nucleus, the olfactory tubercle and the anterior limbus of the internal capsule.

2.2. Examination of nose and paranasal sinuses

In a child, an examiner can view the anterior of the nares by pushing the nasal tip up with his thumb. The anterior septum and tip of the anterior turbinates are then in view. However, with a cooperative child or an adult, a nasal speculum is preferable. The proper technique of **anterior rhinoscopy** is to place the nasal speculum blades under the ala to the desired depth; then the blades are opened down to the nasal floor, to spread nares. With a headmirror or headlight, the inferior turbinate and septum will be in immediate view. If the mucosa is swollen, a vasoconstricting spray, such as oxymetasoline, is administered. The examiner reexamines the nose after few minutes, looking for inflammation or discharge, deviations, masses, and so forth. *Differential diagnosis.* The nasal mucosa should be pink to red in color. Thickened, bright mucosa suggests an inflammatory reaction. Discharge from a viral infection is mucoid and grey. Bacterial infections result in yellow-tinged secretions. Old mucus develops a greenish discoloration. Allergic rhinitis may present as bright red, inflamed mucosa or with a reddish blue or bluish white mucous membrane. Any odor to the drainage, whether noted by the patient or perceived by the examiner, suggests a bacterial infection, such as a dental abscess, chronic nasal infections, or atrophic rhinitis. *Nasopharyngeal examination. The patient should attempt* to breathe through his nose while his tongue is depressed sufficiently to permit a small mirror to slide behind the soft palate. If available, a nasopharyngoscope is helpful. *Sinus examination.* The sinuses are best tested clinically by gentle tapping with the examiner's thumb. An infected sinus will be tender locally (maxillary or frontal sinus). The sphenoid is best examined radiographically. The ethmoids can only be very superficially visualized on nasal examination. *Radiographic examinations of the nose and paranasal sinuses. Sinus X-rays may be helpful* in different diaseases (nasal polyps, foreign bodies, nasal discharge, sinusitis, tumor). The most helpful image is the antero-posterior one when the maxillary and frontal sinuses give good picture. An axial skull view can visualize the sphenoid sinus, and special x-ray may be taken of the ethmoid sinus (the Caldwell view gives best visualization of frontal and ethmoid sinuses). Computerized tomography (CT) and **not** magnetic resonance imaging (MRI) is the imaging modality indicated for assesment of the location and

extent of the tumors, polyps and in some entities allow for specific diagnosis. *Nasal and sinus endoscopy* and their development has been one of the most significant advances in recent memory. Endoscopy by different rigid optical devices allows direct visualization of inflammatory and space occupying processes in the sinuses
and nasal cavity and at the same time endoscopic surgery (**FESS**: functional ednoscopic sinus surgery) can be performed. The primary site of its use is the middle meatus-anterior ethmoid complex (**osteomeatal unit**). Among functional diagnostic measures, rhinomanometry - a procedure to asses nasal airway resistence changes - provides with topical diagnosis of anatomical abnormalities responsible for nasal obstruction.

2.3. The nasopharynx.

The nasopharynx anatomically and pathologically is closer to the nose and paranasal sinuses than to the meso-, hypopharynx. The symptoms of nasopharyngeal diseases are similar to those of nasal origin (nasal obstruction, hyponasal speech, nasal discharge).

2.3.1. Anatomy

The nasopharynx is formed superiorly by the inferior surface of the sphenoid sinus, posteriorly by the cervical vertebrae, and anteriorly by the posterior portion of the nasal septum and posterior choanae. The lateral wall of the nasopharynx is the most complex portion. The Eustachian tube orifices are located immediately posterior to the inferior turbinate. The surrounding Eustachian tube cartilages form a "moundlike" structure around the orifice itself. Above these cartilages is a depression known as the fossa of Rosenmüller. In children and young adults adenoids may be present. It is a mass of lymphoid tissue adherent to the roof of the nasopharynx. In some children the adenoid may be enlarged and obstruct the posterior choanae of the nose. The primary disorders involving the nasopharynx include adenoid hypertrophy and chronic adenoiditis **(Fig. 28).**

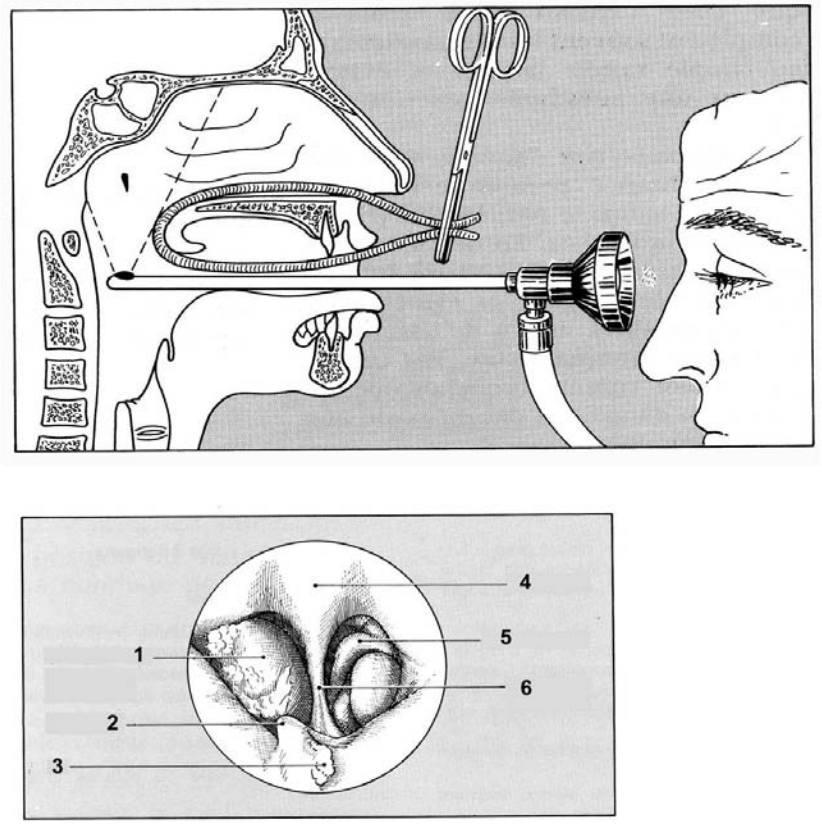


Figure 28. Examination of the nasopharynx (upper panel) through pharyngoscope after elevating the soft palate with a pair of rubber catheters, and the nasopharyngeal fornix as viewed by the pharyngoscope or posterior rhinoscopy (lower panel) showing the anterior (3) and posterior (4) walls, the normal posterior pole of the middle turbinate (5), the posterior edge of the vomer (6), adenoid (2) and bullous hypertrophy of the posterior pole of middle tubinate (1).

2.3.2. Chronic nasal obstruction and adenoidal hypertrophy.

When children are affected by chronic nasal obstruction and adenoidal hypertrophy, it is not possible in most cases to determine which is leading the other. Allergic rhinitis children with chronic nasal obstruction and infection (nasopharyngitis) will develop adenoidal hypertrophy in response to the chronic nasal inflammation. *Symptomes* of adenoid hypertrophy beside nasal obstraction are hyponasal speech, otitis with effusion and recurring nasal infections. *Diagnosis* can be established by manual palpation of the nasopharynx. This is a very unpleasant procedure for children and we may loose further cooperation. Check up examination or allergic rhinitis is recommended *differential diagnostically*. Similarly, nasopharyngeal tumors should be excluded and nasal foreign bodies. The *treatment* is, first, medical. Therapy should include a combination of antihistamines and vasoconstrictors, and short course of antibiotics based on cultures taken from within the nasal cavity. If this course of treatment fails to resolve the problem, a surgical approach should be considered. This includes first an adenoidectomy. Children with otitis with effusion secondary to Eustachian tube obstruction may simultaneously require myringotomy with aspiration of fluid from the middle ear. If fluid is thick and mucinous (gluelike) a pressure-equlization (ventilation) tube (gromet) is placed in the tympanic membrane in order to prevent the reaccumulation of mucus.

2.3.3. Juvenile nasopharyngeal angiofibroma

It is a benign vascular tumor unique to boys and postpubescent adolescent males (10 to 20 years of age) that most often presents with a brisk epistaxis and nasal obstruction. Anterior and posterior rhinoscopy reveals grayish mass which sometimes is similar to polyps. The tumor is much harder by palpation. The *diagnosis* is made by CT scan examination of the paranasal sinuses. That shows that even big tumors respect the bony walls. Angiography demonstrates the heavy vascularization of the mass receiving supply usually from a branch of the internal maxillary artery. Biopsies are not to be done because of risk of excessive haemorrhage. Removal is attempted *surgically*. The surgery is often bloody. Preoperative embolization of the supplying artery prevents severe bleeding. After puberty recurrence is not expected.

2.3.4. Carcinoma

Carcinoma of the nasopharynx (former name: lymphoepithelioma) is not an uncommon disorder. For some reason, as yet poorly understood, the tumor is most common among Chinese. There is a relationship between carcinoma of the nasopharynx and the Epstein-Barr virus; patients with nasopharyngeal carcinoma have been found on serodiagnostic studies to have high titers against EB virus. In 50% of the cases the presenting *symptome* is a lymph node **metastasis** in the neck. These lymph nodes in the posterior cervical chain may be palpated just anterior to the margin of the trapezius muscle (behind the sternomastoid muscle). A second, common presenting symptome is the presence of uniletarel **serous otitis media**. **Treatment.** Involves combined chemoirradiation therapy to the nasopharynx and to the regions of the lymphatic drainage. Tumors in the nasopharynx can not be locally resected and if they fail to respond to irradiation therapy, they will extend through the foramen lacerum into the cavernous sinus and middle fossa, eventually involving the IIIrd, IVth, and VIth cranial nerves. Persistent metastatic tumors in the neck can be removed by block dissection of the neck.

2.4. Trauma to nose, paranasal sinuses and jaws.

Patients with maxillofacial injuries may be suffering from other injuries and diseases and thorough general medical examination is essential. It must be remembered that a fractured base of skull coexists not uncommonly with a fractured maxilla. The most important consideration in maxillofacial injuries is the maintenance of an airway. All foreign bodies, e.g. broken dentures and teeth, must be removed from the mouth and care must be taken to see that the patient is not laid on his back, when the tongue may fall back and asphyxiate him.

2.4.1. Fractures of the nose.

Classification. Type 1. Due to a frontal blow. There is vertical fracture of the nasal septum. The thin distal portion of the nasal bone is depressed or displaced. Type 2. Always due to lateral trauma. The nasal bones are displaced laterally but there is no gross depression. There is a "C"-shaped fracture of the perpendicular plate of the ethmoid. The frontal process of the maxillae may be fractured. *Clinical features.* **Deformity.** External swelling follows quickly. "Black eye" is common (ecchymosis of the periorbital and subconjunctival region). **Pain, epistaxis, nasal obstruction**. **Diagnosis** is radiographic. **Treatment.** Fractures without displacement don't need special care. Fractures with dislocation are treated as follows. 1. Early: If the patient is seen within the first several hours, before swelling, reduce immediately. Local anaesthesia is sufficient. Elevation with a pair of closed forceps or long speculum under the

nasal bones, combined with laterally applied digital pressure is usually effective in achieving realignment. An external splint is necessary. 2. Intermediate: when the swelling is marked has to be left until subsided. 3. Late (1-2 weeks): When the swelling has subsided reduction can be undertaken under general anaesthesia. The fragments have to be mobilized and must be replaced. External splint may or may not be required, depends on the fragments had to be replaced and the haematoma that can internally splint.

2.4.2. Facial fractures.

Frontal sinus fractures are infrequent and involve the anterior wall of the frontal sinus. Usually require open reduction with elevation. Frontobasal fractures may be due to car accidents when the posterior wall of the frontal sinus is affected. The typical fracture line runs through the rhinobasis. Dural tears and brain injuries may be open or closed. Main symptomes are **cerebrospinal fluid rhinorrhoea,** brian prolapse through the nasal cavity, facial hematoma, cerebral concussion, anosmia.

Zygoma fractures are suspected clinically when there is a flattening of the malar eminence, a depressed orbital rim, periorbital ecchymosis, or infraorbital numbness. Confirmation is by X-ray. CT scanning is necessary to evaluate the floor of the orbit. Most of the fractures are best managed by open reduction with wiring across fracture lines.

Blow-out fracture of the maxillary sinus. Elicited by impulse-force violence through the orbital content and resulting in impression fracture into the antrum. The orbital content (inferior rectus and inferior oblique muscles) is entrapped by the fragments. Symptomes are enophthalmos, double vision limited eye-bulb movements. Diagnosis is established by CT scan. Treatment is explorartive Caldwell-Luc surgery, reduction of the fractured fragments and intra-antral support by catheter balloon filled with water through a window in the medial antral wall.

Maxillary fractures may be unilateral or bilateral. The **LeFort** classification is an effort to classify these fractures:

1/ LeFort I. is a transverse fracture just above the teeth

2. LeFort II. is a pyramidal fracture crossing the nasal bones, face of the maxilla, ifraorbital rims, orbital floor, and reaching the zygomatico-maxillary suture line.

3. LeFort III. represents craniofacial dysjunction from fractures through the zygomatico-frontal and nasofrontal sutures, orbital floor, ethmoid and sphenoid sinus (**Figures 29. A-C**). **Diagnosis** of maxillary fractures is suggested by ecchymosis and swelling of the midface. Mobility of the the upper dental arch suggests a LeFort I. fracture. LeFort II and III fractures may be accompanied by

cerebrospinal rhinorrhoea and by movement of the entire face and dental occlusion incapability. **Treatment** of the LeFort I fractures is an intermaxillary fixation using an arch bar.

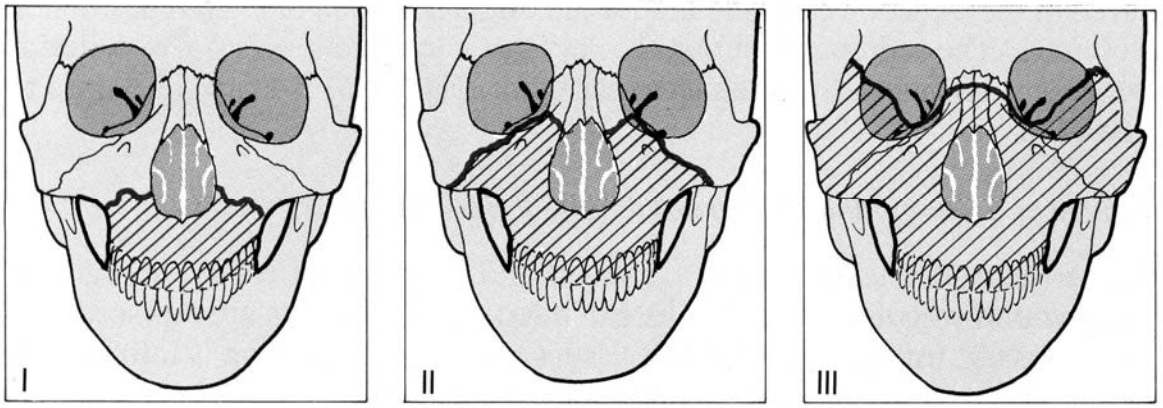


Figure 29. LeFort I., II. and III. fractures

LeFort II fractures are managed by osteosynthesis miniplates through the inferior orbital rim fragments and of the zygomatic process to frontal bone. Occlusion is corrected by intermaxillary fixation using the intact mandible. LeFort III fractures also utilize intermaxillary fixation. Infraorbital and zygomatico-frontal fractures are reduced by miniplates. Also miniplates slung from a stable site above the zygomatico-frontal suture, a head-cap, or other external appliance.

2.4.3. Oro-antral fistula

The maxillary sinus antral cavity communicates with the oral cavity. *Etiology.* Dental extraction, sublabial operations (Caldwell-Luc or similar), erosion by malignant disease, penetrating wounds of the upper jaw. *Clinical features.* Discharge in the mouth; Regurgitation of food particles into the nose; Frequent maxillary sinusitis. **Treatment**. A large alveolar fistule may be closed with a flap from the palate or buccal mucosa.

2.4.4. Nasal septum deviation (deflection) and spurs

Few adults have straight septum. Only gross deflections cause mechanical obstruction. *Etiology.* Trauma is far the commonest cause. Developmental errors also produce bending or spurs. *Symptomes.* Nasal obstruction is always present. External deformity sometimes accompanies. Pressure headaches and nose bleeding may occur. **Treatment.** Submucous septum resection may be performed in adults (after the age of 18 years). Most reliable methode is septoplasty, when the septal deformations are reconstructed by remodeling of the septal cartilage and removal of the bone spurs.

2.4.5. Septal haematoma

A collection of blood beneath the mucoperichondrium of the septum caused by direct trauma, operations or rarely blood dyscrasias. *Featured* by nasal obstruction and septal swelling. **Treatment.** Simple aspiration if the haematoma is small. Incision and drainage, nasal packing and systemic antibiotics.

2.4.6. Foreign bodies in nose.

These are common in childhood. They may enter through the anterior nares, through the posterior choanae or penetrating wounds. *Symptomes.* 1. Unilateral nasal discharge. 2. Nose bleeding. 3. Pain. 4. Sneezing. The treatment is removal directly through the anterior nares or surgically under general anaesthesia.

2.5. Inflammations of the nose

2.5.1. Inflammations of the external nose

1. Nasal furunculosis is a minor staphylococcus aureus infection involving the follicles of the vibrissae of the nares. Redness, swelling and pain are limited to the inner surface of the vestibule. Prompt antibiotic treatment plus incision is required if the infection becomes more than superficial. The reason for special concern is that the infections involving this portion of the nose may seed to cavernous sinus by way of the facial (angular) veins , occasionally leading to a cavernous sinus thrombosis.
2. Recurrent nasal vestibulitis. A chronically irritated nasal vestibule may be the result of recurrent staphylococcal infections. A patient can control vestibulitis by keeping his hands away from his nose and face and by applying bacitracin ointment to the nasal vestibule twice daily with a sterile cotton applicator. Patients with a mustache may have to remove it.
3. Erysipelas. This is a streptococcal dermatitis. *Clinical features.* Red swollen area with sharply defined margins. Pyrexia, lymphadenitis. **Treatment**. Systemic penicillin and locally applied analgesic and antiinflammatory ointment.

2.5.2. Inflammations of the nasal cavity

2.5.2.1. Acute rhinitis.

There are a host of viruses responsible for occurencies of the **common cold**: rhinoviruses, parainfluenza types 1-4 , influenza A and B, respiratory syncytial virus and adenovirus. **Pathology.** During the first 24 hours of a viral cold, there is a significant increase in the IgA level of nasal mucus. During the secretory phase of viral rhinitis there is a necrosis and shedding of epithelial cells accompanied by transudation of serum albumin and IgG. At this point, the nose is most inflamed and obstructed, and bacterial rhinitis and sinusitis may occur secondary to stasis of mucus. The normal flora of the anterior of the nose

include many pathogens that live symbiotically but that under altered conditions of anatomy and physiology can become pathogenic to the host. These include Staphylococcus aureus, Haemophylus influenzae, Streptococcus pneumoniae and haemolytic Streptococci. In case of bacterial rhinitis according to the continuous mucosal lining in the nasal cavity proper and paranasal sinuses and to the nasal obstruction it is better to call the condition as **rhinosinusitis**. *Symptomes* are headache, nasal obstruction, sneezing, anosmia, watery discharging in the viral phase and purulent discharging after bacterial superinfection. **Diagnosis.** Cultures are taken when the mucus becomes thickened and creamy or tinged with yellow. In small children this is an indication of bacterial rhinoethmoiditis and should be treated with appropriate antibiotics. **Treatment.** Seven to ten days of antibiotic treatment with analgesics, antihistamines and vasoconstricting nasal drops.

Chronic rhinitis.

1. **Non-specific chronic rhinitis**. (a) **Simple chronic rhinitis.** *Etiology.* Neighbouring infections, such as sinusitis, chronic tonsillitis, adenoids. Vasomotor rhinitis. Chronic irritation from dust, smoke, pollutants, snuff and the abuse of therapeutic vasoconstrictors. Nasal obstruction leading to retention of discharge. *Clinical features.* Nasal obstruction. Postnasal drip; Nose-blowing; Transient anosmia. *Diagnosis.* The soft, swollen mucosa over the inferior turbinates pits with a probe and shrinks with 5-10% solution of cocaine. If morning sneezing is frequent the condition is likely to be associated with vasomotor rhinitis. An excess of eosinophiles in the mucus suggests nasal allergy. Chronic infective sinusitis must be excluded by radiographs. **Treatment.** Correction of any predisposing factors, where possible (tobacco, alcohol). Locally a slightly alkaline nasal douche helps to remove the sticky mucus. Mild vasoconstrictors used as sprays to which an antibiotic may be added according to bacterial culture. Topical steroids will help in some cases.

(b) **Hypertrophic rhinitis**. *Etiology.* This represents an advanced stage of the simple chronic rhinitis. The causes are similar, but the patients frequently used to spray them with topical decongestants. *Pathology.* Permanent hypertrophic changes accompany the inflammatory oedema and cellular infiltration of the mucosa. The mucosa so becomes thick and nodular. The posterior end of the inferior turbinate shows the mulberry-like enlargement. Fibrosis can cause venous and lymphatic obstruction. If the resulting passive oedema occurs in a situation where the mucosal stroma is loose, polypi may form. Nasal polyposis, however, indicates an allergic or vasomotor origin of the rhinitis, in which the polyposis results from increased capillary permeability. *Clinical features.* Similar to those of simple chronic rhinitis but are unremitting in character.

Treatment. Principles are reduction of the hypertrophied turbinates and removal of polypi by: electrocoagulation, cryosurgery, surgical trimming.

(c) **Atrophic rhinitis**. *Etiology.* Besides infection possibly endocrine or vitamin disturbances may play a role. Types: **Primary atrophic rhinitis (ozaena)** . *Clinical features.* Today a seldom seen disease, more common in females. It appears after puberty. The foul stench is not noticed by the patient, who is anosmic. Epistaxis may follow separation of crusts. *Diagnosis* is establishes on the clinical picture as to sticky greenish crusts in the nasal cavity and nasopharynx causing nasal obstruction inspite of the big nasal space. **Treatment.** Removal of crusts is best achieved by warm isotonic solutions. Glocose 25% in glycerine drops prevent adherence of fresh crusts. Systemic and local antibiotics can be used initially. Several measures has been used in an attempt to increase the glandular and blood supply of the atrophic mucosa and to narrow the airway. These include potassium iodide therapy, endocrine preparations (such as stilboestrol locally or systemically) surgical measures to reduce the calibre of the airway (by submucosal injection of Teflon paste). Complete surgical closure of the nostrils for periods of several months has given striking improvement in the state of the mucous membrane.

Secondary atrophic rhinitis. Due to deviated septum, syphilis, lupus, excessive operative procedures like repeatedly performed FESS (functional endoscopic sinus surgery). Treatment is similar to the previous form.

2. **Specific chronic infective rhinitis**

(a) **Congenital syphilis**. Two forms can be distinguished: the early form manifests up to the 3rd mont**h (**"Snuffles" are the commonest nasal manifestations. This is a rhinitis which becomes rapidly purulent. Fissuring and crusting of the vestibule and lips)

The late form (3rd year to puberty) can be characterized by deformities similar to those in the tertiary stage of acquired syphilis result from gummatous infiltration of the nasal mucosa.

(b) **Acquired syphilis**. Three stages are distinguished: Primary stage. Onset 3-6 weks after contagion. *Chancre.* Rare in the nose and is a hard painless papule in the vestibule. Secondary stage. *Persistent coryza.* Tertiary stage. A *gumma* of the nose, which causes septal perforations and saddleback deformity of the nose plus nasal obstruction and headache. **Treatment.** General antisyphilitic treatment. Nasal toilet, as by irrigation with warm hydrogen peroxide.

(c) **Scleroma**. A chronic infection of the respiratory tract thought to be due to Klebsiella rhinoscleromatis. *Pathology.* Granulomatous tissue infiltrates the submucosa and characterized by large foam cells (Mikulicz cells) and Russell bodies (plasma cells with eosinophyl-staining cytoplasm). Infiltration starts at the vestibulonasal junction, atrophic degeneration follows and adhesions may

form. *Clinical features.* 1. Atrophic stage: nasal obstruction with crusting, discharge and some adhesions. 2. Tumefactive stage: Formation of soft, red, tumor-like mass in the nasal fossae. The external nose may become bulbous. 3. Cicatrizing stage: An unyielding concentric stenosis of the fossae takes place over the years. **Treatment.** Local and systemic steroid and antibiotic therapy. Prognosis is good for life but plastic repair may be necessary.

2.5.3. Inflammations of the paranasal sinuses (Rhinosinusitis)

Hiba!

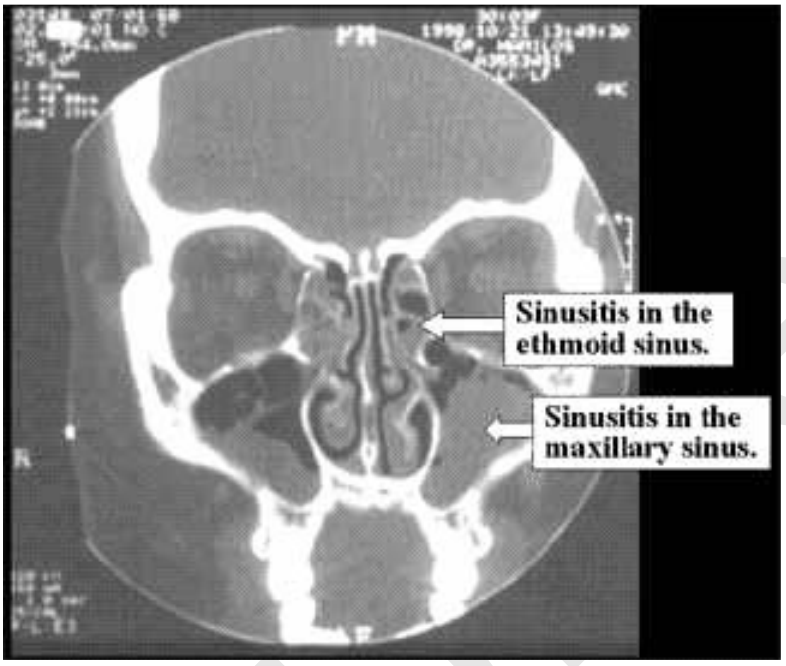


Figure 30. Coronal plane CT scan of ethmoid and maxillary sinusitis.

Acute sinusitis may be restricted to a single sinus or may be present as pansinusitis (unilateral or bilateral). The sinus may be closed if the exsudate can not escape because of closure of the ostium by oedema or the viscosity of the exsudate. Specific infections are rare. *Etiology.* Acute rhinitis due to a common cold may spread to the sinuses, assisted by sneezing and nose-blowing. Rhinitis is the commonest cause of acute sinusitis. Swimming or diving may cause similarly direct spread through the ostium. Dental extractions or infections can enter the maxillary antrum from a dental root. Fractures involving the sinuses may be followed by sinusitis. Predisposing factors are: Nasal obstruction for any cause, obstruction of the sinus ostium by nasal polypi, tumor, vasomotor or allergic swellings, neighbouring infections e.g. tonsillitis or adenoiditis, previous infection in the same sinus, mucociliary disorders, immunodeficiency, irritating atmospheric conditions. The commonest causative organisms are: Pneumococcus, Streptococcus, Staphylococcus, Haemophilus influenzae.

Pathology. The mucosal changes in the affected sinus are: Hyperaemias, oedema, cellular infiltration, increased mucus production, exsudation. **Common features are nasal obstruction, nasal discharge, headache.**

Acute maxillary sinusitis. It can be of nasal or dental in origin. *Features.* The maxillary sinusitis causes pain and tenderness in the cheek. Oedema is rare except in children. **Diagnosis**. Discharge in the middle meatus may be evident only after cocainization. Radiographs are helpful: show opacity or fluid level. **Local treatment** means irrigation by puncture through the inferior meatus, with isotonic saline, if medical treatment (antibiotics, antihistamine and vasoconstrictor nasal drops) is tardy. Intranasal antrostomy via endoscopic approach may be necessary in closed infections failing to respond to antibiotics. Acute maxillary sinusitis of dental origin may follow dental extraction if the sinus is opened. It can follow apical abscess of the premolar or molar teeth. Caries of the intervening bone may provide direct communication between the abscess and the sinus. A root-filled or dead tooth with abscess may be painless. The pus in these two latter cases may be malodorous, owing to the anaerobic and staphylococcus organisms associated. Cacosmia is usual. **Treatment** is irrigation of sinus and general therapy. A tooth or root retained in the sinus should be removed. Systemic antibiotics are necessary.

Acute frontal sinusitis

Usually associated with an infection of the homolateral anterior ethmoidal cells and often of the maxillary sinus. The tortuos nasofrontal duct is easily obstructed by oedema. *Clinical features.* Frontal headache which is periodic: starts soon after waking and subsides in the afternoon. Tenderness to pressure on the orbital roof. Oedema of the upper lids is not uncommon. Discharge is seen in the higher portion of the middle meatus. Diagnosis is given by Water's view and Caldwell's view anteroposterior X-ray. **Treatment**. Local decongestant therapy, antihistamines, antibiotics (cephalosporins, amoxycillin) may help. Failure of the medical therapy indicates nasal endoscopic approach to drain the frontal sinus.

Acute ethmoiditis. The ethmoid sinuses are separated from the orbital contents by a very thin plate of bone, the lamina papyracea. As a result, ethmoid sinusitis is occasionally complicated by orbital cellulites mainly in children. Patients with this complication are to be hospitalized for intravenous antibiotic therapy and intranasal endoscopic ethmoidectomy. Orbital cellulitis must be differentiated from cavernous sinus thrombosis (symptomatology and radiography: see in the section of complications of sinusitis). **Diagnosis** is given by CT scanning of the facial skull (**Fig. 30**). **Treatment** is medical therapy with

antibiotics, nasal drops, antihistamins. Orbital cellulites in chidren requires urgent endoscopic evacuation of pus from the affected ethmoid cells.

Acute sphenoid sinusitis

This occurs in conjunction with pansinusitis. However, can present as an isolated and potentially lethal infection mostly in immunosuppressed, diabetic, or elderly debilitated patients but rarely in young individuals also. *Symptomes* include fever, headache referred to the vertex of the skull and some purulent nasopharyngeal secretions. *Diagnosis* is based on CT scan examination. If the symptomes do not resolve rapidly, **endoscopic spheno-ethmoidectomy** should be performed, since there is the risk of central spread of infection through phlebotic veins or by the development of osteomyelitis. Because the lateral sinus wall is contiguous with the optic nerve, internal carotide artery, superior orbital fissure and the cavernous sinus the infection may spread to these areas. The superior orbital fissure syndrome consists of **panophtalmoplegia** involving the IIIrd, IVth and VIth cranial nerves and first division of the Vth cranial nerve. Cavernous sinus thrombosis is associated with spiking fever, exophtalmos, oedema of the orbit and lids, decreased vision, papilledema and panophtalmoplegia.

Chronic maxillary sinusitis. *Presents* with a generalized facial ache in the region of the sinus in addition to intermittent ipsilateral mucopurulent discharge. **Diagnosis** is radiographic (Water's view). **Treatment**. In the absence of a dental root abscess, carcinoma, or thick allergic or hyperplastic sinus mucosa, the condition may respond to a series of antral irrigation procedures along with a 10 days course of puncture and irrigation in every second day with a properly selected antibiotic based upon the antral culture. Otherwise, an opening of the maxillary antrum through the hiatus semilunaris should attempted in intranasal endoscopic control. A Caldwell-Luc operation to remove the sick sinus lining and to establish improved drainage is final modality of choice and often curative, however, today is rarely done.

Chronic frontal sinusitis. It may result from improperly treated acute frontal sinusitis, allergic polypoid sinus disease or obstruction of the nasofrontal duct secondary to scarring or fracture. Patients may *complain* of steady headache associated with local tenderness and purulent nasal or retronasal discharge. The sinus X-ray films of these patients demonstrate opacification of the sinus. Chronic frontal sinusitis may take the form of a **mucocele**, an expanding mucosal cyst filled with mucinous secretion. When infected, this cyst becomes **pyocele**. Mucoceles slowly erode the sinus walls and can reach the dura or expand into the orbit. The treatment for chronic frontal sinusitis or mucopyocele is endoscopic exstirpation of the disease from the sinus.

Chronic sphenoid sinusitis. It occurs as an isolated infaction in the chronically ill and elderly patient. Physical findings are scant, except for headache deep to

the eyes and intermittent purulent nasopharyngeal drainage. CT scan demonstrates thick mucosa, sometimes complete opacification. Cases that are refractory to the medical treatment require endoscopic surgical (FESS) drainage.
Chronic ethmoiditis. Most commonly is a complication of polypoid allergic sinusitis or polypoid hyperplastic sinusitis in which bacteria become entrapped. Patients suffer from nasal discharge and headache. Blockage of the upper portion of the nose produces diminished or absent sense of smell or taste. **Diagnosis** is established by CT scan examination. The complaints can be resolved by endoscopic ethmoid surgery (FESS).
Complications of suppurative sinusitis. The infection can spread directly through the bony wall by osteitis, osteomyelitis (in diploic bone) or osteoporosis (ehtmoidal polypi may cause dehiscences in the lamina papyracea or the floor of the anterior cranial fossa). The infection can spread by venous route: 1. Septic venous thrombosis, which occurs in the diploic veins during osteomyelitis. 2. Thrombosis in minute ways in sinus mucosa. 3. Septicaemia and pyemia. Two more infection routes still exist, the lymphatic spread and spread via perineural spaces (the olfactory nerve to the subarachnoid space).
Osteomyelitis. Infection of the frontal sinus is the usual cause of this uncommon condition. It is most frequent in young adults. Entry of infection into the marrow spaces occurs by direct or venous spread. It is often precipitated by trauma or operation on the anterior wall of the frontal sinus in the absence of prophilactic systemic antibiotics. Spreading thrombosis occurs in the diploic veins. Subperiosteal abscesses form extracranially and extradural abscesses form intracranially. Sequestration occurs later. Thrombosis of venules crossing the suture line allows the whole calvarium to be affected. *Clinical features.* Dull local pain and headache occur at the onset, which is insidious. An oedematous area arises on the forehead, often a short distance above the upper limit of the frontal sinus. This is a Pott's puffy tumor. *Diagnosis.* Radiographs (CT scan) show loss of bone pattern after a week or two, but a spreading area of necrosis with sequestration can be seen later. The bony sinus wall may be seen to be breached. **Treatment.** Prophylaxis is by antibiotic cover at operations. In early cases an acute frontal sinusitis may often be controlled by systemic antibiotics. Usually no operation is needed other than irrigation of the maxillary sinus and possible endoscopic drainage to the nasofrontal duct. Radical surgery is indicated if improvement is tardy or unsatisfactory, if sequestration is present or if intracranial complications threaten.
Orbital complications. All the sinus enter into the boundaries of the orbit. The walls are thin and are reliable to be eroded by an osteitis. Orbital infection is seen more frequently in children as a result of ethmoiditis. Orbital cellulitis can occur with or without the formation of a subperiosteal abscess. Pus easily lifts the periosteum and points through the eyelids. Spread to the cavernous sinus is a further possibility. *Clinical features.* Pain referred to the eye may be

only slight. Chemosis closes the lids. Proptosis, diplopia from displacement of the globe. Engorgement of the retinal veins is the only change seen in the fundus. *Differential diagnosis.* Dacryocystitis. Presents as a swelling deep to the medial palpebral ligament in the absence of sinusitis. Pus leaks through the canaliculi. Cavernous sinus thrombosis. There is early paralysis of the eye muscles and marked fundus changes. Mucocele. Long history of painless swelling is characteristic. "Eggshell cracking " may be felt. Osteoma, which is hard, painless and of long duration. The radiological appearance is charcteristic. Malignant orbital tumor. These tumors are painless, rapidly growing. Biopsy is required to establish the diagnosis. Erysipelas of the eyelids. **Treatment.** Antral lavage is mandatory. Further actions may be required for: Cellulitis, subperiosteal abscess. In children, an orbital abscess due to ehtmoiditis is difficult to distinguish. An emergency CT scan will show if pus formed. Endoscopic ethmoidectomy is then necessary to draining. Orbital abscess or oedema may require radical sinus surgery by an external approach.

Intracranial complications. They include: Pachymeningitis, with or without extradural or subdural abscesses. Leptomeningitis: serous or purulent, localized or diffuse. Thrombophlebitis of the cavernous sinus, sagittal sinus and frontal cortical veins. Brain lesions:

1. Frontal sinus leads to frontal lobe abscess
2. Ethmoiditis can cause diffuse suppurative meningitis
3. Sphenoidal sinusitis can cause diffuse suppurative meningitis and thrombosis of the cavernous sinus
4. Maxillary sinusitis rarely causes intracranial lesion

Secondary effects of suppurative sinusitis. Infection of the nasopharynx may cause: 1. Lateral pharyngitis; 2. Otitis media; 3. Laryngotracheitis; 4. Bronchitis.

Suppurative sinusitis can be associated with bronchiectasis. The relationship between bronchiectasis and sinusitis is not fully understood but there is a common cilial failure in Kartagener's syndrome and a mucus one in cystic fibrosis. One certainly aggravates the other. Sinusitis can be associated with asthma. A radical sinus surgery in these cases may temporarily be benefitial, but recurrence is common. Asthma may be triggered off for the first time after operation as a part of the natural progress of the allergic state. In the absence of gross infection, surgery should be limited to the removal of obstructing polypi.

Focus of infection. A chronic sinus infection may occasionally act as a focus of infection at distant sites, but such a relationship is much more doubtful than was previously supposed. There are, however, a few condutions which do sometimes respond to the elimination of a suppurative sinusitis. These include infective polyarthritis, tenosynovitis and certain skin deseases.

2.6. Tumors of the nose and paranasal sinuses.

Benign tumors. **Papilloma** may occure in the nasal vastibule as a small sessile wart or in the multiple form. After removal its base should be cauterized to prevent recurrence. Carcinoma mast be excluded by histological examination. Another type of papilloma is the nasal fossa and paranasal sinus papilloma. This is an epithelial papillomatous tumor (**transitional cell or inverted papilloma***)*. Gross thickening of the epithelial surface leads to infolding but the basement membrane remains intact. A malignant change occurs in a small proportion of cases. Obstruction is the presenting symptome. The tumor should be removed *in toto* what may require lateral rhinotomy. Recurrence suggests malignancy. Among the fibrous tissue bening tumors, osteoma is the most frequent and mainly occurs in the frontal sinus. *Clinical features.* It remains symptomeless for a long time. Headache, displacement of the eye, empyema or mucocele of the sinus may be presenting. Radiography shows dense mass inside the cavity which sometimes completely fills it. **Treatment** is surgical removal if caused complaints.

Among the rare tumors there are the "bleeding polyp of the septum" which is a capillary hemangioma (epistaxis is the usual symptome and the treatment is laser excision), the craniopharyngioma (probably derived from the Rathke's pouch: its extracranial portion may occupy the sphenoid sinus, while its intracranial portion may cause raises intracranial pressure).

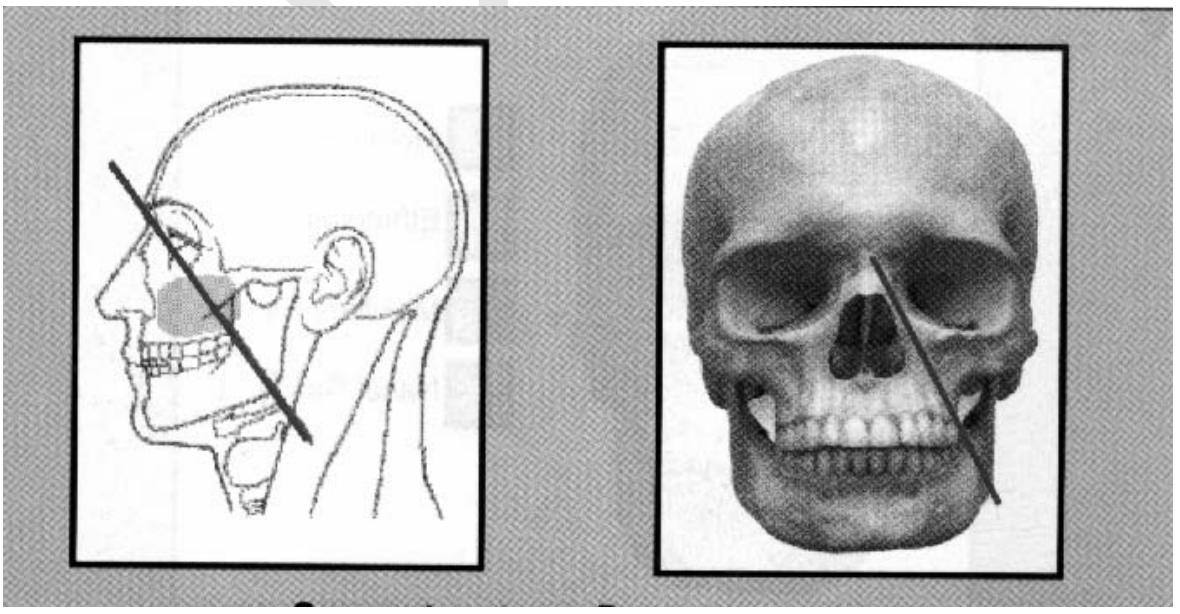


Figure 31. Oehngren's plane. The plane separates the facial skull to anterior-inferior and posterior-superior parts. Former includes maxillary sinus, anterior and middle ethmoid cells and the lower 2⁄3 of the nasal cavity proper. Dicision making in treatment of paranasal sinus malignancies, the location of a tumor according to the plane is of prognostic value. Antero-inferior location dictates combined surgical and chemoirradiation therapy. Postero-superior tumor location requires neurosurgical and rhinologic approach when surgery is choosed otherwise primary chemoirradiation is the modality of choice.

Malignant tumors. **Carcinoma:** Squamous cell carcinoma is the commonest malignant tumor. The tumor infiltrates the soft tissues, destroys bone and ulcerate into the mouth, pharynx. Lymphatic metastasis to the upper deep cervical lymphnodes occurs. Haematogenous metastasis is rare. **Treatment**: Preoperative irradiation, than surgery and again a postoperative irradiation. **Lymphoepithelioma** is found more often in the nasopharynx than in the nose or sinuses. **Adenocarcinoma** arises from the glands of the upper respiratory mucous membrane, particularly in the maxillary antra. The rate of growth is slow but relentless, sometimes crossing the midline and metastases are late. **Adenoid cystic carcinoma** (**cylindroma)**. Columns of cells cut in cross section resemble a series of tubes. They appear as firm, round, encapsulated tumors especially on the alveolus, hard palate, antral floor. They tend to recur locally. Among the connective tissue malignancies, *sarcoma* is the most common. Fibrosarcoma, osteosarcoma and lymphosarcoma occur. The treatment is first of all surgical. When surgery fails to provide result irradiation and chemotheropy can be considered. The most frequently -although they are rare - occuring **odontogenic tumors** are the **ameloblastoma** and **odontoma**. The previous one is a multilocular cyst which causes slow expansion of the jaw. Since it grows infiltratively locally, it should be removed by a radical excision. Odontomas appear like normal teeth sometimes and a calcified masses in others. Should be removed surgically.

Symptomes of malignant tumors in the nose and sinuses. Differ according to the site of origin.

1. Unilateral nasal obstruction, may be due to polypi, thickening of the nasal wall
2. Nasal bleeding and discharge
3. Swelling of the cheek, alveolar margins, nasal bridge or palate.
4. Loosening of teeth
5. Unilateral proptosis can arise by compression of the orbital veins.
6. Pain, facial paraesthesia
7. Severe headache
8. Epiphora Results from involvement of the nasonacrimal duct.
9. Other features include: diplopia, trismus, limitation of jaw movements, anosmia, optic atrophy.
10. Metastasis are first of all lymphnode matastases.

Diagnosis. Radiography (CT scan) and biopsy.

Treatment. The Oehngren's plane (**Fig. 31**) helps in treatment planning and also used for prognostic purposes. Irradiation pre-, and postoperatively (megavoltage: like telecobalt). Intracavity afterloading can also be performed for residual tumors.

Surgical procedures:
1. Sublabial antrostomy (Caldwell-Luc operation): is used for biopsy of suspected neoplasm.
2. Lateral rhinotomy: may be used as an approach to localized growths in the lateral nasal wall and ethmoid.
3. Partial maxillectomy: Usually comprises a palatal fenestration, which is removal of half the hard palate and the upper alveolus (including if necessary the maxillary tuberosity) Suitable for limited growths of palate, alveolus or floor of antrum.
4. Total excision of the upper jaw. Besides allowing excision of the maxilla this approach also permits exploration and exenteration of all homolateral sinuses. The septum and hard palate of the opposite side may also be removed.

2.7. Miscellaneous conditions

2.7.1. Epistaxis.

Anterior septal bleeding from Kisselbach's plexus is primarily venous and is far the most common variety nose bleeding. The possibility of clotting disorders, blood hypertension should be ruled out.
Examination demonstrates venous bleeding from one side of the nasal septum. A check should be made for septal spurs and deviations, ulcerations, septal perforations, granulomas, foreign bodies and tumors. There is a great deal folklore regarding the **treatment** of epistaxis. Patients, and in the case children their parents should be questioned about treatment methods because many old tricks or misconceptions such as lying down, merely serve to prolong bleeding. Taking the time to teach patients the principles of treatment for minor epistaxis will help them have fewer bleeding episodes. The patient should sit up and lean forward to reduce venous pressure in the head and to prevent the swallowing of blood. A small piece of cotton soaked with vasoconstricting nose drop such as oxymetazoline hydrochloride is placed in the vestibule for 5-10 minutes. This stops almost all venous nose bleeding.
If this fails to stop bleeding then the mucous membrane can be anaesthetized by 10% cocain hydrochloride and a silver nitrate (10%) stick can be applied to the membrane over the bleeding site. Occasionally a small artery will also fail to stop to the above manipulations when electrocautery should be done and small packing into the nasal vestibule for 24 hours. Chronic nasal ulcerations due to septal bony spurs, or septal deviations may require *submucous septal resection or septoplasty*.

Arterial epistaxis is a less common variety and involves primarily middle aged or elderly adults. The patient's history may include hypertension, trauma or surgery. Examination for arterial nose bleeding requires head mirror and nasal suction. Treatment for arterial bleeding usually means nasal pack.

Nasal packing has several subvarieties:
1. Nasal tamponad: gause stripes layered on the floor of the nasal cavity and fill completely. They may be regularly soaked with vasoconstrictive agents like nasal spray and antibiotic solution. The tampon stripes should be removed after 5 days as latest. Another possibility is to use nasal balloons, one end of which is in front of the choana and the other end is in the nasal vestibule. Patients should be hospitalized with nasal anterior or posterior packing or balloon. When a nasal balloon or packing is not effective, then anterior and posterior nasal pack should be applied (**Fig. 32**). The patient must be well-medicated beforehand and the nose and palate must be anaesthetized. For posterior packing, a soft rubber catheter is inserted through the side of the nose that is bleeding until the end is seeing in the oropharynx; it is then grasped with a clamp and brought out through the mouth. Two ends of the sutures of the pack are secured to the catheter, which is now withdrawn from the nose as the pack is placed into the posterior choana. The phisician can use fingers to facilitate placement of the pack into the choana.
Surgical options. The surgical options are the following: 1. Transantral ligation of the internal maxillary artery and ligation of the anterior and posterior ethmoidal arteries are almost uniformly succesful and stop bleeding. 2. In case of failure a ligation of the external carotid artery must be attempted but it has lower succes rate because of the large collateral blood supply in the head and neck. 3. Selective internal maxillary arteriography and embolization is an elegant and very benefitial method and can also be attempted in bleedings derived from Osler's disease (generalized capillary haemangiomatosis).

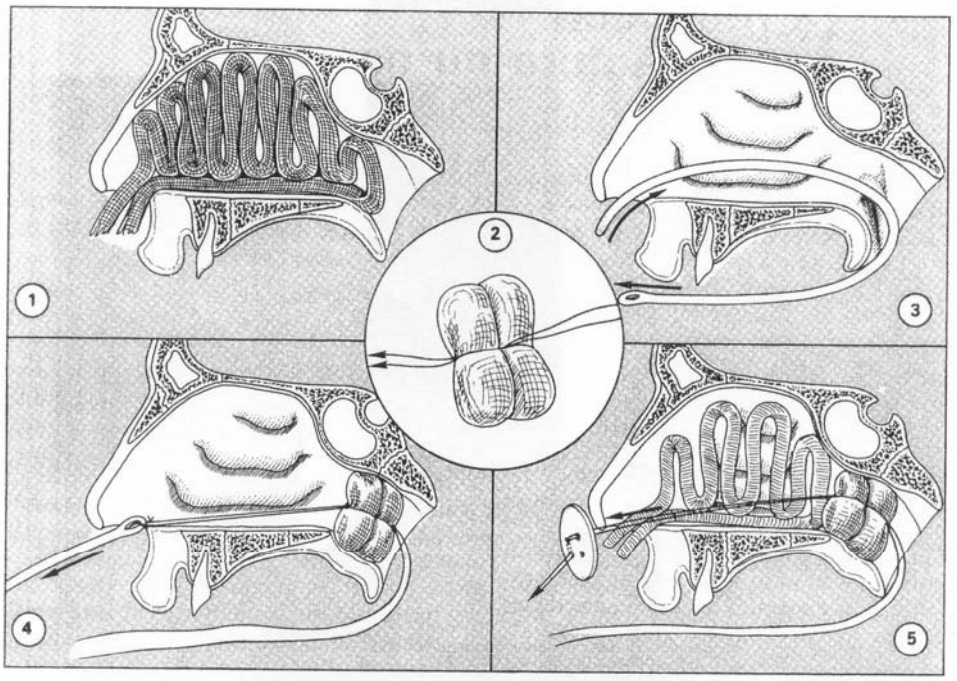


Figure 32. Upper panel: Anterior (1) and posterior nasal pack (2-5) A gauze sponge is folded in half, trimmed, rolled and tied with sutures (2). A rubber catheter is passed through the bleeding nostril and brought out through the mouth (3-4). The pack is tied to the catheter by sutures. The catheter is withdrawn from the nose and the pack is guided and pressed by the index finger into the posterior choana (4). Finally an anterior nasal pack is placed into the nasal cavity layer by layer (5).

2.7.2. Vasomotor rhinitis.

This term denotes a combination of nasal obstruction, watery rhinorrhoea and sneezing of unknown etiology. The symptomes appear to be due to predominance of parasympathetic activity. *Predisposing factors.* Heredity, infections and emotional factors (fear produces a vasoconstriction, while anxiety leads to engorgement of the nasal mucosa). Endocrine influencies may be more important. Vasomotor rhinitis is common at puberty; during menstruation and pregnancy; with sexual excitement ("honeymoon rhinitis") and in old age (senile rhinitis). Drugs can cause oedema of the nasal mucosa (hypotensive drugs: adrenergic blocking agents, methyl dopa; anticholinesterases such as neostigmine; oral contraceptives high in oestrogen). *Overuse of local applications tend to produce "rhinitis medicamentosa". Precipitating factors.* Usually act as a trigger on a hyperreactive mucosa. Atmospheric conditions (changes in humidity and temperature), fumes, dust, alcohol may provoke a hypersensitivity. Reflex sneezing on walking and excercise may act as a factor. Commonest picture is the hypertrophy of the inferior turbinate responsiveless to vasoconstrictor agents such as α-adrenergic agonists. *Clinical features.* The chief complaints are: 1. Sneezing which is paroxysmal. 2. Rhinorrhoea which is spasmodic, profuse and watery. 3. Nasal obstruction, which is often variable and may alternate from side to side. 4. Postnasal discharge. 5. "Nasal tip dew-drop" in elderly persons. **Diagnosis** is based upon symptoms and exclusion of allergic rhinitis. **Differential diagnosis**: cerebrospinal liquorhoea should be excluded. **Treatment.** Known precipitating factors should be avoided. Antihistamines are useful when sneezing and rhinorrhoea are present. Iprotropium bromide is especially useful for watery rhinorrhea. Local application of capsaicin and desensibilization of the parasympathetic neurons are newest trials. Cryosurgery to the surface of anterior turbinates reduces population of mucous glands. Its effect is, however, short lived. Submucosal diathermy to swollen inferior turbinates reduces the bulk of the submucosa. The effect lasts for about 6-9 months. Removal of polypi is mandatory if large and obstructive. Correction of septal deflections should be considered to relieve an obstructed airway. Vidian neurectomy rarely may be justified in severe cases where sneezing and rhinorrhoea are the chief complaints. Symptomes frequently return within 2 years.

2.7.3. Nasal allergy.

Allergy is an abnormal reaction of the tissues to certain substances, the allergens. These are antigens, capable of making the body to produce amtibodies. In allergic subjects, a special form of antibody (IgE) is produced. These fix easily on tissue cells, including those of the nasal and bronchial mucosae. Similar reactions can be produced by non-specific factors (vasomotor rhinitis) thereby causing confusion. *Etiology.* There are three main phases. l. IgE is formed by lymphocytes. In normal individuals there is an IgE suppressor factor which keeps IgE synthesis in control. In allergic individuals there is an IgE helper factor which appeares to promote production at times of exposure to an allergen. 2. IgE is bound to mast and basophyl cells. An interaction between the cell-bound IgE and the allergen initiates the secretion of pharmacologically active mediators such as histamine that leads to the clinical manifestations. 3. Changes occur as in acute inflammation, capillaries become permeable, the ground substance viscosity is reduced by enzymes such as hyaluronidase and oedema occurs. Serous alveolar glands are stimulated directly or via the autonomic reflex nervous patterning to produce excess watery secretion. *Predisposing factors.* They may coexist;
1. Hereditary. The term "atopy" is used to define the inherited tendency.
2. Physical. Changes in humidity of the inspired air may render the nasal mucosa more susceptible. 3. Infections. *Precipitating factors.* Allergens may be grouped as: 1. Exogenous, coming from outside the body:
Inhalants:dusts, pollens, animal emanations, feathers and fungal spores. *Foods*: (especially important in children) egg, strawberries, nuts or fish, milk or wheat. *Contacts to nasal mucosa*: face powders, hairs from electric rasors. 2. Endogenous coming from within the body. These include tissue proteins (from injured tissue exsudates or transudates). *Pathology.* Oedema, infiltration with eosinophyls, thin watery discharge, vascular dilatation, polypi in the ethmoid cells. The sinuses are involved, and can be shown out as thickening of the mucosal lining. Clinically two types are the seasonal (hay fever) and non-seasonal (perennial) nasal allergies. *Symptomes.* Nasal obstruction, rhinorrhoea, sneezing and anosmia. **Diagnosis.** Most helpful beside the case history and clinical examination are tests like checking eosinophyles in the nasal secretions and skin tests. These are confirmatory. They may show single or multiple sensitivities. Nasal provocation tests by a drop of solution containing the putative allergens induce nasal watery discharge and sometimes lacrimation. **Treatment.** The precipitating factors should be avoided if possible. Desensitization is sometimes benefitial. It may be indicated if medical treatment is useless and the allergen can not be avoided. It is only of value in those patients who are sensitive against one allergen. There is a risk of anaphylaxis. Antihistamine drugs and topical steroid nasal sprays give best results with

sodium chromoglycate. Surgary may be necessary for the relief of gross obstruction (removal polypi, adenoids, tonsils, reduction of inferior turbinates).

2.7.4. Nasal polypi

These are oedematous hypertrophy of the submucosa. Might be ethmoidal cell or maxillary antrum in origin. The latter may be single and should be distinguished from meningocele (meningocele shows pulsation synchronously with hard beating). *Symptomes* . Nasal obstruction and subsequent snoring, anosmia, postnasal catarrh. Antrochoanal polypi might cause marked obstruction without any visible ethmoidal polyp. Sneezing and rhinorrhoea are common symptomes. **Treatment.** Conservative: antihistamine preparations, local decongestants and topical steroids as aerosol. Surgical: Ethmoidectomy may be performed by three routes: intranasal, transantral (since the anterior ethmoidal cells can not be reached by this approach, it must be combined with intranasal surgery). Complications of the polyp removal may be: anosmia, damage to the optic nerve, meningitis (by penetration of the floor of the anterior cranial fossa).

2.8. Headache and facial pain

Many ENT diseases cause pain in the head. The presentation, duration, character of the pain are often helpful to establish the possible pathology in the background. Headaches of non oto-rhino-laryngologic origin commonly appear as diffuse pain. These are due to **hypertension** or hypotension, drugs, febrility and **intracranial manifestations** (cerebral infarct, stroke, subarachnoid hemorrhage, inflammatory intracranial diseases: meningitis, encephalitis etc., disorders in the CSF drainage). Neurological and internal medical check up examinations are recommended in all those cases when the clear cause of the pain is uncertain.

Vasomotor headaches can be bilateral (vasomotor cephalalgia) or strictly unilateral (migraine, erythroprosopalgia). Former is often diffuse, dull pain sometimes is maximal above the forehead. Migraine is characterized by periodic attacks and associates with visual symptoms, nausea and vomiting. Erythroprosopalgia (cluster headache, histamine cephalalgia) can be presented also as supersensitivity of the scalp. It can be relieves by histamine or nitroglycerine.

Eye diseases often associate with pain. Most frequent are the refraction and accommodation disorders. These typically are presented after or during long term reading. The pain is dull or pressing. Acute and chronic glaucoma present

with sudden retrobulbar or forehead pain. Optic neuritis and keratitis cause persistent pain. Latter can be interrupted by analgesic eyedrops.

Nasal or paranasal sinus diseases can onset as local or diffuse facial pain. Furuncle of the nasal tip is recognized by physical examination. Nasal obstruction of any cause (inflammation, foreign body, septal deviation, tumor, synechia, vasomotor rhinopathy) can be responsible for headache. Localized pain features different sinusitis. Forntal sinusitis causes not only forehead pain but pressure sensitivity above the forehead, as well. Pain to maxillary sinusitis is presented in the cheek, upper jaw and forehead. Medial canthus and root of the nose are involved in ethmoid sinusitis. Occipital headache are typically due to sphenoid sinusitis.

Otogenic headache characterizes otitis externa, herpes zoster oticus, acut otitis media, mastoditis, skullbase osteomyelitis, ear tumor, petrositis. The presenting other symptoms (aural discharge, hearing loss, skin vesicles) or the localized pain are helpful to call the attention to the origin of the pain.

Pharyngeal headaches are manifested in acute tonsillitis, peritonsillar and retropharyngeal abscess, elongated styloid process (Little's syndrome) and oro-hypopharyngeal tumors. Common features are dysphagia and odynophagia.

Unerupted teeth, pulpitis, periodontitis and inflammation of the jaw are **dental** causes of facial pain. Osteomyelitis of the mandible may cause trismus as well as unerupted tooth.

Temporomandibular joint diseases e.g. arthrosis, arthritis appears as localized pain during mastication which can be persistent later on.

Inflammation or calculosis of the **salivary glands** result in diffuse dull or intensive localized facial pain. Epidemic parotitis is a bilateral disease versus the exclusively unilateral purulent sialadenitis of the parotid or submandibular gland. Local redness or even fluctuation above the abscess determines the disease.

Headache can be due to **cervical spondylosis**, whiplash injury, cervical myalgia.

Large group of headaches are **neuralgia**s. These are extremely intensive pain attacks. Neuralgias can be characterized by trigger zones or even points. **Trigeminal neuralgia** is mainly presented in the area of the second division. Usually it is unilateral, lancinating pain. The trigger zones of the n. V./2 are the foramen infraorbitale, cheek, upper jaw. The ethmoidal nerve may be affected. Its trigger zone is the junction of the nasal bone and cartilage. The trigger zone for the third division (mandibular nerve) is the mental foramen and the tongue. The first division (ophthalmic nerve) is rarely affected and the pressure point is the supraorbital foramen and the forehead. Trigeminal neuralgia is characteristically presented as the "tic douloureux" and may be evoked by eating, contacts, speaking. Lasts couple of minutes. **Glossopharyngeal**

neuralgia is unilateral pain in the tongue, tonsil, hypopharynx and ear. It is an intensive attack elicited by eating or speaking. Unilateral pain in the neck from the ear down to the larynx is the **vagus neuralgia**. The trigger zone is thyrohyoid membrane. Sluder's neuralgia (pterigopalatine ganglion neuralgia) is presented in the nasal root or orbital cavity. This is an episodic intensive pain especially at night and often combined with sneezing attacks. This is usually the disease of women.

3. The mouth and pharynx

3.1. Anatomy

The **mouth** extends from the lips to the anterior pillar of the fauces and consists of two parts: the vestibule and the mouth proper. The mouth cavity proper posteriorly communicates with the pharynx by means of the oropharynx isthmus which is bordered by the palatal arches and the uvula. The mylohyoid muscle is the main muscle of the floor of the mouth and is a thin sheet arising from the whole length of the mylohyoid ridge to be inserted into the hyoid bone and median raphe. The oral cavity is filled with the tongue. The dorsum of the tongue is covered by a modified epithelium which contains papillae (filiform, fungiform, foliate and vallate at the tip, lateral edge and posteriorly, respectively) for **taste receptors**. A V-shaped terminal sulcus borders the tongue proper from the base of the tongue. The central point of the sulcus is the **foramen cecum** which is the rudiment of the thyroglossal duct and can be the place of an only functioning ectopic thyroid gland presenting as a tumor at the base of the tongue. The main mass of the tongue is given by muscles: genioglossus muscle, hyoglossus muscle, palatoglossus and styloglossus muscle. Some muscles without bony attachments run in the 3 dimensions and provides with improved mobility. At the floor of mouth is found the openings of the Wharton's duct (from the submandibular gland) and the Bartholini's duct (from the sublingual gland). **Blood supply** to the tongue (lingual artery) and mouth (sublingual artery for the floor, facial artery for cheeks, descending palatine artery for the soft palate and ascending pharyngeal artery for the tonsillar fossa and palatal arches) is derived from the external carotid artery. Motor **nerve supply** to the tongue is provided by the hypoglossal nerve (n. XII) whereas the sensory supply comes from vagus nerve. Taste buds are innervated by the chorda tympani (from n. VII) at anterior two third and by the glossopharyngeal nerve (n. IX) at the posterior one third of the tongue.

The cavity of the **pharynx** is the upper part of the respiratory and digestive passages. It extends from the base of the skull to the level of the sixth cervical vertebra. The cavity is devided into three parts:

1. **Nasopharynx** (epipharynx) opens anteriorly into the nasal fossae. It is bounded above by the skull base, below the soft palate. Lower opening of the Eustachian tube is situated in the lateral wall about 1 cm behind the posterior end of the inferior turbinate.This bounded above and behind by the tubal elevation (torus tubarius) which is formed by the tubal cartilage covered by mucous membrane. Pharyngeal recess (fossa of Rosenmüller) lies behind the tubal elevation. Nasopharyngeal tonsil (adenoid) sits at the junction of the roof and posterior wall of the nasopharynx.

2. **Oropharynx** (mesopharynx) is bounded above by the soft palate, below by the upper border of the epiglottis. Palatine tonsils are situated in its lateral wall, between the anterior and posterior pillars of the fauces.

3. **Laryngopharynx** (hypopharynx) opens anteriorly into the larynx. It is bounded above by the upper border of the epiglottis, below by the lower border of the cricoid cartilage. Pyriform fossae are small recesses lying on each side of the laryngeal inlet. Each is bounded by the aryepiglottic fold medially and by the thyroid cartilage and thyrohyoid membrane, laterally. The internal division of the superior laryngeal nerve runs beneath the mucous membrane of its floor. Valleculae glossoepiglotticae are paired shallow recesses lying between the base of the tongue anteriorly and the anterior surface of the epiglottis posteriorly. They are separated by the midline glosso-epiglottic fold and bounded laterally by the pharyngo-epiglottic folds.

The structure of the pharynx. The pharynx is a fibromuscular tube and has four layers. 1. *Mucous membrane.* Ciliated columnar epithelium in the upper half of the nasopharynx; stratified squamous epithelium in the oro-, and hypopharynx. *Subepithelial lymphoid tissue of pharynx.* Scattered collections of lymphoid tissue distributed beneath the pharyngeal mucosa form the **Waldeyer ring**. They have efferent lymph vessels but no afferent vessels. They consist of: *palatine tonsils* on each side of the oropharynx; *nasopharyngeal tonsils* (adenoids) between the roof and the upper part of the posterior wall of the nasopharynx; *lingual tonsils* at the upper surface of the base of the tongue; *tubal tonsils (Gerlach's tonsils)* lie in the fossae Rosenmülleri; *plicae tubopharyngeae* are located in the two sides of the nasopharynx and mesopharynx just behind the posterior palatal arches. Some scattered small spots of lymphoid tissue is situated in the laryngeal ventricles either.

2. *Pharyngeal aponeurosis*. An incomplete connective tissue coat in the lateral and posterior walls of the pharynx between the muscular layers.

3. *Muscular coat* has two layers: (a) external (Superior, middle and inferior constrictor pharyngis muscles (b) stylopharyngeus and palatopharyngeus muscles.

The **vascular supply** is given by the ascending pharyngeal artery, the ascending palatine artery, the facial artery and the maxillary artery. **Venous drainage** is provided by the facial internal jugular vein, whereas the **lymphatic drainage** is directed to the deep parajugular lymphnodes (epi-, mesopharynx) and to the paratracheal lymphnodes (hypopharynx). Motor and sensory **nerve supply** arrives from the n. VII, n. IX, n. X, n. XII.

Immune function of the Waldeyer's ring.

Surpisingly the tonsils have only efferent lymph vessels which transport lymphocytes to these lymphoepthelial organs. The lack of the afferent vessels means that the antigen exposed lymphocytes are not transported back to the reginal lymphnodes. Conversly, the tonsils secret the topically immune-stimulated lymphocytes into the pharynx which then by swallowing enter the digestive tract and being absorbed in the intestines will be presented in the mesenterial lymphnodes. The number of these lymphocytes is 100 million daily. They provide with the antigen surveillance in the upper aero-digestive system keeping informed the organism about the antigen environment. The hyperplasia of the lymphoepithelial tissue in early childhood is the sign of active defensive mechanism and consequently the enlargement of e.g. the palatine tonsils does not mean a continuous or chronic tonsillitis by itself. It recalls an active and physiological immune response of the infantile organism. A chronic tonsillitis which can be indication for tonsillectomy is characterized by hypertrophy of the connective tissue in the lymphatic organ with reduction of the lymphatic tissue (the result is a less mobile, hard, fibrotic tonsil) . In other words, the chronic tonsillitis is a term for a lymphatically non-functioning tonsil which however maintains a persisting infection.

Parapharyngeal space. This lies outside the pharynx and is of great importance surgically. It extends from the base of the skull above to the superior mediastinum below. The space and its contents may become infected. Its anteromedial wall is formed by the buccopharyngeal fascia. Posteromedial wall consists of the transverse processes of the cervical vertebrae, covered by the prevertebral fascia and muscles. The lateral wall is formed by the mandible, pterygoid muscles, parotide and by the sternomastoid muscle. Its contents: great vessels of the neck (internal carotid artery, internal jugular vein), ascending palatine and ascending pharyngeal arteries, deep cervical lyph nodes, the last four cranial nerves and the cervical sympathetic trunk. Infection can spread

from the skullbase to the mediastinum in it. **Retropharyngeal space**. Lies behind the pharynx and contains the retropharyngeal lymph nodes. **Blood supply of the pharynx**. Ascending pharyngeal and palatine artery and tonsillar branches of the facial artery, branches of the internal maxillary and of the lingual artery. Veins drain into the facial and internal jugular veins. **Nerve supply**. Derived chiefly from the pharyngeal plexus (IXth and Xth cranial nerves). Motor innervation is provided by the IXth cranial nerve. The nasopharynx is supplied mainly by the Vth cranial nerve. Lymphatic drainage is directed into the deep cervical nodes **(Fig. 33)**.

3.2. Examination of the pharynx. and mouth

The examination of the nasopharynx is described with the examination of the nose. The oropharynx can be viewed by depressing the tongue. Inspection may reveal redness, swelling and ulceration of the mucosa, enlargements of the tonsils. The orifices of the parotid and submandibular glands should be checked. Asymmetry in the palatal arches between the two sides calles the attention for peritonsillar or tonsillar processes. The posterior mesopharyngeal wall can show postnasal drip. The consistency of the tonsils should be described as well as the result of the expression test when an attempt is made to compress pus out of the crypts to verify chronic tonsillitis. In conjuntion with examination of the tonsils the jugulodigastric lymphnodes should be palpated for another sign of chronic tonsillitis.

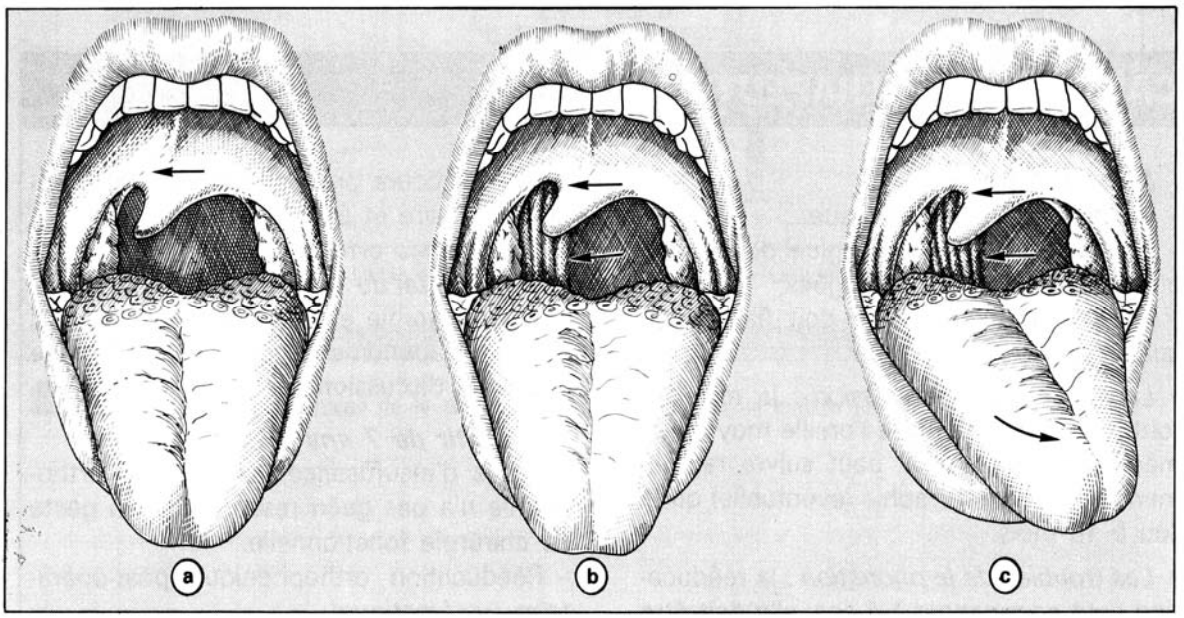


Figure 33. Neurology of the soft palate. (a) isolated paralysis of the n. X and XI. The uvula is dislocated to the affected side. (b) Curtain sign of Vernet in the case of paralysis of the n. IX and X and the pharyngeal constrictor muscle is also paralysed. (c) Velopharyngeal and tongue paralysis. Notice that the tongue is deviated to the contralateral side of the paralysis of n. IX, X and XII.

Palpation is necessary if malignancy is suspected in the tongue base. The hypopharynx is examined with laryngeal mirror, fiberscopic laryngoscopy or direct laryngoscopy (with laryngoscope under general anaesthesia). Examination of the pharynx should be completed by examination of the cervical lymph nodes.

Videolaryngoscopy does not only visualizes the larynx but gives important and invaluable informations about hypopharynx and the pyriform recesses either.

Computed tomography and **magnetic resonance imaging** should be performed in all surgical tumor cases.

3.2.1. Gustometry

A qualitative test can be the application of different test solution to the specific areas of tongue dorsal surface by cotton probe. These solutions are 20% sugar solution, 10% salt solution, 5% citric acid solution and 1% chinin solution. A table with names of these four main tastes are held in front of the patient who should show to the examiner what taste is recognized with open-mouth prolonged after application of the test solution. Both tongue sides should be examined separately.

Electrogustometry gives better and quantitave estimate of taste disorders (ageusia). The taste buds are stimulated electrically and current intensity elicites taste sensation are measured. An adult threshold is between 1-8 μA.

An objective gustometry is still in experimental state.

3.3. Inflammatory diseases of the mouth and the pharynx

3.3.1. Stomatitis

Inflammatory lesions of the oral mucosa are produced by a variety of causes which may be local or systemic. **Recurrent ulcerative (aphtous) stomatitis**. A common condition of unknown cause (viral, hormonal, autoimmune factors and vitamin deficiency are suggested). *Symptomes*. Small vesicles may occur at any portion of the buccopharyngeal mucosa. Ulceration then soon occurs. The ulcers have sloughing base with a marked ring of hyperaemia. Pain may be extreme and lasts for several days. Recurrence is a characteristic feature of the ulcers. Treatment is local application of hydrocortisone, anaesthetics. Aspirin relieves pain promptly. **Behcet's disease**.

Comprising oropharyngeal and genital ulceration with iritis and hypopyon. Symptomes are painful ulcers which heal within few days but recur. Neurological manifestations may appear after 2-5 years and may resemble acute encephalitis. Blindness may result from ocular lesions. Treatment is nonspecific (steroid: 20-40 mg prednisolon daily). *Thermal, chemical, physical injuries and irradiation can also cause stomatitis as well as metals (mercury:black line around the gingival margin; lead, gold)) and drugs* (Epanutin, an antiepileptic drug; antibiotics, antidepressants, bromides, iodids and salicylates). Stomatitis can be associated with viral diseases like measles (characteristic appearance of the Koplik's spots: whitish specks surrounded by an erythematous area), herpes simplex (small vesicles with shallow painful ulcers). The **acute pyogenic stomatitis** is usually a gingivitis that is superimposed upon a chronic inflammatory condition already present in the gums. Strict attention to the oral hygiene is necessary and systemic antibiotics are required. Another infective stomatitis type is the **acute ulcerative (Vincent's) stomatitis**. It is now thought that virus is the precipitating factor but Vincent's organisms are always present. Clinically ulcers are found and foetor is present. **Gangrenous stomatitis** is a rare disease, the cause of which is still obscure and often is fatal. Its predisposing factors are malnutrition and the same microorganisms found in Vincent's stomatitis (spirochaetae and fusiforms). Symptomes are ulcerations in the buccal mucosa that spread rapidly and gangrene supervenes with distruction of the cheeks. **Acquired immune deficiency syndrome (AIDS)**. First recognized in 1981. Infection by the human immunodeficiency virus (HIV) spreads quickly. Aphtous ulceration which persists, thrush, gingivitis, angular cheilitis and scaly red dermatitis in young male suggest immunosuppression and are known as AIDS-related complex (ARC). Kaposi's sarcoma or life threatening infection like pneumocystosis caused by Pneumocystis carinii make the diagnosis certain. Saliva and blood tests should be taken from such patients for HIV. The incubation period from infection to development of AIDS is variable and can extend to many years. AIDS is lethal disease.

3.3.2. Epipharyngitis

The acute form is usually accompanies acute inflammations of the subepithelial lymphoid tissue. The infection may be viral (influenza, parainfluenza, rhinovirus and adenovirus) or bacterial (streptococci, pneumococci and Haemophilus influenzae). A dry, hot sensation is often the first symptom. The clinical features include localized pain and discomfort, exaggerated by swallowing, fever and cervical adenitis. Treatment is expectant and paracetamol.

Chronic nasopharyngitis is associated with an inflammation of the nose or oropharynx. Aggravating factors are nasal obstruction; exposure to dust and fumes; alcohol and tobacco, overuse of adstringent nasal medications. *Clinical features.* Postnasal irritation, inspiratory snor ing, congestion of the mucosa. The treatment is of conservative measure and attention to any predisposing cause.

3.3.3. Inflammation of the mesopharynx and hypopharynx

Acute non-specific pharyngitis is caused by the same viruses and bacteria such as causative in nasopharyngitis. It is a common prodromal manifestation in measles, scarlet feaver, glandular fever, smallpox and rarely of typhoid. In **simple case**s sore throat, earache, enlargement and tenderness of the cervical lymph nodes with low fever can present. **Treatment** is antibiotics, symptomatic treatment and pharyngeal diet (avoidance of alcoholic drinks, sparkling mineral water, spicy foods, smoking, hot and cold drinks and foods). In severe cases high pyrexia (38.9-40.5 ^{o}C) and rigor may initiate the attack that accompanies slow pulse, oedema of the soft palate. Complications are common in children. They include otitis media and oedema of the glottis. The soft tissues of the submandibular region may be involved, leading to a firm swelling: **Ludwig's angina**. This is often a fatal infection primarily of the sublingual space which extends to the neck and mostly due to untreated carietic teeth with periapical granuloma. The condition is attributed to lymphadenitis and perilymphadenitis. The tongue is oedematous and is often dislocated. Pus points to the mouth. *Symptomes*: Lasts for few weeks. High temperature and leukocytosis (10-35000). Boardlike swelling of the submental-submandibular space. Trismus. Stiffness in tongue movement. Pharyngeal spreading may involve the base of the tongue and the pre-epiglottic space resulting in breathing problem. When pus formed it usually results a floor of the mouth abscess (which is rather more a phlegmone-like). Differential diagnosis: glandular fever (Pfeiffer's); Vincent's angina; Diphteria (may be excluded by swabs); blood dyscrasias, like agranulocytosis (can be excluded by blood count). **Diagnosis** is achieved by physical examination. Ultrasound imaging of the submental-submandibular region shows accumulation of pus. Puncture and aspiration of pus confirms diagnosis and pus can be reffered to bacterial culture. **Treatment**. Systemic antibiotics and symptomatic treatment. Heat should be applied locally as hot packs to localize the infection. If pus forms incision and drainage are mandatory. Incisions should be made both **intraorally and transcervically** below and parallel to the mandible through the deep fascia to the depth of the submandibular gland. Vertocal incision may be necessary from the hyoid bone

to the chin and the mylohyoid muscles and geniohyoglossus muscles should be splitted apart.

Acute membranous pharyngitis (Vincent's angina). An acute ulcerative lesion involving one or both tonsils and spreading to the fauces, soft palate and gums. The infection is characterized by a Gram-negative fusiform bacillus and a spirillum (Spirochaeta denticola) often with a secondary streptococcus. *Clinical features* are: pain, foetor oris, high fever, cervical adenitis, grey, membranous slough soon separates with considerable loss of tissue. The acute symptomes subside in 4-7 days, but the ulceration persists for several weeks. Treatment is systemic antibiotics particularly metronidazole and antiseptic mouthwashes.

Acute diphtheritic pharyngitis. A severe infection due to the Corynebacterium diphtheriae. The incidence of faucial diphtheria has fallen markedly in the last four decades due to immunization but recently the morbidity again arises in poor countries. Symptomes are: sore throat is the first; enlargement and tenderness of cervical glands; pyrexia is rarely above 38.5 ^{o}C; toxaemia; vomiting; proteinuria; nasal discharge which is serosanguinous or purulent; patches of false membrane are present on the tonsils, faucial pillars, soft palate, posterior pharyngeal wall. It is usually firmly attached and when detached, leaves a bleeding surface on which it tends to reform. Treatment is antitoxin which should be given immediately. Systemic penicillin.

Chronic non-specific pharyngitis. This is a common condition and there are many causative and predisposing factors: recurrent attacks of acute pharyngitis; nasal obstruction; excess of alcohol and tobacco; dry and dusty atmosphere; infected gum and teeth. It may be presented as catarrhal, hypertrophic (small nodules of lymphoid tissue are scattered over the pharyngeal wall, the lateral pharyngeal bands may be very prominent), follicular (small yellowish cysts are seen, commonly in the valleculae and on the tonsils), atrophic (the pharyngeal mucosa is dry and glazed). *Symptomes* are constant hawking, irritation of the throat and snoring. **Treatment**. Causes must be eradicated. Cautery to the lymphoid patches is sometimes indicated. **Chronic pharyngo-oesophagitis (Plummer-Vinson syndrome; Chronic hypopharyngitis)**. This is a chronic atrophic type of inflammation of the mucous membrane, involving the cricopharyngeal region of the laryngopharynx and the upper part of the oesophagus. Iron deficiency is probably the basic cause. Half the patients have a pyridoxine deficiency. Gastrectomy increases the risk of developing an upper oesophageal web eightfold. It occurs almost exclusively in women, usually over 40 years of age. *Symptomes.* Increasing dysphagia. There may be also a complaint of "lump in the throat". Pallor due to anaemia. Fissures at the angles of the mouth. Koilonychia (spoon-shaped nails). Loss of weight. Superficial glossitis ("bald tongue"). Treatment: Iron and vitamins should be given in large doses; non irritating soft foods are desirable; repeated endoscopic dilatation is

indicated. Regular observations are necessary, good results to iron therapy are frequent.

3.3.4. Inflammations of the pharyngeal lymphoid tissue

Adenoids (pharyngeal tonsils). A hypertrophy of the nasopharyngeal tonsil is sufficient to produce symptomes, most commonly between the ages of 3 and 7 years. Symptomes: 1. Nasal obstruction leads to mouth-breathing, difficulty in eating, snoring and toneless voice. Later the "adenoid facies" may develop, with pinched nostrils and prominent incisors. 2. Eustachian tube obstruction leads to deafness by the presence of fluid in the middle ear (otitis with effusion or secretory otitis media). 3. Nasal discharge, postnasal drip and cough. 4. Acute otitis media is the most serious effect and may present as recurrent acute attacks. 5. Rhinitis and sinusitis may fail to resolve. 6. Cervical adenitis. **Diagnosis** may be established by direct palpation into the nasopharynx or if succesfully performed by posterior rhinoscopy. **Treatment**. In mild cases decongestant nasal drops may give relief. Sinusitis and nasal allergy should be treated. When the symptomes are marked, adenoidectomy is indicated in general anaesthesia **(Fig. 34)**. Nasal obstruction, recurrent otitis media, otitis with effusion and sinusitis are the chief indications. In secretory otitis media myringotomy and possibly insertion of a tympanometal ventilation tube (in glue ear) will be done at the time of adenoidectomy. Removal of adenoids at the same time as antral lavage is usually more effective in maxillary sinusitis than either of these manoeuvres alone. *Complications of adenoidectomy*: 1. Haemorrhage: it may occur at any time during the first 24 hours after operation (prevention: no operation should be done on patients with blood dyscrasias, jaundice or acute infection; a dry field should be obtained before the patient is allowed to leave the operating table). Treatment of haemorrhage is postnasal pack and blood transfusion if indicated. 2. Acute otitis media; 3. Recurrence is not an uncommon sequel:in certain children an inherent tendency for regrowth of lymphoid tissue exists. Further removal is indicated. 4. Trauma to the uvula or soft palate and Eustachian cushions.

Acute and chronic (palatine) tonsillitis.

Acute tonsillitis is most often caused by haemolytic streptococcus. In the acute follicular type, the crypts of the tonsils are filled with infected fibrin, and the openings of the crypts contain pus, leading to the characteristic spotted appearance called **follicular tonsillitis**. Occasionally, these follicular exsudates coalesce, giving the appearance of a whitish-jellow false membrane. This condition is called **confluent tonsillitis**. *Symptomes* are: 1. sore throat 2. pain on swallowing 3. pyrexia tends to be high in children 4. earache 5. malaise.

Other complaints are headache, high pulse rate. Acute tonsillitis occurs in scarlet fever, in which the tongue has a strawberry appearance and soon develops a punctate erythema.

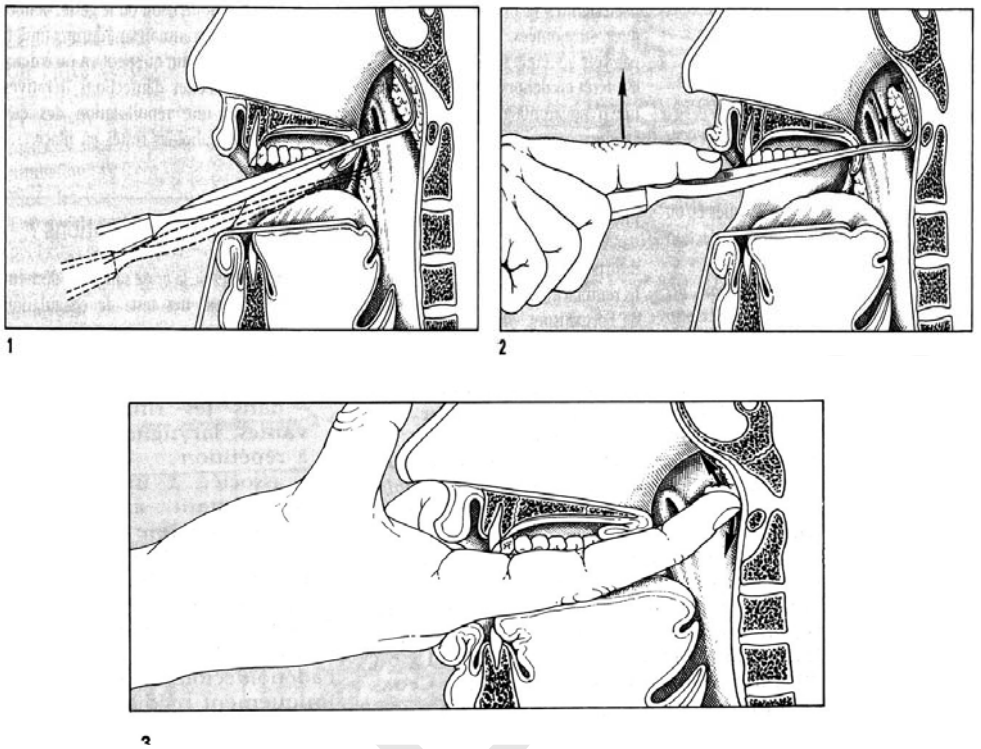


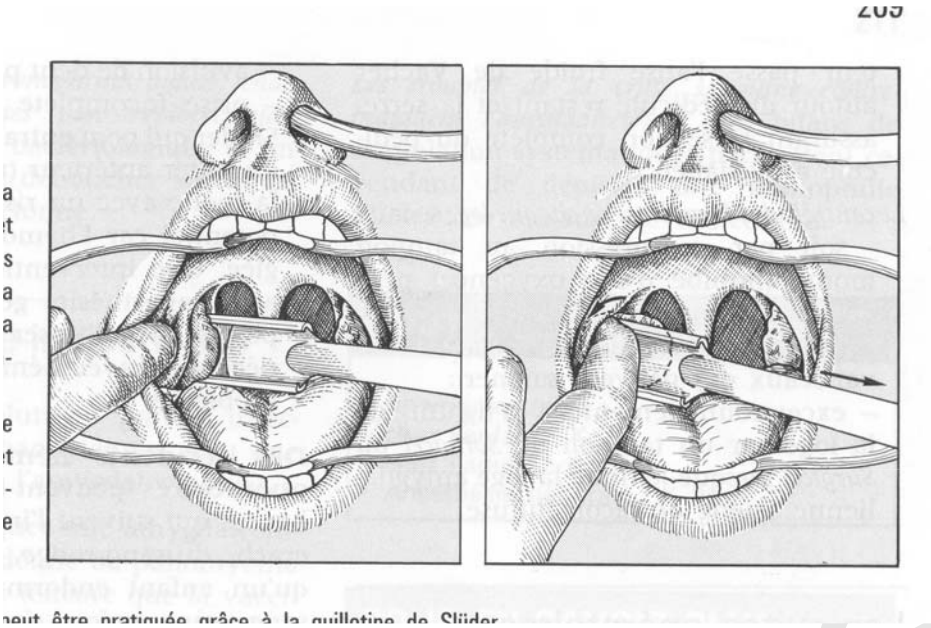
Figure 34. Correct performance of adenoidectomy. The adenotome is positioned through the oral cavity into the fornix of the epipharynx (1). The adenotome is then pulled down with a lever-like hand movement shown by the arrows (2). After this maneuver a palpation has to be done by finger into the epipharynx to verify correct removal (3).

Complications are uncommon in the antibiotic era, the most importants are: Peritonsillar abscess; parapharyngeal and retropharyngeal abscesses; oedema of the larynx; acute rheumatism; acute nephritis; endocarditis; septicaemia; chronic tonsillitis. *Differential diagnosis*: 1. Diphtheria (bleeding pseudomembrane, caused by the Loeffler's bacterium) 2. Infectious mononucleosis (Pfeiffer's fever) (hepatosplenomegaly and positive Paul-Bunnel's serology) 3. Agranulocytosis (blood count) 4. specific tonsillar infections (TBC, syphilis) 5. hematological disorders (lymphomas, leukemias). **Treatment** is systemic penicillin for at least 5 days, aspirin, bed rest and ample fluid intake.

Chronic tonsillitis usually follows acute or subacute attacks and may present a focus of infection with effect on the entire body. There is a chronic inflammatory hypertrophy associated with enlargement of the nasopharyngeal tonsil. **Clinical features** are: Persistent sore throats; **retromandibular lymphadenitis** (jugulodigastric group); tonsillar enlargement; injected anterior faucial pillars; irritating cough; evacuation of **pus from crypts by depressing** the tonsils; occurrence of acute attacks more than 3 annualy; elevated AST (*a*nti*s*treptolysin *t*iter). Treatment is tonsillectomy in local or general anaesthesia **(Fig. 35)**, if the symptomes persist or systemic complications (arthritis, glomerulonephritis, endocarditis) onset.

Indications of tonsillectomy*:* 1. An attack of peritonsillar abscess (quinsy) 2. Repeated attacks of acute tonsillitis 3.Chronic tonsillitis 4. Gross obstruction to breathing and eating 5. As a preliminary stage to operations for cleft palate 6. For avulsion of the glossopharyngeal nerve and/or removal of the styloid process for neuralgia, by the pharyngeal route 7. Sispicious focal disease.

Contraindications. 1. Allergic rhinitis or asthma, unless there is convincing evidence of true tonsillar infection. 2. Bleeding tendency: cryosurgical reduction should be considered in such cases. 3. Epidemics: tonsillectomy should not be performed during an epidemic of any infectious disease. 4. Pharyngitis sicca, 5. agranulocytosis, 6. leukemia. **Complications of tonsillectomy***:* 1. Haemorrhage, can be reactionary within the first 24 hours after the operation (slipping ligature, failure to ligate all vessels, failure of a vessel to contract after crushing, rise of blood pressure) or secondary between the 3rd and 10th postoperative days, usually caused by separation of the slough; 2. Otitis media; 3. Oeema of the soft palate; 4. Cervical lymphadenitis 5. Parapharyngeal abscess; 6. Septicaemia.



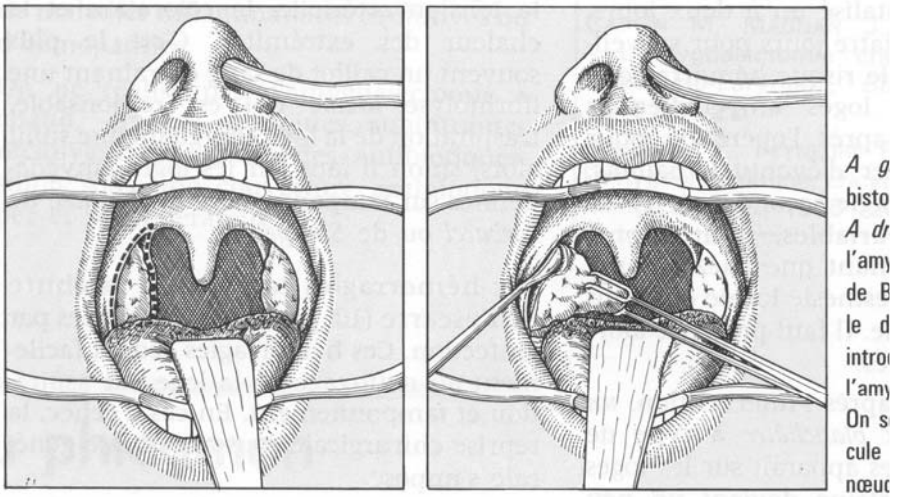


Figure 35. Tonsillectomy. Upper panels show the guillotine (Sluder) tonsillectomy which is today of only historical interest. Lower left panel demonstrates the sharp incision lines (dotted lines) along which the pulled out tonsils (lower right panel) can be excised.

Peritonsillar abscess, parapharyngeal abscess, retropharyngeal abscess. Peritonsillar abscess (quinsy). An abscess between the tonsil capsule and the adjacent lateral pharyngeal wall (palatopharyngeal muscle and superior constrictor muscle). Peritonsillar infection may follow an acute tonsillitis. The size of the tonsils has no bearing on the condition, since it may also occur in patients with a post-tonsillectomy "remnant". Usually it is unilateral and lies above the tonsil near the soft palate. There is first a cellulitis, later pus. *Symptomes* include severe pain in the throat, pyrexia up to 40 ^{O}C, malaise, headache, trismus (making examination difficult), earache, intense salivation, feator oris, cervical lymphadenitis, marked hyperaemia and oedema of the tonsillar and palatal region. **Obligatory sign** is that the uvula is oedematous and pushed to the uneffected side (staphylitis). Aphagia followed by stridor is an indication for urgent drainage. **Treatment** is conservative in early stages of cellulites, meaning that pus can not be obtained by puncture: systemic antibiotics in large doses. If there is no response during the first 48 hours the antibiotics may be changed. Soluble aspirin , bedrest and ample fluids intravenously. When considerable swelling is present drainage is advisable. Incision of the abscess is the standard treatment in topical anaesthesia. Abscess-tonsillectomy is advised in systemic antibiotic cover within the first 24 hours after incision or 1 month later.

Parapharyngeal abscess. This is a suppurative infection of the parapharyngeal space spreads from the tonsils, the tonsillar fossa, a penetrating foreign body or from a lower wisdom tooth. The *clinical features* are: painful throat, trismus, pyrexia, swelling of the neck and medially pushed pharyngeal wall and tonsil. Should seriously be considered because of the potential complications: acute oedema of the larynx, thrombophlebitis of the internal jugular vein with pyemia and direct spread of infection to the mediastinum (mediastinitis). **Diagnosis**. Cervical CT scan examination and ultrasound examinations help to identify localization of pus. **Treatment**. Systemic antibiotics should be given and incision is made. This may be done through the pharynx or through the neck, depending on the point of maximum swelling.

Retropharyngeal abscess. The abscess lies in the potential space between the buccopharyngeal and prevertebral fasciae. The acute abscess is caused by suppuration in the retropharyngeal lymph nodes, which become infected from the nasopharynx or oropharynx. Symptomes may be misleading as the patient is often a suckling infant, and at least one-half of the patients are under the age of 1 year. This is due to the tendency of lymph nodes to atrophy in childhood. Clinical features: difficulty in breathing and suckling; croupy cough; nasal obstruction; stiffnes of the neck or torticollis; pyrexia and toxaemia; lateral

swelling of the posterior pharyngeal wall, oedema of the larynx. Spontaneous rupture of the abscess may occur occasionally and can cause sudden death from aspiration. Treatment: 1. Incision of the abscess is made vertically through the open mouth; 2. Systemic antibiotics; 3. Tracheostomy may become necessary when laryngeal obstruction threatens or supervenes.

The chronic retropharyngeal abscess is caused by tuberculous infection. This is seen in older children and in adults. Local symptomes may be minimal, dysphagia is slight. Sore throat and cough may be complained of. "Cold" abscess is present on the posterior pharyngeal wall. Enlarged cervical lymph nodes are present in the deep cervical group. Radiography reveals vertebral disease or calcification in tuberculous lymph nodes. Treatment is incision made through the neck in antibiotic cover of Rifampicin, ethambutol, PAS, INAH.

3.4. Tumors of the mouth and Pharynx

Benign tumors are hemangiomas and lymphangiomas, that are usually congenital. The tumor may be so large that threatens life due to bleeding and obstruction to breathing and feeding. Removal is usually by cryosurgical approach.

Malignant tumors are in great majority squamous cell carcinomas. Any ulcer that does not heal rapidly and hyperkeratotic areas should be considered as malignancies. In the early stages there is no pain. As the tumor increases, the patient complains of pain and infiltration of the underlying tissues and regional lymphnode adenopathy, in the submandibular triangle and the jugulodigastric group. Treatment is surgery. The tumor must be removed with a wide margin in three dimensions. Radical neck dissection has to be carriend out when lymph node metastasis is supected. Soft tissue defects due to resection of the mandible or large tumors can be closed by pedicled flaps of the forehead, the chest or also pectoralis or latissimmus dorsi myocutaneous flaps.

Carcinoma of the tonsils has to be surgically resected after mandibulotomy and also treated by irradiation therapy in combination when necessary.

Hypopharyngeal carcinoma is distinguished by its localization: piriform sinus, posterior pharyngeal wall or postcricoid region. First **symptome** is commonly a regional lymph node metastasis. Hoarsness, breathing and swallowing difficulties follow. Tumors of the piriform sinus are most common. **Diagnosis** is established by direct laryngoscopy and pharyngoscopy and when necessary by oesophagoscopy. Neck CT scan and MRI help in imaging the extension of the tumor. **Treatment**. Irradiation therapy may be served as palliation. Curative treatment is only the surgical removal of the tumor (**Fig. 47**), this may include total laryngectomy with pharyngeal resection and removal of the aperture of the oesophagus if necessary. Reconstruction of the pharynx and oesophagus is often required: mobilization of the oesophagus, gastric pull-up can be done. Prognosis

of a hypopharyngeal carcinoma is, however, poor. About 40 % of cases obtain 3-year survival, the remaining 60% is lost within 3 years.

Trismus, a relevant associated symptome to mouth infections, is divided to 3 grades on the bases of the distance of the upper and lower incisors. Total trismus indicates complete loss of active movements of the mandible. Causes are:
(a) Inflammation of the teeth or mandible. Unerupted dentition, stomatitis, pulpitis, osteomyelitis of the upper jaw, peri-, and submundibular abscess
(b) Inflammation of the temporo-mandibular joint, as arthritis, arthrosis, polyarthritis
(c) Acute inflammation around the temporomandibular joint: these are peri-, and retrotonsillar abscesses, sialadenitis of the parotid and submandibular glands, parapharyngeal abscess, furunculosis in the external auditory meatus
(d) Facial trauma to the zygomatic arch, dislocation of the temporomandibular joint
(e) Muscle-spasms due to epilepsy, meningitis, cerebral tumors, tetanus, hysteria.

3.5. Dysphagia

Dysphagia is difficulty in moving the food from mouth to the stomach. Swallowing is the entire act of deglutition from placement of food into the mouth through the oral, pharyngeal and oesophageal phase of swallowing. It can associate to many different conditions e.g. elderly ages, gastroesophageal reflux etc. Dysphagia has a huge impact on the quality of life. It is very important from nutritional state and may indicate sinister pathological condition.

Stages of swallowing:

1. oral preparatory
   Food is manipulated in the mouth: masticated and consistency is optimized (cranial nerves involved are: V., VII., IX., XII).
2. oral
   Tongue propels food posteriorly until the pharyngeal swallow is Triggered (cranial nerves involved: V., VII., IX., XI., XII.)
3. pharyngeal
   Pharyngeal swallowing triggered and the food slides down to the oesophagus (from this stage n. X. cranial nerve is important
4. oesophageal

In all 4 stages pathologies may cause dysphagia.

1. Structural causes are congenital or aquired. Among former cleft lip/palate, laryngeal cleft, tracheoesophagea fistula are to be mentioned. Aquired causes are inflammatory conditions (pharyngitis, oesophagitis), trauma (foreign body, dentition), neoplasms in any location of the upper digestive tract and some miscellaneous pathologies, e.g. strictures, GORD (gastro-esophageal reflux disease, diverticulum).
2. Neurological causes can similarly classified. Among aquired conditions those which can be anticipated are cerebrovascular attacks, head traumas, Guillian-Barré sy., poliomyelitis, cervical spinal cord injury. Neurodegenerative disorders (e. g. muscular dystrophies, parkinsonism, ALS, dementia).
3. Medical treatment like nonsteroid anti-inflammatory drugs, anti-cholinergics and sympathomimetics.
4. Other causes are cervical spine osteophytes, rheumatoid arthritis.
5. Psychological abnormalities, e.g. psychosomatic dysphagia, globus sensation.

Presenting *symptoms*: difficulties in place the food into the mouth or control the food in the mouth, caughing before, during after swallowing, recurring pneumonia, gurgly voice, loss of weight, otalgia, GORD in the history *Diagnosis*. Radiocontrast material swallowing and direct laryngo-pharyngo-oesophagoscopy. When positive, CT and MR should be taken. Other imaging modalities may be used are ultrasound examination of oral cavity and tongue movements, videolaryngoscopy to visualize pharynx and larynx as to anatomical variations, videofluoroscopy to examine pharyngeal swallowing. Oesophagus stricture should be biopsied to axclude cancer in the scar. **Treatment**. After exclusion of malignant tumor, the other causes should be treated appropriate to structural or functional abnormality.

3. 6. Snoring and the *o*bstructive *s*leep *a*pnea *s*yndrome (OSAS)

Snoring is noisy breathing during sleep. OSAS is upper airway obstruction during sleep. Normally the nasal and oral airways are opened towards the pharynx and larynx. Snoring patient has an anomaly with the soft palate-uvula complex in term, that they collapse against rear wall of throa and so closes the nasal airways. The oral airway remains open. In sleep apnea, soft palate closes the nasal airway whereas tonguebase closes the oral airway as slides backwards. The blood oxygen content decreases. Above age 60 the 60% of males and 40% of females snore. Its loudness is measured to be 93 dB (Walkert, 1993). For comparison this is the loudness of a jet aircraft at about 300 m altitude: very loud.

Predisposing conditions for snoring are facial abnormalities, micrognathia, overweight, thick neck, heavy drinking, enlarged adenoids. Predisposing factors for OSAS are snoring and blood hypertension.

Diagnostic work up includes examination of the nasal cavity proper for abnormalities in nasal airway, nasenednoscopy. Sleep should be studied first by endoscopy during sleep and by polysomnography wich includes blood gas analysis, pulse rate, electrocardiography and spirometry.

Treatment: Conservative treatment is very effective in most cases: alcohol, food and tobacco abuse should be stopped.. Medical treatment is not really effective. Mandibular splint can be recommended. Surgical treatment in OSAS is largely contraindicated. A whole variety of spectrum of different surgical interventions are described in snoring. These are septoplasty, uvulo-pharango-palatoplasty. The latter can be performed by laser. Adenoidectomy and tonsillectomy is suggested when part of the obstruction. Tonguebase surgeries are lingual tonsil removal by laser, partial glossectomy and there are maxillomandibular advancement procedures. Surgical procedures have immediate succes rate of 80%, however, long lasting results are poor (30% at 2 year).

4. Salivary glands

The major salivary glands include the paired parotis, submandibular and sublingual glands. The salivary gland unit consists of an acinus, a secretory tubule, and a collecting duct. The parotid acini are composed of serous cells, the sublingual acini are mucous cells whereas the submandibular is mixed it is composed of both cell types. The parotid gland has a larger superficial portion and smaller deep lobe, between which run the branches of the facial nerve. The parotide duct (Stensen's) opens into the mouth on the buccal surface, adjacent to the second upper molar tooth. The submandibular gland drains via Wharton's duct, which opens into the anterior floor of the mouth on each side of the midline. The sublingual gland lies in the floor of the mouth and empties via a number of small ducts. The main duct is called Bartholini’s duct. The function of the glands is to maintain the oral environment during mastication of food. Multiple antibacterial systems exit in the saliva although their role in oral hygiene is still questionable. Saliva contains biologically active polypeptides of cytoprotective activity, growth factors. The daily volume of the saliva is normally between 1000 and 1500 ml. The regulation of the salivary flow and composition is under the control of the autonomic nervous system. Many factors influence, however, this control. These are psychologic factors, circadian

rhythm , age, hormones (thyroid, adrenal, hypophysis), diet and drugs. **Autonomic innervation**. The salivary glands contain both parasympathetic and sympathetic nerve fibers. Sympathetic fibers arise from the superior cervical ganglion. Parasympathetic fibers travel from the salivary nuclei in the brainstem and enter the gland by the auriculotemporal nerve to the parotid and by the chorda tympani-lingual nerve to the submandibular and sublingual glands. **Functions of the salivary glands and saliva**. 1. Digestive function. Soluble food substances are dissolved by the saliva and makes redy for for taste receptors. Amylase initiates carbohydrate digestion. 2. Protective function. It includes mechanical protection of the oral mucosa, anticariogenic activity, antibacterial activity (secretory IgA) and antineoplastic activity. 3. Homeostatic function. This is the thirst regulation by the sensation of dryness. Many substances appear in the saliva by excretion: lead. mercury, sulphur, iodide, morphin, many antibiotics. 4. Hormonal function. Parotin was described as parotide hormone, which decreases the serum calcium level by 15%. Renin (as the member of the renin-kallikrein system) was isolated from the submandibular gland. Nerve growth factors were also isolated from saliva.

4.1. Examination

Physical examination includes palpation of the glands externally, inspection of the appropriate duct orifice. The type of secretion produced at the orifice is noted to be mucoid, watery or purulent. Stensen's or Wharton's duct may be probed using the dilators. Regular x-ray film may demonstrate calculi in the gland or duct. Sialography may differentiate inflammatory from neoplastic masses. **Ultrasound** investigations can describe fluid filled cysts, abscesses or tumors and **ultrasound-guided fine needle biopsy** is an extremely useful diagnostic tool for establishing the diagnosis of malignant tumors. The environment of suspected malignant tumors are best imaged by CT and MRI.

4.2. Inflammatory salivary gland disorders

Mumps (epidemic parotitis) is a common infectious disease. The cause is mumps virus. There is incubation period of 2-3 weeks. Clinically featured by swelling and pain of one or both parotid glands, with fever and difficulty in opening the mouth. The Stensen's duct's orifice is usually inflamed without purulent secretion. The white blood count is low, indicating viral disease. **Treatment** is symptomatic, with fluids, heat of the glands, analgesics and bedrest. Antibiotics have no place. While the great majority of cases recover within 7-10 days, complication may arise, especially in adults, including inflammation of testes and ovaries. Other complications are

meningoencephalitis, sensorineural hearing loss or diabetes secondary to pancreatic fibrosis. Mumps vaccinae are available.

Acute suppurative sialadenitis. This is a bacterial infection of the major salivary glands. Retrograde bacterial infection via the duct in a dehydrated, debilitated patient is a common source. Obstruction of a duct due to calculus or stricture predisposes to infection. Staphylococcus aureus is the most common organism cultured in acute parotitis. *Clinical features*. Swelling, tenderness and signes of inflammation of the involved gland. Pus can usually be expressed from the duct orifice. Abscess formation may occur due to progress of infection with fluctuance on palpation. Failure to improve on medication may be indication for surgery. **Treatment.** Antibiotics are given in large doses pending cultures and sensitivity results (methicillin or cephalosporine are good choices). Hydration with intavenous fluids is important. With abscess formation, incision and drainage is carried out. Local heat helps to localize the infection.

Chronic recurrent sialadenitis. This refers to recurrent suppuration of a salivary gland. This condition may fallow acute episode or may occur where ductal obstruction due to a mucous plug or calculus. Symptomes are recurrent swelling of a salivary gland, aften after eating. There is pain. The gland is swollen and may be tender. Pus may be expressed from the duct orifice. With recurrences, there is chronic enlargement. **Treatment**. In the acute phase, the treatment is similar to the acute sialadenitis. When the acute infection subsides, the duct should be probed and dilated. Chronic recurrent sialedenitis of the submandibular gland is indication of removal of the gland.

4.3. Calculus of the salivary gland (Sialolithiasis)

It occurs most frequently in the submundibular glands and in the parotis. The stone is usually single but tends to recur. The stone is chemically calcium phosphate. Calculi originate in the gland and are carried to the duct, where they increase in size by deposit of bacteria and phosphates. *Symptomes:* there is a history of swelling of the gland while eating, which usually subsides within an hour or two. The attacks are recurrent. Bimanual investigation often reveals a stone, especially when it is located in the duct. Majority of stones are radiopaque and can be demonstrated by x-ray. **Treatment.** If the gland is actually inflamed, the patient starts with antibiotics, hydration and heat. As soon as the acute infection quiets down, the calculus may surgically be removed via an intraoral approach under local anaesthesia. A calculus located in the gland requires external surgical approach with removal of the entire gland.

4.4. Tumors of the salivary glands

4.4.1. Epithelial tumors

4.4.1.1. Benign tumors

Pleomorphic adenomas (mixed tumors) grow slowly. Predilection site is the parotid gland. The facial nerve function is retained in even big tumors either. The tumor sometimes grow within a capsule and is multiloculated. May show malignant transformation after recurrencies. Treatment is surgical excision.

Papillary cystadenoma lymphomatosum (Whartin tumor). The tumor causes nontender swelling and is unilateral in vast majority of cases. Usually develops in the inferior part of the parotid gland. It arises from segments of the salivary ducts which are included in extraglandular lymphnodes in the embryonic developmental process. Treatment is surgical removal. Malignant degeneration is extremely rare.

4.4.1.2. Malignant tumors

Malignant mixed tumors. The mixed tumor is present for years when suddenly starts rapidly growing. The previously silent "swelling" of the parotid gland now becomes painful which irradiates often to the ears. Diagnosis is based on hystory and fine needle-biopsy. Treatment is radical paridectomy.

Mucoepidermoid carcinoma. This is a slowly growing tumor. Some tumors, however, show high-grade malignancy and rapidly causes facial nerve paralysis and blood-born distant metastasis. The degree of malignancy can be determined by the ratio of epidermal and mucoid cells in histological sections. Those tumors of having high number of mucous cells have better prognosis. The tumor is located mostly in the parotid gland. Treatment is surgical removal of the tumor.

Adenoid cystic carcinoma. A slowly growing, painful tumor which often causes facial palsy. Regional lymphnode metastasis and distant metastasis are common. Treatment is radical parotidectomy. Prognosis is determined by local findings (infiltration the environment and skull base) and presence of metastasis.

Adenocarcinoma and squamous carcinoma. Both are highly malignant tumors with the symptomes of rapidly growing swelling, pain, firm infiltration

and exulceration, facial paralysis (if parotid is included) cervical lymphnode metastasis. Prognosis is poor if diagnosis is late.

4.4.2. Supportive tissue tumors

4.4.2.1. Benign tumors: Lymphangioma, haemangioma; Fibroma, lipoma, neuroma;
4.4.2.2. Malignant tumors: sarcoma
Symptomes: A swelling is noted by the patient, the lump is usually asymptomatic. Pain, tenderness, facial paralysis may be late signs of a parotid tumor. MRI may help to determine if a lesion inflammatory or neoplastic, if it is within or adjacent to a salivary gland and whether it is invasive or encapsulated. Precise diagnosis should be made by the pathologist on needle biopsy specimen. **Treatment**. Malignant tumors must be widely excised sacrificing branches of the facial nerve when necessary. In some cases the hypoglossal or spinal accessory nerve may be used as a nerve transfer. Radical neck dissection may be carried out where cervical nodes are involved. Postoperative irradiation is useful with malignant lymphoepithelioma.

5. Larynx

The most important function of the larynx is protection of the lower air passages. When food is swallowed the laryngeal inlet and glottis close. The phonation develops later in the evolution of the larynx. Voice is produced by vibration of the vocal cords. The sound so produced is amplified selectively by the resonating chambers of the mouth, pharynx, nose and chest. The larynx plays a part in the respiratory mechanism by reflex adjustments of the glottic aperture. These movements of abduction during inspiration and adduction during exspiration also contribute to the regulation of acid-base balance in the body.

5.1. Anatomy.

The larynx **(Fig. 36)** is situated in the midline of the neck at the meeting of the digestive and respiratory passages. It lies from the level of the third to the sixth cervical vertebrae. It consists of a framework of cartilages connected by ligaments and membranes, lined by mucous membrane and moved by muscles. The male larynx increases in size at puberty. All the cartilages enlarge and the

projection of the thyroid cartilage produces the "Adam's apple". The hyoid bone is a structure pertaining to the tongue.

Laryngeal cartilages (Fig. 37). 1. *Thyroid cartilage.* This is the largest. Each half consists of lamina which is almost a square plate and begins to ossify at the age of 25 and may be completely converted to bone by 65. The two laminae meet in the midline forming an angle of about 90^{o} in men, about 120^{o} in women. The point of junction of the upper borders of the laminae is indented by the V-shaped thyroid notch. The superior cornu arises from the posterosuperior angle of the lamina, the

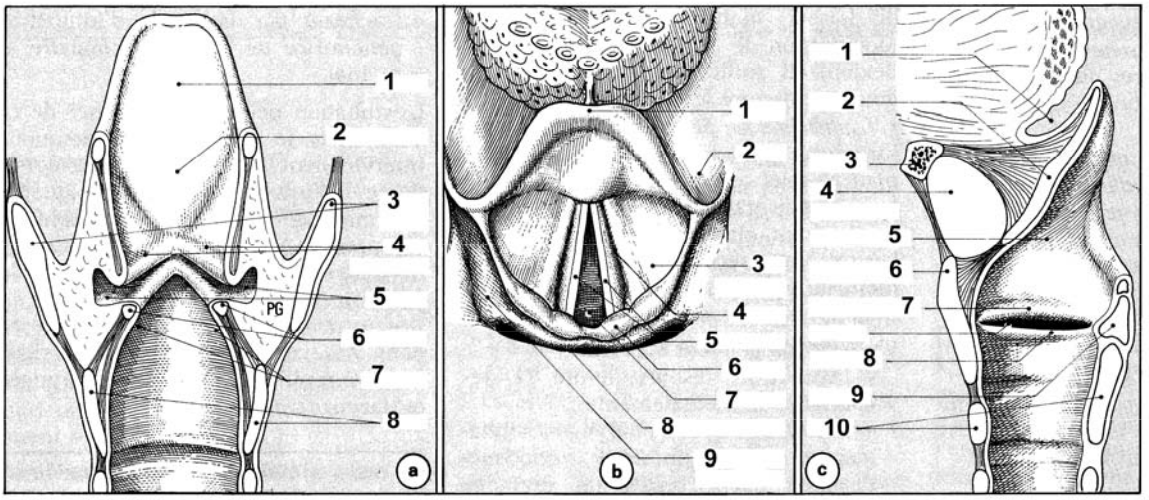


Figure 36. Anatomy of the larynx. (A) Posterior coronal aspect: 1. Epiglottis 2. Laryngeal inlet 3. Thyroid cartilage 4. Ventricular fold 5. Ventricle (Morgagni's pouch) 6. Vocal cords 7. subglottis 8. Cricoid cartilage. (B) Superior aspect (as it looks in the mirror during indirect examination of the larynx)): 1. epiglottis 2. ventricular fold 3. ventricle 4. vocal cords 5. arytenoid cartilage 6. retrocricoid space 7. pyrifom recess. (C) Lateral view: 1. vallecule 2. epiglottis 3. hyoid bone 4. laryngeal inlet 5. thyroid cartilage 6. ventricular fold 7. ventricle 8. vocal cord 9. arytenoid cartilage.

inferior cornu arises from the posteroinferior angle of the lamina. 2. *Cricoid cartilage.* It resembles a ring. It consists of a lamina posteriorly, an arch anteriorly. 3. *Cartilage of epiglottis.* Rises up behind the tongue. It is a thin leaf-like sheet of elastic fibrocartilage. The stem, directed downwards, is long and attached to posterior surface of the thyroid laminae at their junction. The free border, directed upwards, is broad and rounded. The anterior surface is free in its upper part but is separated from the hyoid bone and thyrohyoid membrane by some fatty tissue in its lower part (pre-epiglottic space). The posterior surface is indented by several small pits, in which mucous glands are embedded. 4. *Arytenoid cartilages.* These are the largest paired cartilages. The posterior surface is triangular and concave. The anterolateral surface extends forward into a vocal process. The inferior surface, the base, is concave and articulates with the cricoid cartilage. 5. *Corniculate cartilages (Santorini's cartilages)* are small and articulate with the apices of the arytenoid cartilages. They give attachement to the upper fibres of the oesophagus. 6. *Cuneiform (Wrisberg's) cartilages.* There is one in each ary-epiglottic fold. They do not articulate with any other cartilages.

Laryngeal joints (Fig. 37). 1. Cricothyroid joint. Between the inferior cornu of the thyroid cartilage and the facet of the cricoid cartilage. Two movements occur: rotation through a transverse axis and gliding. 2. Crico-arytenoid joint, between the base of the arytenoid cartilage and the facet on the cricoid cartilage. It allows rotation of the arytenoid on a vertical axis and gliding (arytenoids move towards or away from each other). Two prominent membranes unit the cartilages of the larynx to one another. An intrinsic one is the median cricothyroid ligament, and an extrinsic one is the thyrohyoid membrane.

Laryngeal muscles (Fig. 37). *Abductors of the vocal cords.* There is only one on each side. The posterior cricoarytenoid muscle opens the glottis. It originates from the posterior surface of the cricoid cartilage, goes upwards and backwards and inserts into the back of the muscular process of the arytenoid cartilage. Contraction of the muscle causes the muscular process of the arytenoid cartilage to be rotated backwards around a vertical axis. Hence the vocal process, together with the posterior end of the vocal cords, moves outwards. *Adductors of the vocal cords.* There are three on each side: 1. Lateral crico-arytenoid muscle; 2. Transverse portion of interarytenoid muscle; 3. External portion of thyro-arytenoid muscle.

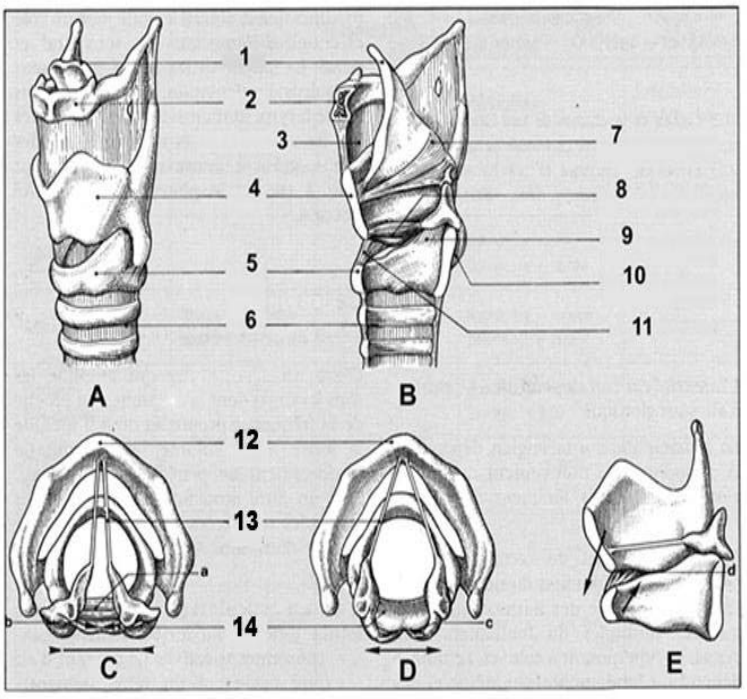


Figure 37. **A** Cartilages of the larynx. (1) epiglottis (2) hyoid bone (3) thyrohyoid membrane (4) thyroide cartilage (5) cricothyroid cartilage (6) first tracheal ring **B** laryngeal muscles in a sagittal view: (9) external cricothyroid muscle (10) posterior cricoarytenoid muscle (11) lateral cricoarytenoid muscle (8) thyroarytenoid muscle (vocal muscle) **C** adduction and **D** abduction of the vocal cords: (12) thyroid cartilage (13) vocal cords (14) posterior cricoarytenoid muscle (15) cricoid cartilage **E** the cricothyroid muscles regulate the tension of the vocal cord

They close the glottis. *Tensors of the vocal cords.* 1. Crico-thyroid muscle; 2. Internal portion of thyroarytenoid muscle (vocalis). These muscles contribute to the fine adjustment of tension of the vocal cords and regulate the pitch of the tones. The laryngeal inlet is opened by the thyro-epiglottic muscle and closed by the ary-epiglottic muscle and oblique portion of the interarytenoid muscle. The *extrinsic muscles* are pharyngeal muscles (stylopharyngeus, palatopharyngeus and inferior constrictor muscles) and neck muscles (sternothyroid and thyrohyoid muscles).

Cavity of 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 devided into three parts. 1. Supraglottis extends from the inlet to the true vocal cords. It is subdivided by the false vocal cords into the vestibule and ventricle of the larynx. The false vocal cords are the ventricular bands, 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 vestibule is bordered by the epiglottis in front, the arytenoid cartilages behind and the ary-epiglottis folds laterally. The ventricle of the larynx (Morgagni's recess) is a recess between the false and true vocal cords and lined by a mucous membrane. 2. Glottis (rima glottidis) is the interval between the true vocal cords in its anterior two thirds and the vocal processes of the arytenoid cartilages in its posterior one third. From practical aspect the glottis consists of the true vocal cords. 3. Subglottic space lies between the true vocal cords and the lower border of the cricoid cartilage.
Blood supply of the larynx. 1. Laryngeal branches of superior thyroid artery; 2. Laryngeal branches of inferior thyroid artery; 3. Cricothyroid branches of superior thyroid artery.
Nerve sypply of the larynx. The larynx is supplied by the vagus nerve. *Superior laryngeal nerve* has two branches. The internal branch is entirely sensory and supplies the cavity of the larynx. The external branch supplies the cricothyroid muscle and part of the anterior subglottis. *Recurrent (inferior) laryngeal nerve* provides motor innervation of all intrinsic muscles (abductors, adductors and tensors) of the larynx except the cricothyroid muscle.

5.2. Examination of the larynx.

Palpation may reveal broadening or tenderness, as in perichondritis and crepitation what is a physiological sign of unaltered mechanics in the joints and excludes tumor invasion of the thyro-cricoid joint. **Indirect laryngoscopy** is

examination of the larynx from above by a laryngeal mirror **(Fig. 38)**. The various structures should be examined: 1. epiglottis: usually presents a slightly curved upper edge but sometimes is conical or Ω-shaped. It may hang backwards and obscure a view of the vocal cords in the relaxed state. It rises upwards and forwards during phonation. 2. aryepiglottic folds must be inspected for swelling or ulceration. 3. Interarytenoid area may be thickened, mamillated 4. False cords may show swelling or ulceration. 5. Vocal cords: should be examined for colour, movement, surface, edge.

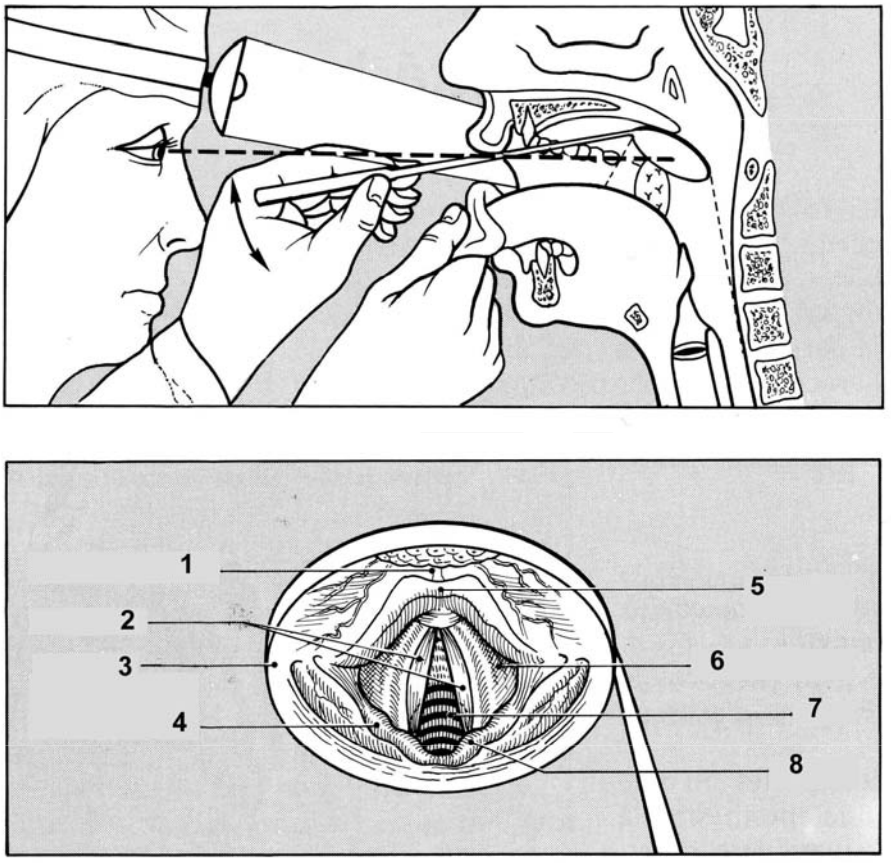


Figure 38. Indirect laryngoscopy and the laryngeal structures seen in the mirror: (1) vallecule (5) epiglottis (2) vocal cords (6) ventricular folds (4) aryepiglottic fold (7) subglottis (8) arytenoid cartilage

Direct laryngoscopy. Endoscopic examination of the larynx and refers to direct visualization for diagnostic or therapeutic purposes **(Fig. 39)**. The hypopharynx can also be examined. This is an operating room procedure requiring topical or local anaesthesia. The head must be extended in the neck. Direct laryngoscopy is necessary for examination in most children and for biopsy or endoscopic treatment at all ages. **Microlaryngoscopy** is a technique for direct examination of the larynx during which the laryngoscope is suspended mechanically, permitting the operator to have both hands free for manipulation. The operating microscope greatly facilitates inspection. Illumination comes from a fiberoptic light source. General anaesthesia is used. Latest development is "jet-ventilation narcosis" which allows surgeon to have full-view of the larynx since no tube is inserted. Microlaryngoscopy associated with laser surgery is an often used technique with therapeutic measure. Most widely used is the CO_2 laser in laryngology practice.

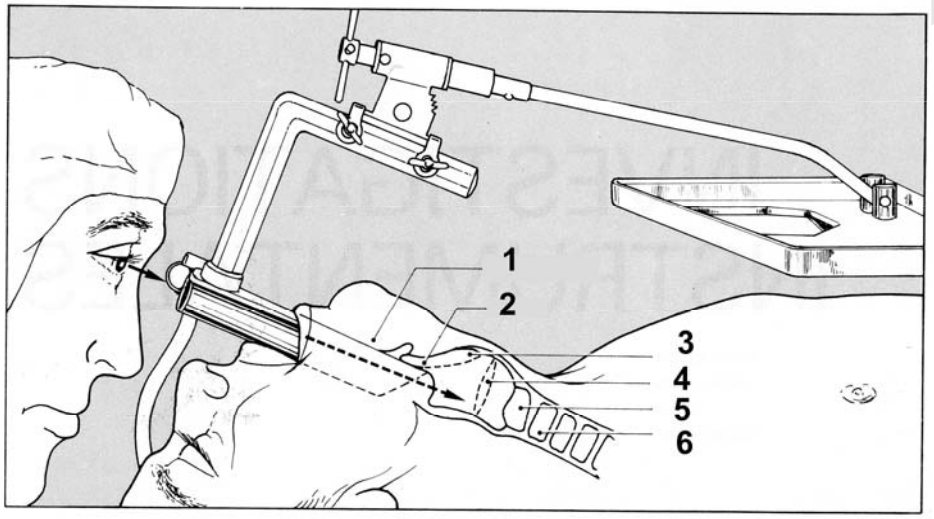


Figure 39. Procedure of direct laryngoscopy which makes possible direct visualization of the larynx and to perform microscopic endolaryngeal manipulations 1: oral cavity; 2: epiglottis; 3: thyraid cartilage; 4: thyrohyoide membrane; 5,6: trachea rings

Stroboscopy and video recording have been used for the analysis of vocal cord movements. **CT scan and MRI** appear to have significant advantage in demonstrating the extent of laryngeal neoplasms because of its tissue differentiation characteristics and ability to produce pictures in any plane.

5.3. Congenital anomalies of the larynx.

C**ongenital laryngeal stridor** (laryngomalacia) is the most common laryngeal abnormality of the newborn due to unusual flaccidity of the laryngeal tissues. Symptome is an inspiratory stridor noted soon after birth with suprasternal and intercostal retraction. Diagnosis requires direct laryngoscopy, which reveals a flaccid epiglottis which is drawn over the glottis on inspiration. The vocal cords are normal. No treatment is required since the stridor invariably disappers by 12 to 16 months of age. In rare cases epiglottectomy or tracheotomy is required. **Congenital laryngeal cysts** occur most commonly in the supraglottic area or associated with laryngocele, producing inspiratory stridor and weak cry. Diagnosis is made by laryngoscopy and the treatment is an endoscopic removal. **Congenital subglottic stenosis** refers to unusual narrowing at the level of the conus elasticus, several millimeters below the vocal cords. This may result from a respiratory infection with inflammatory swelling of subglottic tissues. Usually requires a tracheotomy. **Laryngeal web** is due to an arrest in development and consists of fibrous tissue in the anterior half of the glottis. It causes hoarsness and inspiratory stridor. When stridor is severe a tracheotomy should be performed. The web may be evaporized by laser.

5.4. Traumatic disorders of the larynx.

Direct (compression) injuries are due to blows or strangulation. Submucosal haemorrhage can involve any part of the larynx. Adhesions, stenosis may develop. Perichondritis results if infection supervenes. Fracture of the cartilages may accompany. Clinically these cases refer with dyspnea (mediastinal emphysema), dysphagia, hoarsness, tenderness, external swelling and submucosal haemorrhages. **Treatment**: tracheostomy may be urgently required, systemic antibiotics are necessary. Laryngofissure (vertical split of the thyroid and cricoid cartilage in the midline) and reconstruction of the cartilage frame may be necessary if fractures are present to hold the displaced fragments outwards. **Inhaled foreign bodies** are rare in the larynx and almost immediately fatal when impacted. **Intubation injuries (Fig. 40 C)** occur in patients having prolonged respiration in Intensive Care Units and mostly due to rough intubation, prolonged presence of the tube between the vocal cords or using of

too large tubes. Usually appears as a granulomatous formation around the vocal processes. Symptomes are typically hoarsness and dyspnea. Endoscopic removal gives final resolution. **"Singer's nodes" (Fig. 42. A)** occur in persons who use the voice excessively (singers -sopranos and tenors-, actors, teachers). Pathologically these are localized hyperkeratoses. The site is constant, at the junction of the anterior third and posterior two thirds. Symptomes are increasing hoarsness, vocal fatigue and bilateral, symmetrical nodules. The nodules should be removed by microlaryngoscopy. Speech therapy helps to re-educate voice production. **Contact "ulcers"** occur almost exclusively in male adults. It occurs in singers, street vendors as a result from hammering of one vocal process of the arytenoid cartilage to the other. Vocal abuse is an important contributory factor. Laryngoscopy shows thickening of the tissues around the vocal process usually on one side only. A saucer-shaped depression may occur over one vocal process. Clinical features: discomfort in the throat, huskiness, vocal fatigue, referred otalgia. Vocal rest is an important part of the therapy for 2 to 3 weeks. Systemic steroids and removal of the thickened epithelium by microlaryngoscopy give good result.

5.5. Inflammatory diseases of the larynx.

Acute epiglottitis. An infection caused by Hemophilus influenzae. The *symptomes* onset suddenly as an acute respiratory illnes: fever, sore throat and excessive saliva in the mouth. There is respiratory stridor, retraction, pale cyanosis. The epiglottis is red and swollen. **Treatment.** Intravenous antibiotic (cephalosporine or semisynthetic penicillin) cover. Supportive measures are high humidity oxygen tent if necessary. Most cases respond to medication alone. In children intravenous corticosteriods are given to control inflammatory swelling. If signs of respiratory distress develop, tracheotomy may be required. Nasotracheal intubation during the period of airway obstruction must not last more than 48 hours. In Scandinavian countries immunization is introduced against the Hemophilus influenzae.

Acute infectious laryngitis. This is acute inflammation of the laryngeal mucosa. Bacterial causes are the same as in epiglottitis. The primary symptome is hoarsness accompanies to low-grade fever, cough and nasal obstruction. The larynx mucosa is red and vocal cords are swollen. **Treatment.** Strict voice rest, elimination of tobacco and alcohol are necessary. High humidity environment, increased fluid intake and analgesics like aspirin benefitial. Systemic antibiotics should be given. Penicillin is the drug of choice. In children, treatment should be more vigorous and include the antibiotics. **Acute laryngeal oedema.** Marked laryngeal swelling may

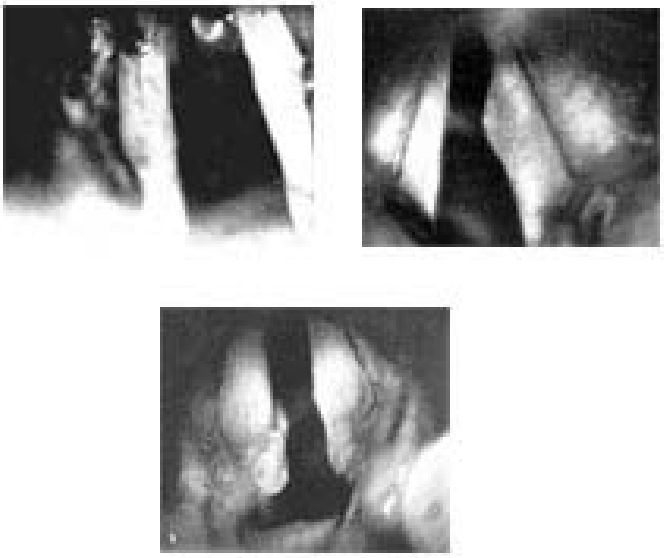


Figure 40. Benign space occupying lesions of the larynx, Upper pictures are polyps of the vocal cords. Lower picture is intubation granuloma.

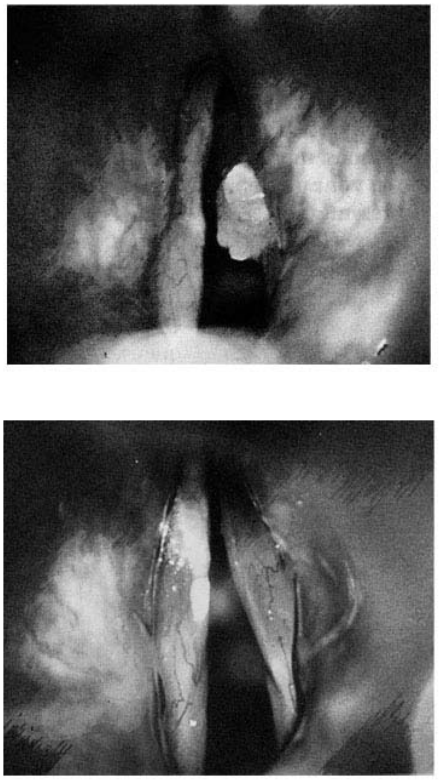


Figure 41. Premalignant disorders of the larynx. Pachydermia, (lower panel) Leukoplakia (upper panel)

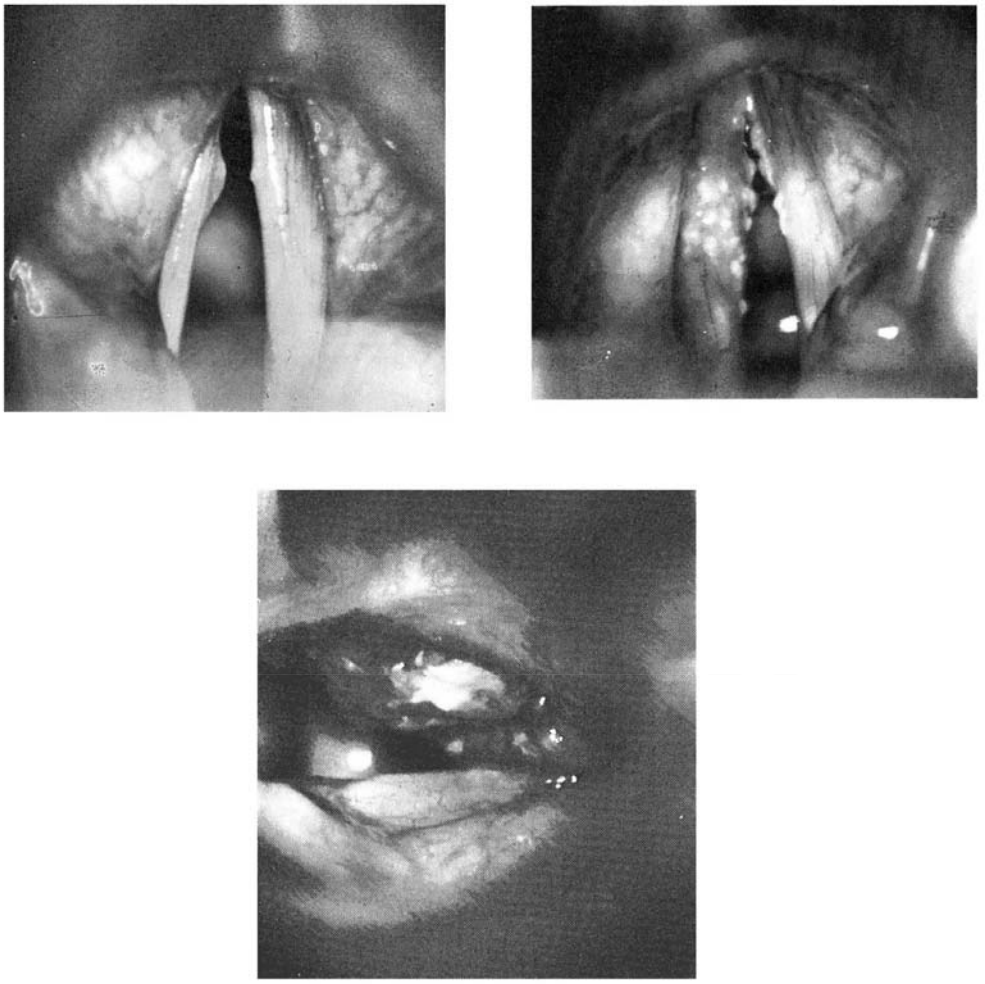


Figure 42. Singers node of the right vocal cord (upper left), chronic laryngitis (upper right) cancer of the right vocal cord (lower)

result from trauma, inflammation or allergy. The swelling represents angioneurotic oedema often due to a drug, including penicillin, horse serum or aspirin. There may be a family history of a similar problem. *Symptomes* are hoarsness of sudden onset, croupy cough, increasing respiratory stridor, cyanosis and possibly death. An asthmatic attck may be differentiated by expiratory wheezing. If the oedema is supraglottic, mirror examination of the larynx reveals pale, boggy swelling of the epiglottis and the ary-epiglottic folds. If the oedema is subglottic, often occuring in children, mirror examination is not diagnostic. A correct diagnosis may be established by the sudden onset, presence of inspiratory stridor and apperent lack of infection. **Treatment.** Intravenous corticosteroids should be given in large doses and repeated in 4 to 6 hours. Milder cases should respond to intravenous calcium alone. If respiratory distress does not improve promptly with treatment, tracheotomy is indicated. **Acute laryngotracheobronchitis (Subglottic laryngitis)**. Commonly called croup/pseudocroup. This is acute infectious disease of young children involving the subglottic larynx, with extension to the tracheobronchial tree. Etiology: viral infection accounts for most cases as suggested by a mild preceding viral upper respiratory infection and low white blood count. Bacterial etiology is suggested in those cases where cultures taken reveal one of the respiratory pathogens, such as haemolytic streptococci or Staphylococci, pneumococci or Haemophylus influenzae. The disease is seen most commonly in children below the age 4. The decrease of the ionized calcium level in blood is commonly assumed to be responsible for the pillow-like ring shaped swelling of the subglottic mucous membrane. The clinical features involve a croupy cough, usually begins at night following a mild rhinitis. Other symptomes are fever, inspiratory stridor. The voice, however, remains pure, indicating pathology of below the glottic region. **Treatment.** The child is placed in a high humidity oxygen tent. Intravenous calcium injection may prompt relieve symptoms. In intractable cases an injection of hydrocortisone in every 6 hours at the first 2 days are mandatory. Decreasing doses of steroids may be given orally over the next several days. Careful observation of the child is required, since continued respiratory distress will necessitate endotracheal intubation or tracheotomy which, however, today is rare.

Chronic laryngitis. **(Fig. 42.B)** This refers to chronic inflammatory changes of the vocal cords which result from the following etiologic factors: 1. vocal abuse; 2. excessive smoking; 3. excessive alcohol intake; 4. extremely dry or irritative environment.

Symptomes are hoarsness as the primary symptome, dry cough and a good voice which gets weaker as its is used throughout the day. Both vocal cords are thickened along their entire length, with patchy pink areas. The epithelium is granular in appearance, but without ulceration. **Treatment.** The prime treatment is to eliminite the etiologic factors. Humidification of the the environment is

important, especially in the winter months. Systemic medications have little effect, except for an antitussive where cough is prominent. Direct laryngoscopy with biposy is not required, except if area of leukoplakia has developed. In cases of chronic laryngitis with hypertrophy of the anterior vocal cord edges, careful stripping of the oedematous tissue by microlaryngoscopy is indicated. Complications are: polyp formation, atrophic laryngitis. **Atrophic laryngitis** is a form of chronic laryngitis characterized by atrophy of the mucosa and mucosal glands. Its etiology is unknown, but atrophic laryngitis often follows the fibrosis of chronic hypertrophic laryngitis. Symptomes are hoarsness and marked dryness of the throat. Indirect laryngoscopy reveals a dry, dull appearance of the laryngeal mucosa with yellowish or greenish crusts which adhere in the posterior comissure of the larynx. **Treatment**. The condition can hopefully be prevented by vigorous treatment of chronic hypertrophic laryngitis before development of atrophic changes. Treatment of atrophic laryngitis with crusting is based on creating a high-humidity enviroment. Nebulization with liquifying medication should be given several times a day. Iodides are given by mouth to counteract the dryness.

5.6. Vocal cord polyp.

This is a common type of benign tumor (not true neoplasm) which may occur in one **(Fig. 40)** or both vocal cords. Polyps may be oedematous, fibrous or vascular. Predisposing factors are irritation (smoking), inflammation or vocal abuse. The relevant symptome is hoarsness. Laryngeal indirect mirroring reveales a sessile or pedunculated smooth-surface, oedematous mass, usually unilateral, attached to the vocal cord. Treatment is surgical removal by microlaryngoscopy.

5.7. Laryngeal papillomatosis.

Two different manifestations are the juvenile which is a benign desease, and the adult which is a a precancerosis and characterized by multiple papillomas of the laryngeal mucosa which recur vigorously despite removal. They are viral (HPV: Human Papilloma Virus) in origin. Most patients show complete remission by puberty. In the rare cases when remission does not occur, the risk of malignant transformation is high. The symptome is absent or weak cry accompanied by inspiratory stridor. Indirect laryngoscopy (in adult) or direct laryngoscopy (in children) reveals the presence of multiple, soft, mucosal projections which tend to narrow the laryngeal airway. **Treatment.** Removal

by microlaryngoscopy should be repeated because of recurrence. Laser removal has given longer periods of remission. Effectiveness of human α-interferon is doubtful.

5.8. Laryngeal premalignant disorders.

Pachydermia (Fig. 41.) is characterized by epithelial hyperplasia of the mucosa of the posterior third of the vocal cords and interarytenoid space. Etiology and symptomes are same as in chronic laryngitis. Treatment is microlaryngoscopy with biopsy to rule out tumor. Avoidance of vocal abuse, cessation of smoking and decreased alcohol intake are advised. **Leukoplakia (Fig. 41.)** This is characterized by hyperplasia of the vocal cord epithelium with cornification. The lesion is white. Etiology: this is a chronic inflammation due to etiologic factors listed for chronic laryngitis.. The leading symptome is hoarsness. Biopsy must be taken by microlaryngoscopy to exclude malignant disease. The patient should be examined periodically (every half a year).

5.9. Laryngocele

It is an air-filled diverticulum originating from the laryngeal ventricle. There are two types: 1. External laryngocele: the commoner form, where the sac penetrates the thyroid membrane and presents as a mass in the neck. 2. Internal laryngocele, less common, where the sac dissects internally. The etiology is unknown. Symptomes: External laryngocele may be symptomless except for swelling in the neck which increases in size with increased intralaryngeal pressure. Internal laryngocele causes hoarsness and dyspnoe as it increases in size. The external laryngocele is palpable and enlarges on positive Valsalva maneuver. On compressing the swelling air is noted to enter the larynx. Internal laryngocele is noted as swelling on indirect laryngoscopy. Diagnosis is confirmed by x-ray. **Treatment.** This is a surgical removal via an external approach in the case of an external laryngocele. Internal laryngocele is removed in endolaryngeal approach via microlaryngoscopy.

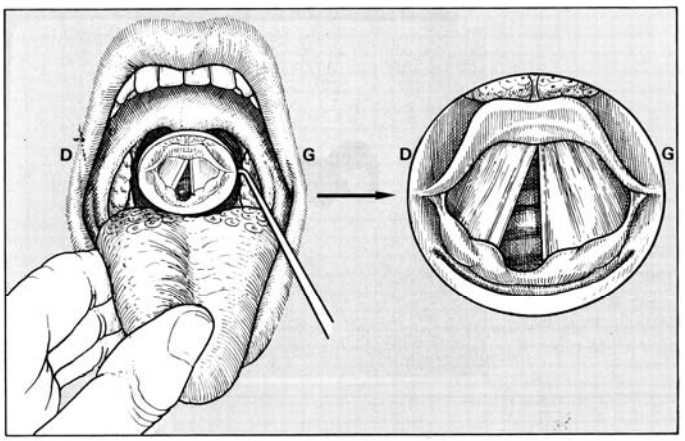


Figure 43. Paralysis of the left vocal cord. Note the paramedian position of the affected cord.

5.10. Laryngeal paralysis.

Paralysed vocal cords are best described by their position: median, paramedian, intermediate and extreme abduction (lateral). In median position, the paralysed cord remains in midline **(Fig. 43)**. This is a frequent position of a paralysed vocal cord, since the abductor muscles are weaker and more vulnarable than the adductor fibres. The intermediate position, often called cadaveric, is midway between the midline and position of complete abduction. Paramedian is between the midline and intermediate. Vocal cord paralysis may be temporary when the involved recurrent nerve has been stretched or traumatized rather than severed. The voice may improve after vocal cord paralysis due to a shift towards the midline by the paralysed cord and/or attempt at compensation by the uninvolved cord. Vocal nerve (Xth cranial nerve) involvement with vocal cord paralysis may be associated with other cranial nerve interruptions. From the nucleus ambiguus in the medulla to several centimeters below the jugular foramen, the last (IX-XIIth) cranial nerves are close together and may be involved by a single lesion. These can cause **associated laryngeal paralysis to other functional deficits**. Bulbar lesions may involve IXth, Xth, XIth and XIIth cranial nerves. Jugular foramen involvement (**Vernet's syndrome**) results in paralysis of the soft palate, pharynx, larynx, sternomastoid and trapezius muscles; anaesthesia of the soft palate; and loss of taste at the posterior third of the tongue. A parapharyngeal space lesion involves IXth, Xth, XIIth and sympatetics. **Avellis' syndrome** refers to any lesion involving the mucleus ambiguus and spinothalamic tract, with ipsilateral paralysis of the soft palate and larynx, and contralateral loss of of pain and temperature sensations over the trunk and extremities. Interruption of the cervical sympathic trunk results in **Horner's syndrome**, characterized by

miosis, enophtalmos, ptosis and anhydrosis. Thrombosis of the posterior inferior cerebellar artery (**Wallenberg's syndrome**) combines the findings of Avellis' and Horner's syndrome. *Anaesthesia of the larynx* results from involvement of the internal branch of the superior laryngeal nerve. Causes are peripheral neuritis, syphilis, tumor, multiple sclerosis and cerebrovascular accidents. The treatment and prognosis are dependent on the etiology. **Abductor paralysis of the larynx**. Most commonly results from interruption of the recureent nerves during thyroid surgery (iatrogenic). May be unilateral **(Fig. 43)** or bilateral. The voice is good with bilateral abductor paralysis cases, where the vocal cords are in the midline. The patient is however dyspneic. With unilateral paralysis mild hoarsness is present. Left unilateral vocal cord paralysis is often due to cardiac surgery or compression of vagus by the arcus aortae (**Oertner's syndrome**). Indirect laryngoscopy reveals the vocal cords to be immobile and fixed in midline position. **Treatment.** Bilateral abductor paralysis requires tracheotomy or laterofixation of the vocal cords to the neck. As the latter is a reversible lateralization the ligation can be released when electromyography reveals reinnervation of the cord. Widening of the glottic airway by removal of one vocal cord and the ipsilateral arytenoid cartilage is beneficial and the tracheotomy can then be closed. **Adductor paralysis of the larynx**. This is rare, due to trauma, neuritis and central nervous system lesions. The voice is weak. The involved cord remains in abductor position. A functioning interarytenoid muscle may close the glottis posteriorly. If only the arytenoideus is paralysed, the vocal cords will come together in phonation, except for a triangular chink posteriorly. Treatment is **phonosurgery**. Previous years a teflon-paste injection into the involved vocal cord was performed. Recently in internus paralysis cases an autologous fat homogenate (from hip or belly) is preferred. It gives long-term good results.

5.11. Cancer of the larynx

Almost always is squamous cell type and one of the commonest malignancies **(Fig. 42.C)** in males. Classification is outlined in the table.
Leading *symptome* is hoarsness. Accompanies referred otalgia (in supraglottic cancer), difficulty in swallowing, dyspnea, enlarged cervical nodes. Every patients with hoarsness of 2 weeks duration should have a mirror examination of the larynx. The lesion appears as raised, irregular, ulcerated growth which may occur on any part of the larynx. **Vocal cord mobility** should be determined, since prognosis and treatment are dependent on it. The neck is carfully palpated for presence of enlarged neck lymph nodes. During indirect or

direct laryngoscopy (microlaryngoscopy) biopsy specimen should be taken for histological diagnosis. Treatment is dependent on extension of the tumor. **T_1 tumors** can be treated primarily by irradiation or surgery (chordectomy, epiglottectomy, laser surgery). **T_2 supraglottic and glottic tumors** can be treated by partial laryngectomies while **T_3 and T_4 glottic, supraglottic tumors** and **subglottic tumors** should be treated with total laryngectomy. Postoperative irradiation is recommended in subglottic and T_4 tumors of any location. Where cervical lymph nodes are palpable radical neck dissection is carried out. Chemotherapy is used only in patients with far advanced disease, or occasionally, in conjunction with radiotherapy. Agents include cis-platinum, cyclophosphamide, bleomycine, methotrexate and 5-fluoro-uracil.

Supraglottic	Glottic	Subglottic
T. Primary tumor T_1 Tumor confined to one anatomical region within the larynx (laryngeal surface of the epiglottis or to ary-epiglottic fold or ventricular cavity or ventricular band	Tumor is confined to vocal cord and mobility of cord is normal **T_{1a}** only one vocal cord id sffected **T_{1b}** both vocal cords are affected	Tumor limited to one side of the subglottic region, exclusive of the undersurface of cord.
T_2 Tumor extends to the glottis or is confined to one anatomical region within the larynx but involves more subregions (epiglottis, ventricular folds or cavities)	Tumor extends to the supraglottis or subglottis with normal or decreased mobility	Tumor extends to glottis or both sides of subglottic region
T_3 Tumor extends to glottis immobilizing vocal cords	Tumor extends from the vocal cords to subglottic region or to supraglottic region and immobilizes vocal cord/s	Tumor involves the cords with immobility.
T_4 Tumor extends extralaryngeally	Tumor extension to extralaryngeal regions	Tumor extension to the trachea or postcricoid region.

5.11.1. Surgical treatment of laryngeal tumors

Cordectomy is chosen in those cases when tumor is localized in the free edge of one of the vocal cords **(Fig. 44.)**. Removal of the cord with the arytenoid cartilage is then an ablastic measure. Cordectomy is done in general anesthesia via microlaryngoscopy. The removal can be attepted by laser, electrocoagulation or surgical knife.

Hemilaryngectomy (**Fig. 44.** and **Fig. 44.)** is a vertical partial laryngectomy when the thyroid alae are splitted apart via a horizontal vertical incision in the midline and paramedially. The endolaryngeal mucosa, muscles with the perichondrium are removed from vertical half of the larynx including true vocal cord, ventricular fold, arytenoid cartilage. This surgery is suggested for T_2 glottic tumors. A modified version of this surgery can also be performed with resection of hypopharyngeal tumors **(Fig. 47)**.

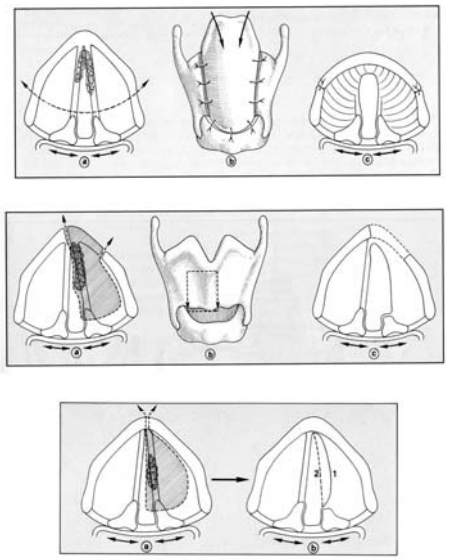


Figure 44. Cordectomy, performed through a thyrotomy (splitting the thyroid cartilage in the midline). Cordectomy is done preferably in endoscopic or endolaryngeal way. Note that the arytenoid cartilage is not removed. (lower panel) Frontolateral partial laryngectomy or hemilaryngectomy. (middle panel) Extended frontolateral partial laryngectomy.(upper panel) Dotted lines show resection lines, dark areas show resected parts of the larynx.

Horizontal supraglottic laryngectomy (Fig. 45). This is a horizontal partial laryngectomy when the supraglottis including the epiglottis, ventricular folds and aryepiglottis folds is removed. The vocal cords are left in place. Can be performed in T_1 and T_2 supraglottic tumors.

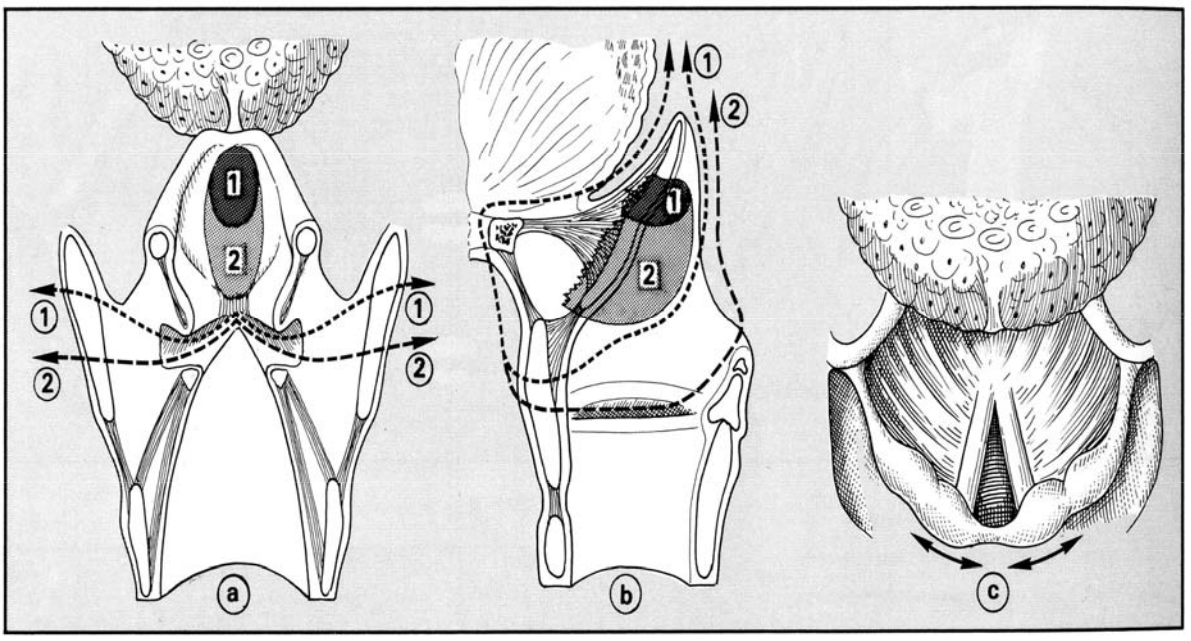


Figure 45. Horizontal partial laryngectomy (Supraglottic laryngectomy). (a) and (b) show posterior and lateral view of the larynx with dotted lines indicating the resection lines. (c) superior view of the glottis after the surgery is done (the epiglottis, the ventricular folds are missing)

Supracricoid horizontal laryngectomy (Cricohyoido-epiglottopexy) (Fig. 46). A horizontal subtotal laryngectomy. Only the cricoid cartilage is left in place with one vocal cord if possible. The hyoid bone and epiglottis is saved and used for improve deglutition and speech. Can be afforded for transglottic tumors, when tumor extends to supraglottis and glottis but mobility of the vocal cords is preserved. The advantage of this surgery is that the tracheal cannula can be removed and tracheostoma is closed postoperatively.

Total laryngectomy. It is for T_3 and T_4 laryngeal tumors. The whole larynx is removed. The trachea of the patients is opened to the neck skin. The hypopharyngeal pouch is sutured to the base of the tongue. Main handicap of these patients is the loose of the natural speech. Possibilities are, however, today numerous. An esophagus voice may be attempted to learn postoperatively which is developed from belching and can produce a very good and intelligible speech.

Those who can not learn esophagus voice can be implanted with voice-prosthesis. This is a teflon valve, which can be implanted in between the esophagus and trachea. Another possibility is an electrical microphone which produces a monotonic voice but gives understanable speech.

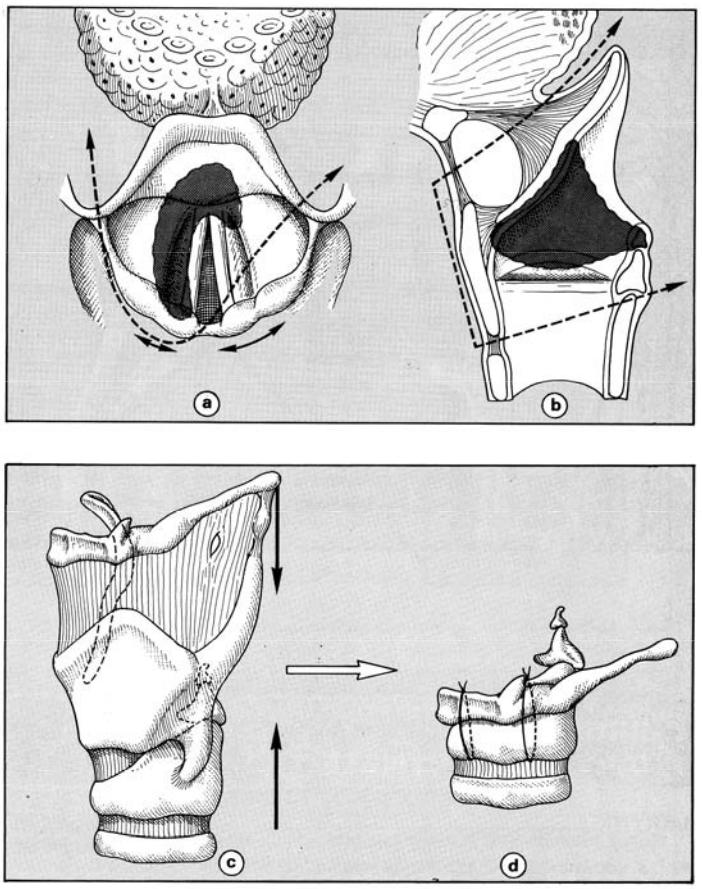


Figure 46. Subtotal horizontal laryngectomy with crico-hyoido-epiglottopexy. (a) and (b) are superior and lateral views of the larynx with indications of the resection by dotted lines. Dark areas are those which are excised during surgery. (c) and (d) show the cartilagenous skeleton of the larynx before (c) and after (d) the surgery is done.

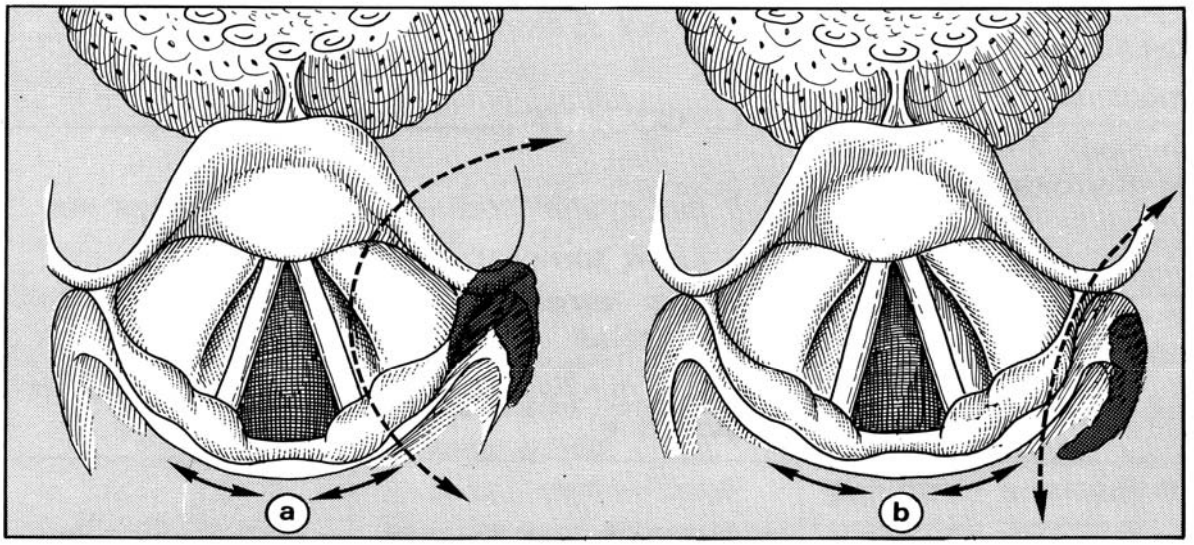


Figure 47. Resection of tumors in the pyriform recess. (a) the tumor extends to the medial wall of the pyriform recess and removal includes the affected side of the supraglottis. (b) the tumor extends to the lateral wall of the pyriform recess

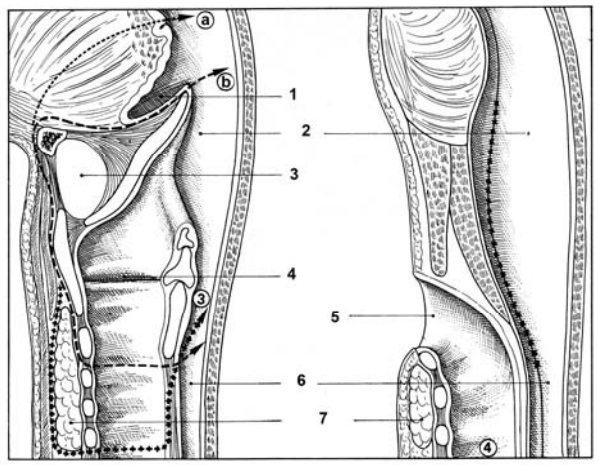
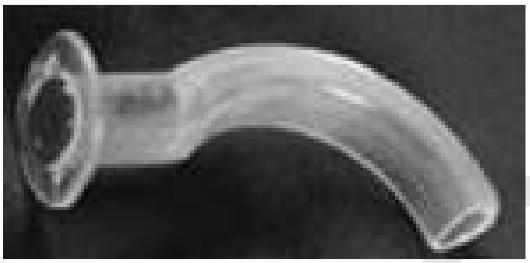


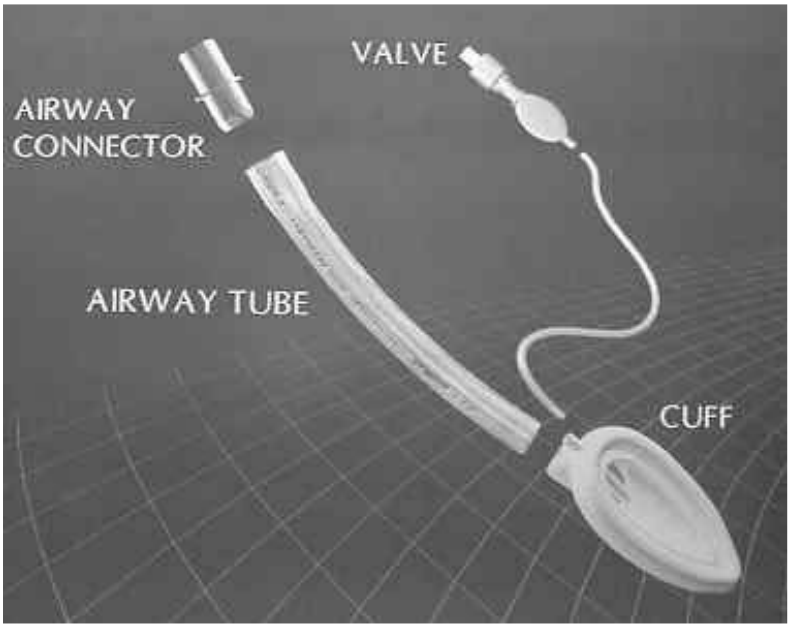
Figure 48. Total laryngectomy, and the larynx and laryngo-pharynx as it looks before (A) and after (B) laryngectomy. Dotted line shows the resection line from lateral aspect (A). (a) epiglottis (b) laryngopharynx (c) hyoid bone (d) vocal process of the arytenoid cartilage (e) tracheostome (f) opening of the esophagus (g) thyroid gland.

5.12. Decision making in acute airway obstruction

The number one life threatening urgency is acute airway obstruction. First task is to prove with secure airway by intubation, tracheotomy or cricothyrotomy (conicotomy).

Case history if could be taken may clarify previous thyroid surgery, medication. The possible causes are strongly related to the age of the patient. **Newborns** present congenital anomalies of the larynx (laryngeal stenosis, vocal cord paralysis, laryngomalacia). Direct laryngoscopy reveals abnormalities when tracheotomy should be considered. Normal larynx suggests choanal atresia or neurological disease. **Children's** airway obstruction may be due to foreign body





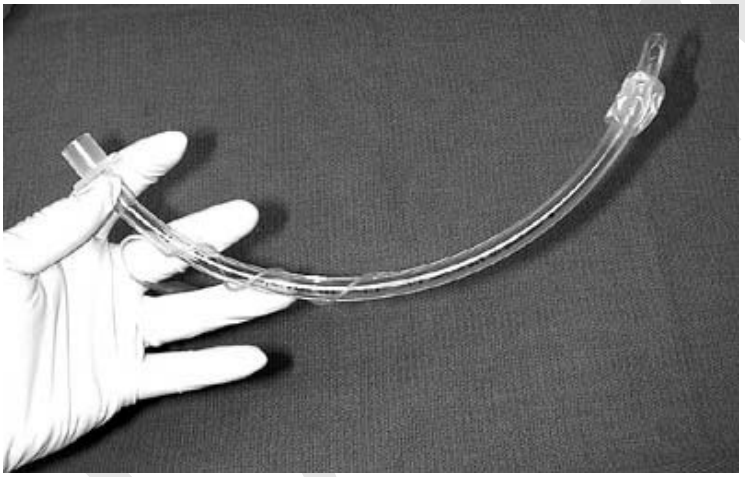


Figure 49. Tubes for secure airway in acute upper airway emergencies. Uppermost is oropharyngeal tube, upper middle: nasopharyngeal tubes, lower middle is laryngeal mask and lowermost is endotracheal tube.

(Heimlich maneuver), epiglottitis or subglottic laryngitis In the latter 2 cases medical treatment is usually sufficient (steroid, calcium, antibiotics). The diagnostic protocol depends on the obstruction whether it is compete or incomplete. Incomplete cases allow chest X-ray.

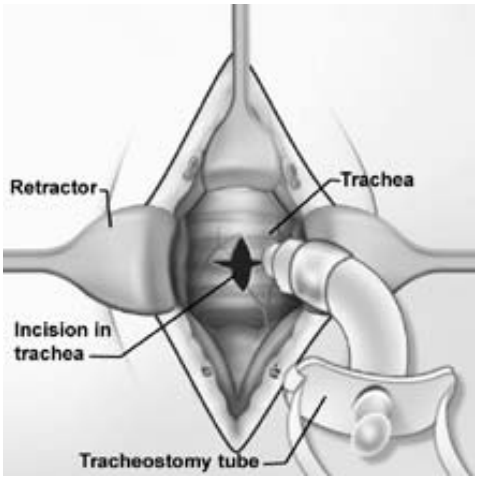
The main causes in adults are: 1. foreign body, 2. trauma, 3. oedema, 4. infection, 5. neoplasm

1. Impending loss of airway due to foreign body dictates conicotomy if Heimlich maneuver is unsuccessful and there is no time for direct laryngoscopy.
2. Traumatic cases are most dangerous when associated with laryngeal fractures, which produce multiple or single extended subperichondrial hematomas and decrease critically the airway, although usually the obstruction is incompete. Surgical reconstruction of the laryngeal cartilages broken should be attempted in tracheostomy cover.
3. Allergic or hereditary laryngeal oedema can be controlled by medical treatment (steroid). Case history usually is orienting.

1. Infections in adults are deep neck infections (Ludwig's angina), lingual tonsillitis. Although these may be presented as an acute urgency, tracheotomy often can be avoided. Diphtheria is a rarity today.
2. Neoplasms are the most frequent casuses of acute airway problems in untreated cases.

5.13. Tracheostomy

A surgical opening made into the cervical trachea **(Fig. 50)**. Indications are:



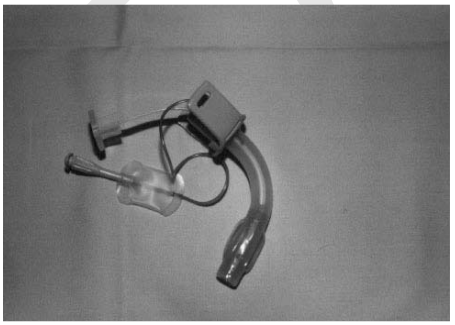


Figure 50. Tracheotomy. Incision on the trachea is done in the 2nd or 3rd tracheal cartilage-ring. Middle panel shows an uncuffed trachea canule, lower panel demonstrates a cuffed trachea canule.

1. Laryngeal obstruction
(a) acute inflammatory conditions:
diphtheria,acute epiglottitis, subglottic laryngitis
(b) allergic oedema
(c) trauma
(d) congenital anomaly (atresia, web)
(e) bilateral vocal cord paralysis
(f) neoplasms
2. Retained tracheobronchial secretions:
(a) unconscious patient: head injury, cerebrovascular accident
(b) to permit controlled breathing by respirator
3. Respiratory insufficiency: to reduce dead space
4. Paralysis of respiratory muscles: poliomyelitis, cervical cord lesions
5. Pharyngeal or hypopharyngeal lesions: neoplasms
6. Prophylactic: prior to extensive surgery of mouth, pharynx, larynx

Complications of tracheostomy: Bleeding, subcutaneous emphysema, apnea, infection, crusting, stenosis. **Cricothyrotomy (conicotomy)** is an opening made higher in the neck, at the level of the cricothyroid membrane, and is used in extreme emergencies. This procedure can be performed in less than one minute through a horizontal incision between the thyroid and cricoid cartilages. After the situation is under control, a standard tracheostomy should be placed at the usual site and the conicotomy opening is closed within 24 hours.

5. 14. Dysphonia

Any impairment in the ability to speak normally as from spasm or strain of the vocal cords. Not necessarily the vocal cord abnormality is the cause of quality change in speech. The term is almost synonymous with hoarseness. There are many benign causes. The pathological background can be devided to 2 groups: functional dysphonia and morphological causes.

Any patient with **hoarseness of 2 weeks** duration must undergo visualization of the vocal cords.

The history should be carefully worked up as to previous trauma of the larynx, intubation episode, pulmonary diseases, gastrointestinal problems (GORD, laryngopharyngeal reflux), autoimmune diseases (rheumatoid arthritis), endocrine abnormalities (hypothyroidism).

Different **neurological disorders** cause dysphonia:
1. Hypoadduction: Parkinson's disease, myasthenia, muscular dystrophy

Abductor spasmodic dysphonia

2. Hyperadduction: Huntington's disease, adductor spasmodic dysphonia
3. Mixed ab- and adductor: multiple sclerosis (MS), ataxic dysphonia (cerebellar), amyotrophic lateral sclerosis (ALS)
4. Vocal tremor: Parkinson's disease, spasmodic dysphonia, palatopharyngeal myoclonus

Some s**urgical interventions** are responsible for hoarsness e.g. skullbase procedures, carotid endarterectomies, thyroidectomies, repair of aorta aneurysm. Lot of different therapeutic agents, **drugs** can induce hoarseness e.g. antihistamines, decongenstants, diuretics, antidepressants, phenothiazines). Social history should clarify alcohol or tobacco abuse and professional voice use.

Associated symptomes are:

1. breathiness (vocal fold paralysis, vocal fold mass disease)
2. vocal fatigue (vocal fold atrophy or paralysis)
3. choking (cerebrovascular accident)
4. odynophonia (vocal fold granuloma, muscular tension dysphonia)
5. laryngospasm (gastroesophageal reflux disease)
6. stridor (bilateral vocal fold paralysis, laryngeal stenosis, paradoxical vocal fold motion)
7. dysphagia (laryngeal cancer, unilateral vocal fold paralysis, cerebrovascular accident)
8. globus (laryngopjaryngeal reflux, neurologic disease)
9. velopharyngeal insufficiency (myasthenia gravis, ALS, vagal paralysis)

Diagnosis. Indirect and direct laryngoscopy and videolaryngoscopy are necessary. CT and MR imaging may elucidate tumor in the chest, brain or head and neck region. Direct pharyngolaryngoesophagoscopy.

Most probable causes of dysphonias are: vocal cord pathology, neurological, muscular causes.

Special attention is payed to **recurrent laryngeal nerve** pathologies (nucleus ambiguus can be involved in stroke).

1. Neck lesions, tumors, trauma can equally cause nerve injury
2. Congenital vocal cord pathologies: any laryngeal abnormality e.g. laryngeal web, laryngeal cleft
3. Inflammatory diseases: laryngitis, epiglottitis, pharyngitis.
4. Traumatic: direct trauma (intubation granuloma), vocal nodules due to vocal abuse
5. Laryngopharyngeal reflux: granuloma
6. Neoplasma: benign and malignant laryngeal and pharyngeal tumors
7. Voice abuse and smoking: Rienke's oedema
8. Radiation injury: postirradiation dysphonia

All these morphological alterations can be used surgically. **Functional dysphonia** is idiopathic or psychosomatic manifestation. It is due to abnormal vibration pattern of the vocal cords or abnormally increased (**spasmodic dysphonia**) or decreased passage of air through the glottis which causes hoarseness rather than a clear tone. Functional dysphonias can be classified as **hyperkinetic** or **hypokinetic** disorders. **Psychogenic aphonia** is a typical psychosomatic disease. Dysphonia should be examined by endoscopy, stroboscopy and high speed cinematography. Phoniatric speech conditioning is provided by speech pathologist or phoniatrician. Spasmodic forms can be treated by botulinus toxin local injections. The results are only temporary.

6. The trachea and tracheobronchial tree

6.1. Bronchoscopy

It is the examination of the tracheobronchial tree for diagnostic and therapeutic purposes. The trachea is a cylindrical tube, slightly flattened posteriorly, which extends from the larynx above to the level of the second costal cartilage below, where it bifurcates into the right and left main bronchi. The tracheal mucosa is very pale and glistening, with circular cartilaginous rings seen through it. The carina is a sharp, keel-like structure representing the dividing line between rigth and left bronchi. It runs vertically and is freely movable on respiration. The rigth main bronchus is shorter than the left and comes off the trachea at a less severe angle than the latter. Endoscopically viewed, the right main bronchus gives off the rigth upper lobe bronchus at a rigth angle laterally, the middle lobe bronchus anteriorly and continues as the lower lobe bronchus. The left main stem bronchus gives off the upper lobe bronchus at a right angle laterally, then continues as the lower lobe bronchus. There is no middle lobe of the left lung. The normal tracheobronchial tree is freely mobile with the diameter of the various bronchi becoming wider on inspiration and narrow on exspiration.

Most bronchoscopic procedures are done today by flexible bronchoscopes. These are illuminated by fiberoptic bundles and small enough in diameter to be passed into segmental bronchi, which can not be seen by open, rigid bronchoscopes. Disadvantage of the flexible instrument of its smaller diameter that limits its applicability in taking larger biopsies, removal of bigger foreign bodies. The flexible bronchoscope is used for almost all diagnostic cases. The

rigid bronchoscope is still in use for biopsies of tumors in the trachea, for aspiration of thick secretion as with atelectasis, to remove foreign bodies. *Indications for bronchoscopy.*
1. To inspect disease process in the tracheobronchial tree. 2. To collect secretions for laboratory analysis including bacterial smear, culture and sensitivity. 3. To biopsy tumors. 4. To remove retained secretions (atelectasis, bronchiectasis) 5. To remove foreign bodies 6. To remove obstructing granulomas 7. To dilate strictures.

6.2. Bronchography

This is the radiographic examination of the tracheobronchial tree utilizing radiopaque material instilled via a catheter inserted into the trachea. *Indications:* 1. Suspected bronchiectasis; 2. Haemoptysis of unknown etiology; 3. Suspected neoplasm distal to the area covered by bronchoscopy. *Contraindications:* 1. Active infections; 2. Patient in respiratory distress; 3. Active tuberculosis.

Bronchography is always done in local anaesthesia, but has to be done under general anaesthesia in children. There may be an allergic reaction to iodine contained in the contrast material.

6.3. Foreign bodies

Those which passed through the larynx lodge most commonly in the right main bronchus. There may be no history of inhalation, for children may be too frightened to mention it, adults may inhale when asleep. Caugh and dyspnea occur at the time of the accident. Bloodstained expectoration is sometimes present. Great variety of non-vegetable foreign bodies have been recorded. Atelectasis occurs if the lobe of the lung is completely obstructed, with subsequent danger of lung abscess. An obstructive emphysema occurs if a lobe is only partially obstructed. Hard or metallic objects may be present for many months or years without causing suspicious symptoms. **Vegetable foreign bodies**. Include nuts and pips, fruits and vegetables. There is always an intense inflammatory reaction of the mucosa. This may be a specific allergic reaction to the vegetable oil liberated by the swelling object. Symptomes are acute tracheitis or bronchitis. An emphysema results when it is partial. Atelectasis and lung abscess follow later when obstruction is complete. **Treatment** is removal through bronchoscope. Foreign bodies can rarely recover by the flexible bronchoscope.

6.4. Inflammations of the tracheobronchial tree

Acute inflammations of the trachea and bronchi frequently accompanies to laryngitis. Common symptomes are cough, dyspnea, retrosternal pain. In severe cases systemic antibiotics are given. **Tracheitis sicca.** Always associated with atrophic rhinitis or laryngitis sicca and common disease of laryngectomized patients. Dry crusts on the tracheal wall are irritating. Treatment is aimed at making the secretions more moist, especially by the use of a humidifier and soda-bicarbonate spray. Obstructing crusts may require frequent removal after laryngectomy. **Bronchiectasis**. Often associated with chronic sinusitis. **Kartagener's syndrome** is the combination of bronchiectasis, chronic sinusitis and dextrocardia. Bronchial foreign bodies, tumors or strictures may cause bronchial obstruction. Bronchoscopy is necessary for diagnosis and treatment. **Lung abscess:** two main types are: 1. Broncial abscess due to dilatation of a bronchus and filling with pus. 2. Solitary lung abscess develops in atelectatic area. The treatment includes systemic antibiotics, postural drainage and breathing exercise, bronchoscopic aspirations, external drainage or lobectomy.

6.5. Neoplasms

Benign tumors are rare: papillomas and adenomas may occur. *Adenocarcinoma and squamous cell carcinoma* of the trachea are not common. The growth is usually ulcerative and covered with slough. Cough, haemoptysis dyspnea are characteristic symptomes. Voice change indicates involvement of the recurrent laryngeal nerve. It may be the first sign. **Treatment.** Tracheostomy is required when dyspnea is present. The tube must be long enough to reach beyond the tumor. Excision of the affected segment together with the paratracheal lymph nodes may be possible. The ends are mobilized and reunited. Irradiation therapy provides long periods of relief. *Carcinoma of the bronchus.* There are some suspected aetiologic factors: 1. carcinogenic hydrocarbons, which are present in tobacco smoke; 2. radioactive substances, encountered by uranium and cobalt miners; 3. pneumoconiosis; 4. chronic bronchitis. The tumor may histologically be anaplastic, squamous cell and adenocarcinoma. The primary, secondary and tertiary bronchi are the site of origine. Direct spread in the lung occur by local extension, peribronchial infiltration and submucosal spread. Lymphatic spread to the hilar nodes is usual and often early. Haematogenous spread, blood-borne metastasis are common, especially in the liver, bones and brain. *Clinical features.* 1. Cough; 2. Sense of constriction in the chest due to endobronchial obstruction; 3. Pain; 4. Atypical pneumonia may be first sign of the presence of carcinoma; 5. Haemoptysis; 6. Dyspnea; 7. Voice change if the tumor invades the recurrent laryngeal nerve. *Radiography* is the most valuable diagnostic measure. The changes are: hilar

shadow, atelectasis, infiltration of the lung, obstructive emphysema, pleural effusion and abscess cavities. CT scan gives precise informations. Other diagnostic measures are cytology of sputum or of bronchial aspirate, bronchography, bronchoscopy and biopsy or needle biopsy. **Treatment.** Lobectomy or pneumonectomy with complete removal of the related mediastinal lymph nodes. Radiotherapy may be a useful palliative measure for releiving the bronchial obstruction. Cytotoxic drugs given systemically can be of some value in oat-cell carcinoma.

7. The oesophagus

The oesophagus is a muscular tube, 25 cm long. It extends from the lower border of the cricoid cartilage above at the level of the sixth cervical vertebra, to the cardiac orifice of the stomach below. There are three constrictions: 1. 15 cm from the upper incisor teeth, at its commencement. 2. 25 cm from the upper incisor teeth, where it is crossed by the aortic arch and left main bronchus. 3. 40 cm from the upper incisor teeth, where it pierces the diaphragm. The oesophagus has four coats: mucous, submucous, muscular (internal circular, external longitudinal) and fibrous. **Blood supply**. Arteries arise from the inferior thyroid artery, in the cervical part. The descending thoracic aorta in the thoracic part and the left gastric artery, in the abdominal part. Veins drain to inferior thyroid vein, the azygos vein, and left gastric vein. **Nerve supply** derives from the Xth cranial nerve (parasympathetic) and from the sympathetic trunk.

Examination of the oesophagus can be performed radiologically and endoscopically. *Radiography* of the chest and lateral views of the neck should always be done before oesophagus is examined endoscopically. In most cases barium swallow examination is also indicated. If these precautions are not taken an unsuspected pharyngeal pouch mey be entered and even pierced, or an aneurysm of the aorta perforated. Oesophageal obstruction from strictures, neoplasms, or other causes is also demonstrated. *Endoscopy. Oesophagoscope* is used when the whole length of the oesophagus must be examined. The region of the arch of the aorta can be recognized by its redder, rugose mucosa. *Bronchoscopy* should always be done in cases of oesophageal neoplasms. Erosion of the trachea or left main bronchus may be seen.

7.1. Congenital anomalies

Congenital atresia is rare, causes regurgitation of feeds usually becomes manifest on the second day of life. Closure of fistula with end-to-end anastomosis must be performed immediately a diagnosis has been made (atresia with tracheo-oesophageal fistula). It can be performed by a transthoracic approach. Congenital shortening is usually associated with a throcic stomach. The oesophagus ends at the level of the seventh thoracic vertebra (instead of the eleventh). Clinical symptomes are dysphagia, vomiting, pain caused by reflux oesophagitis. Transthoracic operations may be performed to mobilize the stomach. An anastomosis is sometimes made between the two parts of the stomach. Section of the left phrenic nerve is performed with some succes.

7.2. Traumatic damage to the oesophagus

Rupture occurs during violent vomiting in males of alcoholic disposition, most commonly. Symptomes are severe pain, vomiting, dyspnea and collapse**. The treatment** is surgical drainage through thoracotomy, gastrostomy and intravenous antibiotics. **Direct injuries** are burns, scalds, corrosive injuries, instrumentation from bouginage, biopsy or removal of foreign bodies. Clinically features by pain which is severe and immediate in onset; dysphagia, shock, dyspnea. Barium swallow locates the site of the trauma. Immediate repair of tears or ruptures should be attempted by an external operation. **Foreign bodies**. The impaction of a foreign body depends chiefly on its size and shape. Sharp articles may stick in any part. A large article or bolus of food may become impacted in a normal oesophagus. Small articles may be impacted at a site of the normal narrowing. In contrast to the trachea and bronchi, here the non-vegetable foreign bodies are more dangerous. Commonest locations of impactions are the thoracic inlet and the cardia. Symptomes are complained immediately, but some objects may be impacted for weeks or months. There may be no obvious history of foreign body, especially in children (i.e. a piece of glass in the milk). Pain, dysphagia, regurgitation of food are the commonest. Opaque objects are shown lying in the coronal plane, i.e. coins. Non-opaque objects may be visualized by barium swallow. Endoscopic examination may be recommended in general anaesthesia.

7.3. Inflammation and ulceration of oesophagus

Acute oesophagitis is uncommon. A relatively mild degree of oesophagitis occurs in many cases of dilatation of the oesophagus. A **"reflux" oesophagitis** occurs with certain types of short oesophagus and with certain lesions of the cardiac end of the stomach.

Ulceration may be due heterotopic peptic tissue in the lower third of the oesophagus, and even in the upper third. A typical peptic ulcer may occur. Haemorrhages, perforation and malignant changes are recorded. Stricture formation is the commonest complication. This may lead to aquired shortening of the oesophagus. Ulceration may fallow diphtheria, scarlet fever and typhoid. Tuberculous and syphilitic ulcers are also recorded.
Plummer-Vinson syndrome (see in 3.3.3. section).

7.4. Neoplasms of the oesophagus

Benign growths are rare (leiomyoma, papilloma, fibroma, angiofibroma, lipoma. Their removal by endoscopic means is recommended when results in dysphagia. *Carcinoma*. Majority of cases are males above 50 years old age. The half of the cases present tumor in the middle third of the oesophagus. Symptomes are dysphagia, cough, cachexia and pain. Diagnosis should be established by radiography and endoscopy. **Treatment**. Oesophagectomy, a transpleural excision of the growth, with anastomosis of the stomach or intestine to the severed oesophagus above. The best form of palliation is an irradiation therapy

7.5. Miscellaneous conditions of the oesophagus.

Strictures. Strictures might be congenital or post-traumatic such as swallowing of corrosives or foreign bodies. The symptomes are dysphagia, regurgitation of food, loss of weight, cough. **Treatment is dilatation** by different techniques. Gastrostomy must be performed when obstruction is severe. **Achalasia**. There is a marked dilatation of the lower two-thirds of the oesophagus. The muscular wall may be hypertrophied. Etiology is uncertain. Most popular theory postulates a failure of relaxation of the cardiac orifice from degeneration of Auerbach's plexus. It affects young persons. Fullness after meals, dysphagia, regurgitation are characteristic. Radiography after barium swallow is typical: There is an enormous dilatation of the oesophagus, with smooth rounded lower border at the cardia. **Treatment**. 1. Lavage with a stomach tube as a preliminary to other treatments. 2. relaxation may be obtained by amyl-, or octyl-nitrite inhalation. 3. Dilatation of lower end by bouginage. 4. Cardioplasty operation 5. Anastomotic operations (between stomach and oesophagus) **Diverticulum.** May be congenital. They are of the pulsion type and must be distinguished from the traction type of acquired origin. In the latter case, there is a traction pouch. It usually occurs at the level of the bifurcation of the trachea and is the result of tuberculosis of the tracheobronchial lymph nodes. Rarely, the pouch may be large, from spontaneous rupture of a mediastinal

abscess or cyst into the oesophagus. Usually symptomless but there may be vague "dyspepsia" or dysphagia. **Acquired tracheo-oesophageal fistula**. Due to carcinoma of the oesophagus, rupture of a para-oesophageal abscess, perichondritis of the tracheal rings, trauma from foreign bodies. Cough is marked, especially on taking foods. Bronchopneumonia develops early. **Treatment** is stomach tube through the nose. Gastrostomy must be performed if the tube can not be passed. Repair may be attempted in traumatic cases. **Hiatus hernia**. l. Sliding: the cardiac part of the stomach follows the oesophagus in line through the diaphragm. 2. Para-oesophageal: the cardia retains in normal position while the fundus of the stomach herniates into the thorax alongside the oesophagus. Symptomes are heartburn and pain, which is due to acid reflux into oesophagus. Both aggravated by lying down, releived by alkalis, and occur more usually in the sliding type in which the cardiac sphincter mechanism is ineffective. Bleeding and peptic ulcers may also be the consequences. Diagnosis may be established by barium swallow. **Treatment**: In most cases weight reduction, alkalis by mouth, and sleeping propped up with pillows will give some relief. Surgical treatment with replacement of the stomach in the abdomen and repair of the diaphragm is usually reserved for severe cases in which medical treatment has failed.

8. Differential diagnosis of masses in the neck

The main lymphnode localizations in the neck is schematically drawn in the **Fig. 51**.

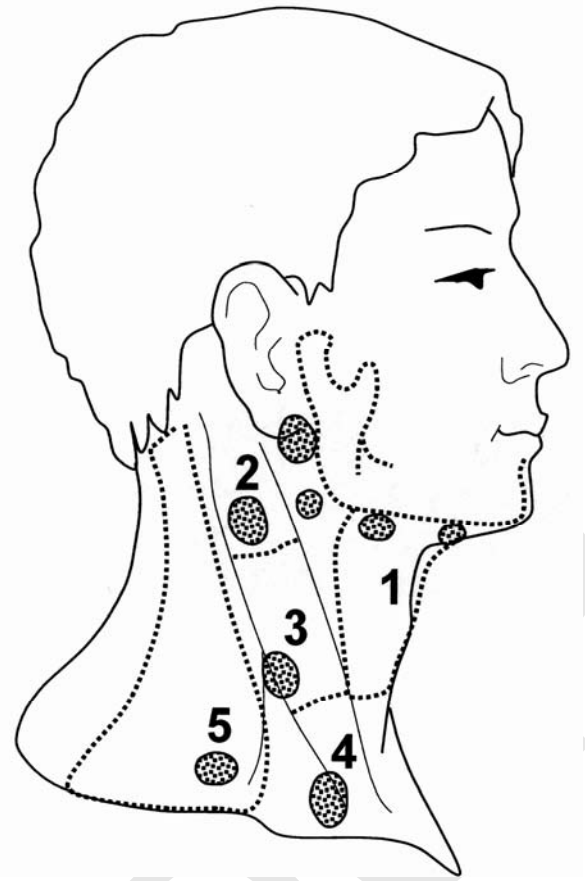


Figure 51. Lymphnodes in the neck. 1. anterior compartment (submental) 2. upper deep parajugular 3. Middle parajugular 4. Lower parajugular 5. posterior compartment (Supraclavicular and retrosternocleidomastoid).

(a) Inflammatory neck nodes are common in young individuals. Adenopathy in association with mononucleosis and chronic adenotonsillar infections are characteristically bilateral and multiple. Unilateral adenopathy can be associated with dental pathology, scalp infections, infected congenital cysts of the neck, and cat scratch fever. Fluctuance, tenderness, fever, systemic toxicity all favour a diagnosis to infection. Tenderness may be misleading because neoplastic nodes can be secondarily infected. Lymphoma - as systemic disease - should always be excluded. **Lateral cervical cysts and fistulae** . The opening of the fistula is always at the anterior border of the sternomastoid muscle. The discharge may be milky or purulent, permanent or episodic. the cause is genetic or external toxic agents (smoking, acohol, drugs). Introduction of contrast material through the fistula shows the branches. If the fistula has a pharyngeal opening, patient tastes the contrast material. Treatment is complete dissection. Cysts are presented in childhood, as firm, elastic and fluctuant masses. Since epithelial cysts may undergo malignant degeneration, their ablastic removal is mandatory. **Thyroglossal duct cysts and fistulae.** These are located in the midline in the level, above or below the hyoid bone. These are remnants of the thyroglossal ducts and present as median cervical cysts. The site is so much typical, that alone ensures the diagnosis. An important point in surgical treatment that body of the hyoid bone should be removed with cysts to prevent recurrence.

(b) Unexplained neck adenopathy, especially in the older individuals with risk factors for head and neck malignancies (smoking, ethanol, poor oral hygiene), should have a thourough examination of all mucosal surfaces of the upper aerodigestive tract for asymptomatic carcinomas. This includes examination of the nasopharynx, base of tongue, pyriform sinus, tonsillar fossae, larynx, and hypopharynx.

(c) Low supraclavicular nodes are frequently associated with primary tumors below the clavicles (gastric, ovarial). A fine needle aspirate should be taken to obtain histological diagnosis.

(d) Cervical nodes in the upper and middle deep parajugular cervical regions have better than 90% chance of being metastatic squamous cell carcinoma from the head and neck (larynx, pharynx, paranasal sinuses, thyroid glands, salivary glands, mouth). Examination workup has to include sinus x-ray, oesophagogram and panendoscopy (nasal, oesophageal, pharyngeal, laryngeal and bronchoscopy). If this workup is unproductive, biopsy of the node should be performed. Imaging modalities are CT and MR examinations. **Unknown primaries in the head and neck are best searched by PET scanning.**

(e) Inflammatory lymph nodes (tuberculuos, upper airways viral infections, tonsillitis, adenoiditis) should also be considered in the diagnosis

(f) in the midline, thyroid gland nodules can make the picture more colorful.

(g) Actinomycosis produces very hard infiltrations in the submundibular region, and diagnosis is given only by culture of the pus from this multiloculated honeycomb-like structures. These may be intractable even against high doses of systemic penicillin therapy.

(h) swelling of the submandibular glands due to inflammation or calculosis should be considered

(i) rarely occuring anomaly may be the aneurysm of the carotid artery: diagnosis might be established by its pulsating character, sometimes gives, however, a very obscure picture.

(j) as rare possibility, we have to mention foreign bodies in the neck (e.g. bullets) that can persist in this location for decades.