Chronic middle suppurative otitis. Nonsuppurative pathology of the chronic ear: sensoneural deafness.

Zaporozhye - 2011

The actuality of the theme.

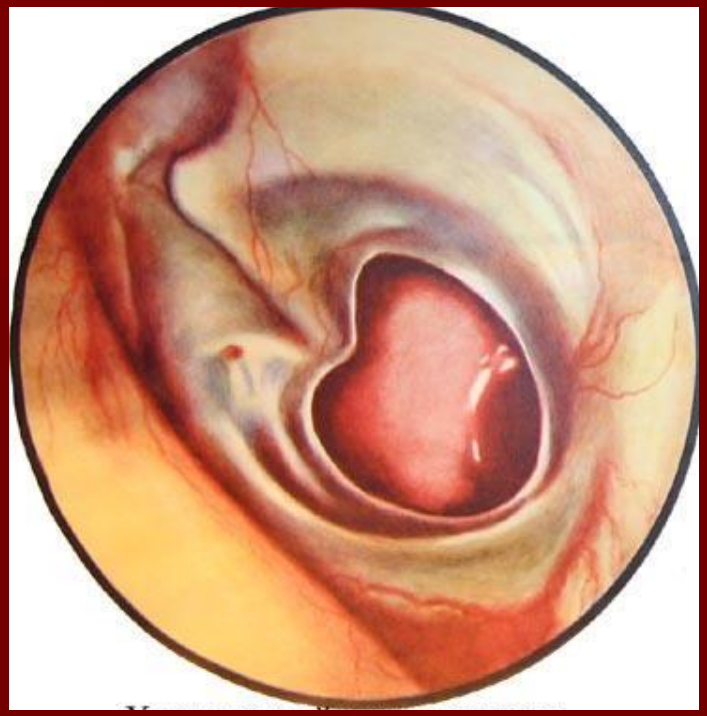
Chronic purulent middle otitis is the most frequent disease of the ear and you can meet it in 20-25 per cents of cases among the all pathology of ENT organs. But unsymptomatically taking chronic otitis, especially epitympanitis, can suddenly causes the hard intracranial complications (meningitis, sepsis, brains abscesses, etc).

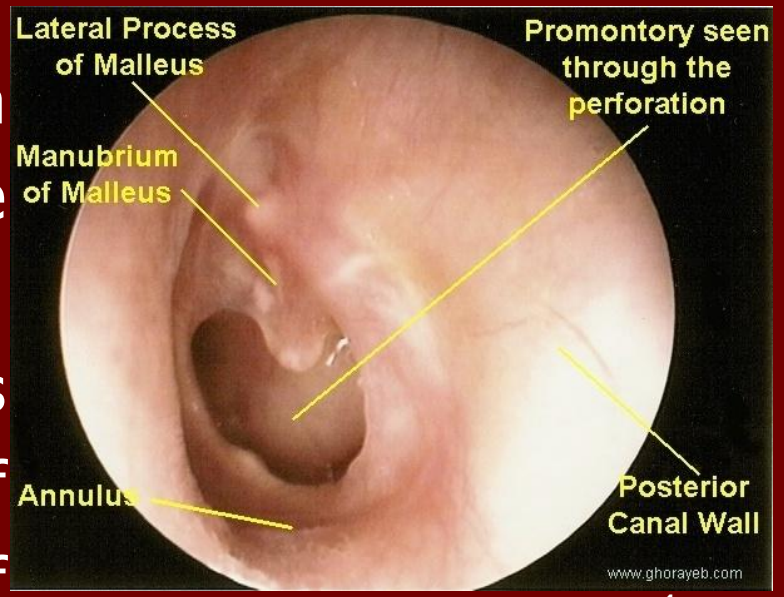
An expressed hardness of a hearing, the unpurulent ear's diseases are in 98 percents of observations. The loss of hearing is accompanied by agonizing noise in the ears and reflected on the human ability to work, his moral condition. A child, who lost in hearing early, usually can't study to speak. When he grows, he becomes deaf mute. All these factors determine a social importance of the problem of unpurulent

Chronic suppurative otitis media is a common disease. Chronic suppurative otitis media is characterized by persistent perforation of the tympanic membrane, periodic or permanent otopyorrhoea, and hearing loss of various degrees.

Aetiology and pathogenesis. The disease is usually secondary to acute suppurative otitis which can persist during several months for various reasons. Among frequent causes of conversion of acute otitis media into the chronic form is a severe acute pathological process in the middle ear, which depends on virulence and the character of infection, decreased resistance of the body associated with chronic specific or non-specific infection. Pathology of the upper airways is also important for the onset of the disease. According to the clinical course and gravity, chronic suppurative otitis media is classified as mesotympanitis and

Mesotympanitis occurs in 55 per cent of cases with chronic suppurative otitis media. The mucosa of the middle and lower portions of the tympanic membrane, and also of the auditory tube are involved in this form of chronic inflammation of the middle ear. Inflammation of the tubal mucosa associated with pathology of the nasal cavity and the nasopharynx. The degree of pathological changes depends mainly on the activity of chronic inflammation, frequency of





Otoscopy in mesotympanitis reveals intact flaccid part of the tympanic membrane and the presence of a perforation in the tense part. The perforation can be round, oblong, bean-shaped; it can vary in size from punctate to an opening occupying almost the whole area of the tense part, a narrow band remaining by the circumference.

Subjective *symptoms* are indistinct. Patients complain of periodical or constant otopyorrhoea and impaired hearing function. In rare cases the patients complain of tinnitus and vertigo. Pain in the ear arises only during exacerbation or due to development of secondary diseases of the ear, such as diffuse otitis extema or circumscribed otitis extema. Discharge from the ear is mucopurulent. The discharge is usually odourless. It can be meagre or profuse (in exacerbation). The

The discharge from the ear can persist for years without causing any serious complications. Otopyorrhoea can stop spontaneously and recur only during exacerbation caused by common cold, water in the ear, respiratory diseases, diseases of the nose, nasopharynx, paranasal sinuses, etc.

Despite the benign course of mesotympanitis, severe intracranial complications can sometimes occur. They can be caused by caries of the promontorial wall, polyps, and granulation.

Diagnosis is based on the anamnestic, clinical, and otoscopic findings (persistent central perforation). Mesotympanitis should be differentiated from epitympanitis. The distinguishing signs of mesotympanitis are persistent central perforation of the tense part of the eardrum, mucous, mucopurulent, or (less

Prognosis is usually favourable, provided a systematic and rational general and local treatment is given. But it is difficult to improve the hearing function, and in this respect the physician should be careful in his prognosis. Hearing improves in most cases after cessation of otopyorrhoea.

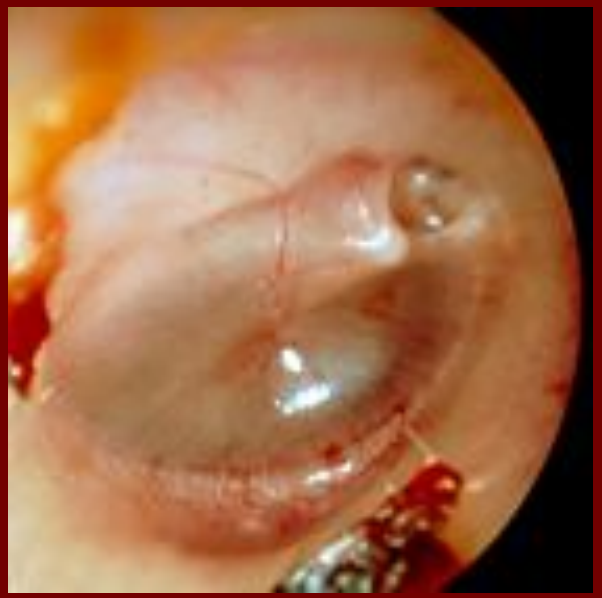
Treatment includes prevention of pus retention in the middle and external ear and action on the microflora and the inflamed mucosa with disinfectants and astringent preparations. Local treatment includes daily irrigation of the ear with the following warm solutions: 3 per cent hydrogen peroxide, and antibiotics, after preliminary testing the microflora for sensitivity to them. In the presence of perforation in the tympanic membrane, endaural administration of medicinal preparations is effective: 1.5-2 ml

If the patient feels the taste of the medicine in the mouth, it indicates that the solution has passed the middle ear and entered the auditory tube.

Local treatment includes also direct instillation of the following solutions: antibiotic solutions, antibiotics should be injected intramuscularly only during exarcerbation.

Minor surgical operations are sometimes necessary: treatment of small granulations or polyps with trichloroacetic acid, a 40 per cent silver nitrate solution;; removal of large granulations using a conchotome, or a curet; and removal of polyps using an aural snare. Physiotherapy is also necessary. It includes UV-therapy and UHF on the ear in the absence of polyps or granulation. General envigorating measures are recommended: rational nutrition, hardening of the body,

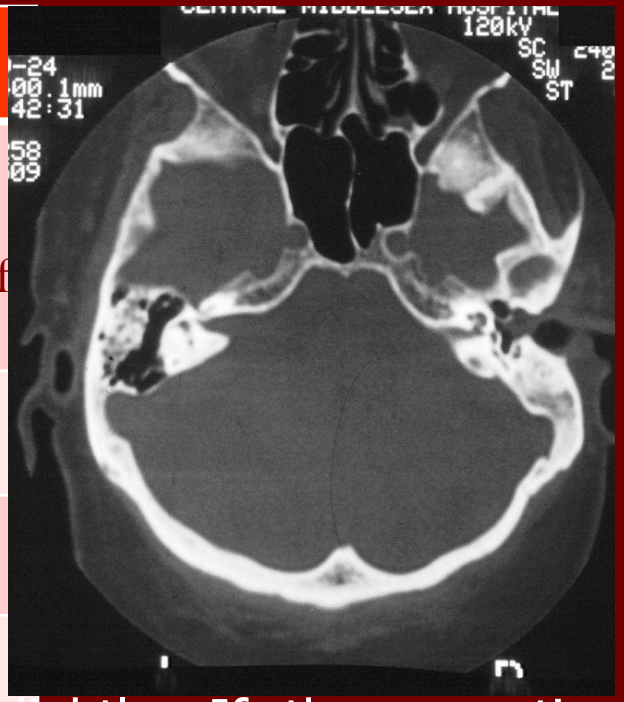
Epitympanitis (atticitis). The inflammation is mainly localized in the epitympanum, the attic of the tympanum. A perforation is usually present in the lateral wall of the epitympanum. Atticitis is characterized by affection of the mucosa and the bony tissue of the middle ear walls and the mastoid process. Caries or cholesteatoma can destroy the wall of the middle ear thus causing a severe intracranial or general complication. The main otoscopic sign of the pathology is persistent marginal perforation in the upper (flaccid) portion of the tympanic membrane. If the process is destructive,



A sample of cholesteatoma or pus can be extracted from the attic on the tip of the probe. Probing detects the presence of granulation (and determines its location) and can also reveal the presence of labyrinthine fistula.

Cholesteatomatous epitympanitis. Cholesteatoma causes vast destruction in the temporal bone. Cholesteatomatous masses can sometimes be seen during otoscopy through a perforation in the tympanic membrane. Cholesteatoma increases in size gradually and constantly due to desquamation of the epidermis, fills in the attic and the antrum and then destroys the bone. As a result the cholesteatoma can reach the meninges, destroy the bony capsule of the labyrinth, the wall of the canal for the facial nerve, almost the entire mastoid process, and thus expose the cerebellar meninges and the wall of the sigmoid sinus

Sings	Mesotympanitis	Epitympanititis
Path morphologic changes.	Inflamation of mucous membrane of tympanic cavity.	Inflamation of mucous, caries of ossicous formations of middle ear.
Excretions from the ear	Serous – mucous	Purulent with putrid smell
Localization of perforation.	Central	Marginal
Test with probe	Negative	Positive



The hearing function often decreases only slightly. If the neurotic component joins, hearing can be impaired significantly at later stages of the process.

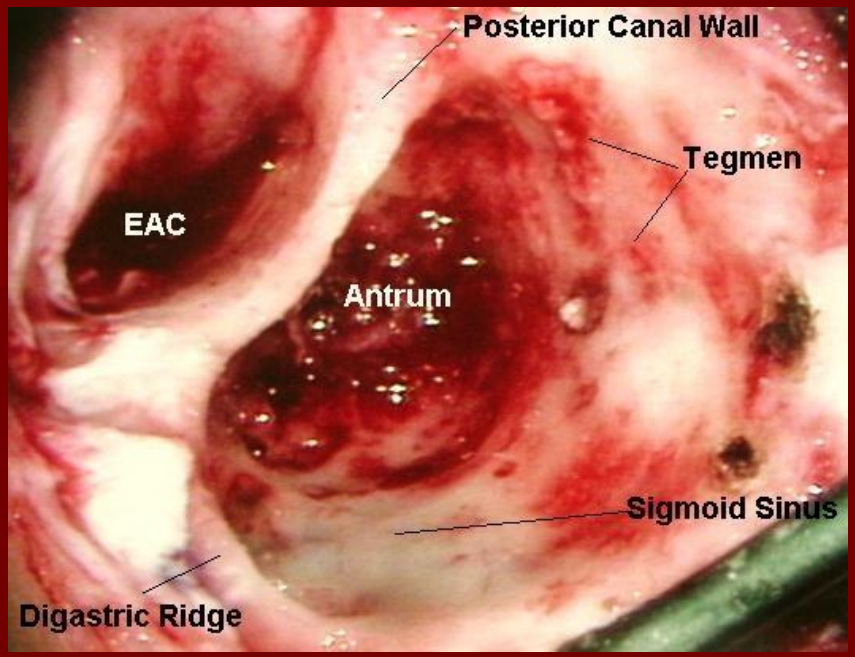
X-ray examination of the temporal bone (Schuller, Mayer, Stenvers position) is a valuable *diagnostic* method. The X-ray picture of the attico-antral region reveals a distinct defect of the bone in the form of structureless clear cavity surrounded by a thin opacity (the wall of the cavity). The X-ray picture of a caries-affected bone reveals indistinct

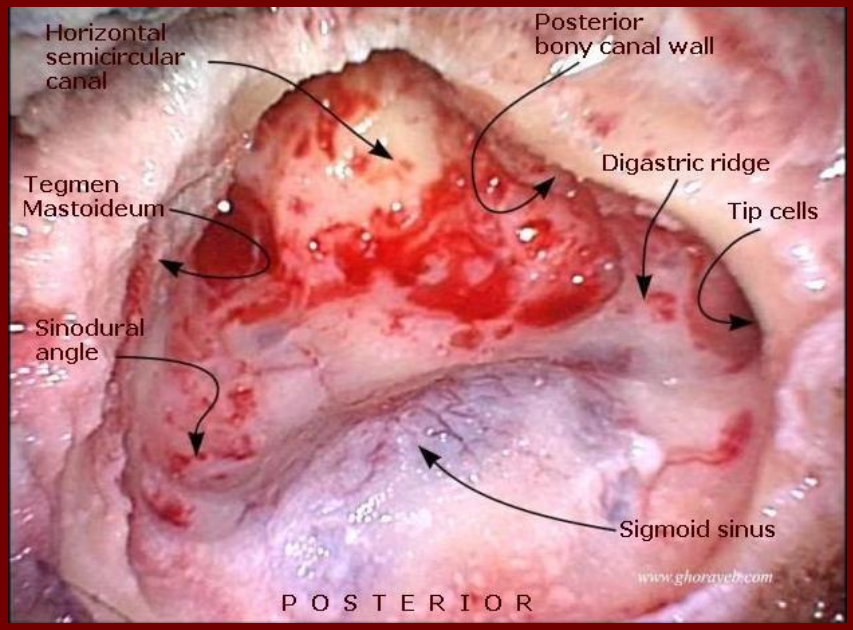
Sings.	Sympoms.
Obligatory. Caries of the walls of tympanic caviity and acoustic bones.	Purulent excretions with annoying smell; Progressive hardness of hearing; Gradual increasing of perforation.
Growth of granulative tissue in the region of attic.	Purulent – blood excretions. Formation of granulations, polypi.
Development of cholesteatoma.	Pus with stinking smell, containing scales of epitelium. There is frequent tendency to lowering of hearing.
Destroying of labyrinth wall of tympanic cavity with formation of labyrinthus fistula and paresis of facial nerve.	Giddiness, headache in the morning, unsteadiness of the step, nystagmus, positive pressor test, lagophtalmos, smooth of nasolabial fold, prolapsus of mouth angle.

Treatment of chronic suppurative epitympanitis is more difficult than of chronic suppurative mesotympanitis. Conservative treatment is effective in cases with anterior epitympanitis. Local treatment includes daily irrigation of the attic by attic needle with the following warm solutions: …. Conservative treatment is usually ineffective in cases with the medial and posterior location of the marginal perforation in the superior parts of the tympanic membrane. A surgical intervention is necessary in such cases.

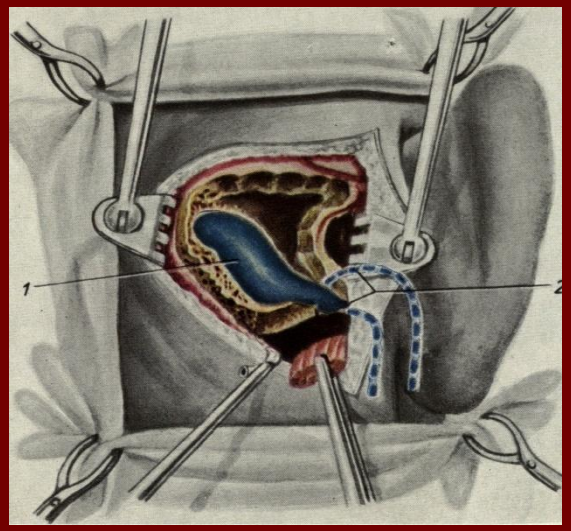
The *radical operation.* The radical operation essentially consists in the tympanic cavity, the epitympanic recess, the antrum with the remaining mastoid cells and the external auditory meatus being thrown into one wide cavity. A thorough removal of carious bone and the cholesteatoma will

The operation begins with opening the antrum, as in mastoidectomy; next follows the removal of the upper section of the posterior bony wall of the external auditory meatus and the external wall of the attic. Here, in the depth of the operative cavity, great care must be taken to avoid injury to the facial nerve, as the descending knee of the facial nerve canal is located in the depth of the posterior bony wall of the auditory meatus. The concluding stage of the operation is removal of all necrotic auditory ossicles apart from the stapes. Polyps, granulations and

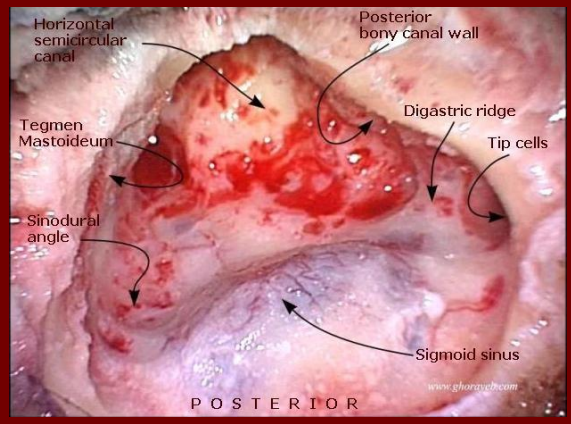




The operation is rounded off with a plastic repair in order that the walls of the operative cavity may later be overgrown with epidermis. For this purpose one or two flaps are cut out of the skin of the posterior wall and roof of the external auditory meatus and are transplanted on to the lower or upper parts of the wound.

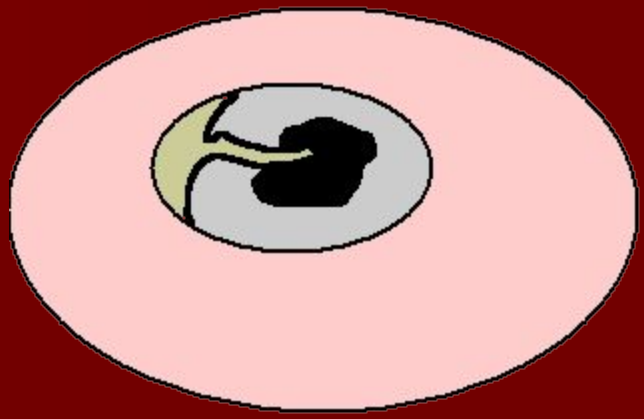


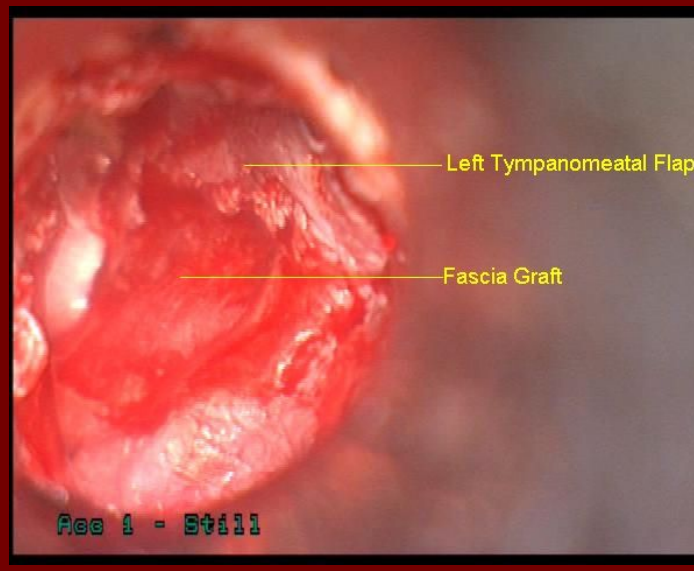
The operation area is packed with a tampon soaked in iodoform or antibiotic solution. Dry dressing is first applied on the sixth to eighth day following the operation, provided there is no fever or pain in the wound. The postoperative treatment is



Tympanoplasty is the surgical reconstruction and building of new sound conduction apparatus. It is an operation to eradicate disease in the middle ear and to reconstruct hearing mechanism. Type of middle ear reconstruction depends on the damage present in the ear. Transformation should be improved in (a) dry perforation of the tympanic membrane; (b) disruption of the ossicular chain; (c) dry adhesive process in the tympanic cavity. The procedure may be limited only to repair of tympanic membrane (myringoplasty), or to reconstruction of ossicular chain (ossiculoplasty), or both (tympanoplasty).

myringoplasty





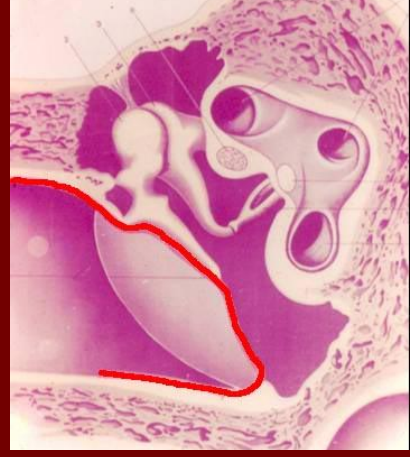
Types of tympanoplasty.

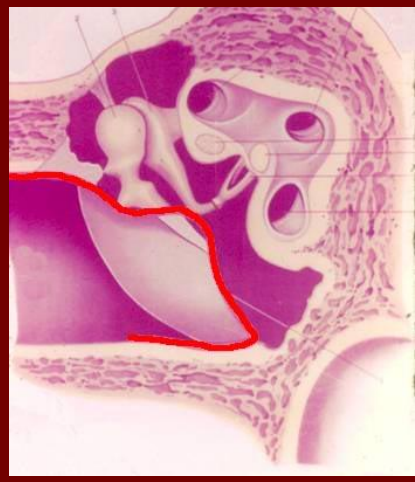
Wullstein classified tympanoplasty into five types.

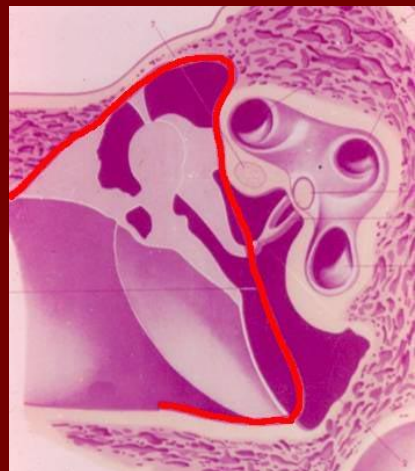
Type I Defect is perforation of tympanic membrane which is repaired with a graft. It is also called myringoplasty.

Type II Defect is perforation of tympanic membrane with erosion of malleus. Graft is palced on the incus or remnant of malleus.

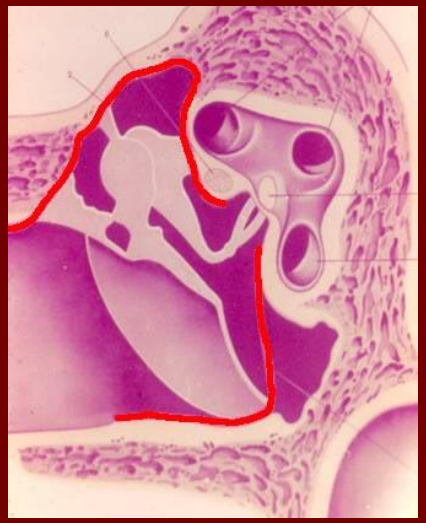
Type III Malleus and incus are absent. Graft is placed directly on the stapes head. It is







Type IV Only footplate of stapes is present. Graft is placed directly on the footplate and round window separated; sound waves in this case act directly on the footplate.



Myringoplasty. It is repair of tympanic membrane. Graft materials of choice are temporalis fascia or the perichondrium taken from the patient.

VESTIBULOCOCHLEAR (COCHLEAR) NEURITIS.

Neuritis of the vestibulocochlear (auditory) nerve is a collective term implying affection of any part of the auditory apparatus, beginning with the neuroepithelial cells of the spiral organ to the transverse temporal (Heschl's) gyri. It may be present at birth (congenital) or start later in life (delayed onset or acquired). Common causes of acquired SNHL include :

1. Infections of labyrinth. Most common causes of the disease are infectious diseases such as influenza, measles.

2. Trauma to labyrinth or VIII-th nerve, e.g. fractures of temporal bone.

3. Noise induced hearing loss (acoustic, vibrational.

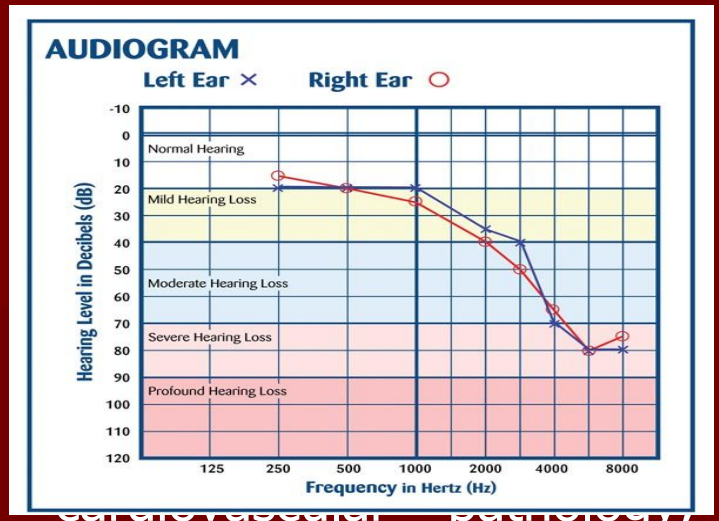
4. Ototoxic drugs or industrial poisoning.Degenerative changes in the cells of the organ of hearing prevail in toxic

4. Presbycusis.

6. Acoustic neuroma.

7. Sudden hearing loss (vessel etiology).

8. Familial progressive SNHL.



9. Systemic disorders, e.g. diabetes, [illegible] pathology), hypothyroidism, kidney disease, autoimmune disorders, multiple sclerosis, as.

A. INFLAMMATIONS OF LABYRINTH

1. Viral labyrinthitis**.** Viruses usually reach the inner ear by blood stream affecting stria vascularis and then the endolymph and organ of corti. Several other viruses, e.g. rubella, herpes zoster, herpes simplex, influenza and Epstein-Barr are clinically known to cause deafness but direct proof of their invasion of labyrinth is lacking.

2. Bacterial. Bacterial infections reach labyrinth through the middle ear (tympanogenic) or through CSF (meningogenic). Sensorineural deafness following meningitis is a well known clinical entity.

B. FAMILIAL PROGRESSIVE SENSORINEURAL HEARING LOSS

It is a genetic disorder in which there is progressive degeneration of the cochlea startingin late childhood or early adult life. Deafness is bilateral with flat or basin-shaped audiogram but an excellent speech discrimination.

C. OTOTOXICITY

1. Aminoglycoside antibiotics. Streptomycin, gentamicin and tobramycin are primarily vestibulotoxic.They selectively destroy type I hair cells of the crista ampullaris but, administered in

They cause selective destruction of outer hair cells, starting at the basal coil and progressing onto the apex of cochlea.

Symptoms of ototoxicity - hearing loss, tinnitus and/or giddiness, may manifest during the treatment or after completion of treatment (delayed toxicity).

2. Diuretics. Furosemide and ethacrinic acid are called *loop diuretics* as they block transport of sodium and chloride ions in the ascending loop of Henle. They are known to cause oedema and cysticchangesinthe stria vascularis of the cochlear duct. The effect, in most cases, is reversible but permanent damage may occur.

3. Salicylates. Symptoms of salicylate ototoxicity are tinnitus and bilateral sensorineural hearing loss particularly affecting higher frequencies.

Hearing loss due to salicylates is reversible after the drug is discontinued.

4. Quinine. Ototoxic symptoms due to quinine are tinnitus and sensorineural hearing loss, both of which arc reversible. The symptoms generally appear wilh prolonged medication but may occur with smaller doses in those who are susceptible. Congenital deafness and hypoplasia of of cochlea have been reported in children whose mothers received this drug during-the first trimester of pregnancy.

5.Topical ear drops. Topical use of drugs in the middle ear can also cause damage to the cochlea by absorption through oval and round windows. Deafness has occurred with the use of chlorhexidine which was used in the preparation of ear canal before surgery or use of eardrops containing aminoglycoside

D. NOISE TRAUMA. Hearing loss associated with exposure to noise has been well-known in boiler makers, iron- and copper-smiths and artillary men. Lately noise trauma has assumed greater significance because of its being an-occupational hazard, the compensations asked for, and the responsibilities thrust upon the employer and the employee to conserve hearing. Hearing loss caused by excessive noise can be divided into two groups:

Acoustic trauma. Permanent damage to hearing can be caused by a single brief exposure to very intense sound, e.g. an explosion, gunfire or a powerful cracker. Noise level in rifle or a gun fire may reach 140-170 dB SPL. Sudden loud sound may damage outer hair cells, disrupt the organ of Corti and rupture the Reissner's membrane. A severe blast may

F. PRESBYCUSIS. Sensorineural hearing loss associated with physiological aging process in the ear is called presbycusis. It usually manifests at the age of 65 years but may do so early if there is hereditary predispostion, chronic noise exposure or generalised vascular disease. Patients of presbycusis have great difficulty in hearing in the presence of background noise though they may hear well in quiet surroundings. They may complain of speech being heard but not understood. Curtailment of smoking and stimulants like tea and coffee may help to decrease tinnitus.

Symptoms. Vestibulocochlear neuritis is characterized by two main symptoms: permanent noise of varied pitch in the ears due to inflammatory and degenerative process and vascular disorders, and impaired hearing which is characterized

Less frequently the patients complain of permanent or transient buzzing (ringing) noise in the ears (tinnitus). If neuritis further progresses, impaired hearing can turn into complete deafness. Complete deafness is a total loss of auditory sensitivity. A rapidly progressing hearing loss is often attended by symptoms of irritation of the vestibular apparatus; these are, first of all, vomiting, vertigo, and absence of the sense of balance. A spontaneous nystagmus can develop.

Diagnosis. A thoroughly collected anamnesis and also clinical findings are important for diagnosis of vestibulocochlear neuritis. Tuning-fork and audiometric tests are of leading importance in topical diagnosis. Hearing disorders associated with neuritis should be differentiated from perceptive disorders due to brain tumor, haemorrhage into the internal ear, and

Characteristics of sensorinural hearing loss are :

1. A positive Rinne test, i.e. air conduction better than bone conduction.
2. Weber lateralised to better ear.
4. More often involves high frequencies.
5. No gap between air and bone conduction curve on audiometry
6. Loss may exceed 60 dB. 7. Speech discrimination is poor.

Treatment of infectious neuritis should be aimed at elimination and neutralization of causes of the disease. We should prescribe the most rational treatment, which is able to remove the consequences of actions on to the internal ear. All the remedies are effective only in the first few weeks from the beginning of the disease before degenerative changes in the cochlea. That's why patients with acute

A doctor prescribes to these patients a confinement to bed, a limit of salt and a liquid food, sedative remedies and active etiotropic treatment.

1) Complex vitamins In — B1, B6, B12, vitamins A and Є;

2) Cocarboxylase (50—100 Mg I./v. or i/m daily N 10-2

3)Agents which improve microcirculation (Angio protektors and Disagreegants): Trentalum, Cavintonum.

4) Agents which improve conductivity of a nervous tissue (Anticholesterase preparations): galantaminum (0,5 % 1,0 subcutaneously N 10).

5) Antihistamine preparations (Suprastinum, Tavegilum)

6) Anticoagulants. In the first days of treatment use a heparin on 5 000 from i/m 2 times for days, then a dose depends on indicators coagulogrem.

7)Corticosteroids. 60 mg of Prednisolonum a day throughout 2-3 weeks with daily dose depression.

Treatment of toxic neuritis first of all includes prevention of further ingress of toxins into the body and their immediate withdrawal from the body. Diuretics and sudorifics should be given. In cases with acute streptomycin intoxication unithiol should immediately be administered in combination with vitamins B group. Unithiol should be injected intramuscularly or subcutaneously, 1 ml of a 5 per cent solution per 10 kg body weight of the patient.

During the first day unithiol is administered 3-4 times; during the second day, 2-3 times; and during the next seven days, 1-2 times a day.

Prognosis. Fortunately about half the patients of idiopathic sensorineural hearing loss recover spontaneously within 15 days. Chances of recovery are poor after 1 month. Younger

Meniere's disease

This is a non-suppurative disease of the inner ear characterized by the classical triad: (1) attacks of systemic labyrinthine vertigo attended with nausea and vomiting; (2) unilateral hearing loss; (3) noise in the involved ear. The disease was first described by Prosper Meniere, a French physician, in 1861.

Fluctuation of hearing is a leading diagnostic sign of the auditory disorder: the hearing can improve considerably between attacks against the background of a gradually progressing deafness. During the initial stage of the disease, the hearing function can be restored completely thus indicating the absence of organic changes in the vestibulocochlear nerve during this period. Meniere's disease occurs mostly in the young. Its onset is characterized by the noise in the ear which is followed (in a few hours or years) by attacks of systemic vertigo and vegetative disorders. An important point is that the auditory, rather than vestibular, disorders are typical for the onset of the disease. When establishing a diagnosis, it is necessary to take into account the periodicity of attacks, their short duration, good subjective condition of the patient during remission, etc.

The disease should first of all be differentiated from the vascular and vestibular syndrome, arachnoiditis, and tumour of the cerebellopontine

Treatment. The polyaetiological origin of the disease accounts for the multitude of methods of treating it. Surgical methods of treatment have been widely used in the recent decade (the operation for decompression of endolymphatic sac).

Treatment at acute vestibular dysfunction

The patient lays down in a bed in convenient position. Bright light and sharp sounds is not supposed.

2. To feet of the patient the heater lays down, and on a cervicooccipital site Sinapismuses are imposed.

3. Medicamental therapy is referred on reduction of intralabyrinthine pressure and normalisation of a parity of processes of nervous excitation and inhibition: i/v. enter 20 ml of 40 % of a solution of a glucose, 5 ml of 0,5 % of a solution of Novocainum; i/v enter 2 ml of 2,5 % of a solution of Pipolphenum or 1 ml of a solution of aminazine of 1 %; Subcutaneously enter 1 ml of 0,1 % of a solution of atropine (or 2 ml of a solution Platiphilinum) and 1 ml of 10 % of a solution of caffeine.

4. Highly effective method of elimination of an attack of illness Meniere is meathotimpanium novocainic blockade.

Treatment in the period between attack

1. I/v. driply pour 4 % a solution of a hydrocarbonate of sodium of 150-200 ml, on a course of 15 injections.
2. Prescribe Disagreegants and Angioprotectors or preparations, which beter microcirculation
3. Throughout last years in an arsenal of preparations for treatment of illness Meniere the appreciable place was occupied with preparation Betaserc. Thanks to microcirculation improvement it is immediate in blood vessels of a cochlea and modulation of excitation of neurones of medial vestibular kernels. The preparation is prescribed on 16 mg (2 tablets) by 3 times per day throughout 1-2 months

Surgical treatment

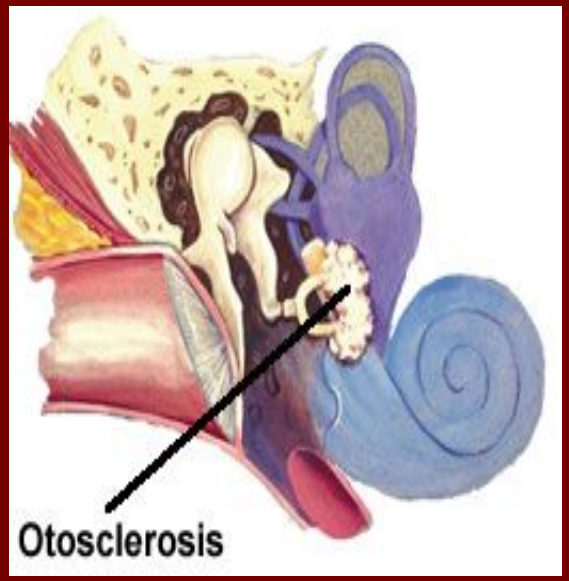
It is used only when medical treatment fails.

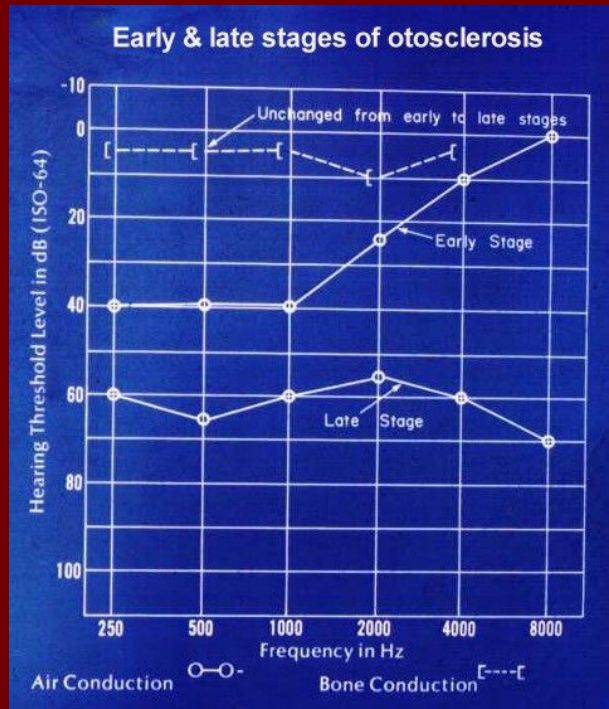
Conservative procedures. They are used in cases when vertigo is disabling hut hearing is still useful and needs to be preserved. They are: *decompression of endolymphatic sac., ultrasonic destruction of vestibular labyrinth.* Cochlear function is preserved. 2. Destructive procedures. They totally destroy cochlear and vestibular function and are thus used only when cochlear function is not serviceable.

Patients with Meniere's disease should abstain from work with moving mechanisms or in conditions of vibration and noise exceeding 70 dB. Work at high altitudes is also prohibited.

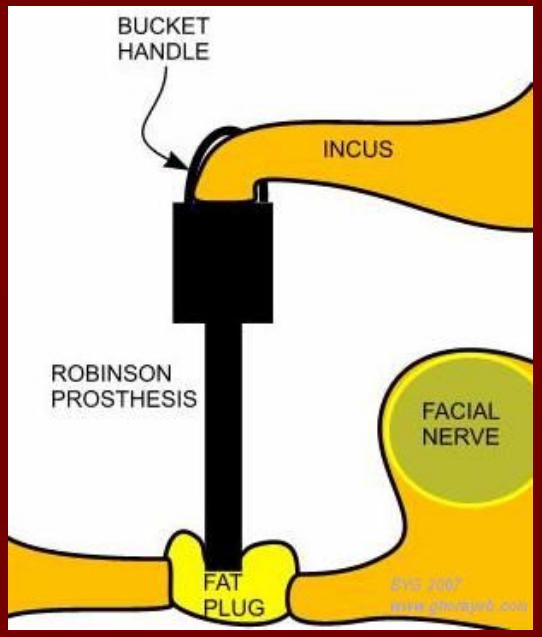
OTOSCLEROSIS Otosclerosis is a frequent cause of deafness (it occurs in more than 0.5 per cent of cases). The morphological substrate of otosclerosis is a circumscribed osteodystrophic process manifested by small single foci of newgrowths in the bony walls of the right and left labyrinths. These foci are relatively symmetric in the bony capsules of the internal ear. They grow to replace gradually the wall of the labyrinthine capsule by a spongioid or dense bone with a different structure. In most cases the otosclerotic focus is localized anteriorly to the oval window; as it grows, the focus extends to the stapedovestibular junction, the anterior limb of the stapes, which impairs mobility of the stapes thus affecting the hearing function and causing noise in the ear. Hearing is first impaired in one ear; then, following months or years, the other ear is involved. This form of otosclerosis is called clinical. If otosclerotic foci are localized outside the windows of the labyrinth, the form is called histological; it can only be

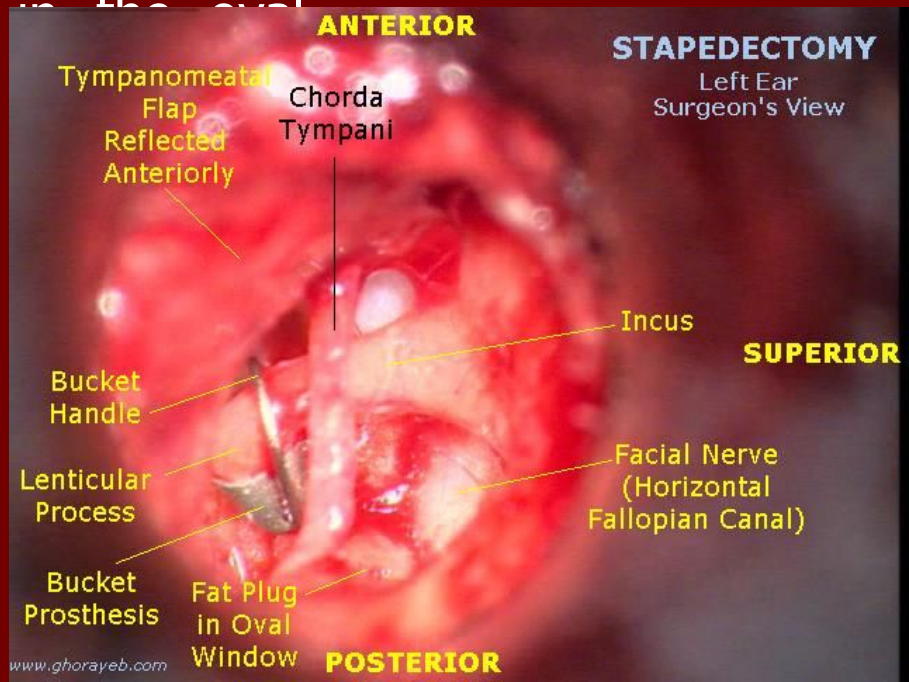
Otosclerosis is usually associated with dystrophic changes in all tissues of the temporal bones. There are tympanic, cochlear and mixed form of this diseases. Otosclerosis occurs mostly in women (in 80-85 per cent of cases). In 70 per cent of cases the disease begins at the age from 20 to 40. Otosclerosis is a hereditary disorder. Various intrinsic and environmental factors can also be important for the onset and the course of the disease. The main audiological sign of otosclerosis is considerably increasing thresholds of air conduction in both ears. Bone conduction thresholds usually increase to a considerably smaller extent.





Treatment of otosclerosis is surgical. It is actually symptomatic because it does not eliminate the pathogenic factors of the disease and only removes to a lesser or greater extent the symptom-deafness and tinnitus. The operation is aimed at reconstruction of the sound transmission system, from the ossicles to the perilymph. The impaired due to the growth of the mobility of the base of the stapes in the oval otosclerotic focus into the annular window is ligament and the base of the stapes (usually at its anterior pole). The following operations aimed at improving the hearing function are now widely used: Stapedoplasty with partial or complete stapedectomy, and





At a late term postoperative period 80 per cent of the operated patients preserve socially adequate hearing, which is an evidence of the high efficacy of surgical treatment of otosclerosis. But operations on the oval window are fraught with great danger to the function of the internal ear, both in the early and late postoperative periods. The operation is therefore performed usually on one ear only. If the hearing function of the operated ear is completely lost, the nonoperated ear can be assisted by a hearing aid.