1

MINISTRY OF HEALTH OF UKRAINE
ODESA NATIONAL MEDICAL UNIVERSITY
Department of OTORHINOLARYNGOLOGY

CONFIRMED by

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METHODICAL AID OF LECTURE ON THE EDUCATIONAL DISCIPLINE

Course IV Faculty Medical

Academic discipline: **Otorhinolaryngology**

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Lecture № 1

Topic: Bioethics in otolaryngology. Acute otitis media. The peculiarities of acute otitis media in children. Kinds of mastoiditis, clinical symptoms, diagnosis, treatment. l otitis. Chronic middle purulent otitis. The contemporary methods of surgical treatment of chronic purulent middle otitis (sanative and hearing restore operations). Labyrinthitis. Otogenic intracranial complications. Otogenic sepsis. Non purulent pathology of the ear: sensoneural hearing loss, otosclerosis , Meniere's disease, chronic catarrh of the middle ear. Deaf-mute and dumb. Rehabilitation of the hearing-impaired.

Actuality of theme. Acute purulent middle otitis is called inflammatory infectious disease of mucous layer of air containing cavities of middle ear.

Today acute middle otitis occurs quite frequently within the population of different age groups and particularly frequent in early child age due to anatomic peculiarities of structure of middle ear in this age, as well as tendency towards infectious diseases, which are complicated by diseases of ear. Therefore doctor of many different specialities come across with contingent of such patients.

Suffered acute otitis may be the reason of stable hardhearing, of development of chronic inflammation of middle ear, threatening intracranial complications. Probability of the latter is related with no diagnosis at right time, as well as with mistakes in treatment tactics of acute purulent middle otitis.

Above mentioned facts form the base of importance of aim of study, placed before students. These knowledge of the topic may be used during study of infectious, paediatric, nervous diseases and in practice of doctor of general profile.

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).

Otogenic intracranial complications is one of the most hard and complicated problems of the modern clinical medicine because of the hardness of its current, difficulty of diagnostics and treatment and very high lethal outcome. That's why the knowing of etiology, pathogenesis, clinics and diagnostics of these complications is necessary for doctors of different types (otolaryngologists, neuropathologists, therapeutists, infectional doctors).

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. Vestibular disorders are hard too. Its lead to a long loss of ability to work and even to disability. All these factors determine a social importance of the problem of unpurulent ear's diseases.

Aim:

Learning aim. To give an idea of the prevalence and social significance of acute and chronic purulent inflammation of the middle ear, the variety of external and internal factors that lead to the development of chronic purulent otitis media; the relationship of ear pathology with diseases of other organs; modern methods of research of the auditory analyzer;

The student should know:

- etiology, pathogenesis, clinic, methods of diagnosis and treatment of acute and chronic purulent inflammation of the middle ear and labyrinth;

The student should be able to:

- to establish the stage of acute purulent otitis media;
- to establish the clinical form of chronic otitis media;
- to evaluate the data of X-ray examination;
- to make a differential diagnosis between labyrinthitis and cerebellar pathology.

Educational purposes. Acute and chronic purulent inflammation of the middle ear is one of the most important moments of modern clinical medicine due to the peculiarities of their course,

difficulties in diagnosis and treatment. From these positions at the lectures, students are brought up an awareness of high professional responsibility. The lecturer controls the psychological readiness of the student to work as a doctor (social significance of the doctor's activities, high morality, social activity).

Basic concepts: acute and chronic purulent otitis media, labyrinthitis, mastoiditis, purulent meningitis, thrombosis of the sigmoid sinus and otogenic sepsis, sensorineural deafness, Meniere's disease

Plan and organizational structure of the lecture

№.	The main stages of lectures and their contents	Time
1	Preparatory stage	3
2	Determination of learning objectives.	2
	Providing of positive motivation	2
3	The main stage. Bioethics in the formation of an otorhinolaryngologist.	7
	Etiology, pathogenesis of acute and chronic purulent inflammation of the middle ear.	6
	Classification and clinic of acute and chronic purulent otitis media (mandatory signs, two forms of the disease, clinical characteristics of mesotympanitis, epitympanitis)	10
	Treatment of patients with acute and chronic purulent otitis media.	10
	Labyrinthitis (acute and chronic, limited and diffuse, serous, purulent and necrotic, their clinic). Treatment.	5
	General characteristics, etiology, pathogenesis of otogenic intracranial complications.	10
	Middle ear catarrh	5
	Sensorineural hearing loss	10
	Otosclerosis	5
	Meniere's disease	5
	The final stage	
4	Lecture summary, general conclusions.	2
5	Lecturer's answers to possible questions	2
6	Self-study tasks for the student	6

Content of the lecture material

Bioethics in the formation of an otorhinolaryngologist

The formation of ethical principles of behavior, a high culture of communication, along with the formation of professional knowledge, is one of the main moral principles of the formation of the personality of a medical student. At the current moment, along with the medical rehabilitation of patients, considerable importance is attached to the socio-psychological, spiritual, ideological, cultural, national and religious aspects of medical interventions. These issues are especially important in a market economy, when the patient has the right to choose his doctor, to whom he is imbued with trust and respect. On the other hand, a doctor should be self-critical of his ability to treat one or another type of pathology. In relation to the "narrow" specialty of otolaryngology, there are also types of specializations such as phoniatrics, ENT Oncology, allergic diseases of ENT organs, microsurgical interventions on the cavity of the nose, larynx, middle and inner ear, pediatric otolaryngology, otoneurology amateurish mastery of which can lead to lack of therapeutic effect, and sometimes disability of the patient. Taking into account his own abilities, the doctor should establish personal contact with the patient and his relatives. Providing truthful information about the state of health requires during a deep assessment of the emotional and psychological state of the patient, especially with cancer, loss of hearing, voice, cosmetic defects of the face and neck. In order to ensure the safety of the patient and the legal protection of the health worker before the mandatory receipt of informed consent, the doctor must carefully explain to the patient how much the level of improvement in the

patient's condition exceeds the risk of complications. The final decision must be made by the patient himself or his guardian.

Bioethics issues should be discussed in clinical studies with students and doctors of postgraduate education, lays the foundation for a humane attitude of specialists towards patients.

Acute Catarrh of the Eustachian Tube. Inflammations of the nasal and nasopharyngeal mucosa, as in acute coryza, influenza and other diseases, are very likely to extend to the mucous membrane of the Eustachian tube which, together with the middle ear cavity, forms a kind of nasopharyngeal diverticulum.

An inflammatory swelling of the tubal walls causes obstruction of air passage to the tympanic cavity in swallowing. Tubal obstruction also occurs in edematous hypertrophies of the posterior ends of the inferior conchae, nasopharyngeal tumours; tubal obstruction is particularly frequent in children with adenoid hyperplasias, as well as in a number of other diseases.

The *symptoms* of obstruction of the Eustachian tube detected by otoscopy may result from changes in the tympanic cavity. Cessation of air supply or inadequate ventilation will result in the tympanic cavity air being partially absorbed by the mucosa at the expense of oxygen, which is followed by an air pressure loss in the middle ear. The disturbance of pressure balance on both sides of the drum will cause the latter's retraction.

The subjective symptoms are loss of hearing acuity, a feeling of fullness in the ear and a crackling sound heard when swallowing; the patient may sometimes imagine he hears the echo of his own voice; this is known as autophony. Where there is transudate in the middle ear, the patient will complain of a sensation of fluid in the ear. Body temperature is usually normal, ear pain is slight or completely absent.

Otoscopical examination of drum retraction reveals that the handle of the malleus takes a more horizontal position and looks shorter in perspective, the short process sticks out sharply, the anterior and posterior folds leading from it have a distinct outline. The light cone changes in form and becomes shorter to appear as a dot or disappear altogether. An acute obstruction of the Eustachian tube is often followed by hyperemia of the mucous membrane ex vacuo and appearance in the tympanic cavity of transudate whose level may sometimes be observed in otoscopy.

Treatment. This consists in the removal of the basic cause of tubal obstruction. Tumours, hypertrophies and adenoid hyperplasias in the nasopharynx are removed by surgery. Acute inflammations of the nasal and nasopharyngeal mucosa are treated with various vasoconstrictive and antiinflammatory remedies.

Cocaine-ephedrine drops are prescribed for instillation into the nose. Simultaneously local heat treatment is given through the application of hot compresses to the ear and its irradiation with a "sollux" lamp. This treatment restores nasal respiration, serves to reduce swelling in the tubal mucosa; hence the transudation in the middle ear resolves. Restoration of the tubal function and normal pressure in the tympanic cavity is helped by inflations which are best used as soon as the acute inflammation in the nose has subsided.

Acute inflammation of the middle ear

Acute inflammation of the middle ear is quite common. Acute otitis media involves not only the tympanic cavity but also the other parts of the middle ear, such as the auditory tube, the antrum, and the cells of the mastoid process.

The direct cause of acute otitis media is infection of the middle ear with streptococci, staphylococci, pneumococci, and less frequently other microbes; mixed flora is sometimes responsible for the onset of the disease. Acute otitis is often secondary. It can be a complication or a manifestation of a systemic infection, for example, infection of the upper airways and influenza; scarlet fever, measles, diphtheria and some other diseases provoke acute otitis media in children. It can be due to acute and chronic inflammation of the pharynx and the nose. The main pathological factor is mechanical compression of the pharyngeal orifice of the auditory tube and impairment of its ventilating and draining functions. Among such diseases are hypertrophies rhinitis, adenoids, choanal polyp, hypertrophies pharyngitis, polyps of the nose,

tumors of the pharynx. Less frequently otitis is secondary to injuries to the ear.

In addition to the mentioned pathological factors, the leading role in the etiology of this disease belongs to the decreased local and general reaction of the body often associated with general viral and microbial infections.

Infection usually enters the middle ear through the auditory tube. Less frequently infection gets into the middle ear through an injured tympanic membrane or through the damaged mastoid process. In rare cases infection penetrates into the middle ear by haematogenic routes (in infectious diseases).

Three periods are distinguished in a typical course of acute suppurative otitis media. The first period is characterized by the onset and development of inflammation in the middle ear, infiltration and exudation, and development of minor symptoms, such as hearing loss, noise, earache, hyperemia of the tympanic membrane, protrusion of the membrane due to the thrust of the exudate, and some general symptoms such as elevation of body temperature to 38-39 °C, deranged appetite and sleep, indisposition.

The second period is perforation of the tympanic membrane and discharge of pus. All reactions subside. Otopyorrhoea lasts 4-7 days. Perforation of the tympanic membrane sharply changes the course of acute otitis: earache subsides and disappears, temperature normalizes quickly, palpation of the mastoid process becomes less painful, and the general condition of the patient improves.

Inflammation subsides in the third period. Purulent discharge discontinues, perforation closes, and the anatomical and functional condition of the middle ear is restored.

The first period of acute otitis media can sometimes be very grave and attended with hyperpyrexia, severe headache, vomiting, vertigo, and drastic impairment of the general condition, painful palpation of the mastoid process. Changes in the blood of patients with otitis during the first days of the disease are characterized by high leukocyte count with a considerable shift to the left. After perforation of the tympanic membrane and discharge of pus, the blood picture gradually normalizes.

If the disease runs a typical benign course, the patient usually recovers with resolution of the inflammation and complete restoration of the hearing function. If the disease runs an atypical course, the outcomes can be different, with adhesions and commissures between the tympanic membrane and the medial wall of the middle ear and impairs hearing (adhesive otitis media); persistent dry perforation (dry perforating otitis media); conversion of acute disease into its chronic form with persistent perforation and periodic otopyorrhoea; complications, such as mastoiditis, petrositis, labyrinthitis, paresis of the facial nerve, intracranial complications, etc.

Dynamics of basic symptoms of AMO in 3 stages of development of process.

Symptoms	I stage (before-perforate)	II stage(perforation or pus flow)	III stage (scaring or healing)
Pain in ear	sharp	insignificant	absent
Noise in ear	moderate	less expressed	absent
Decrease in hearing	sharply	decreased	restores
Excretions	no	serous-blood, mucous-purulent	stops
Changes in tympanic membrane	infiltrated, hyperemised, protruded	perforation, pulsate reflex	tympanic membrane becomes distinct, appear recognising points (signs), at the beginning short process of malleus and at the end - light cone; scars of perforation of tympanic membrane

Temperature of body	high	subfebril	normal

Differentiate symptoms of AMO from external otitis.

Symptoms	AMO	External otitis
Pain in ear	Sharp, pulsate, irradiate; accompanied with head ache, heaviness and pressure in ear	Strong, sometimes irradiate, not accompanied by headache; increases during chewing, movement of jaw
Decrease of hearing	Moderate	Hearing is not changed
Noise in ear	Of sharp intensity	Absent. May arise during sharp infiltration of skin of auditory passage and its felling with pus
Character of excretion in acoustic meatus (auditory passage)	Mucous-purulent, serous; blood.	Purulent
Touching of acoustic meatus and tragus	Painless	Sharply painful
Change in tympanic membrane	Depending upon stage of process	Unchanged

Treatment includes sparing conditions at home or at hospital. The diet should be easily and rich in vitamins to ensure the normal function of the gastro-intestinal tract.

Vasoconstrictors or astringents should be instilled into the nose for restoration or improvement of ventilation and drainage of the auditory tube(naphtyzini, halasolini, sanorini etc.)

In cases of shooting pains and marked redness of the drum, otipax should be used.

If acute otitis media runs a severe course with marked general and local symptoms, antibiotic is injected intramuscularly for at least 5-6 days. It is necessary to remember that streptomycin, gentamycin, kanamycin and monomycin are contraindicated for local and general use because of their toxic effect on the cochlear and vestibular apparatus. The antibiotic therapy should be combined with nystatin and vitamins.

Analgesics and antipyretics should be given for severe headache and pyrexia. Warming compresses should be placed on the mastoid process. Compresses should be prepared as follows: gauze should be folded four or five times and soaked in alcohol diluted with water (1:1). The compress should be changed at 4-5-hour intervals. A UV-lamp is recommended for warming up the ear.

In rare cases, when this treatment fails and severe pain in the ear persists, the body temperature remains high and the tympanic membrane bulges outside, it is necessary to incise the tympanic membrane. Paracentesis is positively indicated for irritation of the middle ear or meningeal irritation which are manifested by vomiting, vertigo, severe headache, and other signs. Paracentesis is more frequently indicated for children because their tympanic membrane is thicker (especially in nursing infants) and it resists rupture stronger than in adults, while the local and general symptoms (pain, pyrexia) are more pronounced.

Paracentesis. The tympanic membrane is incised using a special needle and observing the rules of asepsis. When performing paracentesis in children, not only the head but the whole body must be immobilized. The incision is made on the drum bulge, well-lit, kept under direct observation and carried downwards in the posterior-inferior quadrant of the drum.

Special conditions must be provided for unobstructed drainage of pus from the ear after paracentesis. This can be attained by inserting a special turunda. The external acoustic meatus must be cleaned thoroughly using sterile hygroscopic cotton with 3% hydrogen peroxide. The ear may be suringed once or twice daily under low pressure along the posterior wall of the auditory meatus. After them the medicinal preparations can be administered into the middle ear

through the external acoustic meatus (transtympanic administra-tion).To that end, the mentioned mixture (1 ml) should be instilled into the acoustic meatus and forced into the tympanic cavity by gently pressing the tragus into the external orifice of the acoustic meatus. The medicinal solution can pass the middle ear, the auditory tube, and enter the mouth and nose.

The blowing with balloon of Politcer, catheterisation of the auditory tube facilitates drainage of the middle ear and removes air rarefaction which always attends acute otitis media; blowing is also used to insufflate medicinal preparations. Moreover, this procedure normalizes the function of the auditory tube and has a favorable effect on the course of inflammation. Blowing through a catheter is effective during the third stages of acute otitis media. The procedure should be performed once a day, during 3 or 4 days. A suspension of hydrocortisone mixed with antibiotics should be administered into the middle ear through a catheter.

Prevention includes a combination of measures such as control of infectious diseases, timely treatment of acute and chronic diseases of the nose, paranasal sinuses, and the nasopharynx.

Acute **otitis media in children.** Acute otitis media in neonates and infants occurs much more frequently than in adults. Its course is specific. The special character of the symptoms is determined by the absence of general and local immunity, the morphology of the mucous in the middle ear and the structure of the temporal bone (residues of myxoid tissue, the nutrient medium for infection growth, are present in the tympanic cavity). Inflammation of the middle ear in neonates often develops due to penetration of amniotic fluid into the middle ear through the auditory tube during birth. The infection mechanism in nursing infants is the same, but in addition to infection penetrating from the nose and nasopharynx, food can also pass into the middle ear during regurgitation.

It is more difficult to establish the *diagnosis* of acute otitis media in a nursing infant. But the behavior of a baby with a diseased ear differs substantially from that of a healthy baby. The baby has bouts of inconsolable crying, refuses the breast because of pain during swallowing, rubs his diseased ear against the mother's hand. The main symptoms of the disease are painful palpation of the tragus (because of the absence of the bony part of the acoustic meatus) and high body temperature (39.5-40°C). A baby with otitis media is almost always depressed and sleeps a lot; his gastrointestinal function is upset; vomiting develops and wasting ensues. Meningeal symptoms with dimmed consciousness are possible. As distinct from meningitis, this condition is called meningism and is caused by toxaemia (without inflammation of the meninges). Meningism subsides immediately after perforation of the tympanic membrane and evacuation of pus from the middle ear.

The stages of acute otitis media in a child are the same as in adults, except that the child can more frequently recover without perforation of the tympanic membrane because of its higher resistance, high absorbing power of the mucous in the tympanic cavity and easier drainage of the middle ear through the wider auditory tube.

Treatment of otitis media in a child is the same as in adults, but paracentesis at earlier terms is indicated.

Acute otitis media concurrent with infectious diseases runs an especially severe course in septicotoxic forms of scarlet fever, especially in the presence of necrotic affections of the fauces and changes associated with measles and influenza.

The course of such otitis is especially severe because the patient's immunity is weakened by the pathogenic agent of the infectious disease, which penetrates the ear mostly through the auditory tube and, less frequently, by the haematogenic routes.

Two forms of acute otitis concurrent with infectious diseases are distinguished: (1) late (secondary) otitis arising during the late period of infection, and (2) early otitis developing during the initial stage of the infectious disease and having the same signs as the main disease.

Influenzal otitis occurs usually during viral influenza epidemics. The virus penetrates directly into the ear by the haematogenic route or from the upper airways through the auditory tube. Specific influenzal otitis is characterized by haemorrhagic inflammation which is

manifested by a pronounced dilatation of the vessels in the external acoustic meatus and the middle ear with extravasation (haemorrhage) under the epidermis in the bony part of the external acoustic meatus and the tympanic membrane. Extravasation appears as haemorrhagic blisters (bullae) in the mucous membrane of the middle ear.

Influenzal otitis is localized mainly in the supratympanic space. Its course is often very severe, because inflammation develops in the presence of general toxemia, sometimes with involvement of the internal ear.

Otitis concurrent with scarlet fever and measles usually does not differ substantially from otitis associated with other infections. The necrotic form of otitis deserves mentioning.

Necrotic otitis in scarlet fever and measles usually develops during the initial stage of the disease, more frequently in the presence of necrotic affections of the pharynx and the nose; in measles, otitis develops simultaneously with rash (or before it). The causative agent of this form of otitis is hemolytic streptococcus. Pathology in the ear develops unnoticed in the septicotoxic forms of scarlet fever and measles. Pain is often absent which can be explained by the necrotic affections of the tympanic membrane; the only manifestation of the disease is profuse purulent discharge from the ear (with pungent putrefactive odour if the bone is involved).

Perforation of the tympanic membrane is vast, to complete destruction. Perforation often occurs during the first days of the disease and persists for a long time. Carious process tends to exacerbation.

Necrotic otitis is characterized by a permanent hearing loss (mixed type). Symptoms of labyrinthine affections sometimes join.

Treatment includes measures directed at eradication of the main disease and its local manifestations. Timely and correct use of antibiotics for scarlet fever and measles has reduced significantly the incidence of purulent otitis associated with these diseases. Severe forms of otitis are very rare now.

Acute mastoiditis is a complication of acute otitis media. This is inflammation of the bony tissue of the mastoid process which occurs in malignant course of acute suppurative otitis media. The inflammation easily extends from the tympanic cavity onto the cells of the mastoid process through the entrance to the antrum due to the high virulence of the microbes

Primary mastoiditis occurs in rare cases associated with injury to the mastoid process, tuberculosis, syphilis, actinomycosis and metastasis in general septicaemia.

Incorrect use of antibiotics therapy for acute otitis and also unreasoned abstention from paracentesis, blowing of tube auditive can cause secondary mastoiditis.

Changes in the mastoid process associated with typical mastoiditis vary depending on the stage of the disease. Mucoperiostal (I) and bone-alterative (II) stages of mastoiditis are distinguished.

Symptoms. The clinical signs of mastoiditis can be local and general. The general symptoms are impairment of the patient's general condition, fever, changes in the blood, etc. They do not differ substantially from those of acute suppurative otitis media.

The subjective symptoms are pain, noise in the ears, and hearing loss. Examination of a typical mastoiditis patient reveals hyperaemia and infiltration in the skin overlying the mastoid process (due to periostitis). The pinna is displaced either anteriorly or inferiorly.

The mastoid process, especially the apex, and sometimes its posterior margin, are very tender to palpation. Inflammation in the mastoid process can be activized causing subperiosteal abscess due to passage of pus from the mastoid cells to the periosteum. The differential blood count shifts to the left; the leukocyte count is moderately high; the ESR gradually increases.

The specific otoscopic symptom of mastoiditis is sagging soft tissue of the posterior-superior wall of the bony part of the external acoustic meatus at the tympanic membrane (the anterior wall of the antrum). Otopyorrhoea is often pulsating and profuse. The consistency of pus is often creamy. Pus can fill the acoustic meatus immediately after its cleaning.

Zygomatic abscess. It is due to infection of zygomatic air cells situated at the posterior root of zygoma. Swelling appears in front of and above the pinna. There is associated oedema of

upper eyelid. Pus in these cases collects superficial or deep to temporalis muscle.

The apex- cervical forms of mastoiditis:

Bezold's abscess. It is seen when pus breaks through the tip of mastoid into the sheath of sternomastoid muscle. A swelling is seen in the upper part of neck.

Citelli's abscess. In this case pus breaks through inner table of mastoid tip and travels along posterior belly of digastric muscle. Swelling is seen in the digastric triangle of neck.

Orleansky. Pus spread to the parapharyngeal space through the stylomastoid foramen.

Mure. Pus spreads through the medial plate of the mastoid tip to the retropharyngeal space.

Masked (latent) mastoiditis

It is a condition of slow destruction of mastoid air cells but without the acute signs and symptoms often seen in acute mastoiditis. There is no pain, no discharge,no fever and no mastoid swelling but mastoidectomy may show extensive destruction of air cells with granulation tissue and dark gelatinous material filling the mastoid. It is not surprising to find erosion of the tegmen tympani and sinus plate with an extradural or perisinus abscess.

Aetiology. The condition often results from inadequate antibiotic therapy in terms of dose, frequency and duration of administration.

Clinical features. Patient is often a child, not entirely feeling well, with mild pain behind the ear but with persistent deafness.

Tympanic membrane appears thick with loss of translucency. Slight tenderness may be elicited over the mastoid. Audiometry shows conductive hearing loss of variable degree. X-ray of mastoid will reveal clouding of air cells with loss of cell outline.

***PETROSITIS*.** Spread of infection from middle ear and mastoid to the petrous part of temporal bone is called petrositis.

Like mastoid, petrous bone may also be pneumatised but only in about 30% of individuals. Two groups of air cell tracts lead from mastoid and middle ear to the petrous apex.

Gradenigo's syndrome is the classical presentation and consists of a triad of external rectus palsy(VI th nerve palsy), deep-seated orbital or retro-orbital pain (V th nerve involvement) and perisistent ear discharge.

Persistent ear discharge with or without deep-seated pain inspite of an adequate cortical or modified radical mastoidectomy also points to petrositis.

Fever, headace, vomiting and sometimes neck rigidity may also be associated

Diagnosis. Roentgenography of the temporal bone is very important for diagnosis. An X-ray picture shows diffuse reduction of pneumatization and shaded antrum and the cells. During later stages of the disease the bony septa can be destroyed with formation of clear sites on X-ray pictures (due to destruction of bone and accumulation of pus).

Treatment. Depending on the stage of acute otitis media and mastoiditis. Conservative treatment includes administration of antibiotics and sulpha preparations (locally and intramuscularly). The patient should first be tested for sensitivity to these preparations; their effect on the microflora in the ear should also be tested. Desensitizing preparations and physiotherapy (UHF, SHF, wanning compresses on the ear and the mastoid process) are used. The condition of the nose, the paranasal sinuses and the nasopharynx should be thoroughly examined in each particular case, especially in children.

If conservative treatment fails, objective symptoms intensify, and complications develop in the areas adjacent to the middle ear, surgical intervention is necessary.

Basic differential diagnostic symptoms of AMO and mastoiditis.

Symptoms	AMO	Mastoiditis
General (overall) condition	Improves	Inspite of treatment deteriorates
Pain in ear	After perforation decreases	Inspite of perforation does not decrease

Noise in ear	Gradually decreases	Inspite of treatment does not decrease
Hearing	Improves	Does not improve
Excretion from ear	Stands less, after then disappears. From serous - blood and mucoid-purulent stands mucoid	Purulent; purulent-blood in very big quantities
Palpation of mastoid process	Painless, may be painful during the first days of disease (mastoidal reaction)	Sharply painful
Skin of postauricular region	Unchanged	Infiltrated, swollen mastoid process, smoothness of postauricular fold
Change in tympanic membrane and external acoustic meatus	Correlative to stages	Infiltrated, thickened (mastoidal type); hanging of posterio-superior wall of acoustic meatus
Percussion of mastoid process	Painless	Painful

Differential symptoms of mastoiditis and furuncle of external acoustic meatus.

Symptoms	**Function of external acoustic meatus**	**Acute mastoiditis**
Spontaneous pain	Increase during chewing (mastication)	Does not increase while chewing (mastication)
Pain caused by pressing	Maximum while pressing on tragus	Maximum while pressing on mastoid process
Pain cased by pulling the auricle	Extremely painful	Painless
Condition of external acoustic meatus	Swelling of skin of cartilaginous part	Swelling of bony part (hanging of posterior wall)
Tympanic membrane	Normal	Changed
Hearing	Normal	Decreased
Temperature	Normal or slightly increased	Increased nearly always

The operation on the mastoid process, known as mastoidectomy, is performed under local and sometimes under general anesthesia.

Indication:

1. Acute coalescent mastoiditis.
2. Incompletely resolved acute otitis media with reservoir sign.
3. Masked mastoiditis.
4. As an intial step to perform:

(a) endolymphatic sac surgery

(b) decompression of facial nerve

(c) translabyrinthine or retrolabyrinthine procedures for acoustic neuroma.

Patient lies supine with face turned to one side and the ear to be operated upper most. A curved incision is made behind and following the attachment of the auricle. The incision extends from a point on a level with the upper margin of the pinna to the mastoid tip. In infants and children up to 2 years, the incision is short and more horizontal. This is to avoid cutting facial nerve which is superficial in the lower part of mastoid. Incision cuts through soft tissues up to the periosteum. Temporalis muscle is not cut in the incision. Periosteum is scraped from surface

of mastoid and posterosuperior margin of osseous meatus. Tendinous fibres of sternomastoid are sharply cut and scraped down. The lips of the wound are drawn apart with retractors to keep the mastoid surface open for examination. Should a fistula be darkened and soft portions of bone be discovered, the operation must be started at this place. Should a fistula be absent, the operation must be started in a typical place determined by landmarks. The upper border of the operative area is the temporal line; the anterior border is the spine above the external auditory meatus and the latter's posterior wall. Trephination is begun by attacking the bone right behind the spine on the *planum mastoidenum* to the antrum. In an adult antrum lies 12-15 mm fromthe surface. Horizontal semicircular canal is identified.

All the carious and soft bones should be removed carefully until the antrum has been exposed. The antrum is then widened somewhat with a small curette, and the granulations are thoroughly scraped out with utmost care. Care must be taken in opening the mastoid process to avoid injury to the sigmoid venous sinus, the dura mater, the middle cranial fossa, the facial nerve and the external semicircular canal. Lateral wall of the mastoid tip is removed exposing muscle fibies of posterior belly of digastric. Zygomatic cells situated in the root of zygoma, retrosinus cells lying between sinus plate and cortex behind the sinus are removed.

The operation is usually concluded by filling the wound with antibiotic powder and packing it lightly with tampons. Sometimes mastoid cavity is thoroughly irrigated with saline to remove bone dust and the wound closed in two layers. A rubber drain may be left at the lower end of incision for 24-48 hours in cases of infection or excessive bleeding. A meatal pack should be given to avoid stenosis of ear canal. Mastoid dressing is given.

Antibiotics started pre-operatively are continued post-operatively for at least one week. Culture swab taken from the mastoid during operation may dictate a change in the antibiotic.

Complications:

1. Injury to facial nerve.
2. Dislocation of incus.
3. Injury to horizontal semicircular canal. Patient will have post-operative giddiness and nystagmus.
4. Injury to sigmoid sinus with profuse bleeding.
5. Injury to dura of middle cranial fossa.
6. Post-operative wound infection and wound breakdown.

Prognosis is favourable provided the patient applies to the doctor in due time and is given effective treatment.

Prophylaxis consists in early and rational treatment of acute otitis media.

Mastoiditis (antritis) in children. The mastoid process is underdeveloped in neonates and nursing infants; only a prominence can be found at the place of its future location. There is an antrum in this prominence, into which the purulent process extends from the middle ear. A subperiosteal abscess is likely to develop if the petrosquamous and tympanomastoid fissures are not closed.

The local *symptoms* are few. The otoscopic picture is characterized by indistinct topography of the tympanic membrane; its color can be pink or slightly yellowish. X-ray pictures of temporal bones reveal decreased transparency of the antrum in some cases.

Antritis is always associated with a vigorous general reaction of the child's gastrointestinal tract, the respiratory and nervous systems. The child's conduct varies from flaccidness to excitation; he cries, does not sleep; the symptoms of meningitis are not infrequent. Appetite is very poor, stools are frequent and liquid, and the baby loses his weight. The skin is pale-grey and moist; the heart sounds are dull, the pulse is frequent; tachypnoea develops. The temperature reaction does not always agree with severity of the condition. Body temperature can be normal, subfebrile or be as high as 38-39°C. The blood picture is characterized by neutrophilic leucocytosis; the ESR is accelerated.

Treatment includes local therapy and intramuscular injections of antibiotics. UV-therapy is helpful.

Surgical treatment includes antral puncture, antrotomy, and mastoidotomy (in children after three ears age).

Chronic suppurative otitis media is a common disease. In view of its high incidence and the danger to the hearing function and even to life, it deserves great attention on the part of practicing physicians and nurses.

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, diseases of the blood, rickets, diabetes mellitus and some other diseases. Pathology of the upper airways is also important for the onset of the disease. Inefficient therapy of acute otitis media is among the provoking factors.

According to the clinical course and gravity, chronic suppurative otitis media is classified as mesotympanitis and epitympanitis.

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, upsets the function of the auditory tube which, in turn, becomes the permanent source of infection that affects the mucosa of the middle ear. The degree of pathological changes depends mainly on the activity of chronic inflammation, frequency of exacerbations, the specific properties of the patient's body, and some other reasons.

Otoscopy in mesotympanitis reveals intact flaccid part of the tympanic membrane and the presence of a perforation in the tense part. Perforation varies in location, shape and size. The presence of a permanent central perforation, not reaching the tympanic ring (anulus tympanicus) is characteristic. 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; it can be sangui-purulent in the presence of granulation and polyps. The discharge is usually odourless. It can be meagre or profuse (in exacerbation).

The hearing function is impaired as in affection of the conduction system.

The course of mesotympanitis is usually uneventful. 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 frequently) purulent odourless discharge. The odour indicates involvement of the bone (malignization of the disease).

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, 3 per cent boric acid, furacin (1:5000) and antibiotics, after preliminary testing the microflora for sensitivity to them. In the presence of local signs of allergy (oedema of the mucosa of the tympanic cavity, watery discharge, etc.), a hydrocortisone suspension should be added to the antibiotic solution.

In the presence of perforation in the tympanic membrane, endaural administration of medicinal preparations is effective: 1.5-2 ml of medicinal solution is instilled in the external acoustic meatus and the tragus is then pressed rhythmically by the finger for 10-15 seconds to pump the liquid into the middle ear. 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. A Siegle's speculum or a Politzer's bag can be used for the purpose. The medicine can be administered through a catheter and through the auditory tube.

Local treatment includes also direct instillation of the following solutions: antibiotic solutions, a 1 per cent zinc sulphate solution, a 2-3 per cent protein silver solution, a 0.5 per cent colloid silver, and other 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 (through a cone) and UHF on the ear in the absence of polyps or granulation (with adequate withdrawal of pus). General envigorating measures are recommended: rational nutrition, hardening of the body, climatotherapy, and the like.

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. Perforation is called marginal if bone is a part of the perforation margin.

Inward propagation of caries involves large portions of the temporal bone, the labyrinth capsule included. If the process is destructive, pus has a putrid odour specific for epitympanitis.

A curved end of the probe is passed into the attic through the perforation and the surface of the bony wall is examined. Rough surface indicates caries. 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. These appear like a white tumour (with a pearly lustre) consisting of stratified keratinized epithelium filled with pus, debris, and bacteria. A common cholesteatoma of the ear (secondary cholesteatoma) arises due to extension of the epidermis from the acoustic meatus through the perforation into the middle ear. This epidermis is tightly attached to the bone and is an envelope (matrix) for the cholesteatoma. This should not be mistaken for a true cholesteatoma which occurs in rare cases, when it develops from the embryonal precursors. 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. Purulation of cholesteatomatous mass can extend to the intracranial tissues to cause intracranial pathology.
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 margins of the defect.

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

Pathomorfologic sings of epitympanitis and its symptoms

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 granula tions, polypi.
Development of cholesteatoma.	Pus with stinking smell, containing scales of epite lium. 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: 40% alcohol, 3% alcohol solution of boric acid, 0,25% solution of formaldegide. 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. If chronic inflammation is confined to the attic, atticotomy is performed. Attico-antrotomy is necessary if the process extends to the antrum. Both operations are sparing; the hearing function is preserved. If caries extends to the cavities of the middle ear, the diagnosis is confirmed otoscopically, roentgenographically, and by other instrumental examinations, a radical surgery is necessary. The presence of signs of intracranial complications (sinus thrombosis, meningitis, cerebral abscess and abscess of the cerebellum) is the absolute (vital) indication for a radical operation on the ear in suppurative otitis media. The operation should be performed

immediately. Surgical intervention is also required in the presence of sings of mastoiditis, paresis of the facial nerve, and labyrinthitis. In the other cases the extent of operative intervention should be determined with consideration of the auditory and vestibular functions of the ear.

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. Therefore, this operation is also known as radical mastoidectomy. A thorough removal of carious bone and the cholesteatoma will ensure free pus drainage through the auditory canal and prevent possible intracranial complications.

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 carious bone are carefully removed with a curette. 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 flaps serve as a source of epidermis for the whole trephination field. The skin wound behind the ear is either sutured or left open if the meninges are exposed. 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 rather complicated and normally continues for at least six to eight weeks. In some cases, tympanoplasty is performed if there is no affection of the middle ear together with signs of an intracranial complication. The aim of this operation is not only to remove pathological tissue from the middle ear but to repair the drum. Not infrequently the hearing also improves as a result of this operation.

Chronic suppurative otitis media in children differs in some respects from this disease in adults. The differences are associated with the anatomy of the temporal bone in children and the specific reaction of a child's body to various stimuli. Special attention should be paid to children under 3 years of age, in whom the conversion of acute inflammation into the chronic form is associated with malnutrition (hypotrophy) and exudative diathesis due to hypersensitivity to various stimuli. Immunity is weak at this age.

Chronic suppurative otitis media in children has two forms:

mesotympanitis and the necrotic form. The latter is usually secondary to exudative diathesis. If a cholesteatoma develops in the middle ear, it progresses rapidly to destroy the mastoid process and penetrate into its soft tissues through the cortical layer.

Treatment. In addition to anti-inflammatory treatment of otitis media, correct nutrition is also important. Vitamins A, B, C, D and PP should be given in sufficient quantity. Surgical procedures on the temporal bone of a child must be very sparing and preserving the hearing function.

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. It may be combined with mastoidectomy if disease process so demands. 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).

Contraindications for tympanoplasty are the following: (a) exacerbation of chronic otitis and especially the presence of labyrinthine, intracranial or septicopyemic complications; (b) considerable affection of the sound perception apparatus; (c) obstruction of the auditory tube.

Reconstructive surgery of the ear has been greatly facilitated by development of operating microscope, microsurgical instruments and biocompatible implant materials.

From the physiology of hearing mechanism, following principles can be derived to restore hearing surgically: *An intact tympanic membrane* to provide large hydraulic ratio between tympanic membrane and stapes footplate. *Ossicular chain* to conduct sound from tympanic membrane to oval window. *Two functioning windows,* one on the scala vestibuli (to receive sound vibrations) and the other on the scala tympani (to act as a relief window). If it is only one window, as in stapes fixation or closure of round window, there will be no movement of cochlear fluids resulting in conductive hearing loss. *Acoustic separation of two windows* so that sound does not reach both the windows simultaneously. It can be achieved by providing an intact tympanic membrane, preferential pathway to one window (usually the oval) by providing ossicular chain and by the presence of air in the middle ear. *Functioning eustachlan tube* to provide aeration to the middle ear. A *functioning sensorineural apparatus,* i.e. the cochlea and VIIIth nerve.

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 also called myringostapediopexy or columella tympanoplasty.

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.

Type V Stapes footplate is fixed but round window is functioning. In such cases another window is created on semicircular canal and covered with. a graft. Also called fenestration operation.

Several modifications have appeared in the above classification and they mainly pertain to the types of ossicular reconstruction.

Myringoplasty. It is repair of tympanic membrane. Graft materials of choice are temporalis fascia or the perichondrium taken from the patient. Sometimes homografts such as dura, vein, fascia or cadaver tympanic membrane are also used. Repair can be done by two techniques - the underlay or the overlay. In underlay technique, margins of perforation are freshened and the graft placed medial to perforation supported by gelfoam. In overlay technique, the graft is placed lateral to fibrous layer of the tympanic membrane after carefully removing all squamous epithelium.

Ossicular reconstruction. It is required when there is destruction or fixation of ossicular chain. Most common defect is necrosis of the long process of incus; the malleus and the stapes being normal. In others, there is additionally the loss of stapes superstructure leaving only a mobile footplate and malleus. Yet in others only the footplate is left; all other ossicles, the malleus, incus and stapes superstructure are destroyed.

Repair of ossicular chain can be achieved by the use of autograft incus or cartilage, homograft ossicles, or the prosthetic implants *made of* ceramics or teflon. The techniques commonly employed in ossicular reconstruction in such cases are the incus transposition or a sculptured ossicle.

Most common ossicular fixations are the ankylosis of stapes footplate as in otosclerosis, and the congenital or acquired fixation of head of malleus in the attic.

Ankylosis of stapes can be corrected by removal of the fixed stapes and its replacement by a prosthesis while the attic fixation of malleus head entails removal of the head of malleus and entire incus and then establishing contact between handle of malleus and the stapes.

Labyrinthitis. Inflammation of the internal ear is a very grave complication of suppurative otitis which always involves a severe disturbance of equilibrium, impairment, and

sometimes full loss of hearing in the affected ear. The routes of infection extending from the middle ear into the labyrinth may be through the oval and round windows, as well as through a direct destruction in the labyrinthine bony wall. The latter route is more likely to occur in chronic suppurations in the middle ear complicated by cholesteatoma which gives rise to a fistula in the external semicircular canal. Infectious toxins may also spread into the labyrinth even in case of intact bone and membranes covering the labyrinthine windows. This kind of disease is known as induced labyrinthitis. According to the clinical picture labyrinthitis is divided into the purulent and serous types.

The purulent type occurs more often in chronic suppurative otitis, and sometimes in cases of acute scarlet fever and influenzal otitis. The destructive effect of cholesteatoma *is* a particulary frequent cause of labyrinthitis. Purulent labyrinthitis may occur in a localised (circumscribed) and diffuse form.

Symptoms. Labyrinthitis has very typical signs. The disease begins suddenly with the so-called labyrinthine attack, namely, severe giddiness and disturbance in equilibrium followed by fits of nausea and vomiting recurring frequently during several days. The temperature is usually normal, but if there is a considerable rise during a labyrinthine attack this will suggest an incipient inflammation of the meninges.

The serous forms of labyrinthitis cause a drastic deterioration of hearing and of the vestibular function, and in cases of diffuse purulent labyrinthitis both functions are lost. Circumscribed labyrinthitis, however, is not accompanied by total deafness. The nystagmus arising at the very onset of the disease is at first towards the affected ear, and in case of a full failure of the labyrinthine function it changes to the opposite, unaffected side. If there is no onset of meningitis, all disturbances in equilibrium and other symptoms gradually disappear within three to four weeks. Diffuse purulent labyrinthitis results in permanent loss of hearing.

Serous forms of labyrinthitis are in most cases induced by the effect of toxins on the labyrinth through the intact membranes of the round and oval windows. These forms occur in intense inflammations of the middle ear and sometimes may follow an injury made during a radical mastoidectomy.

The clinical signs of serous labyrinthitis will be the same as in the purulent form, though somewhat milder. Recovery from serous labyrinthitis is followed by a partial restoration of hearing. The most frequent and mild form of labyrinthitis is apparently the so-called *circumscribed labyrinthitis.* In the majority of cases, this form is observed in chronic purulent otitis complicated by cholesteatoma and accompanied by a fistula in the bony capsule of the labyrinth, more often in the area of the external semicircular canal.

The diagnosis of circumscribed labyrinthitis is facilitated by the so-called compression nystagmus which may be evoked in the patient by alternate compression and rarefaction of the air in the auditory canal of the affected ear. When the air is compressed the nystagmus will be towards the affected side, and in case of its rarefaction the nystagmus will reverse in the opposite direction. This kind of nystagmus will indicate a fistula in the external semicircular canal (fistular symptom).

Treatment. If there is no associated intracranial complication, the treatment of labyrinthitis should be conservative. The patient must be strictly confined to bed and kept perfectly quiet. Antibiotic treatment should be continued for two weeks. Dosage depends on the gravity of the disease. If there are indications for an operation on the ear, it should be carried out when severe labyrinthine symptoms have subsided. In the presence of mastoiditis, a suppurated cholesteatoma or intracranial complications the operation is performed immediately. The mode of surgical interference will depend on the symptoms of the existing complications and the pathological changes discovered at operation.

OTOGENIC INTRACRANIAL COMPLICATIONS

Otogenic intracranial complications arise due to extension of the pathological process from the middle and internal ear into the cranium. The complications include epidural (extradural) and subdural abscesses, sinus thrombosis, intracranial abscesses (temporoparietal and cerebellar),

meningitis, and arachnoiditis. Lethal outcome is 24 per cents at otogenic intracranial complications.

Streptococcus and staphylococcus are the main causative agents in the aetiology of otogenic intracranial complications. Sometimes inflammatory intracranial processes occur in acute influenzal otitis media.

Infection spreads from the middle and internal ear into the cranial cavity by (1) contact extension (the most common route); (2) by the vascular route; and (3) by preformed routes. The specific character of these routes explains the possibility of concomitant intracranial complications and inflammation foci on the side opposite to that of primary inflammation.

The infection most frequently spreads from the middle ear to the cranial cavity by the contact route, through the upper walls of the tympanic cavity and the antrum of the mastoid process, which form the floor of the median cranial fossa. The upper wall of the middle ear of neonates has a fissure which closes with age. Infection can also spread through the anterior wall of the middle ear and the medial wall of the auditory tube and extend onto the carotid artery which passes in the vicinity.

The inflammation usually extends from the middle ear onto the meninges, the venous sinuses and the medulla not through healthy bone tissues, but through carious bones. The major part of the labyrinth is adjacent to the tympanic cavity and the inflammation can thus spread from the middle ear to the labyrinth and the canal of the facial nerve, and further, through the internal acoustic meatus, to the posterior cranial fossa.

The bloodborne infection is important for the onset of intracranial complications in acute otitis media. The suppuration focus is as a rule located far away from the primary focus.

Spreading of infection by preformed routes stands the third in the list of incidence. Infection spreads by the bone canals for blood and lymphatic vessels, perivascular space of the internal acoustic meatus, the aqueduct of the cochlea and the aqueduct of the vestibule.

Otogenic **meningitis** is the most frequent complication of acute and chronic otitis media. All cases of otogenic meningitis can be classified as primary (due to infection spreading from the middle ear by various routes) and secondary (due to other intracranial complications, sinus thrombosis, subdural or cerebral abscesses). All otogenic meningites should be considered as suppurative and should be differentiated from irritation of the meninges in other intracranial complications, such as thrombosis of the cerebral sinuses and brain abscess.

Symptoms. General symptoms of infectious disease, meningeal signs and sometimes focal symptoms can be differentiated in the clinical picture. The general symptoms are elevated body temperature, changes in the internal organs (cardiovascular, respiratory, and alimentary systems) and impairment of the general condition. The onset of the disease is usually characterized by elevation of body temperature to 38-40°C. Temperatures curve is usually of constant character. Since meningitis develops during exacerbation of chronic or acute suppurative otitis, is pyrexia usually observed against the background of subfebrile temperature or appears as a repeated elevation of temperature.

Among the meningeal symptoms are headache, vomiting, and disordered consciousness. Headache is attended with nausea in 90 per cent of cases and vomiting occurs in not less than 30 per cent of cases.

Membrane's or meningeal symptoms:

- rigidity of muscles of occiput is expressed in tension of back cervical muscles at the attempt to band the patient's head passively to the front. A chin should touch the check at closed mouth;
- symptom of Kernig - resistace, painfulness at unbending of shank in knee-joint in position lying on the back;
- Brudzinsky's symptom (upper) - bending of legs in coxal and knee-joint in answer on bending of the head to the forward;
- Brudzinsky's symptom (middle) -the same leg's mooving at pressure on puleic joint;
-Brudzinsky's symptom (lower) or contrlateral-unbending of the led in knee-joints, is accompanied by bending of another led.

-Symptom of "suspending"-there is bending of extremities in copal taking him under hands, Bekhterev's sign (pain inside the head or blepharospasm associated with striking on the zygomatic arch with a hammer), hypertension, and photophobia can be vivid on the very first days of the disease, and become even more pronounced in 2 or 3 days. In sharply expressed cases you can't take out the patient's head from horizontal position at his paying position. In more sharp form rigidity leads opisthotonees, i.e. head is always in throwing back position (this symptom is positive at other intracranial complications, especially in back cranial fossa). This is meningitis pose of "gun-dog".

Consciousness then becomes dimmed. Psychomotor excitation can arise which then changes to mental distress and sleepiness.

The blood changes are in all cases characterized by neutrophilic leucocytosis. Leucocyte counts are as high as 33-34 x 10^9 (usually 10-15 x 10^9) per litre with shifts to the left. Single juvenile forms (myelocytes, 1-2 per cent) sometimes appear. Rod (nucleated) cells are from 5 to 30 per cent and the segmented cells are 70-73 per cent. ESR increases from 30-40 to 60 mm per hour. **In** some cases of otogenic meningitis the fundus oculi is not changed.

The cerebrospinal fluid pressure is always high: from 300 to 600 mm of the fluid (normal 150-200 mm). The colour of the cerebrospinal fluid changes from slight opalescence to milky or cloudy greenish-yellow purulent. Cell count varies from 1000 to 30000. Neutrophils prevail in all cases (80-90 per cent). The amount of protein increases from 0.66 to 6.6 g/l and in some cases to 9.6 g/1, but the increase does not always agree with pleocytosis.

Treatment. Treatment of otogenic meningitis includes (1) aetiological; (2) pathogenetic; and (3) symptomatic measures.

Aetiological treatment implies sanation of the focus and antimicrobial therapy. Elimination of the infectious focus (the spreading radical operation on the ear)is an obligatory first measure, irrespective of the gravity of the patient's condition or the extent of changes in the ear. A severe condition is not a contraindication for operation because the purulent focus remains a constant supplier of pathogenic microbes to the subarachnoid space and is thus a source of toxaemia.

Antibacterial therapy begins simultaneously with sanative operation. There are many schemes for treatment of otogenic meningitis with antibiotics (with respect to their combinations, doses, and routes of administration). Permeability of the blood-brain barrier increases 5-6 times in acute meningitis.

Cefalosporines used for endolumbar injections. Aetiological treatment should concur with pathogenetic therapy (dehydration, detoxication, decreasing the permeability of the blood-brain barrier). The intensity and continuation of this therapy depend on the condition of the patient. Mannitol (30-60 g/day in 300 ml of isotonic sodium chloride solution) should be injected as a dehydrating measure. Frusemide (2-4 ml/day) should be injected intravenously, 10 ml of a 25 per cent of magnesium sulphate solution intramuscularly, and 7 ml of glycerol per os are prescribed for the same purpose. Depending on the general condition and the state of the cardiovascular system, the patient can be given symptomatic treatment (cardiac glycosides, tonics, analeptics).

Extradural (epidural) abscess is accumulation of pus between the dura and the cranial bone. It occurs due to extension of inflammation from the mastoid process and the middle ear to the cranial cavity and is localized either in the posterior cranial fossa (the sigmoid groove, Trautmann's triangle) or in the middle cranial fossa. Extradural abscess is a complication of acute otitis media and of exacerbated chronic suppurative otitis. It often concurs with cholesteatoma, pus in the tympanic cavity, destruction of the roof of the tympanic cavity, and suppurative labyrinthitis (if the process is localized in the posterior cranial fossa).

Symptoms are only few and the diagnosis is often established only during operation. The general symptoms are not pronounced.

The main symptom of all intracranial complications is headache. Headache is usually constant in extradural abscess but it is not severe. In perisinuous abscess, pain occurs in the frontal and occipital parts of the head. If the abscess is localized in the middle cranial fossa, pain is felt in the squamous part of the temporal bone. Headache is sometimes attended with nausea,

vomiting, and sleepiness. The general condition can be satisfactory or severe. The meningeal symptoms are sometimes observed.

Treatment is only surgical. Local headache, impairment of the general condition, focal symptoms, and the presence of a purulent process in the ear are indications for the operation.

Subdural abscess is a rare intracranial complication. It occurs as a complication of chronic suppurative otitis media, especially cholesteatoma, and less frequently, of acute otitis media. The abscess is localized in the middle or posterior cranial fossa. Abscess in the posterior cranial fossa usually develops in suppurative labyrinthitis or thrombosis of the sigmoid sinus.

Cerebral abscess. The clinical picture of otogenic cerebral abscess includes three groups of symptoms: general signs of infection, general cerebral symptoms, and signs of local affection of the brain depending on the site of abscess. The course of brain abscess is divided into four stages: initial, latent, apparent, and terminal.

Symptoms differ depending on the stage of the disease. Brain abscess develops through four stages: 1.Stage of invasion (initial encephalitis). 2.Stage of localisation (latent abscess). 3.Stage of enlargement (manifest abscess). 4.Stage of termination (rupture of abscess).

It is very important to assess correctly the general cerebral symptoms such as impairment of the general condition, high temperature, bradycardia, and blood changes.

The initial stage, which lasts 1-2 weeks, is marked by a slight headache, elevated temperature (to 37.5-38°C), nausea, vomiting, and indisposition. This condition often coincides with that of the postoperative period (after a sanative operation on the temporal bone) and is therefore often overlooked. The symptoms of the latent period are few. Flaccidity, paleness, absence of appetite, and regular headaches are possible. The body temperature can be normal and there may be no changes in the blood. Then the apparent stage comes, which sometimes is quite unexpected because of the seemingly satisfactory general condition. The process tends to deteriorate, although the course can be undulant with periods of improvement and impairment. The general condition of the patient during this stage is usually grave. The patient is flaccid, sleepy and indifferent, the skin is pallid (sometimes with grey or yellow hue), the face expresses suffering. Appetite is usually poor, the tongue is dry and coated with a brown fur; constipation is characteristic.

The erythrocyte count and haemoglobin content do not usually change in brain abscess. But moderate hypochromic anaemia with haemoglobin reduced to 600 g/1 and erythrocyte count decreased to 3-3.5 x 10^{12}/l, with signs of hypochromia and poikilocytosis can be seen in patients with non-complicated brain abscess. Various quantitative and less frequently qualitative changes are possible in the differential blood count. Neutrophilic leucocytosis occurs most frequently.

General cerebral symptoms. Headache is a frequent symptom. It can be attended by nausea and vomiting. Vomiting is usually associated with diffuse headache and is an evidence of intra-cranial hypertension.

Meningeal symptoms depend on the localization of the abscess, its closeness to the meninges and the ventricles of the brain, and the presence of perifocal oedema of the brain medulla and the meninges. The meningeal symptoms are indications for a lumbar puncture. The pressure of the cerebrospinal fluid in brain abscess is usually high, but it does not usually exceed 300-350 mm of the fluid. The cerebrospinal fluid in a non-complicated brain abscess is clear and sometimes opalescing; in complicated abscess it is turbid, and if the abscess opens into the subarachnoid space, the fluid contains pus. The cerebrospinal fluid is almost always sterile, even in cases with significant pleocytosis.

The main local symptom of the abscess of the left temporal lobe in right-handed persons is aphasia. In the presence of amnestic aphasia, the patient fails to name an object but can repeat speech. Spontaneous speech is monosyllabic and poor; verbs prevail and nouns are almost completely absent from speech.

Hemianopsia (hemianopia) is a very important symptom of the abscess in the temporal lobe. It indicates involvement of the optic tract which passes through the temporal and occipital lobes.

Focal symptoms of cerebellar abscess. Abscesses of the cerebellum occur much less frequently than those of the temporal lobe. Vertigo is a very valuable diagnostic sign for otogenic abscesses. The most important focal symptoms are muscular hypotonia on the involved side, upset coordination, and spontaneous nystagmus. Upset coordination of the limbs is always obvious on the involved side. This can be manifested by failure to perform finger-nose and heel-knee tests, and adiadochokinesia. Among ophthalmological signs are changes in the visual acuity, optic neuritis, papilloedema, and changes in the field of vision.

Additional methods of examination: echoencephalography, electro-encephalography, lumbar puncture, angiography, pneumo- and ventriculography, and scanning are used in cases where the necessity arises, as indicated by the clinical picture.

Treatment is surgical. It includes vast radical operation on the ear, finding the abscess and its opening. In addition to a common surgical intervention, a vast radical operation includes exposure of the dura and the middle and posterior cranial fossae. Depending on the findings of examination, the temporal lobe of the brain, the sigmoid sinus or the cerebellum are punctured. In cases when the abscess of the brain or cerebellum has been revealed and drained, the prognosis markedly improves. The overwhelming majority of patients recover, but if encephalitis develops around the purulent focus, prognosis is worsened. In addition to the surgical treatment, active antibacterial and anti-inflammatory therapy is also required.

Arachnoiditis of the posterior cranial fossa often complicates chronic suppurative otitis. Arachnoiditis of this location is explained by the special anatomical features which promote infection spreading from the internal ear to the posterior cranial fossa. The onset of arachnoiditis coincides with exacerbation of otitis; the patient's condition is later worsened by intercurrent infections or exacerbated otitis if no sanative operation is performed.

The *clinical picture* is characterized by headache, vertigo (often systemic), forced position of the head, vomiting, and focal symptoms of affection of the cerebellopontine angle. The main symptoms are nystagmus (which develops during the attack), vertigo, and Romberg's sign (uncertain standing and walking). Coordinated movements of the limbs are almost never upset. Slight dysfunction of the trigeminal nerve manifests itself by hypoaesthesia on the face and a weak corneal reflex. Elevated cerebrospinal fluid pressure causes changes in the fundus oculi. The optic papillae are swollen to a various degree during various stages of the disease and in some cases cause secondary atrophy of the optic nerves. The visual acuity is almost always normal. The composition of the cerebrospinal fluid is often characterized by a slight protein-cell dissociation (0.66 g/1); less frequently the composition is hydrocephalic, with decreased protein content (to 0.26-0.099 g/1).

Treatment is surgical and antibacterial. A vast radical operation on the ear is required; courses of antibiotic therapy are periodically repeated depending on the degree to which the symptoms are pronounced. In the presence of a vast cystic process in the posterior cranial fossa, a neurosurgical intervention is required.

Sinus thrombosis and sepsis. As a rule, sinus thrombosis is caused by otitis media, with extension of inflammation to the mastoid process. Caries of the petrous part of the temporal bone, cholesteatoma, and other inflammatory diseases of the bones of the skull can cause sinus thrombosis. Thrombosis of the bulb of the jugular vein is usually secondary to thrombosis of the sinus, but it can also be a primary affection due to infection spreading from the tympanic cavity.

Pathology The pathological process can be divided into following stages:

Formation of perisinus abscess. Abscess forms in relation to outer dural wall of the sinus. Overlying bony dural plate may have been destroyed by coalescent bone erosion or cholesteatoma. Sometimes it remains intact when infection spreads by thrombophlebitic process.

Endophlebitis and mural thrombus formation. Inflammationspreadsto innerwall of the venous sinus with deposition of fibrin, platelets, and blood cells leading to thrombus formation.

Obliteration of sinus lumen and intrasinus abscess. Mural thrombus enlarges to occlude the sinus lumen completely. Organisms may invade the thrombus causing intrasinus abscess which may release infected emboli into blood stream causing septicaemia.

Extension of thrombus. Though central part of thrombus breaks down due to intrasinus abscess, thrombotic process continues. Proximally it may spread to confluence of sinuses and to superior sagittal sinus or cavernous sinus, and distally into mastoid emissary vein, to jugular bulb or jugular vein.

Local symptoms are especially distinct in infected thrombosis. This condition is marked by hyperaemia and oedema of the soft tissues overlying the mastoid process, pain in this region and tenderness to palpation, dilatation of the superficial veins at the posterior edge of the mastoid process. Changes in the cervical vascular bundle occur in some patients. These changes account for the tenderness of the jugular vein to palpation. At later terms, the vein is palpated as a strained string. The lymph nodes along the course of the vascular bundle become enlarged when the thrombosis extends to the bulb or the jugular vein or the vein itself. Griesinger's sign is due to thrombosis of mastoid emissary vein. Oedema appears over the posterior part of mastoid. Levin's symptom is painfulness on the path of internal jugular vein; Toss's symptom - absence of noise at auscultation under the jugular vein; Kvekkenshtededt's symptom : this is to record CSF pressure by manometer and to see the effect of manual compression of one or both jugular veins. Compression of vein on the thrombosed side produces no effect while compression of vein on healthy side produces rapid rise in CSF pressure which will be equal to bilateral compression of jugular veins.

Body temperature is elevated in more than 50 per cent of cases. Elevated body temperature (hectic type of fever) can be attended with a chill and tachycardia, which indicate the onset of sepsis. In very rare cases sinus thrombosis is associated with slow pulse, which is a symptom for differential diagnosis to exclude brain abscess. General cerebral symptoms such as headache, nausea, and vomiting are due to high intracranial pressure caused by obstruction of the cerebrospinal fluid outflow. Intracranial hypertension accounts for the congestive changes in the fundus oculi, which are usually not pronounced.

X-ray pictures of the temporal bone show considerable destructive changes in the mastoid process, destruction of the cells, and often cholesteatoma which is present in at least 50 per cent of patients. Neutrophilic leucocytosis is frequent; the ESR is always high.

Lumbar puncture reveals elevated cerebrospinal fluid pressure, while the composition of the fluid remains unchanged.

Treatment is surgical. Like in other intracranial otogenic complications, the operation should be performed immediately after establishing the diagnosis of chronic or acute inflammation in the ear, and in the presence of the symptoms of sinus thrombosis and sepsis. An infected clot or intrasinus abscess may be present and must be drained. In such cases sinus dura is already destroyed or may appear unhealthy and discoloured with granulations on its surface. Dura is incised and the infected clot and abscess drained. In very rare cases when the symptoms of sepsis persist after operation, the internal jugular vein should be ligated or the transverse sinus opened. Antibiotics should be administered in maximum permissible. Blood cultures should be incubated repeatedly.

Facts of puncture of spinal fluid in normal condition and in different form of meningitis.

Characteris-tic of liquor	Norm	Otogenic purulent meningitis	Epidemic cerebrospinal meningitis	Tuberculous (serous) meningitis	Virus (serous) meningitis
Pressure	100-250 mm	High	High	Increased	Increased
Transparence	It is transpa-rent like water	From opalescence to darkness	The same, may be xanthochrome	Transparent	At influence can be haemorrhage
Cytosis	0-3-5in 1 mkl	Thousands (80-90% of neutrophils)	Increases quiqly. At first it is neutrophil's one, then it is	Hundreds of cells (mainly these are lymphocytes)	200-300 ctlls in 1 mkl (lymphocytes)

			lymph. one.		
Albumen	150-450 mg\l 0,15-0,45 g\l 0,2-0,3 %	increased	In great number	Its maintenance is early increased	There is a small number
Globulin's reaction	negative	Sharply positive	Sharply positive	Always positive	Positive
Chlorides	118-132 mol\l 720-730 mg\l	Lowered or norm	lowered	Small lowering in compare with meningitis of other aetiology	In norm
Sugar	2,5-4,2 mm\l 0,5-0,75g\l	Norm or lowered	lowered	Sharply lowered	Norm or lowered
Bacteriologic research	-	Streptococcus staphylococcus	Meningococcus is sowed in the first days of disease	Through the twenty-four hours the net of fibrin falls out where you can find bacilles Kochii	negative

Differential diagnosis of otogenic and other meningites

Symptoms	Otogenic meningitis	Epidemic cerebrospinal	Tuberculous meningitis	Serous (virus) meningitis
Beginning is	acute	Acute, sudden	gradual	acute
Current is	Quick, hard	Quick, hard	Slow with remission	Quick, but it isn't hard
Presence of centers	Acute or chronic purulent middle otitis, especialy epitympanitis	Frequently there was ARVI early	Tuberculosis of lungs, bones	Appears of measles, typhus, epidemic paro-titis, influensa
Peculiarities of clinical current	Temperature is high, constant. Meningeal symptoms are expressed sharply. In typical cases there are no local symptoms and changes on the eye's bottom	It is met as epidemic flash. Temperature is high and remittent. Meningeal syndrome is sharply expressed at the beginning of the disease. There are herpes on the lips, haemorrhagic rash on the body, sharply expressed leucocytosis.	Usually in youth. Temperature is not very high,but constant. Rigidity of muscles of occiput and Kernig's symptom grow gradually. Headache is little	Clinics is less hard then at other meningites. Neurologic symptoms regress in short period and finish without leaving a trace.

Differential diagnosis of abscess of cerebellum and labyrinthitis.

The sings of the disease	Labyrinthitis	Abscess of cerebellum
Spontaneous nystagmus	Small-sweeping, always horizontaly-rotatory, its both components are clearly distinguished. At labyrinth's irritation it is directed to the injured side and showed during bouts of giddiness, its force shanges and de-pends on position of a head. At tur-ning off labyrinth it is directed to the health side and quickly is exhausted	Big-sweeping, horizontal. Quick and slow components aren't clearly distinguished. Turning off nystagmus is character to the injured side.
Spontaneous miss	Two-sides, always in direction which is opposite to nystagmus. It quickly disappears at turning off labyrinth.	There is on the side of center (there is miss only by ill hand on the injured side). It doesn't depend on nystagmus.
Spontaneous fall	Depends on position of a head (changes direction of the fall) and depends on direction of nystagmus (a patient deflects to the side of the slow component of nystagmus)	To the side of injury of cerebellum. This concurs with direction of nystagmus. It doesn't depend on the head's position. It is observed for a long time.
Deflection at walking	A patient deflects to the side of the slow component of nystagmus.	A patient deflects to the side of cerebellum's abscess.
Flanking gait	It is easily fulfiled to the both sides.	A patient deflects to the side of cerebellum's abscess.
adiadochokinesis	Is absent	Is observed. There is lag of a hand on the injured side.
Giddiness	Is very intensive and becomes stronger at change of head position	Doesn't connect with change of head position
Nausea, vomiting	There are on the height of bout of giddiness, gradually lowers.	Gradually increases
Headache	Is absent	Acute headache in occipital or frontal region
Slowing down of a pulse	Is absent	There is frequently
Hearing	Is lowered or is absent depending on the form of labyrinthitis	Doesn't change
Experimental irritation of labyrinth	There is increased excitability or absence of vestibular excitability	There is normoreflexia of vestibular analizer if abscess is unlabyrinthogenic.
Stagnant optic teat	There is no	Can be.

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 (the organ of Corti) to the transverse temporal (Heschl's) gyri.

The aetiology of affection of the auditory apparatus is quite varied. . 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, viral, bacterial or spirochaetal. Most common causes of the disease are infectious diseases such as influenza, measles, scarlet fever, typhus or malaria.
2.Trauma to labyrinth or VIIIth nerve, e.g. fractures of temporal bone or concussion of labyrinth or ear surgery.
Noise induced hearing loss (acoustic, vibrational, barotrauma)
3.Ototoxic drugs or industrial poisoning. . Degenerative changes in the cells of the organ of hearing prevail in toxic neuritis caused by medicamentous poisoning (streptomycin, monomycin, kanamycin).
4.Presbycusis . 5.Meniere's diease. 6.Acoustic neuroma. 7.Sudden hearing loss (vessel etiology). 8.Familial progressive SNHL. 9. Systemic disorders, e.g. diabetes, cardiovascular pathology, hypothyroidism, kidney disease, autoimmune disorders, multiple sclerosis, blood dyscrasias.

SPECIFIC FORMS OF HEARING LOSS

A. INFLAMMATIONS OF LABYRINTH

It may be viral, bacterial or syphilitic.

1. Viral labyrinthitis**.** Viruses usually reach the inner ear by blood stream affecting stria vascularis and then the endolymph and organ of corti. Measles, mumps and cytomegaloviruses are well documented to cause labyrinthitis. 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). Labyrinthitis as a complication of middle earinfection is discussed on page 102. Sensorineural deafness following meningitis is a well known clinical entity.

3. Syphilitic**.** Sensorineural hearing loss is caused both by congenital and acquired syphilis.

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 large doses, can damage the cochlea also.

Neomycin, kanamycin, amikacin, sisomycin and dihydrostreptomycin are cochleotoxic. They cause selective destruction of outer hair cells, starting at the basal coil and progressing onto the apex of cochlea.

Patients particularly at risk are those

- having impaired renal function,
- elderly people above the age of 65,
- concomitantly receiving other ototoxic drugs,
- who have already received aminoglycoside antibiotics.

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 vascu la ris of the cochlear duct. The effect, in most cases, is reversible but permanent damage may occur.

3. Salicylates. Symptoms ofsalicylate ototoxicity are tinnitus and bilateral sensorineural hearing loss particularly affecting higher frequencies. Site of lesion testing indicates cochlear involvement, but light and electron microscopy have failed to show any morphologic changes in

the hair cells. Possibly they interfere at enzymatic level. 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. Ototoxic effects of quinine are due to vasoconstriction in the small vessels of cochlea and stria vascularis.

*5. Chloroquin***.** Effect is similar to that of quinine and permanent deafness can result.

6.Cytotoxic drugs. Nitrogen mustard and cisplatin can cause cochlear damage. They affect the outer hair cells of cochlea.

*7. Miscellaneous***.** Isolated cases of deafness have been reported with erythromycin, ampicillin and chloramphenicol, indomethacin, phenylbutazone, ibuprofen, tetanus antitoxin, propranolol and propylthiouracil.

Alcohol, tobacco and marijuana also cause damage to the inner ear.

*8. 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 anitbiotics, e.g. neomycin and gentamycin.

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:

*1. 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 concomitantly rupture tympanic membrane and disrupt ossicular chain.

2. Noise induced hearing loss(NIHL). Hearing loss, in this case, follows chronic exposure to less intense sounds than seen in acoustic trauma and is mainly a hazard of noisy occupations.

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. Recruitment phenomenon is positive and all the sounds suddenly become intolerable when volume is raised. Tinnitus is another bothersome problem and in some the only complaint.

Patients of presbycusis can be helped by a hearing aid. They should also have lessons in speech reading through visual cues. 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 by inadequate perception of high-pitch sounds and shortened bone conduction. 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 tumour, haemorrhage into the internal ear, and some other affections. The main differentiating sign of vestibulocochlear neuritis is bilateral deafness or amblya-cousia.

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.
3. Bone conduction reduced on Schwabach and absolute bone conduction tests.
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. Therapeutic measures should therefore be immediately taken. 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 hardness of hearing need in urgent hospitalization. It is necessary to make intensive therapy too. A doctor prescribes to these patients a confinement to bed, a limit of salt and a liquid food, sedative remedies and active etiotropic treatment.

The therapy of infectious neuritis includes mainly measures aimed at elimination of inflammation and eradication of the routes of infection ingress. The bed rest and antibiotics should be administered. Steriod therapy. Pednisone 60 inflammatory and relieve oedema. They have been found useful in SHL of moderate degree. Inhalation of carbogen (5% CO_2 + 95% 0_2*)*. It increases cochlear blood flow and improves oxygenation. Vasodilator drugs. Low molecular weight dextran (hemodes, neohemodes, neogluman etc.). It decreases blood viscosity. It is contraindicated in cardiac failure and bleeding disorders.

Indicated also is stimulation therapy: aloe, 1 ml a day, 25-30 injections and subcutaneous injections of a corpus vitreum preparation, 2 ml, 20 injections for a course. Vitamins C and B are necessary to treat vestibulocochlear neuritis of any aetiology. Intravenous injections of a 20 per cent glucose solution are also effective. Infectious neuritis should also be treated by physical methods. Most effective of them are electrophoresis of a 5 per cent potassium iodide solution on the mastoid process (15 sessions) and d'Arsonvale current. Ringing and buzzing noise (tinnitus) in the ear can be decreased by intracutaneous novocain block (1 per cent novocain solution is injected intracutaneously into the external acoustic meatus in a dose of 0.5 ml, 1-1.5 cm from the entrance to the meatus). The course includes 12 injections.

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.

Rp.: Sol. Unithioli 5%, 5.0

D. t. d. N.10 in amp.

S. Subcutaneous injections of 5 ml 3-4 times a day

Good effect is attained with cocarboxylase, 50 mg daily, during 30 days, in combination with apilac (a tablet for sublingual intake contains 0.01 g of the preparation; the tablets should be taken 3 times a day after meals, for 30 days). The metabolic processes in the nerve tissue can be improved by intramuscular injections of ATP (adenosinetriphosphoric acid) in a dose of 1-2 ml of a 1 per cent solution for a month.

When a lowering of a hearing develops slowly and because of a breach of vascular nutrition of internal ear, doctors usually prescribe a complex of medicines, that consists of a spasmolytic and vascular broadening remedies (sturgeon, cinnarizine), nicotinamide, complamine, no-spa, cavinton, otoneurine); remedies, promoting a dissolution of atherosclerotic congestions (prodectin); remedies, rising a flow of erythrocytar mass through the narrow capillaries (Trental etc.). In some cases there is an effect of a treatment of vertebrobasilar insufficiency, appearing because of cervical osteochondrosis.

In some patients acupuncture is an effective means to reduce (or remove) noise in the ear.

Prognosis. Fortunately about half the patients of idiopathic sensorineural hearing loss recover spontaneously within 15 days. Chances of recovery are poor after 1 month. Severe hearing loss and that associated with vertigo have poor prognosis. Younger patients below 40 and those with moderate losses have better prognosis.

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.

Attacks of vertigo occur amid complete health and are attended by nausea and sometimes vomiting. As a rule, noise in the affected ear intensifies during an attack. The patient feels as if his ear is stuffed or he is deafened. The objective sign of an attack is spontaneous nystagmus which disappears soon after the attack is abated. The patient loses his sense of balance during attacks and tries to assume a horizontal position, often with his eyes closed. Any attempt to change the position impairs the patient's condition and intensifies nausea and vomiting. Attacks can occur at any time of the day, but mostly at night time or in the morning. A physical or psychic overstrain can be the provoking factors. Some patients feel the approaching attack a few hours or even days before the actual onset of the disease. Noise in the ear or slight loss of balance are precursors of the forthcoming attack.

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 angle.

Variants of Meniere's disease

Cochlear hydrops. Here only the cochlear symptoms and signs of Meniere's disease are present. Vertigo is absent. It is only after several years that vertigo will make its appearance.

Vestibular hydrops. Patient gets typical attacks of episodic vertigo while cochlear functions remain normal. It is only with time that a typical picture of Meniere's disease will develop.

Lermoyez syndrome. Here symptoms of Meniere's disease are in reverse order. First there is progressive deterioration of hearing followed by an attack of vertigo, at which time the hearing recovers.

Secondary Meniere's disease

Endolymphatic hydrops with clinical picture resembling Meniere's disease has been observed in congenital or acquired syphilis, otosclerosis, Paget's disease and post-stapedectomy cases.

Treatment. The polyaetiological origin of the disease accounts for the multitude of methods of treating it.

Methods causing reconstruction of the vegetative nervous system are widely used. These are as follows:

(1) reflex action of novocain block (intranasal block, the block of the stellate ganglion and the cervical sympathetic trunk);

(2) vitamin B, PP**,** A, and E therapy;

(3) oxygen therapy and habituation (training with controlled increasing strength of rotation);

(4) exposure of the diencephalon (the centre of the vegetative nervous system) and the sympathetic cervical ganglia to X-rays.

Surgical methods of treatment have been widely used in the recent decade (the operation for decompression of endolymphatic sac).

An acute attack of vertigo is eliminated by subcutaneous injection of 1 ml of a 0.1 per cent atropine sulphate solution, intravenous administration of 10 ml of novocain solution and 10 ml of a 40 per cent glucose solution. If this measure is not sufficient, 1-2 ml of a 2.5 per cent aminazine solution should be injected intramuscularly. If the attack fails to be removed completely, administration of atropine, aminazine and novocain should be repeated in 3-4 hours. If vertigo is severe and the mentioned means prove insufficiently effective, 1 ml of a 1 per cent pantopon solution can be administered subcutaneously.

The presence of arterial hypotension rules out the use of aminazine.

Antihistaminics, chloropyramine, and diphenhydramine hydrochloride are effective both during and after the attack. One of these preparations is administered in a common dose subcutaneously.

The following mixture should be given during 10 days following the attack:

Rp.: Atropini sulfas 0.003

Papaverini hydrochloridum 0.2

Aq. destill. 20.0

S. 15 drops two times a day after meals

It is recommended to carry out a course of intravenous injections of a 5 per cent sodium bicarbonate solution, 50 ml a day, for 15-30 days. Positive effect is attained with dehydration: salt intake should be restricted to 0.5 g a day; ammonium chloride should be taken in 3-day courses (3 g, 3 times a day), 2 or 3 courses at 3-4-day intervals.

Surgical treatment

It is used only when medical treatment fails.

1. 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.

Endolymplwtic shunt operation. A tube is put connecting endolymphatic sac wilh subarachnoid space to drain excess endolymph.

Succulolomy. It is puncturing the saccule with a needle through stapes

footplate. A distended saccule lies close to stapes footplate.

Section of vestibular nerve. The nerve is exposed by middle cranial fossa approach and seieclively sectioned. It controls vertigo hut preserves hearing.

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.

Labyrinthectomy. Membranous labyrinth is completely destroyed either by opening lateral semicircular canal or through the oval windows.

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 detected during histological examination of pathological material. 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.

The Willis paracousis symptom is pathognomonic for otosclerosis : the patient hears much better in noisy surroundings (e. g. in traffic, or airplane). This phenomenon can presumably be explained by mobilization of the stapes with strong low-frequency vibrations and jolting, on the condition that the stapes is only moderately fixed in the oval window. Ultrasound testing (98 000 Hz) of the hearing function is important for differential diagnosis of otosclerosis and cochlear neuritis. In otosclerosis ultrasound is perceived at the same intensity as in health, or the intensity can be increased only slightly, while in cochlear neuritis the sound intensity should be increased two or three times compared with the norm.

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 mobility of the base of the stapes in the oval window is impaired due to the growth of the otosclerotic focus into the annular 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 Stapedoplasty by a piston method.

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 non-operated ear can be assisted by a hearing aid.

Chronic Catarrh of the Middle Ear

Chronic catarrhal otitis media is produced by various morbid processes in the nose and nasopharynx which spread up the Eustachian tube and serve to narrow its lumen thereby obstructing ventilation of the middle ear. Repeated acute catarrhs of the middle ear gradually thicken its mucosa and make the drum less elastic. A long-standing obstruction of the Eustachian tube gradually leads to a noticeable and stubborn retraction of the drum followed by ankylosis of the ossicular chain. Frequently lengthwise and crosswise fibres of scar tissue form between the drum and the walls of the tympanic cavity. This condition is known as chronic or adhesive catarrh. The patient complains of progressive deafness and tinnitus. It often happens that the hearing improves at times, particularly in dry weather, and deteriorates when the weather is damp, and in coryza.

Diagnosis. The diagnosis of this condition rests on examination of the drum and functional examination of hearing. The drum is more or less markedly indrawn, dull and sometimes creamy-white in colour. Sharply outlined white spots of variable form are often observed, which are calcareous deposits in the depth of drum tissue. Scars left by suppurative otitis, as well as atrophic areas of the drum appear to be dark and are often mistaken for drum perforations. In atrophy the drum closely adheres to the internal wall of the middle ear, which sometimes creates the impression of complete absence of the drum.

The extent of changes in the drum has no decisive bearing on the degree of hearing. Therefore, the diagnosis should be p. verified by an assessment of the hearing and in many cases by a trial inflation of the auditory tube. The most typical results will be produced by tuning-fork tests where a nearly normal hearing for high tones produced by a C 2048 tuning fork is accompanied by a severe low-tone loss as evidenced by the use of a C 128 tuning fork. Bone conduction is often lengthened.

Trial inflation of the tube often improves the hearing immediately.

Prognosis. This is favourable if the disease is of short duration, and the hearing has markedly improved after tubal inflation.

Treatment. The first task is to restore the patency of the Eustachian tube, that is, to eliminate the morbid condition in the nose and nasopharynx. Ade-noidectomy is a frequent procedure in such cases, particularly in children, while operations on adults are mostly performed for deformities of the nasal septum, for removal of hypertrophic posterior ends of the lower nasal conchae, etc. Sometimes, these measures alone are sufficient to remove the tubal obstruction and largely restore the hearing. But should elimination of the nasal disease fail to produce a lasting improvement of hearing, tubal inflation will be required.

Tubal inflation is carried out by means of a rubber bulb through an olive-shaped composition tip or an aural catheter. The first procedure is based on the fact that in swallowing and pronouncing some consonants and vowels the soft palate rises and fully closes the entrance to the nasopharynx. By pressing on the bulb at this moment the air in the nasal cavities will be compressed and pushed into both Eustachian tubes. Inflation is performed by introducing a composition tip into one of the nostrils which are pinched simultaneously with fingers of the left hand. The patient is directed to take a little water into his mouth and swallow it at the count of three. At this moment the bulb is compressed, and a blast of air penetrates into the Eustachian tubes with a characteristic noise.

Tubal inflation may also be performed without the use of water. The patient is directed to call out numbers, and at the count of three an air blast from the bulb is blown into the ear. To check whether inflation has been successful an otoscope is used. In cases where a rubber bulb and composition tip are inadequate equipment for inflation to be properly performed, or if unilateral inflation has to be made, the tube will be inflated through a catheter following a short nasal anesthesia, if necessary. An aural catheter is a slender 15 to 17 cm long metal tube curved like a beak at one end funnel-shaped at the other, basal end. At the base of the catheter, there is a small ring set on edge in the same plane as the beak. Prior to use, the catheter should be sterilized in boiling water. After the catheter has been slipped in along the nasal floor down to the nasopharynx with its beak pointing downwards, the latter is turned to the middle, and the

catheter is gently pulled back until the beak has touched the back edge of the vomer. Here, on the lateral wall of the nasopharynx, is the mouth of the Eustachian tube. By turning the beak 180 degrees outwards it is slipped into the mouth of the Eustachian tube. This is followed by inflation . The catheter should be introduced with gentle caution and without any pressure. The beak curvature may be altered, if necessary.

When air is blown through the catheter, characteristic sounds may be heard through the otoscope. These may vary according to the patency of the Eustachian tube and its possible mucous contents. A soft blowing sound indicates a patent tube, louder high-pitched sounds are a sign of tubal obstruction, and, finally, the presence of exudate causes characteristic bubbling sounds. Careless insertion of the catheter may injure the mucous membrane and produce nasal bleeding. The blowing of air into torn tissues may cause emphysema.

In severe atrophy of the drum, inflation should be made with great care and sometimes be abandoned for fear of rupturing the drum.

Inflation may improve the hearing for several hours to a few days. Therefore, repeated inflations have to be made every one, two or three days, sometimes up to 5, 10 and 15 times in all. The nasopharynx is simultaneously painted with 1% silver nitrate solution or 0.25% Lugol's solution. In advanced cases, various kinds of thermic procedures, diathermy and mudtherapy are used to resolve commissures and increase flexibility of the ossicles, which unquestionably aid recovery. A pneumatic massage of the drum can also be used in combination with inflation. If a special apparatus is not available, the massage can be made by means of a pneumatic speculum tightly pressed into the auditory canal and compressed with moderate effort up to 60-100 times a minute to produce alternate suction and pressure on the drum membrane. This will make the drum move in and out and set in motion the entire ossicular chain. In recent time, injections of aloe preparations and skin grafting by Filatov's method have been used with favourable results.

Prophylaxis. The best way to avert middle ear catarrh is to ensure normal nasal breathing. The earliest possible treatment of acute catarrh of the upper respiratory tract and timely management of chronic diseases of the nose and nasopharynx will no doubt serve to keep down the rate of severe deafness.

Prophylaxis of amblyacousia (dull hearing) in pre-school and school children demands the utmost attention. Periodical examination of all children of this age always reveals those who are in need of some treatment. The presence of adenoids severely affects the hearing and their timely removal will undoubtedly prevent hearing loss in quite a number of cases.

Table 1. The most frequent distinctive signs of a cochlear neuritis and a diseases of a middle ear.

Anamnesis	Cochlear neuritis	Exudative middle otitis	Otosclerosis
1.Hereditary diathesis	Sometimes – inborn deafness	Absence	Presense of a hardness of hearing at a near relations
2. The main reason of the disease	Infectional diseases, vascular diseases and intoxications, including by antibiotics	The diseases of the nose, pernasal sinuses; inflammation of auditory tube. Tumors in epipharynx.	Unknown. Disease usually progresses after pregnancy and birth.
3. The peculiarities of disease’s current.	Sudden or gradual lowering of the hearing accompanied	Clicking in the ears at deglutition, the sensation of the noise is	The sensation of the noise in the ears; slow lowering of the

	by sensation of noise and sometimes – giddiness.	unnecessary. The lowering of the hearing has inconstant character.	hearing.
4. The character of noise	High frequency (ringing, whistle).	Mostly absent	Low frequency (A noise of wind, rustle of leaves etc.)

1. An acute or gradual lowering of the hearing, accompanied by the high-frequent noise and sometimes giddiness are character features of cochlear neuritis. Vascular diseases and intoxications are on the base of pathology most frequently. The vascular diseases are: thrombosis and embolism, hemorrhages near internal auditory artery; the intoxications by antibiotics of aminoglycosid's group; the infectional diseases are influence, parotitis, typhus, syphilis.
2. Transient changes of the hearing (frequently on a one side) are character for an excudative middle otitis. There is a clear dependence on a condition of respiratory tractus. There is a change of a hearing at change of a head's position (because of moving of exudate) or relatively strong hardness of a hearing on the last stage of the disease because of formation of commisures in tympanic cavity (adhesive otitis).
3. Otosclerosis is characterized with slowly growth of hardness of hearing, accompanied by a sensation of low-frequency noise. The disease begins gradually, but its push is pregnancy. The hardness of hearing often can be hereditary. An unusual symptom is a character-paracusis Willisii, when a patient hears better in noise.

The research of external patient's condition and a results of otoscopy can give also additional facts for a differential diagnosis (Table 2).

The facts of complains, anamnesis, a results of objective research of a patient allow to suppose a breach of a sound conducting, diseases of a cochlea or a central part of auditory analyser. This question is determined with tuning folks in our patient's clinical picture (*Table 2*)

Table 2.

Tuning fork's tests	Otosclerosis, exudative otitis	Cochlear neuritis

Tuning fork 128 Hz Rinne's experimentation of air and osseous conduction Veber's experimentation of the sound at a position of the sounding tuning fork at the middle of a crown. Shvabach's experiment – tuning fork's position is on the mammiform processes. Zhelle's experiment – degree of perception of a sound through the bone of mammiform processes at a change of the pressure in acoustic duct with the help of Zugle's funnel	Negative. A patient hears better through the bone of mammiform processes then by the air. In the side of injured ear, at a breach of a sound conduction from both sides – in a side of less hearing ear. There is no changes. Variations of pressure in acoustic duct because of stirrup's ankylosis ain't reflect on perception of tuning fork's sound through the bone.	Positive. Perception of tuning fork is low through the air and bone. In a side of a health ear. Perception of tuning fork's sound is shortened through mammiform processes. The tuning fork's sound will be percepted better or less at change of a pressure in acoustic duct.
Tuning fork 512 Hz Federiche's experiment – comparison of perception of tuning fork's sound which is on tragus and bone of mammiform processes	Negative. A patient hears better the sound from a mammiform processes.	Positive. A patient hears better the sound from tragus
Tuning fork 2048 Hz 6.) Perception of tuning fork's sound through the air.	It isn't lowered or lowers unimportanly	It lowers considerably.

Table 3 **Distinctive signs of patient's with otosclerosis, exudative otitis, cochlear neuritis.**

Symptoms	Cochlear neuritis	Exudative otitis	Otosclerosis
1. Color of sclerae	Normal	Normal	Can be blue
2. Amount of ear-wax in acoustic duct	Normal	Normal	Usually there is no ear-wax in acoustic duct
3. A state of tympanic membrane	Normal	Membrane is drawn in; you can see air's bubbles or a level of exudate, which changes depending on head position.	Normal. Sometimes it is partly thin, pink spot appears through (promontory).

The Hearing loss and Deaf

Children with profound or total deafness fail to develop speech and have often been termed as *deaf-mute* or *deaf and dumb*. However these children have no defect in their speech producing apparatus. The main defect is deafness. They never heard speech and therefore do not develop it. Lesser degrees of hearing loss result in defective speech. The period from birth to 5 years of life is critical for the i development of speech and language. Therefore, there is need for early identification and assessment of hearing loss.

Finding the cause

This may require a detailed history of prenatal, perinatal or postnatal causes, family history, physical examination and certain investigations depending on the cause suspected.

Suspicion of hearing loss. Hearing loss is suspected if the child sleeps through loud noises unperturbed or fails to startle to loud sounds, fails to develop speech at 1-2 years. A partially hearing child may have a defective speech and perform poorly in school and be labelled as mentally-retarded. It is essential that all children *at risk* for hearing loss should be screened. Factors which put the child at risk are:

1. Family history of hearing loss.
2. Prenatal infections or use of ototoxic drugs.
3. Birth weight less than 1500 g.
4. Child with stigmata ofsyndromal deafness (deformed pinna, cleft palate, cranio-facial deformities, etc.)
5. Bilirubin level exceeding 20 mg%.
6. Meningitis, especially due to Haemophilus influenzae.
7. Severe asphyxia with seizures or coma in neonatal period.

Testing for hearing loss. Assessment of auditory function in neonates, infants and children demands special techniques. They are grouped under following heads:

Behaviour observation audiometry. Auditory signal presented to an infant produces a change in his behaviour, e.g. alerting, cessation of activity, widening of eyes or facial grimacing. *Moro's reflex* is one of them and consists of sudden movement of limbs and extension of head in response to sound of 80-90 dB. In *cudileo-palpebralreflex,* the child responds by a blink to a loud sound. *Incessation reflex,* an infant stops activity or crying in response to a sound of90dB.

A newborn screening device is the *auditory response cradle* where baby is placed in a cradle and his behaviour responses (trunk and limb movement, head jerk and respiration) in response to auditory stimulation are monitored by transducers. It can screen babies with moderate, severe or profound deafness.

Play audiometry (conditioning techniques). The child is conditioned to perform an act (place marble in a box, block in the bucket, ring on a post, etc.) when he hears a sound. It can be done in free-field or using headphones. It is possible to get a frequency-specific audiogram in children 2-4 years of age.

Visual response audiometry is similar to distraction technique. The child is conditioned to turn his head to the direction of sound which is also reinforced by a light. The head turns are then noted in response to sound stimuli.

Objective audiometry. It includes :

Electrocochleography. It can measure auditory sensitivity to within 20 dB. But it is an invasive procedure.

Auditory brain stem response. It is an electro-physiological test and measures sensitivity in the range of 1000-4000 Hz.

Impedance audiometry. Stapedius muscle contracts reflexly in response to a sound of70-100dB HLand this reflex can be recorded. Elevated intensity levels indicate middle ear or sensorineural hearing loss. Tympanometry can also detect and differentiate causes of conductive deafness.

MANAGEMENT

It is essential to know the degree and type of hearing loss and other associated handicaps such as blindness or mental retardation and whether hearing loss is prelingual (before development of speech) or post-lingual. Aetiology of hearing loss remains obscure in about half the cases.

Aims of habilitation of any hearing-impaired child are to develop speech and language, adjustment in society and useful employment in a vocation.

1. Parental guidance. It is a great emotional shock for parents to learn of their child being deaf. They should be dealt sympathetically so as to accept the child. They should be told of child's disability and how to care for it. Habilitation of the deaf demands a lot from parents : care

and periodic replacements of hearing aids, change of ear moulds as child grows, follow up visits for re-evaluation, education at home and selection of vocation.

2. Hearing aids. Most deaf-children have a small but useful portion of residual hearing which can be exploited by amplification of sound. Hearing aids should be prescribed as early as possible. Ifnecessarybinaural aids, one for each ear, can be used. Hearing aids help to develop lip-reading.

3. Development of speech and language

4. Education of the deaf. There are residential and day schools for the deaf. Some children with moderate hearing loss can be integrated into schools for the normal children with preferential seating in the class.

5. Vocational guidance. The deaf are sincere and good workers. Given the opportunity, commensurate with their ability, they can be usefully employed in several vocations.

Types of hearing aids

Air conduction hearing aid. In this, the amplified sound is transmitted via the ear canal to the tympanic membrane.

Bone conduction hearing aid. Instead of a receiver, it has a bone vibrator which snugly fits on the mastoid and directly stimulates the cochlea. This type of aid is specially useful in persons with actively draining ears, cases with otitis extema or atresia of the ear canal when ear-inserts cannot be worn.

Most of the aids are air conduction type. They can be:

1. Body-worn type. Most common type; microphone and amplifier along with battery are in one case worn at the chest level while receiver is a long distance away at ear level.
2. Behind-the-ear type. Here microphone, amplifier, receiver and battery are all in one unit which is worn behind-the-ear.
3. Spectacle type. It is a modification of the "Behind-the-eartype" and the unit is housed in the auricular part of spectacle frame.
4. In-the-ear type. The entire hearing aid is housed in an ear mould which can be worn in the ear. It is useful for mild to moderate hearing losses with flat configuration. Because of the cosmetic appeal they are very popular.
5. Canal type/This hearing aid is so small that the entire aid can be worn in the ear canal without projecting into the concha.

Indications for hearing aid

Any individual who has a hearing problem that cannot be helped by medical or surgical means is a candidate for hearing aid.

1. Sensorineural hearing loss which interferes with day to day activities of a person. Hearing aid may not suit all such persons because of the intolerable distortion of sound in some.

2. Deaf children should be fitted with hearing aid as early as possible for development of speech and learning. In the severely deaf children binaural aids (one for each ear and individually fitted) are more useful. Training in lip reading is given simultaneouly.

3. Conductive deafness. Most of such persons can be helped by surgery but hearing aid is prescribed when surgery is refused or not feasible or has failed.

COCHLEAR IMPLANTS

Cochlear implants have been developed recently and are still in their developmental phase. They are electronic devices which convert sound signals into electrical impulses which thendirectly stimulate the cochlearnerve. Thus they replace the non-functional transducer system of hair cells of the cochlea.

A cochlear implant consists essentially of three components: (a) a *microphone* which picks up the acoustic signals from the environment ,(b) a *speech processor* which converts sound signals into electrical energy.and (c) an *electrode* which stimulates the cochlear nerve. The speech processor and the electrode are connected together by a wire or through an induction coil system. The electrode which stimulates the nerve is either placed in contact with the promontory

(extracochlear), inside the scala tympani (intracochlear) or in the cochlear nerve (intraneural). Further the electrode may be single channel or multiple channel.

Surgery for implantation. Placement of electrode on the promontory, in the scala tympani or cochlear nerve in the modiolus would require surgery of the ear. Currently the most widely used approach is cortical mastoidectomy with access to the round window and promontory through the facial recess. Electrode is also anchored to the mastoid cortex through a suture to prevent displacement. At some centres (Paris model) radical mastoidectomy is performed to expose the cochlea so that electrodes can be implanted in different coils of the cochlea.

Selection of patients

Cochlear implants are more useful in postlingually deaf patients, i.e. those who lost their hearing after acquisition of language. Congenitally deaf patients have not been benefitted as effectively. Criteria for selection of patients for cochlear implant are:

1. Bilateral deafness with average hearing threshold of 95 dB for speech frequencies of 500,1000 and 2000 Hz.
2. Inability to benefit from a hearing aid. All candidates for cochlear implant must undergo a trial of bearing aid.
3. Sound mental and physical health.
4. Motivation and patience on the part of the patient to undergo subsequent rehabilitation programme.

Current status of implants

Multiple channel implants have been found more useful than single channel implants. Postlingually deaf patients are benefited most. Some will develop the ability to understand speech without lip-reading while others enhance their ability to lip-read because of the useful cues. In the prelingually deaf, some benefit is claimed to adults and teenagers but not to the children.

Materials on the activation of students of higher education during the lecture:

A 5-year-old child suffered acute purulent otitis media about 3 weeks ago, was treated on an outpatient basis, and his condition improved somewhat. Two days ago, the body temperature rose again, abundant pus appeared from the ear, pain in the behind the ear area. During the examination, the bulging of the auricle, tenderness during palpation in the area of the mastoid process, and fluctuation under the soft tissues in this area are noted. During otoscopy, the overhang of the back-upper wall of the bony part of the external auditory canal, the tympanic membrane is crimson in color, is barely visible. Creamy pus in the auditory canal. What is the diagnosis?

A 7-month-old child, after suffering SARS, became restless for two days, cries, turns his head, reaches for his right ear with his hand, refuses the breast. Body temperature 39.20 C. During the examination of the mother, it was established that the child had short-term convulsions, vomiting, diarrhea. During otoscopy, the right tympanic membrane is red, bulging, its contours are not defined, pressure on the tympanic membrane is painful. The stiffness of the muscles of the back of the neck is noted. Make a diagnosis.

The patient, 38 years old, complains of pain in the right ear and behind the ear, purulent discharge from the ear, hearing loss, headache, temperature rise to 37.5 0 C. He has been ill for two weeks. Objectively: the external auditory canal is narrowed in the bony part due to overhanging of the back-upper walls, the discharge is mucous-purulent in nature. The tympanic membrane is red, infiltrated, slit-like perforation in the anteroinferior quadrant with pulsating purulence. Swelling in the area of the nipple, loss of its contours is determined. Soft tissues in the behind-the-ear region are swollen, tense, and painful upon palpation. The auricle is exfoliated. Make a diagnosis.

General material and teaching-methodical support of the lecture: Methodical development of the lecture, multimedia presentation, mock-ups, tables, sets of tools, cameras, sets of radiographs and tomograms, audiograms.

Questions for self-control

1. The incidence of acute and chronic purulent otitis media.
2. Etiology of acute and chronic otitis media.
3. Pathological anatomy of acute and chronic purulent otitis media..
4. List the main forms of acute and chronic purulent otitis media.
5. Conduct differential diagnosis between mesotympanitis and epitympanitis.
6. Possible complications of acute and chronic purulent otitis media..
7. Conservative methods of treatment of acute and chronic purulent otitis media..
8. Surgical methods of treatment of acute and chronic purulent otitis media.
9. Ways of penetration of infection into the cranial cavity in CHS.
10. Clinic of purulent meningitis.
11. Differential diagnosis of different forms of meningitis.
12. Stages of abscesses of the brain and cerebellum.
13. Nest symptoms of brain and cerebellum abscess.
14. Clinic of sinustrombosis and otogenic sepsis.
15. Treatment of intracranial complications
16. Classification of labyrinthites.
17. Clinic and treatment of labyrinthitis.

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7. www.bundesaerztekammer.de – German Medical Association

Lecture № 2

Topic: Acute and chronic rhinosinusitis. Rhinogenic intraorbital and intacranial complications.

Actuality of theme. Diseases of the nasal cavity and paranasal sinuses are a type of pathology most often encountered in the practice of otorhinolaryngologists, as well as specialists in related specialties - ophthalmologists, neuropathologists, pulmonologists.

Anatomical features of the structure of the nasal cavity and paranasal sinuses make it difficult to make a diagnosis using only traditional methods of examination (examination, palpation, research of respiratory function and function of smell; anterior and posterior rhinoscopy, diaphanoscopy). Mastering modern diagnostic methods and studying the clinic of the most common diseases of these organs will significantly reduce the frequency of life-threatening rhinogenic complications.

Diseases of the nose and paranasal sinuses and their complications are frequent pathologies in ENT practice. In addition to otorhinolaryngologists, therapists, pediatricians, neuropathologists and other specialists meet with them. These diseases can lead to changes in other organs and systems of the body. From these positions at the lectures, students are educated in a sense of high professional responsibility. The lecturer controls the psychological readiness of students to work as a doctor (social significance of the doctor's activity, high moral, social activity).

Aim:

Learning aim. To give an idea of the prevalence and social significance of acute and chronic purulent inflammation of the nasal cavity and paranasal sinuses, the variety of external and internal factors that lead to the development of acute and chronic rhinosinusitis; the relationship of nose pathology with diseases of other organs; modern methods of research of the paranasal sinuses.

The student should know:

- etiology, pathogenesis, clinic, methods of diagnosis and treatment of acute and chronic inflammation of the nose and paranasal sinuses;

The student should be able to:

- to establish the stage of acute and chronic rhinosinusitis;
- to establish the clinical form of acute and chronic rhinosinusitis;
- to evaluate the data of X-ray examination;
- to make a differential diagnosis between acute and chronic rhinosinusitis.

Educational purposes. . Pathology of the nose and paranasal sinuses takes one of the first places among the ear, throat, nose diseases which often result in temporary loss of the labor ability. Any physician must know the clinical symptoms methods, treatment of nasal diseases. He must also in time diagnose rhinogenic and internal eye-socket and cranium complications which occasionally lead to death. At the lectures students are brought up an awareness of high professional responsibility. The lecturer controls the psychological readiness of the student to work as a doctor (social significance of the doctor's activities, high morality, social activity).

Basic concepts: Acute and chronic rhinitis, rhinosinusitis, etiology, pathogenesis, diagnosis, treatment. Rhinogenic intraorbital complications. Nose injuries. Epistaxes.

Plan and organizational structure of the lecture

№.	The main stages of lectures and their contents	Time
1	Preparatory stage	3
2	Determination of learning objectives.	4
	Providing of positive motivation	3
3	The main stage. Differential diagnosis rhinitis and rhinosinusitis	10
	Etiology, pathogenesis of acute and chronic purulent inflammation of the nose and paranasal sinuses.	10
	Classification and clinic of acute and chronic rhinosinusitis (mandatory signs, two forms of the disease, clinical characteristics)	15
	Treatment of patients with acute and chronic rhinosinusitis.	20
	General characteristics, etiology, pathogenesis of rhinogenic intraorbital	15

	and intracranial complications. The final stage	
4	Lecture summary, general conclusions.	2
5	Lecturer's answers to possible questions	2
6	Self-study tasks for the student	6

Content of the lecture material

Furuncle, or boil, of the nasal vestibule is an acute inflammation of the hair follicle or sebaceous gland; it often concurs with general furunculosis. The main aetiological factor is an injury, local impairment of resistance of the skin or body to staphylococci and streptococci. Some systemic diseases, e.g. diabetes mellitus, metabolic disorders, hypovitaminosis, tuberculosis, or chilling, can provoke furunculosis of the nose. It is important to foresee a possible formation of septic thrombi in small veins in the inflammatory infiltration that surrounds the hair follicle or the sebaceous gland. The infection can be carried by the venous blood to the cavernous sinus with subsequent severe intracranial complications and sepsis.

The clinical picture is characterized by severe pain in the inflammation focus, circumscribed cone-shaped infiltrate covered with hyperaemic skin, whose top in 3-4 days becomes crowned with a yellowish pustular head, which ripens during the next 4-5 days. The general reaction can be either insignificant or absent altogether. If the course of furunculosis is unfavourable and a carbuncle develops, the body temperature can rise, leucocyte counts increase, and the regional lymph nodes become enlarged and tender.

Treatment includes administration of antibacterial preparations, polyvita-mins, and (locally) quartz lamp and UHF. The skin around the furuncle is treated with boric alcohol. It is absolutely prohibited to express the furuncle contents because of the danger of intracranial complications and sepsis. Ichthyol or balm ointments should be applied locally until the furuncle ripens. The patient should be taken to hospital in view of possible complications such as rhinogenic sepsis or thrombosis of the cavernous sinus.

Deformation of the nasal septum. This occurs usually due to injury. Physiological deformation is usually the result of disbalanced growth of the septum and the bony framework into which the septum is included. Three types of deformities are distinguished: deviated septum, septal spur and shelf. The main complaint of the patient with deviated nasal septum is nasal obstruction. Headache, nasal discharge, occasional pain in the ear, dry throat, etc. also occur in some cases.

Treatment is surgical . Difficult respiration through one or both sides of the nose is indication for operation. Submucous resection of the deviated part of the septum should be carried out.

Acute catarrhal rhinitis (common cold) is an acute non-specific inflammation of the nasal mucosa. The aetiology of acute rhinitis is determined by decreased local or general reactivity of the body and activation of microflora of the nose. The disease usually occurs following general or local chilling that interferes with the protective nervous and reflex mechanisms.

The clinic of acute catarrhal rhinitis includes three stages, which are continuous with one another: the first stage is dry irritation, the second stage is characterized by increased mucous secretion, and the third stage (resolution) is characterized by mucopurulent secretion. Acute rhinitis begins with the feeling of dryness, tension, burning, and itching in the nose and often in the pharynx and the larynx; sneezing is annoying. The patient complains of indisposition, chill, discomfort and headache (mostly pain in the forehead). The body temperature is elevated. Nasal respiration becomes difficult-from insignificant impediment to a complete obstruction due to obturation of the nasal meatuses with swollen mucosa. Olfaction is impaired significantly. The sense of taste is also altered. The speech becomes nasal (rhinolalia clausa). Profuse watery discharge from the nose is characteristic of the first day of acute rhinitis. The amount of mucus in the discharge increases later. This can cause hyperaemia and swelling of the skin at the nose vestibule and of the upper lip. The nasal discharge becomes seropurulent in 4 or 5 days. The

amount of nasal discharge decreases gradually during the next few days, swelling of the mucosa subsides, respiration through the nose and olfaction are restored, and the patient recovers in 8-14 days from the onset of acute catarrhal rhinitis.

Treatment as a rule is given on out-patient basis. If rhinitis is severe and is attended with high temperature, the patient is recommended bed rest at home.The course of acute rhinitis can be aborted by thermal, counter-attractive, and sudorific procedures. Hot bath is recommended for the feet and the lumbar region; hot tea, 0.5 g of acetyisalicylic acid is also recommended. UV-therapy, application of mustard plasters to the calves, UHF, or diathermia are also useful. Before nursing an infant, it is necessary to suck off mucus from each side of the nose using a rubber syringe. Two drops of a vasoconstrictive substance should be instilled into each nostril 5 minutes before breast-feeding. Four drops of a 2 per cent colloid silver solution should be instilled. Adults should be given galasoline, or otrivin, and sanorine at all stages of acute catarrhal rhinitis.

Chronic rhinitis. The main forms of chronic rhinitis are catarrhal, hypertrophic, atrophic, vasomotor and allergic. The disease is common.

Chronic catarrhal rhinitis. The onset of chronic rhinitis is connected as a rule with frequently recurring acute inflammation in the nasal cavity (including inflammations associated with various infections), irritating environmental effects such as dust, gas, dry or moist air, variations in ambient temperature, etc.

The main symptoms of chronic catarrhal rhinitis are impeded respiration through the nose and rhinorrhoea; both signs are manifested moderately. Respiration through the nose becomes periodically difficult, mostly due to chilling. The passageway through one side of the nose is usually obstructed permanently. Nasal respiration is even more difficult when the patient lies on his side

Chronic hypertrophic rhinitis. The main signs of hypertrophic rhinitis are impeded respiration through the nose, mucous nasal discharge, and thickened and swollen nasal mucosa, mainly in the entire inferior and middle concha. The mucosa is usually red-blue, gray-blue and covered with mucus. In the presence of mucopurulent discharge, inflammation of the paranasal sinuses should be excluded. The posterior ends of the inferior conchae are usually thickened; application of vasoconstrictor drops don't causes the reduction of nasal concha.

Chronic atrophic rhinitis. Common chronic atrophic rhinitis can be diffuse or circumscribed. Mineral dust (silicates, cement) and that of tobacco produce a strong effect on the condition of the nose. Common symptoms of the disease are crusts in the nose. Meagre tenacious mucus (or mucopurulent discharge) adheres to the mucosa and dries into crusts. The patient complains of dryness in the nose and the pharynx, and impairment of olfaction. Separation of the crusts often causes nosebleed, usually from the Kiesselbach area.

Treatment of chronic rhinitis. Treatment of various forms of chronic rhinitis includes the following: elimination of possible factors which cause and maintain rhinitis; specific medicamentous therapy of each particular form of rhinitis; surgical management for special indications; physiotherapy and climatic treatment.

Astringent substances are used for chronic catarrhal rhinitis. These are a 3-5 per cent protein silver or colloid silver solution and a 3-5 per cent silver nitrate solution. If the mucosa is swollen, it can be treated with an iodine-glycerol solution. The treatment with the mentioned preparations should not continue for more than 10 days. Physiotherapy is also recommended: UHF or microwaves on the nose and UV-therapy endonasally. Courses of instillation of peloidin, inhalations of balms should be alternated. If hypertrophy is insignificant, sparing surgical interventions are recommended: ultrasound disintegration,cauterization with chemical substances (silver nitrate, trichloroacetic acid, chromic acid), electric current, or extreme cold. If hypertrophy is significant and respiration through the nose is impeded, partial resection of the hypertrophied parts of the conchae (conchotomy) is recommended .

Treatment of atrophic rhinitis. The patient should take care of his nose so that crusts and nasal discharge should not accumulate in the nasal cavity. The nose should be cleaned once or twice a day by irrigating the nasal cavity with isotonic sodium chloride solution containing an

additive of iodine (6-8 drops of a 5 per cent iodine tincture per 200 ml of the solution). Irritants should periodically be used: the mucosa should be treated with an iodine-glycerol solution once a day in the course of 10 days, this stimulates the secretion of the glands in the nasal mucosa. A 30 per cent potassium iodide solution (8 drops 3 times a day, for 2-3 weeks) should be given per os for the same purpose.

Ozaena is a pronounced atrophy of the nasal mucosa and the nasal bones marked by formation of fetid crusts which produce a firm layer on the nasal mucosa. Metaplasia of the columnar ciliated epithelium into squamous epithelium associated with ozaena is characteristic for the major part of the nasal mucosa. It mainly occurs in women and begins in the young, its cause is unknown. The disease persists during the whole life. Ozaena patients complain of marked dryness in the nose, intensive crusting, and fetor. The respiration through the nose is impeded. Olfaction is lost completely. *Diagnosis* is established by the fetid odour from the nose, the presence of many crusts and atrophy of the nasal mucosa and bony walls of the nose.

Allergic and vasomotor rhinitis.

The aetiology of the ***allergic*** form depends basically on the allergen. Allergic rhinitis can be seasonal or permanent (non-seasonal). Seasonal allergic rhinitis recurs regularly at the same time of the year, when the specific plant is in blossom. Permanent (non-seasonal) rhinitis is caused by many various substances (allergens) with which the patient often comes in contact, e.g. house dust, fur of domestic animals, pillow feathers, book dust, some foods, various microflora.

Vasomotor rhinitis occurs due to disordered nervous mechanisms accounting for the normal physiology of the nose. Sympathetic stimulation causes vasoconstriction and shrinkage of mucosa, while parasympathetic stimulation causes vasodilation and engorgement. The long application of the vasoconstrictor drops, the deformation of the nasal septum may also cause this disease.

The main symptom of both forms of rhinitis is paroxysmal sneezing attended by nasal hydrorrhoea and difficult nasal breathing. This triad of symptoms is more or less pronounced in all cases. The rhinoscopic signs of rhinitis are oedema and pallor of the mucosa, and cyanotic or white spots on it.

The allergic form of the disease is characterized by increased eosinophil counts and appearance of eosinophils in the nasal mucus.

Treatment depends on the findings of the allergological examination and includes elimination from the patient's environment of allergens, purulent foci or microbial allergy. Treatment includes specific and non-specific hyposensitization of the patient, local procedures, including surgery and action on the nervous system.

Specific hyposensitization is conducted in conditions of an allergological laboratory because severe allergic reactions are possible following administration of the allergens. The identified allergen should be highly diluted and administered to the patient in gradually increasing microdoses (subcutaneously or into the nose, on the mucosa, regularly during the course of several weeks). The body can thus produce protective antibodies to the allergen.

Non-specific desensitization is used in both allergic and vasomotor forms of rhinitis. Antihistaminics (suprastine, tavegyle, diazolyn, klaritin) and hormones (hydrocortisone, prednisolone, prednisone) are used for the purpose. Topical steroids such as beclomethasone, dipropionate and flunisolide acetate used as aerosols are very effective in the control of symptoms. Topical steroids have fewer systemic side effects but their continuous use beyond 3 weeks is not recommended. Sodium chromoglycate stabilises the mast cells and prevents them from degranulation despite the formation of IgE antigen complex. It is used as 4% solution for nasal drops or aerosol powder. It is useful both in seasonal and perennial allergic rhinitis.

Preparations of calcium, sulphur, and vitamins are also helpful. Local methods of treatment, including endonasal novocain block, submucous administration of corticosteroids, cauterization of the reflexogenic zones of the nasal mucosa with strong acids, silver nitrate, intranasal physiotherapy, sclerotherapy are used for treatment of both forms of rhinitis.

Electrophoresis of various medicinal solutions is the most common method of physiotherapy for rhinitis. Endonasal electro-phoresis with a 2 per cent calcium chloride solution is used most frequently. Long-standing vasomotor rhinitis often increases the volume of the conchae and imposes permanent difficulties in nasal breathing. Surgical treatment (sparing inferior conchotomy, submucous destruction of the inferior conchae with ultrasound) is most rational in such cases.

Inflammatory diseases of paranasal sinuses

Acute and chronic inflammatory diseases of the paranasal sinuses are frequent. They make 25-30 per cent of the hospitalized patients with diseases of the ear, nose and throat.Maxillary sinusitis stands the first in the list of incidence. Next comes ethmoiditis, then frontitis and finally sphenoiditis_ Sometimes all paranasal sinuses are affected (pansinusitis) or the sinuses of one side (hemisinusitis).

Acute inflammation of the sinuses is caused by acute respiratory diseases, influenza, common cold, general microbial infections, and injuries Chronic sinusitis can be secondary to protracted or frequently recurring acute diseases in the presence of various local and general harmful factors such as decreased reactivity and general weakening of the body, impaired drainage of the sinuses in the presence of hypertrophy or polyps of the mucosa in the region of the orifices, deviated septum, and diseases of the teeth. The suppurative forms of the disease are usually caused by streptococci and staphylococci or other micro-organisms.

Classification of sinusitis:

1.Acute sinusitis: a)catarrhal; b) suppurative.

2.Chronic sinusitis: a) exudative (catarrhal, serous, suppurative, vasomotor, allergic) b) polipous; c) polipous-purulent; d) hypertrophy; e) atrophy (cholesteatomal, caseous, necrotic, ozaenous)

Acute maxillary sinusitis. Signs of acute inflammation of the maxillary sinuses can be local and general. The local symptoms are pain in the region of the involved sinus, forehead root of the nose, and the cheek bone. Headache can be diffuse. Impeded respiration through the involved side of the nose is a common symptom. Nasal discharge is usually unilateral, and is first liquid serous, but then it becomes cloudy, tenacious, and purulent. Olfaction is affected as a rule, but the severity of other symptoms masks this disorder. The general symptoms are elevated temperature of the body, indisposition. The temperature reaction can begin with a chill and be intensive during the entire disease.

The objective symptom of acute maxillary sinusitis is a narrow strip of purulent discharge from the maxillary sinus into the middle nasal meatus, which is especially evident if the head is inclined to the opposite side. Some additional examinations should be earned out: X-ray examination of the paranasal sinuses, diagnostic antral puncture and irrigation of the maxillary sinus; contrast X-ray and echography, and some other techniques can also be used.

The Kulikovsky needle is commonly used for antral puncture. The sinus wall is punctured by the needle and the sinus contents are aspirated; then, the sinus is irrigated with a disinfectant solution, e.g. furacillin. The liquid is passed into the sinus through the needle, while the sinus is drained through the natural orifice. The patient leans downward so that the washings are withdrawn through the nose without entering the nasopharynx. The presence of pathological contents in the sinus is a direct indication of the specific pathology; the absence of pathological matter in the washings does not exclude completely the disease of the sinus. A radiopaque substance (iodolipol) should then be injected into the sinus and an X-ray picture taken in two projections.

Treatment includes local use of vasoconstrictors drops, physiotherapy, and general antibacterial therapy in the presence of high temperature and intoxication of the body. If these measures fail to give the rapid effect, the sinus should be punctured and irrigated and a mixture of antibiotics, steroid hormones, protheolitic enzyme are instilled. The acute suppurative inflammation ends in 5-6 days. UHF, laser therapy of the maxillary sinuses should then be carried out daily. UV-therapy should be used locally and generally.

Chronic maxillary sinusitis. Chronic inflammation of the sinus is as a rule a sequel of acute sinusitis, which is recurrent in some patients. Acute inflammation persisting for more than 3 weeks should be considered as long-standing. If such inflammation does not terminate by the end of the 6th week, the disease can be considered chronic. Sometimes chronic maxillary sinusitis is associated with spreading of pathology from a caries-affected tooth.

A common symptom and complaint of patients with the exudative forms of chronic maxillary sinusitis is discharge from one side of the nose, which can be copious during exacerbation and scarce in remission. The purulent discharge in patients with maxillary sinusitis can be thick or liquid and have a specific odour. The mucopurulent discharge is tenacious and it dries in crusts. Catarrhal sinusitis is marked by tenadous mucous discharge which is often retained in the nasal cavity, and dries in crusts. The discharge in serous, or allergic maxillary sinusitis accumulates in the sinus and drains in portions when the patient assumes a certain position facilitating drainage of the sinus through the nasal meatus. An unpleasant odour is sometimes the main complaint of the patient who feels the smell himself. In bilateral chronic pathologies in the maxillary sinuses patients always complain of decreased sense of smell. Local or diffuse headache usually develops only during exacerbations or in obstructed drainage of the sinus. During remission, the general objective and subjective condition of the patient is satisfactory. Exacerbation of a chronic process can be attended with elevated temperature, worsening of the patient's condition, painful swelling of the cheek, oedema of the eyelid and local or diffuse headache.

Serous-catarrhal maxillary sinusitis facilitates formation of polyps which usually grow from the middle nasal meatus.In rare cases, in the presence of dental granuloma, cysts and fistulae in the sinus, a cholesteatoma can form from the cells of the squamous epithelium.

True (retention) cysts of the sinus form due to obstruction of the mucous glands. Pseudocysts can also develop in the sinus, but they differ from true cysts by the absence of the inner epithelial coat. The main symptom of a cyst is headache arising due to compression of the endings of the trigeminal nerve. Amber-coloured liquid can at times issue from one side of the nose, after which the headache subsides. This is a sign of spontaneous drainage of the cyst.

The pathological discharge from the nose and sinus (taken during antral puncture) is examined in the laboratory for the presence of microflora and for sensitivity to antibiotics. Diagnostic puncture of the maxillary sinus is widely used in older children. Pathology of the maxillary sinus should be differentiated from frontitis, ethmoiditis, and in rare cases from sphenoiditis. In adults it is necessary to rule out the odontogenic nature of the disease, especially in the presence of a suppurative process in the roots of the upper teeth (4, 5, 6), whose apices are in the immediate vicinity of the floor of the maxillary sinus.

Conservative treatment. Treatment should begin with elimination of causes of the disease. If maxillary sinusitis is odontogenic, the teeth should first of all be treated. It should be noted that radical operations on the sinus will be ineffective if the odontogenic cause remains active. In the presence of adenoids or adenoiditis in children, the tactics should be the same: the nasopharynx should first be treated, and only then should treatment of maxillary sinusitis be started. As a rule, general antibacterial treatment is administered during exacerbation.

Antral puncture and irrigation of the sinus with a disinfectant solution (furadn, potassium permanganate solution, peloidin) or enzymes (chymopsin), and administration into the sinus of a solution of the antibiotic to which the microflora is sensitive. In addition to the irrigation of the sinus, UHF and SHF therapy should be applied to the involved area. If conservative treatment of chronic suppurative maxillary sinusitis fails, a radical operation of the maxillary sinus is indicated.

Patients with the polypous and suppurative-polypous forms of maxillary sinusitis usually require radical surgical treatment which should be followed by conservative treatment to prevent relapses of polyposis. Postoperative conservative treatment includes endonasal electrophoresis with calcium chloride, regular administration of astringent preparations, and if signs of allergy

are obvious, anti-allergic treatment is indicated. Patients with large cysts, cholesteatoma, caseous and necrotic maxillary sinusitis need surgical treatment.

Surgical treatment. Operations on the maxillary sinus are performed with endonasal and extranasal approach. The endonasal technique can be used to open the medial wall of the sinus and to perforate it for drainage and aeration of the sinus. The extranasal approach operation ensures an easy access to all parts of the sinus and the operation is therefore radical. This technique includes incision of the soft tissues under the upper lip, separation of these tissues, and approach to the anterior wall of the maxillary sinus. The sinus is then opened, the pathological matter removed, and a communication with the nasal cavity is made (through the inferior or middle nasal meatus).

Acute frontal sinusitis can be secondary to acute rhinitis and ethmoid sinusitis, general viral infection, acute respiratory disease, or chilling of the body.

The main symptoms of acute frontal sinusitis are pain in the forehead, diffuse headache, and purulent discharge from the involved side of the nose. Pain intensified on palpation or percussion of inferior wall of sinus. The nasal discharge is first serous and liquid; later it becomes purulent, odour is usually absent. Nasal respiration through the involved side is impeded. If the affection is pronounced, the body temperature can elevate to sub-febrile levels. The forehead in the area overlying the frontal sinus can be swollen and the skin hyperaemic. A special cannula is passed into the frontal sinus for diagnostic purposes and for irrigation. But since the approach to the sinus is through a curved frontonasal duct, this manipulation is not always possible. X-ray control is recommended during this operation.

X-ray examination and trepanation puncture of the frontal sinus are used for diagnostic and therapeutic purposes.

Treatment is usually conservative. But if the disease is longstanding and complications develop in the orbit, skull, or other organs, surgery should be performed immediately to eliminate the purulent focus and to restore patency of the frontonasal duct. Local treatment includes application of preparations causing anaemization of the nasal mucosa: vasoconstrictors drops (galasoline, naphtiziine). UHF- and SHF-therapy of frontal sinusitis is indicated only for cases where drainage of the sinus is adequate; otherwise physiotherapy will exacerbate the process. Elevated temperature and headache can be managed parenteral administration of antibacterial preparations in the appropriate doses.The absence of the desired effect is an indication for probing or puncture of the sinus.

Chronic frontal sinusitis. The most common cause of conversion of acute frontal sinusitis into its chronic form is persistent obstruction of the frontonasal duct and decreased reactivity of the body, especially subsequent to general infectious diseases. This process is promoted by hypertrophy of the middle concha, significant deformity of the nasal septum, a narrow or tortuous frontonasal duct, or polyps in the nasal cavity. There may be no complaints from the patient during remissions. A small amount of the nasal discharge often escapes into the nasopharynx to cause chronic pharyngitis, laryngitis, and tracheitis.

Palpation of the walls of the frontal sinus is often painful, especially at the upper internal angle of the orbit, which can be swollen. In the absence of microflora, obstruction of the frontonasal duct sometimes stimulates the accumulation of discharge in the sinus and the formation of mucocele consisting of secretions of the mucous glands. In the presence of infection in the sinus, a subperiosteal abscess can develop for the same reason; a suppurative fistula can also form, usually in the inferior wall, most frequently closer to the inner canthus of the eye

Treatment. In the absence of local and general complications, conservative treatment is indicated. It is directed at providing adequate drainage of the secretion from the sinus using vasoconstrictors which are instilled into the nose, and administration of antibacterial preparations (after preliminary testing of the microflora for sensitivity to these preparations). Trephination puncture of the frontal sinus with removal of its contents and subsequent irrigation and administration of medicinal preparations are effective.

Long-standing and persistent chronic frontal sinusitis (despite active treatment), and also symptoms of developing complications (and complications themselves) are indications for surgical treatment (operation of frontoethmoidotomy).

Acute ethmoid sinusitis commonly follows acute rhinitis, influenza, often in combination with acute inflammation of the other paranasal sinuses. Acute ethmoid sinusitis in children is secondary to an acute respiratory disease, measles, scarlet fever, and other infectious diseases; sometimes it has the character of necrotic osteitis, often in combination with acute maxillary sinusitis.

The symptoms of acute ethmoid sinusitis are pressing pain in the dorsum and the bridge of the nose, headache of various localization, and significant impediment of nasal respiration. The first days of the disease are marked by copious serous discharge from the involved side of the nose which later becomes muco-purulent or purulent. The discharge is usually odourless. Oedema and hyperaemia of the internal angle of the orbit and the adjacent parts of the lower and upper eyelids, and also conjunctivitis are frequent findings in children. Hypoosmia are also frequent.The temperature is usually between 37.5 and 38 °C and persists for a week.The diagnosis can be confirmed by X-ray examination. The nasal discharge should be studied for microflora and its sensitivity to antibiotics which will help assess the severity of the infection, prescribe the appropriate antimicrobial therapy.

Treatment is conservative. If any complications develop, surgical treatment is indicated. Vasoconstrictors are instilled into the nose. The same preparations are applied under the middle concha. UHF or SHF on the area of the ethmoidal sinus are indicated. If the body temperature is elevated, antibacterial preparations are given. If a closed empyema or ophthalmic complication develops, the cells of the ethmoidal labyrinth should be opened to gain access to the purulent focus in the orbit.

Chronic ethmoid sinusitis. The disease is often secondary to the affection of the other paranasal sinuses. Chronic ethmoid sinusitis therefore often concurs with frontal sinusitis, sphenoid sinusitis, and more frequently, maxillary sinusitis. The catarrhal-serous, catarrhal-suppurative and polipous forms of chronic ethmoid sinusitis prevail..

The symptoms depend on the activity of the disease. During remission, the patient complains of occasional headache, mostly in the region of the nose root and bridge; headache is sometimes diffuse. In serous-catarrhal ethmoid sinusitis, the nasal discharge is clear and copious. The suppurative form is characterized by a meagre discharge that dries to form crusts. Involvement of the posterior cells of the ethmoidal labyrinth promotes accumulation of the discharge in the nasopharynx, usually in the morning. Olfaction is impaired to some degree.

Treatment of non-complicated forms is usually conservative. Sometimes it is combined with endonasal operations (polypotomy, opening of cells of the ethmoidal labyrinth, partial resection of the conchae, etc.). Opening of the cells of the ethmoidal labyrinth and polypotomy with an endonasal approach are the most common operations.

Acute and chronic sphenoid sinusitis. Isolated affection of the sphenoidal sinuses is rare. The inflammation is usually combined with lesion of the posterior cells of the ethmoidal labyrinth.

Acute sphenoid sinusitis is marked by severe oedema of the mucosa. The most common subjective symptom of acute sphenoid sinusitis is headache in the occipital region and inside the head; the pain is sometimes felt in the orbit. Nasal discharge is often absent because it passes from the superior nasal meatus into the nasopharynx and further along the posterior wall of the pharynx, where it can easily be seen during pharyngoscopy and posterior rhinoscopy. The body temperature is usually subfebrile; the general condition is satisfactory; the patient can complain of weakness, discomfort, and irritability.

X-ray examination is an important diagnostic tool. If the clinical picture is obscure, the sphenoidal sinus can be punctured through its anterior wall.

Treatment is usually conservative: local treatment with vasoconstrictors and general antibacterial treatment. If the disease lasts longer than 2 weeks, the sinus should be irrigated or

opened endonasally. Symptoms of complications (septic, intracranial, ophthalmic) are indications for emergency operation on the sphenoidal sinus.Chronic sphenoid sinusitis is provoked by the same conditions as chronic affection of the other paranasal sinuses.

RHINOGENIC COMPLICATIONS

Orbital complications include:

(a) *Inflammatory oedema of lids.* This is only reactionary. There is no erythema or tenderness of the lids which characterises lid abscess. Eyeball movements and vision are normal. Generally, upper lid is swollen in frontal, lower lid in maxillary, and both upper and lower lids in ethmoid sinusitis.

(b) *Subperiosteal abscess.*Pus collects outside the periosteum. A subperiosteal abscess from ethmoids forms on the medial wall of orbit and displaces the eyeball forward, downward and laterally; from the frontal sinus, abscess is situated just above and behind the medial canthus and displaces the eyeball downwards and laterally; from the maxillary sinus, abscess forms in the floor of the orbit and displaces the eyeball upwards and forwards.

(c) *Orbital cellulitis.* When pus finds its way into the orbit, it spreads between the orbital fat, extraocular muscles, vessels and nerves. Clinical features will include oedema of lids, exophthalmos, chemosis of conjunctiva and restricted movements of the eye. Vision is affected causing partial or total loss which is sometimes permanent. Patient may run high fever. Orbiti cellulitis is potentially dangerous because of the risk of meningitis and cavernous sinus thrombosis.

(d) *Orbital abscess.* Intraorbital abscess usually forms along lamina papyracea or the floor of frontal sinus. Clinical picture is similar to that of orbital cellulitis. Diagnosis can be easily made by CT scan or ultrasound of the orbit. Treatment is antibiotics and drainage of the abscess and that of the affected sinus (ethmoidectomy or trephinalion of frontal sinus).

(e) *Superior orbital fissure syndrome.* Infection of sphenoid sinus can rarely affect structures of superior orbital fissure. Symptoms consist of deep orbital pain, frontal headache, and progressive paralysis of CN VI, III and IV, in that order.

(f) R*etrobulbar neuritis of CN I.* Inflammation of the posterior cells of the ethmoidal labyrinth and the sphenoidal sinus spreads to the orbit impairing the visual acuity, narrowing the field of vision, and intensifying scotoma.

Treatment is surgical with simultaneous general anti-inflammatory treatment. In children, the paranasal sinuses, especially cells of the ethmoidal labyrinth, should be opened by extranasal approach.

Intracranial complications. Rhinogenic intracranial complications are very dangerous. In 75 per cent of the cases, rhinogenic intracranial complications arise due to chronic inflammation in the sinuses, and in 25 per cent of the cases, they are secondary to acute sinusitis.The infection can spread by the contact, haematogenic and lymphogenic pathways.Frontal, ethmoid and sphenoid sinuses are closely related to anterior cranial fossa and infection from these can cause: (a) Meningitis and encephalitis, (b) Extradural abscess, (c) Subdural abscess, (d) Brain abscess, (e) **Cavernous sinus thrombosis**. Orbital veins have no valves and freely communicate with the cavernous sinus and for this reason infection from the orbit or paranasal sinuses can easily spread to the cavernous sinus. Thrombosis of the cavernous sinus is characterized by pronounced local symptoms which develop due to difficult venous outflow. The affection is characterized by swelling of the eyelids and the adjacent tissues, dilatation of superficial veins and hyperaemia of the orbital veins, cyanosis of the orbit, and exophthalmos. These symptoms are supplemented by papilloedema, oedema and thrombosis of the retinal veins. Focal symptoms are also characteristic. Since the first branch of the trigeminal nerve and the oculomotor nerves (3rd, 4th and 6th pairs) pass along the sinus wall, the patient suffers from neurological pain in the region of innervation of the first branch of the 5th pair (orbital and infraorbital neuralgia), analgesia of this region, including comeal anaesthesia, and decreased or lost corneal reflex. Affection of the oculomotor nerves causes internal and external ophthalmoplegia with paralysis of the eyeball of various type and gravity. Unilateral thrombosis of the cavernous sinus can extend to the other

side. Signs of toxaemia, high fever (40°C) with chills, and meningitis are also present. Blood culture should always be done. CT scan is very helpful in diagnosing intracranial abscesses. Treatment includes I/V antibiotics, anticoagulants and drainage of any abscess..

Theatment of rhinogenic intracranial complications requires emergency surgical intervention with subsequent dehydratation, anti-inflammatory and symptomatic treatment. A radical operation will be required with surgical interference in the cranial cavity, the common rules of brain surgery must be employed.

Injuries to the nose, Common injuries of the skin are contusions, bruises, abrasion, or wounds. Injuries to the nose often concur with concussion of the brain. If such suspicion arises, the nasal bones should be examined with X-rays and the patient should be subjected to neurological examination. Contusion and a bruise do not require special treatment except application of cold immediately after the injury. Crepitation sounds indicate fractures of the ethmoid bone and rupture of the mucous and air passes via the injured tissue to bulge the face skin. The fracture of the cribriform plate is manifested by liquorrhoea from the nose.X-ray examination usually reveals the character and extent of injury to the nasal and facial bones in the presence of severe oedema and infiltrative swelling of the soft tissues.

Treatment of nasal fractures is most effective during the first hours following the injury. Bleeding from injured tissues should be arrested as soon as possible to prevent massive loss of blood, tetanus antitoxin should be administered according to the accepted scheme. If the anamnestic and objective findings indicate possible concussion of the brain (degree II or III), the patient should be given neurological treatment and only later (in one or more days) correction of the displaced bones and more extensive surgical operations should be performed. The primary surgical treatment of the wound should be carried out in such cases: the wound margins should not be excised, and only non-viable tissues should be removed. The face wounds usually heal rapidly because of the intensive blood supply.

Laterally displaced bone fragments should be repositioned using the thumb of the right hand to correct displacement to the left, and of the left hand to correct the displacement to the right . A significant effort of the finger is required for repositioning of a bone fragment. A specific crunch is heard when the bone fragment is replaced. No anaesthesia is usually required, since the operation lasts only 2-3 seconds. Novocain solution can, however, be injected into the field of injury or the operation can be performed under short-lasting anaesthesia.

Posteriorly displaced bone fragments should be repositioned using nasal elevators. In most cases, the replaced bone fragments have to be fixed by anterior tamponade of one or both sides of the nose. If fractures are multiple, a firmer and longer immobilization is required. The fractured nasal bones are usually repositioned on the day of the injury, but if the patient attends doctor at later terms, the operation can also be performed, although the correction becomes more difficult. If the fragments are replaced to the nasal cavity we use endonasal method, which needs the next front tamponade and fixing of fragments. If soft tissues are broken, we carry out a primary surgical treatment and make skin titches. As per to the constructions fixing bandages are different: plasters, universal splints, made of colloidal bandages, stens, gyps. They are put on form 7 to 14 days. Such bandages are formed according to the shape of nose. Their upper part is fixed to the forehead by the bandage, and the lower one covers the nose and is fixed by the splint.

Injuries of the paranasal sinuses. Injuries to the frontal sinuses stand the first in the list of incidence; next follow the maxillary sinuses and the ethmoidal labyrinth; the sphenoidal sinuses are injured in rare cases. As a rule, the injury to a paranasal sinus is combined with injuries to some other bones of the face and skull. Mechanical or gunshot wounds of the frontal sinus are often attended by the injury to the frontal lobe of the brain, to the ethmoidal labyrinth, the cribriform plate, the superior and interior parts of the orbit. Injuries to the paranasal sinuses are attended by the concussion syndrome more frequently than injuries to the nose. Nasal bleeding is common at the moment of injury and afterwards.

Injuries to the paranasal sinuses with penetration into the skull usually cause general symptoms of brain injuries: loss of consciousness, dizziness, psychic derangement, vomiting, congestive changes in the fundus of the eye, and cardiovascular dysfunction. The local change at the site of injury is characterized by accumulation of blood in the injured sinus. In some cases, the only sign of bone fracture is subcutaneous emphysema of the orbit, eyelid, cheek, or forehead. Emphysema can be diagnosed by the specific crepitation which can be heard during palpation of the swollen site. Air can penetrate into the cranium through the infractions in the skull bones; this is due to the difference between the pressure inside the cranium and the atmospheric pressure. The injury to the ethmoidal labyrinth is usually attended by the injury to the olfactory receptor. Constant hypo-osmia or anosmia thus develops.

Treatment is aimed at arresting bleeding and eliminating shock. These are the first and emergency measures in injuries to the paranasal sinuses. In the presence of brain concussion of degree **I,** and also in the absence of signs of concussion, the injured site should be given a complete primary treatment including repositioning of the bone fractures. Shock and brain concussion of the second and third degrees require maximum sparing conditions for the patient. The primary treatment of the wound should therefore include arrest of bleeding, suturing the wound, and administering antitetanic serum.

Nasal bleeding is a symptom of a local nose injury or of a systemic disease. Causes of nasal bleeding are therefore classified as local and general. The most frequent site of bleeding is the anteroinferior part of the nasal septum (Kiesselbach's area). Haemorrhage into this area is usually mild and presents no special danger. The superior and posterior parts of the nasal walls are the sites where bleeding can be profuse.

The most common local cause of nasal haemorrhage is injury which can be slight and thus cause only insignificant bleeding. General causes of nasal bleeding are diseases of the blood and the circulating system. Relapsing nasal bleeding often occurs in patients with hypertension and nephronecrosis or contracted kidney. Nasal bleeding can also be caused by blood congestion in heart diseases, lung emphysema, diseases of the liver and spleen, and in pregnancy.

Severe nasal bleeding occurs in haemorrhagic diathesis, including haemophilia, haemorrhagic thrombasthenia, thrombopenic purpura, haemorrhagic vasculitis, capillary toxicosis, and telangiectasia (Osler-Rendu syndrome). In some cases bleeding is caused by disorders in the blood coagulation system, and in others by the affections of the vascular walls. Diseases of the haemopoietic system (leucosis, reticulosis, haemocytoblastosis, etc.) can also be attended with bleeding from the nose and the mucosa of other organs.

Various other factors, such as hypo- and avitaminosis, especially vitamin C deficiency, vicarious menstruation (instead of normally expected menstruation), and also low atmospheric pressure, physical overstrain, exposure to heat and some other factors, can also cause nasal haemorrhage.

Clinical picture. It should be remembered that blood can get into the nose from other parts of the upper airways, e.g. from the pharynx, larynx, trachea, oesophagus, the lung and sometimes even from the middle ear through the auditory tube. The diagnosis is established by rhinoscopy, pharyngoscopy, and inspection of the other related organs.

Mild, moderate, and profuse nasal bleedings are distinguished. Mild nosebleed usually originates from the Kiesselbach area. The bleeding is and only a few millilitres are lost. Such bleedings stop spontaneously. Moderate nasal bleeding is characterized by discharge of larger amount of blood , which, however, does not exceed 200 ml in adults. Measures should be taken in such cases to arrest bleeding rapidly and completely. If blood enters the pharynx and is swallowed, profuse haematemesis can occur with a fall of arterial pressure and tachycardia.

In profuse haemorrhage (from anterior and posterior ethmoidal artery), the blood loss exceeds 200 ml a day. In severe cases one litre and more of blood can be lost. Such haemorrhage is a direct danger to the life of the patient.

Treatment includes the arrest of nasal bleeding. Whenever necessary, the circulating blood volume should be replenished. The protein, electrolyte, and acid-base balance of the body should be corrected.

Insignificant nasal bleeding can in most cases be easily arrested by putting for 15-20 minutes a sterile cotton ball soaked in a 3 per cent hydrogen peroxide solution into the anterior part of the involved side of the nose. The cotton in the nostril should be compressed by the finger against the nasal septum. The patient should be seated upright and ice applied to the nose. If insignificant bleeding from the anterior parts of the nose recurs, the bleeding site should be infiltrated with a 1-2 per cent novocain solution or cauterized with strong trichloroacetic acid, silver nitrate, or chromic acid. Recurrent bleeding from the Kiesselbach area can be managed by separating the mucosa in the area between two incisions. If this measure fails, or if bleeding originates from deeper structures, anterior tamponade is required. A 10 per cent lidococaine or a 2 per cent dicaine solution can be used (2 or 3 times) for anaesthesia. Anterior tamponade of the nose is performed by means of a 60-70-cm long turunda, nasal forceps, haemostatic paste, or emulsion.

A turunda is prepared from a 4-cm wide and 1-1.5-m long strip of gauze or roller bandage. The sterile turunda is taken with two forceps and unrolled into a container filled with a haemostatic solution. The tamponade of the nose is performed by placing the turunda on the floor of the nasal cavity, from its vestibule to the choanae. The turunda is taken by the forceps at a distance of 6-7 cm from its end and is placed on the floor of the nasal cavity to the choanae. The forceps is then used to press the turunda to the floor of the nasal cavity. Then the next loop of the turunda is placed, and so on. The anterior pack should be removed in 24 hours after preliminary wetting it with a hydrogen peroxide solution. In cases of severe bleeding, the tampon should be left in place for 3-4 days, but it should be wetted each day with antibiotic.

A finger of a rubber glove is often used for anterior tamponade (instead of gauze). The glove finger should be stuffed with foam rubber. One or more such rubber fingers are inserted into the bleeding nasal cavity to ensure its tight filling. Inflatable balloon (with a breathing pipe passed inside) is also used for the purpose. Foam rubber encased in a rubber sheath can be used for anterior tamponade as well.

If nosebleed is profuse and does not stop, posterior tamponade is indicated. The blood group of the patient and his Rhesus factor should be established for immediate blood transfusion.

These measures prove ineffective in some cases. The external carotid artery should then be ligated not only on the involved but also on the opposite side. Destruction of cells of the ethmoidal labyrinth is an effective surgical method of arresting profuse nasal bleeding. In some cases, for example, in the presence of the Osler-Rendu syndrome, this operation should be done on both sides.

Posterior tamponade is done with special sterile tampons. Gauze is folded several times into 3 x 2.5 x 2 cm tampon which is then tied up crosswise with two 20-cm long silk threads. One end is cut off, while the other three ends remain. The posterior tamponade is begun with passing a thin rubber catheter into the bleeding side of the nose until its end enters the nasopharynx to appear in the middle of the pharynx. The end of the catheter is taken with a forceps and pulled outside through the mouth. Two threads of the tampon are tied up to this end of the rubber catheter and pulled back through the nose. The second finger of the right hand should be used to help to seat the tampon behind the soft palate in the nasopharynx and press it tightly to the corresponding choana. The next manoeuvre is to pull the two threads through the nose. The threads should be held strained while the nose is packed with the turunda and the thread ends are then tied tightly over a gauze pad at the nasal vestibule. The thread in the mouth will be used to withdraw the tampon. Its free end is fixed on the cheek with an adhesive tape.

Posterior tampon is removed in 24 hours. But if bleeding resumes, the tamponade should be repeated and the tampon remains for 3-4 and in some cases for 7-8 days. Antibioticotherapy and antiseptic solution should be used to wet the tampon. It should be remembered that the

drainage of the auditory tube is impaired in posterior tamponade and inflammation of the auditory tube and the middle ear can develop.

When the anterior and posterior tamponades are used in combination, it is necessary to see that the tampon closing the entrance' to the nose should not compress too tightly the wing of the nose, otherwise necrosis can develop due to impaired blood supply. Antibacterial preparations should be administered in common doses immediately after tamponade is applied. Vitamins K (or vikasol), C and P, rutin, dicinon, solution of aminocaproic acid, and calcium gluconate should be given per os or injected to increase blood coagulation. A 10 per cent calcium chloride solution should be injected intravenously (3-5 per cent solution to children). Blood transfusion is a strong haemostatic means. It should also be conducted as a replacement therapy. Oxygen therapy is indicated, because oxygen deficiency develops in the body after blood loss.

If bleeding originates from a vascular tumour in the nose, it should be removed. A bleeding malignant tumour should be removed with underlying healthy tissue, and with subsequent radio- and chemotherapy of the main disease.

Materials on the activation of students of higher education during the lecture:

Task 1. The patient experienced severe hypothermia, felt general malaise, headache, and subfebrile temperature in the evening (37,50). Difficulty in nasal breathing, more on the left side, abundant muco-purulent discharge from the left half of the nose.

Objectively: pronounced hyperemia of the mucous membrane of the left half of the nose, a strip of pus in the middle nasal passage. What is the patient's diagnosis? What examination methods do you offer for diagnosis? Your tactics of treating the patient?

Task 2. The patient developed a headache, mucous secretions from the nose, general weakness, and an increase in temperature up to 37.60 after the flu.

Objectively: upon examination, the nasal mucosa is hyperemic, swollen, and there is abundant mucous discharge in the middle nasal passage. On the x-ray of the paranasal sinuses, there is a decrease in pneumatization of the frontal sinuses. What complications developed in the patient after the flu? Your tactics of treating the patient.

Task 3. A patient who had the flu had abundant purulent discharge from one half of the nose. Headache in the forehead, pain in the left cheek, nasal congestion also on the left. The temperature rose to 38-39.00C. What is the reason for the deterioration? What methods do you offer for diagnosis? Your treatment tactics.

General material and teaching-methodical support of the lecture: Methodical aid of the lecture, multimedia presentation, mock-ups, tables, sets of tools, sets of radiographs and tomograms, simulators.

Questions for self-control

1.What are the tracts of spread of infection in the sinuses.
2.Symptoms and methods of treatment acute and chronic rhinosinusitis.
3.Significance of sinusitis in infant.
4.Etiology, evolving the chronic sinusitis.
5.Rhinoscopic picture at different forms of sinusitis.
6.Which type of maxillitis have conservative and operative ways of treatment.
7.What are the non-typical forms of location of polyposis during different kinds of sinusitis.
8. Allergic rhynosinusopathy, clinics, diagnosis, treatment.
9.Basic principles of surgical treatment of sinusitis.
10.Etiology of cyst formation in sinuses.

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6. www.gmc-uk.org- *General Medical Council* (*GMC*)
7. www.bundesaerztekammer.de – German Medical Association

Lecture № 3

Topic: Acute and chronic tonsillopharyngitis and their complications. Acute secondary tonsillitis at infection diseases and blood system diseases. Hypertrophy of the lymph tissue of the throat. Acute and chronic laryngitis. Types of the chronic form of laryngitis (hyperkeratosis, pachydermia, leukoplakia). Acute and chronic stenosis of the larynx. Infection granulomas of the upper respiratory tract and ear. Benign and malignant tumors of the upper respiratory tract and ear. Foreign body of the ENT organs. Burns of the esophagus.

Actuality of theme. Parynx and Larynx being a part of the air conducting tracts of the organism, participates in fulfilling the main functions of breathing, phonation and speech. The violation of the normal anatomical and functional relitionships in the larynx leads to different pathological processes, firstly discovered by the development of the nose , larynx and voice disfunction. The knowledge of topological peculiarities of the larynx is necessary for studying of these organ diseases and working out the methods of treatment. Acute and chronic larynx diseases running with the disturbance of breathing and vocal functions, are often met in the clinical practice. The most important problem is that of acute laryngotracheitis in children. It is one of the key problems not only in children's otolaryngology but also in pediatrics. It is due to great frequency and grave course of the disease - 99% per cent of the acute stenosis of larynx in babies occuring as a result of laryngotracheitis turn out to be acute respiratory virus infections. Sick persons with acute stenosis are subjected to different larynx diseases and need urgent aid which must be provided by any physician. It is necessary to know epidemiology and the symptomts of infectious granulomas of the upper respiratory tract and ear for mating the differential diagnosis.

2. The knowledge of topological peculiarities of the trachea, lower respiratory tract and esophagus is necessary for studying of these organs diseases and working out the methods of treatment. Malignant neoplasms become a frequent cause of death. In connection with the rise of frequency of the upper respiratory tract oncological diseases any physician must be able to diagnose malignant neoplasms and benign neoplasms of the ear, nose and throat, and know the principles of prophylaxis and dispensary treatment of patients. There are many traumas by alien bodies of the nose, throat and esophagus in war and peace time, which require urgent help, therefore any physician must be able to help in such cases. A correct selection of methods of treatment and urgent aid provided in time will help the patients frequently and some times will save their lives.

Aim:

Learning aim. To give an idea of the prevalence and social significance of acute and chronic tonsillopharyngitis, laryngitis, stenosis of the larynx, tumors, infection granulomas; the relationship of throat pathology with diseases of other organs; modern methods of research of the pharynx and larynx;

The student should know:

- etiology, pathogenesis, clinic, methods of diagnosis and treatment of of acute and chronic tonsillopharyngitis, laryngitis, stenosis of the larynx, tumors, infection granulomas;

The student should be able to:

- to establish the stage of stenosis of the larynx;
- to establish the clinical form of acute and chronic tonsillopharyngitis, laryngitis, stenosis of the larynx, tumors, infection granulomas;
- to evaluate the data of X-ray examination;
- to make a differential diagnosis.

Educational purposes.

Acute and chronic tonsillopharyngitis, laryngitis, stenosis of the larynx, tumors, infection granulomas are one of the most important moments of modern clinical medicine due to the peculiarities of their course, difficulties in diagnosis and treatment. From these positions at the lectures, students are brought up an awareness of high professional responsibility. The lecturer

controls the psychological readiness of the student to work as a doctor (social significance of the doctor's activities, high morality, social activity).

Basic concepts: Acute and chronic tonsillitis. Complications of tonsillitis. Acute and chronic laryngitis. Stenosis of the larynx. Tumors of the ENT organs

Plan and organizational structure of the lecture

№.	The main stages of lectures and their contents	Time
1	Preparatory stage	3
2	Determination of learning objectives.	2
	Providing of positive motivation	2
3	The main stage. Most important anatomic position of the pharynx and larynx	7
	Etiology, pathogenesis of acute and chronic purulent inflammation of the pharynx.	6
	Classification and clinic tonsillopharyngitis, laryngitis, stenosis of the larynx	10
	Treatment of patients with acute and chronic purulent otitis media.	10
	Labyrinthitis (acute and chronic, limited and diffuse, serous, purulent and necrotic, their clinic). Treatment.	5
	General characteristics, etiology, pathogenesis of otogenic intracranial complications.	10
	Middle ear catarrh	5
	Sensorineural hearing loss	10
	Otosclerosis	5
	Meniere's disease	5
	The final stage	
4	Lecture summary, general conclusions.	2
5	Lecturer's answers to possible questions	2
6	Self-study tasks for the student	6

Content of the lecture material

CLINICAL ANATOMY OF THE PHARYNX

The **pharynx** is a part of the alimentary and respiratory tracts. The pharynx connects the nasal and oral cavities with the larynx superiorly and with the oesophagus inferiorly, it passes into the oesophagus below the sixth cervical vertebra. The pharynx is divided into three parts: nasopharynx (epipharynx), oropharynx (mesopharynx) and the laryngopharynx (hypopharynx). Seven orifices open into the pharynx: two openings of the choanae and two openings of the auditory tubes are in the nasopharynx; the fauses opens into the oropharynx; and the inlet of the larynx and the oesophagus are found in the laryngopharynx.

The **nasopharynx** performs only the respiratory function. Two choanae are found anteriorly. Funnel-shaped openings of the auditory tubes are located on the lateral walls, at the level of the posterior ends of the inferior conchae. Posterior to the openings of the auditory tubes found are the tubal tonsils. At the border between the superior and posterior walls of the nasopharynx is the pharyngeal tonsil. The pharyngeal tonsil is well developed only in children. During sexual maturation, the tonsil diminishes. The border between the superior and middle parts of the pharynx is an imaginary plane passing at the level of the hard palate.

The **oropharynx** is the part of the pharynx through which air and food pass; the alimentary and respiratory tracts meet in this region. Anteriorly the oropharynx opens into the mouth (fauces), while the posterior wall of the oropharynx borders on the third cervical vertebra. The fauces is confined in the space between the edge of the soft palate, the uvula, the anterior and posterior palatine arches, and the root of the tongue. In the soft palate itself a muscle is found which elevates the palate to bring it in contact with the posterior wall of the pharynx. Contraction of this elevator muscle widens the lumen of the auditory tube. The other muscle of the soft palate strains and stretches the palate thus widening the opening of the auditory tube but narrowing its

lumen in the rest of it.

Palatine tonsils are found in triangular recesses (tonsillar fossae) between the palatoglossal and palatopharyngeal arches. The histological structure of the lymphoid tissue of the pharynx is uniform: a mass of lymphocytes with spheric formations known as follicles is located between connective-tissue fibres. The structure of the palatine tonsils is important from the clinical standpoint. Their free surface is exposed to the pharyngeal cavity and is lined with mucous membrane with stratified squamous epithelium. The tonsil has 16-18 deep pits known as lacunae, or crypts. The total summ of the surface area of the crypt is about 300sm^2, when the area of the mucous layer covering of the pharynx does not exceed 45sm^2. The outer surface of the tonsils is connected with the lateral wall of the pharynx by a firm fibrous membrane called the capsule. The lacunae penetrate into the depth of the tonsil where they ramify. Located underneath the epithelium of the crypt are diffuse lymphoid tissues and groups of follicles, which are differentiated into: a) so called primary follicles, which are made up only of lymphocytes; b) bigger sized secondary follicles with an germinal centre, surrounded by lymphocytes. In the past years the anatomical unit of the tonsils has been isolated – cryptophon, which is made up of the crypt lumen with its contents, the crypt epithelium, the lymphoepithelial tissue of cryprts and secondary lymph nodules.

The basic cell element of the tonsil is the lymphocyte. T-lymphocytes (about 25%) appear in the palatine tonsils only after the formation of the thymus. They are mostly represented by T-helpers and T-suppressors. A small number of T-helpers located in the secondary follicle. In addition the tonsils also have plasmatic cells, the so called normal killers; immunoglobuline synthesizing cells of the classes G, A, M, U, D, small lymphocytes with a relatively massive nucleus – the carrier of encoded information. These are the so called memory cells.

In the defense function of the tonsils an important role is played by the synthesis of a factor of local immunity of mucous membranes IgA, which repels the attachment of microbes to the epithelial cells and is an inhibitor of the adsorbtion and reproduction of bacterial cells on mucous membranes, blocking the surface receptors of the bacteria; produce a powerful factor of nonspecific immunity, mainly antiviral defense – interferons, and also the lysosomes.

The structure of the palatine tonsils foresees the continuous prolonged contact of the antigens with the lymphoid cells which migrate into the lacunar lumen; a more intense migration of lymphocytes takes place in regions where the connective tissue is absent. In these places the epithelial cells seen to move away, forming the so-called “physiological wounds”. This contact in itself helps the lymphoid cells in obtaining antigenic information. With the formation of clone cells in tonsils tissues which are specific in relation to the given antigen. The former assure the informative function of the tonsil tissue, carried out by the smaller lymphocytes (memory cells), who are capable of giving out a fast secondary immunological answer.

The tonsils also have a tolerance functions – they are not stimulated by the cryptal saprophytic flora – streptococc, which plays a role similar to that of E. coli in the intestines. – sterptococc saprophytes along with other conditional pathogenic cocci and anaerobic microbes. This function enables the sustainment of the normal mictoflora. In such a manner, the tonsils, mainly, are responsible for carrying out 3 biologically important functions: defensive, informative and sustenance of the bacteriological homeostasis.

Lymphoid tissue is also found on the posterior wall of the pharynx where it is present in the form of small (punctate) granules or follicles; it is also found posterior to the palatine arches, on the lateral walls of the pharynx. Small accumulations of lymphoid tissue are also found at the entrance to the larynx and in the piriform pharyngeal recesses. The lingual tonsil (IV) is located on the root of the tongue. The following lymphoid formations thus form a sort of a ring: two palatine tonsils, two tubal tonsils, one pharyngeal tonsil, one lingual tonsil, and the fine accumulations of lymphoid tissue. All these tonsils are called the throat ring (Pirogov-Waldeyer tonsillar ring).

The laryngopharynx. The superior edge of the epiglottis and the root of the tongue form the border between the oropharynx and the laryngopharynx. The lower end of the

laryngopharynx narrows into a funnel and is continuous with the oesophagus. The entrance to the larynx borders the laryngopharynx anteriorly and inferiorly. Along the sides of the entrance to the larynx, between the entrance and the lateral walls of the pharynx, are found cone-shaped diverticula known as the piriform recesses. Food moves to the oesophagus by these piriform recesses. The pharyngeal wall consists of four layers. The main layer is a fibrous membrane, which is lined with mucosa on the inside (from the side of the pharyngeal cavity), and with muscles on the outside. The outer surface of the muscles is lined with a layer of loose connective tissue, the adventitia, owing to which the pharynx can move relative to the surrounding anatomic structures.

The mucosa of the pharynx (its upper portion) is covered with stratified ciliated epithelium in accordance with the respiratory function of the nasopharynx. Stratified squamous epithelium lines the middle and lower parts of the pharynx. The muscular layer of the pharynx includes striated fibres (circular and longitudinal) which contract and elevate the pharynx. Three constrictor muscles of the pharynx, namely, the superior, the middle, and the inferior constrictor muscles are responsible for its contraction. Two longitudinal muscles elevate the pharynx. As the muscles contract, they ensure the peristalsis-like movement of the pharynx at the moment of swallowing.

A retropharyngeal space is found between the posterior wall of the pharynx and the prevertebral fascia. The space is a flat slit packed with loose connective tissue. The retropharyngeal space is divided sagittally by the median septum into two symmetric parts. In children, there are lymph nodes into which the lymphatic vessels of the palatine tonsils and the posterior parts of the nasal and oral cavities empty. These nodes atrophy with age. The nodes can purulate in children thus causing a retropharyngeal abscess. The parapharyngeal space lined with connective tissue is located by sides of the pharynx. A neurovascular bundle and the deep cervical lymph nodes are found here.

The pharynx is supplied with blood mainly by the branches of the external carotid artery. The lymph is emptied from the pharynx into the deep and posterior cervical lymph nodes. The lymphoid formations of the pharynx (all tonsils included) have no vessels by which lymph is supplied to them. The pharyngeal nervous plexus is located on the external and internal surfaces of the middle constrictor, it is responsible for the motor and sensory innervation of the pharynx.

CLINICAL PHYSIOLOGY OF THE PHARYNX

Through the pharynx, food and saliva pass into the gastro-intestinal tract and air passes into the larynx. The pharynx is involved in the following vital functions: (1) ingestion of food (sucking and swallowing); (2) production of vocal sounds; (3) respiration; (4) protective function (during eating and respiration).

Ingestion of food during the first months of life can only be accomplished by sucking. The passage of food by the pharynx, from the mouth into the oesophagus, is accomplished by a complicated and well coordinated swallowing reflex.The muscles of the tongue, pharynx and the larynx contract in a specific sequence.

The *vocal* (sound-producing) function of the pharynx includes intensification of sounds produced in the larynx by resonance. The voice timbre is formed in the cavities of the larynx, pharynx, nose, paranasal sinuses, and the mouth.

All parts of the pharynx are involved in the *respiratory* function. But if the nasal passages are obstructed, breathing is accomplished through the mouth. In this case, and partly during speaking and singing, air does not enter the nasal cavity but gets into the oropharynx.

The protective function of the pharynx consists in reflex contraction of the pharyngeal muscles when a foreign body or an irritating substance.

Inspired air is first warmed in the nose and then in the pharynx, where it is also cleaned from dust which sticks to the mucous lining of the pharyngeal walls. The physiology of the palatine tonsils is not autonomous. It is part of the function of the entire lymphatic system of the body. It is believed that the lymphoid apparatus of the pharynx, in particular, the palatine tonsils (like similar accumulations of lymphoid tissue in the small intestine) protect the body from the

ingress of micro-organisms. During the first years of life, the lymphoid structures of the pharynx attain maximum growth, but during sexual maturation (at the age of 14-15) they undergo partial and gradual back development.

EXAMINATION OF THE PHARYNX

Inspection and palpation. The regional lymph nodes of the pharynx are palpated: the submandibular nodes, the nodes in the retromandibular fossae, deep cervical, posterior cervical nodes.

Mesopharyngoscopy. Using a spatula (held in the left hand) the anterior two-thirds of the tongue should be pressed down (without touching the root of the tongue, because this will stimulate the vomiting reflex). A normal soft palate is readily movable. The mucosa of the soft palate, of the uvula, and the anterior and posterior palatine arches should then be inspected. Normal mucosa is smooth and pink; the arches are well defined.

The size of the palatine tonsils should be estimated. The mucous membrane of the tonsils should next be examined. Normally it is pink, smooth and moist. The lacunae contents should be examined. To that end, two spatulas are used. Using one spatula, the tongue is pressed down, while the other spatula (in the other hand) is used to press the base of the anterior arch and, through its agency, the tonsil, at its upper pole. A normal tonsil contains non-purulent scarce secretion (epithelial plugs) in its lacunae. The lacunae can contain no plugs at all. The mucosa of the posterior wall of the pharynx is then examined. The normal mucosa is pink, moist and smooth. Granules, approximately 1 x 2 mm in size, occur occasionally.

Epipharyngoscopy. A warmed naso-pharyngeal speculum and a spatula are used for this purpose. The superior parts of the nasopharynx, the choanae, the lateral walls of the pharynx (where the openings of the auditory tubes can be seen at the level of the posterior ends of the conchae) are visible in the mirror. Normal choanae are empty; the mucosa of the superior regions of the pharynx is pink and smooth. The nasopharyngeal tonsil can be seen in the vault of the pharynx. Normally it is lodged on the posterosuperior wall of the nasopharynx without reaching the superior edge of the vomer and the choanae.

Palpation of the nasopharynx. The physician stands behind and to the right of the seated patient. The doctor's right index finger should swiftly pass behind the soft palate into the nasopharynx to feel the choanae, the vault of the nasopharynx,
and the lateral walls. The cheek of a child should be pressed between the upper and lower jaws using the left index finger.

Hypopharyngoscopy. The lower portions of the pharynx should be inspected using indirect laryngoscopy with a warmed laryngeal speculum. The tip of the tongue should be wrapped in a piece of gauze and held by the fingers of the left hand so that the thumb is on the superior and the middle finger on the inferior surfaces of the tongue, the index finger lifting the upper lip. The tongue is pulled slightly forward and downward. The laryngeal speculum should be held by its handle in the right hand and moved into the mouth without touching the root of the tongue and the posterior wall of the pharynx. The mirror surface should be directed downwards. The patient is asked to utter the sound 'ee' and gently breathe in.

INFLAMMATION OF THE TONSILS

Acute tonsillitis is a general infectious disease in which the lymphoid tissue of the tonsils is affected by inflammation. In most cases the palatine tonsils are affected, while the other tonsils are involved less frequently.

Aetiology and pathogenesis. Among many microbes that can provoke acute tonsillitis (coccus, bacilli, viruses, spirochetae, fungi, etc.) the leading aetiological role belongs to beta-haemolytic streptococcus of group A. *Staphylococcus aureus* is another common causative agent of acute tonsillitis. Virological and clinical studies have shown that adenoviruses can also cause various forms of tonsillitis.

The exogenic factor attacks the tonsillar mucosa via airborne and alimentary route, and also by direct contact. Three main forms of the development of common acute tonsillitis are distinguished: (1) ocassional acute tonsillitis manifested as auto-infection due to impaired

environmental conditions, often as a result of chilling; (2) epidemic form arising as a result of infection from a tonsillitis patient; (3) exacerbation of chronic tonsillitis.

The commonly used classification includes the following forms: I-catarrhal; II-follicular; III-lacunar; IV-fibrinous; V-herpetic; VI-phlegmonous (intratonsillar abscess); VII-necrotic (gangrenous); and VIII-mixed forms.

Clinical forms. **Acute catarrhal tonsillitis.** The pathological changes are characterized by pronounced dilatation of small blood and lymphatic vessels in the parenchyma of the tonsil, thrombosis of small veins, and stasis in the lymphatic capillaries. The onset is acute and is marked by dryness, burning and tickling in the throat; then swallowing becomes slightly painful. The patient complains of general indisposition, fatigue, and headache. The body temperature is usually subfebrile; insignificant inflammatory changes in the peripheral blood are found. Pharyngoscopy reveals diffuse hyperemia of the tonsils and the margins of the palatine arches; the tonsils are somewhat enlarged. The regional lymph nodes are often slightly enlarged. The clinical signs are more pronounced in children. The disease usually lasts 3-5 days. We must differentiate this form with ARVI.

Follicular tonsillitis. The disease usually begins with elevation of temperature to 38-39° C. The patient feels strong pain during swallowing. The pain radiates into the ear; salivation is often increased. More severe symptoms can develop in children: febrile temperature is often associated with vomiting; signs of meningism develop. The changes in the blood are often pronounced: neutrophilic leucocytes count from 12000 to 15000; moderate shift to the left and eosinophilia are observed; ESR is often 30-40 mm/h; traces of protein are found in the urine. As a rule, the regional lymph nodes are enlarged; their palpation is painful.

Pharyngoscopy reveals diffuse hyperaemia and infiltration of the soft palate and the arches; the tonsils are hyperaemic and enlarged, with numerous yellowish or yellowish-white spots (1 -3 mm) elevated over the surface. These formations are suppurating follicles. The disease lasts 5-7 days.

Lacunar tonsillitis. Lacunar tonsillitis usually runs a more severe course than follicular. Pharyngoscopic picture is characterized by enlargement of hyperaemic tonsils which are covered with islets of yellowish coat, first in lacunar orifices and then over the entire surface of the tonsils. Toxaemia is severe, and it is therefore necessary to monitor the cardiovascular and respiratory functions.

Fibrinous (fibrinomembranous) tonsillitis. Follicular or lacunar tonsillitis can sometimes develop like fibrinous tonsillitis when a membrane is formed from the ruptured purulent follicles. The fibrinous membrane spreads over onto the sites of necrotized epithelium in the lacunar orifices; it fuses with the adjacent sites of affection to form a confluent patch which can extend beyond the boundaries of the tonsils.

Table of Distinctive Symptoms of Diphtheria and Lacunar Tonsillitis

Symptoms	Tonsillitis	Diphtheria
Swollen tonsils	Less marked than in diphtheria, frequently it is bilateral	More severe, accompanied by edema of the palate arches, uvula and soft palate.May be unilateral
Patches	Spread within free areas	Extend beyond tonsils to palate arches, soft palate and posterior pharyngeal wall
Color of patches	Yellowish	White, grey -white, dirty-grey
Adherence of patches	Patches superficial and peel off easily	Patches deep, with necrosis of mucous; in typical cases

		strip off with difficulty to leave a bleeding surface
Pain on swallowing	Sharp	Not always marked
Regional lymph nodulus	Swollen, individual nodes easily palpated and extremely tender	Markedly swollen nodes on both sides from early days of disease, edema of subcutaneous tissue; flattened out contours of neck
Constitutional disturbance	Less severe than in diphtheria	Increasingly severe in toxic form
Fever	Within 39-40 °C	From subfebrile to 40 °C; more stable
Bacteriological examination	Negative (for Loeffler's bacilli)	Positive in most cases

Treatment. Rational treatment includes sparing conditions, local and general therapy. The patient must remain in bed during the first days of the disease and then abstain from physical work. The patient should be separated from the others; he should use separate dishes and other objects. In very severe cases the patient should be hospitalized. Food should be nutritious, rich in vitamins, soft, and not irritating. Treatment includes also gargling with a warm solution of sodium chloride or hydrocarbonate, furacin, potassium permanganate, calendula or camomile tea. A warming compress should be applied to the neck. Salicylates and antibacte-rial preparations should be used for general treatment.

The choice of antibacterial preparations depends on the gravity of the disease and the danger of complications. The antibiotic is administered usually for 5 days, which is, as a rule, sufficient to normalize body temperature and to improve the patient's condition. In order to eliminate reliably the infectious focus, it is necessary to continue the antibiotic therapy for another 3-5 days, or it is better to replace common by bicillin. If the patient is sensitive to penicillin, broad-spectrum antibiotics should be given in appropriate doses. Nystatin is given to patients to prevent candidiasis. If the course of acute tonsillitis is not aggravated by any factors, sulpha drugs are used instead of antibiotics. Desensitizing preparations such as suprastine, hysmanale, diazoline, etc. are recommended.

Phlegmonous tonsillitis. Intratonsillar abscess is a rare disease. It is associated with purulent destruction of a part of the tonsil. One side is usually involved. The affected tonsil is hyperaemic and enlarged. Its surface is tense; palpation is painful. *Treatment* includes opening of the abscess. Unilateral tonsillectomy is indicated for recurrent affections.

Herpangina. Viral tonsillitis is caused by adenoviruses. The causative agent of herpangina is type A Coxsackie virus. The disease is usually sporadic. The disease is highly contagious.The onset of herpangina is acute. The body temperature rises to 38-40°C, the patient complains of pain in the throat during swallowing, headache, and muscular pain in the abdomen. Vomiting and diarrhoea are also possible. Changes in the blood are moderate: slightly increased leucocyte counts, more often slight leucopenia, insignificant shift to the left. During the first hours of the disease diffuse hyperaemia of the pharyngeal mucosa can be revealed pharyngoscopically. Small reddish vesicles can be seen on the soft palate, tongue, palatine arches, and, less frequently, on the tonsils and the posterior wall of the pharynx.

Necrotic (ulcerous-necrotic) tonsillitis of Simanovsky-Vensana. Symbiosis of *Bacillus fusiformis* and *Spirochaeta buccalis* that is often found in the mouth of healthy people in the

avirulent state is believed to be the pathogenic factor. The incidence of the disease is low and sporadic. The morphological changes are characterized by necrosis of the surface of one tonsil with formation of an ulcer whose floor is covered with a loose fibrinous membrane underlied by necrotized lymphoid tissue.The patient complains of discomfort in the throat during swallowing, fetid breath and hypersalivation. The body temperature is usually normal. The leucocyte count moderately increases. The regional lymph nodes are enlarged on the involved side; they are moderately painful to palpation. Swallowing is usually painless. The disease lasts 1 to 3 weeks but can in some cases persist for several months.

Treatment consists in tending the mouth cavity, cleaning the ulcers from necrotized matter, gargling with disinfectant solutions. The surface of the ulcer is treated with an iodine tincture, silver nitrate or other solution, but neosalvarsan or novarsenol is believed to be the most effective. Novarsenol (0.3-0.4 g at 1-2-day intervals) and antibiotics should be injected intravenously in severe cases.

Lingual tonsillitis. Acute inflammation of the lingual tonsil is a relatively rare disease. The body temperature is febrile, swallowing is severely painful; speech is impaired. Protrusion of the tongue during its inspection and palpation of its root are very painful. Inspection with a laryngeal speculum reveals enlarged and hyperaemic lingual tonsil; punctate patches are sometimes formed. Oedema and stenosis of the larynx are dangerous complications. Treatment is the same as for other acute tonsillites. Abscesses should be opened surgically.

COMPLICATIONS OF TONSILLITIS

Peritonsillitis or Quinsy. Inflammation of the peritonsillar cellular tissue arises due to virulent infection spreading, usually from the palatine tonsil to the peritonsillar cellular tissue in the presence of predisposing local or general factors. In most cases, peritonsillitis is a complication of acute tonsillitis, foreign body or odontogenic ethyology.

Symptoms. The development of the process has three stage: the oedema-infiltrative, purulent and reconvalescent stages. The process is usually unilateral. Tonsillogenic peritonsilites occur several days following a recurrent exacerbation of chronic or acute tonsillitis. A peritonsillar abscess can be found in the anterior or antero-superior (supratonsillar) part, between the tonsillar capsule and the upper part of the anterior palatine arch. The supratonsillar location of the abscess is most common. Posterior peritonsillitis (developing between the tonsil and the posterior arch) may cause oedema of the larynx. Peritonsillitis can also be inferior, with location of the focus between the inferior pole of the tonsil and the lateral pharyngeal wall, or lateral, occurring between the middle portion of the tonsil and the lateral wall of the pharynx. Lateral abscess runs the most severe course because of difficult spontaneous drainage.

The onset of the disease is manifested by severe pain during swallowing. The patient complains of headache and fatigue; the body temperature rises to febrile. Spontaneous pain in the throat becomes more intense, it radiates into the ear, teeth, and becomes so intense during swallowing that the patient refuses food and drinks. Trismus of the masticatory muscles develops. The speech becomes nasal and slurred. Inflammation of the pharyngeal muscles and also cervical lymphadenitis cause pain as the patient moves his head to one side. The leucocyte counts are 10-15 x 10^9 per 1; the blood count is shifted to the left; the erythrocyte sedimentation rate increases. Pharyngoscopy is difficult due to trismus: the mouth usually would open not wider than 2-3 cm. Anterosuperior and anterior peritonsillites are characterized by marked protrusion of the upper pole of the tonsil together with the palatine arches and the soft palate toward the median line. Half of the soft palate, together with the superior tonsillar pole and the upper part of the arches form a sphere whose surface is tense and hyperaemic; the uvula is moved to the opposite side, the tonsil is displaced posteriorly and inferiorly. The tongue is covered with a thick coat, the saliva is tenacious. Fluctuation is observed in the region of the strongest protrusion; the abscess opens at this point, often through the supratonsillar recess or the anterior arch.

Treatment. The patient must be hospitalized, bed rest is obligatory. Antibacterial therapy is indicated for all stages of peritonsillitis. Antibiotics are injected intramuscularly. As soon as

the abscess is ripe (the 3rd or 4th day) it should be opened surgically, without waiting for its spontaneous rupture. The abscess is usually incised without any anaesthesia, or after spraying over the pharynx with a 10 per cent lidocaine or a 2 per cent dicaine solution. The incision should be done at the most prominent site. A dull tool, e. g. a bulbed probe or a packer, is often used to open the abscess, although this method is more painful . Tonsillectomy is indicated in cases when the opening of abscess is impossible (lateral peritonsillitis), abortive attempt to open the abscess, persistent course of the disease, and in the presence of signs of complications, such as sepsis, pharyngeal abscess, phlegmon of the neck, and mediastinitis.

Retropharyngeal abscess. This is a purulent inflammation of the lymph nodes and loose connective tissue found between the fascia of the pharyngeal muscles and the prevertebral fascia. The disease occurs almost exclusively in children because the lymph nodes and the loose connective tissue in this region are well developed up to the age of 4, after which they undergo involution.

The first symptoms are usually pain in the throat during swallowing and impeded respiration. The child refuses food, becomes restless and often cries; sleep is deranged. The temperature rises to 38-39° C. If the abscess is found in the nasopharynx, respiration through the nose becomes difficult, speech is nasal and the voice timbre dull. If the abscess is located in the mesopharynx, a pharyngeal stridor can develop. The voice becomes hoarse and respiration noisy. If the abscess extends onto the inferior parts of the pharynx, asphyxia and cyanosis develop. The entrance to the larynx can be constricted, and the oesophagus and the trachea compressed. The reaction of the regional lymph nodes is usually pronounced; they swell and become tender so that the child has to hold his head in a forced position. Pharyngoscopy reveals bulging and hyperaemic mucosa; the affection is often asymmetric so that only one half of the posterior pharyngeal wall is involved.

The blood reacts to the inflammation: the leucocyte counts increase to 10-15 x 10^9 per 1; the blood count shifts to the left; the erythrocyte sedimentation rate accelerates to 40-50 mm/h. The disease lasts 5-6 days or sometimes longer.

Treatment should be conservative until the abscess develops. Antibiotics and sulpha drugs are prescribed. When an abscess develops, it should immediately be opened; measures should be taken to prevent aspiration of pus. This can be attained by preliminary suction of pus during puncture; the abscess should preferably be opened on a half-lying patient .

Peripharyngeal abscess develops due to various causes, such as extention of infection to the cellular tissue of the peripharyngeal space during acute tonsillitis, often during peritonsillitis; possible injury to the pharyngeal mucosa; purulent discharge from the mastoid process through the incisura mastoidea and the pharyngo-maxillary space.

Symptoms. The patient experiences severe pain during swallowing (the mouth opens with great difficulty). The head is inclined to the involved side; respiration can be difficult. The body temperature is usually elevated, the general condition bad. The leucocyte counts are 12-14 x 10^9 per 1, the erythrocyte sedimentation rate 45-50 mm/h. Inspection reveals infiltration of the sub- and retromandibular region. Fluctuation is sometimes revealed during palpation of the swelling.

Treatment at the initial stage of peripharyngitis includes intravenous injections of big doses of antibiotics, dehydratation, desensetive medisine. Ripe abscess should be opened surgically. There are two approaches : external, by the anterior margin of the stemocleidomastoid muscle, and through the oropharynx.

AFFECTIONS OF THE PHARYNX IN SYSTEMIC DISEASES

Infectious mononucleosis. This infectious disease is probably caused by a special lymphotropic virus which occurs together with *Listerella* genus. It is believed that infection occurs by air-borne droplets or by contact; the nasal cavity and the pharynx are the portals of infection. Children and the young usually develop mononucleosis. The disease is characterized by a fever, tonsillitis-like changes in the fauces, adenosplenomegaly, and changes in the blood (high counts of leukocytes and atypical monocytes). The incubation period lasts 4-5 days (sometimes 10 days). At the onset of the disease the body temperature rises to 38-40° C and

persists at this level from 5 days to 2-4 weeks (for longer periods in rare cases). The symptoms are sometimes alleviated periodically during this stage. An early and permanent sign of the disease is enlarged lymph nodes, first on the neck and then in the groin, armpits, and the abdomen. The spleen and the liver are also enlarged in most patients. Changes in the fauces usually follow the enlargement of the lymph nodes; they are similar to those occurring in catarrhal, lacunar, fibrinous, and less frequently necrotic tonsillitis.

The most characteristic symptom of the disease is a moderate leucocytosis with a predominance of mononuclear cells, which may number 50 to 90 per cent of the total leukocytes, a great number of altered monocytes .

Treatment. Bed rest and high-calorie diet rich in vitamins are prescribed. Antibacterial preparations prevent secondary infection; the causative agent is insensitive to them. Gargling with disinfectant or astringent solutions is useful. Necrotized areas are treated with a 10 per cent silver nitrate solution. General light (UV) treatment is recommended.

Agranulocytosis (agranulocytic angina). Affection of the tonsils is the specific symptom of this disease. Agranulocytosis is considered not as an independent nosological disease but as a response of the haemopoietic system to various pathological factors (such as infection, toxicosis, radiant energy) or as a result of altered haemopoiesis in systemic diseases of the blood. Agranulocytosis occurs mostly in women; it is a rare disease affecting mostly adults.

Symptoms. The prodromal period is characterized by indisposition; it lasts 1-2 days. Fulminant, acute, and subacute forms of agranulocytosis are distinguished. In the former two cases the disease begins with high temperature (to 40° C), chills, and bad general condition. Necrotic and ulcerative changes in the pharynx, mainly in the region of the palatine tonsils, occur simultaneously. Necrosis often spreads onto the mucous of the pharynx, gums, and the larynx. In rare cases, the destructive changes occur in the intestine and the urinary bladder. Necrosis can extend onto deep underlying soft tissues and bones.

The blood is characterized by a very low count of polymorphonuclear leukocytes, or they can be absent.

Treatment is aimed at activizing the haemopoietic system and controlling secondary infection. Exeption of all medicines that can cause agranulocytosis (amidopyrine, sulphanilamide, salvarsan, etc.). Blood transfusion, antibioticotherapy, hormone preparations and other means of treating agranulocytosis are prescribed. The diet should be sparing; the patient must gargle the throat with antiseptic solutions; the necrotized matter should be removed.

Septic angina (alimentary toxic aleukia). The onset of this disease is marked by a sudden fever of 39° to 40 °C, inflammatory and necrotic signs in the throat, petechial eruptions and severe hemorrhage from the nose and mouth.

The anginal stage is not the onset of the disease and follows food intoxication that has been in progress for one to three weeks without any significant signs.

The disease is caused by cereal food such as millet, wheat, rye, barley, buckwheat, and oats, that had been left out in the field during the winter.

Ingestion of this grain, in particular millet, will cause a bitter taste and a burning sensation in the mouth, pharynx, esophagus and stomach, as well as numbness in the tongue. These symptoms are often accompanied by nausea, vomiting, and headache. Yet in other cases, the absorption of this food for only two or three weeks is followed by headache, prostration and weakness. Punctate hemorrhage looking like flea bites appears on the skin. Already at this early period of septic angina, blood analysis will reveal a progressive reduction in the leukocyte count, viz., onset of the period of leukopenia. The whitish or yellowish-brown membrane which appears on the tonsils marks the onset of necrosis which soon, in fact in 24 hours, causes deep ulcers . This ulceration commonly affects not only the tonsils which soon collapse completely but other aggregations of lymphadenoid tissue as well, and may extend to the palatine, pharyngeal and esophageal mucosa and, sometimes, to that of the oral cavity.

Withdrawal of toxic products from food at the initial period of the disease, prior to the onset of anginal symptomps, may often bring recovery, especially if the total amount of toxic

food eaten has been moderate. Advanced septic angina is frequently fatal.

Treatment. At the first signs of the disease, toxic products should be immediately withdrawn from food, and lavage of the stomach undertaken. The patient is then given large doses of magnesium sulfate or sodium sulfate to cleanse the stomach of toxic food residue. The diet must be nourishing and rich in proteins and vitamins, and drink must be given in plenty to help expel toxins from the body. Local treatment, apart from the use of gargles, and anesthetic ointments, is by sprinkling the ulcerated surfaces with streptocide or sulfadimezin powders twice daily.Intramuscular antibiotics injections have been used with success.

CHRONIC TONSILLITIS

Inflammation of the palatine tonsils prevails among chronic inflammations of the other tonsils of the lymphoid pharyngeal ring. Chronic tonsillitis is infectious-allergic diseases of human body. According to some authors, the incidence of chronic tonsillitis is 4-10 per cent among adult population and 12-15 per cent among children.

The factors predisposing the onset of chronic tonsillitis are the anatomo-topographic properties of the tonsils (the presence of crypts, and some others) and their histological properties, the presence of microflora in the lacunae and conditions favorable for its cultivation, and disordered biological and protective-adaptation mechanisms in the tonsil tissue.

In chronic tonsillitis the flora is not polymorphous in deep parts of the lacunae. Monoflora is usually found: various forms of streptococci (especially of haemolytic staphylococcus), adenoviruses (mostly in children), and others. Chronic tonsillitis should be regarded as an infectious disease caused mostly by autoinfection.

Chronic tonsillitis is usually secondary to acute tonsillitis. Acute inflammation of the tonsillar tissue is not followed by complete resolution; it continues and turns into a chronic form. In rare cases chronic tonsillitis can develop without preceding acute inflammation.Permanent autoinfection from chronic foci such as carious teeth, chronic inflammation in the nasal cavity and the paranasal sinuses, or in the pharynx, and also bacterial and local tissue and general autoallergy provoke the onset of chronic tonsillitis.

The pathological inflammatory changes are localized in the epithelial coat of the fauces and in the walls of the tonsillar lacunae, in their parenchyma and stroma, and also in the peritonsillar connective tissue. The squamous epithelium of the crypts comes off in scales to form fetid caseous masses plugging the crypts and containing numerous bacteria and leukocytes. Owing to the expansion of the crypts the tonsils appear porous and spongy, and the faucial pillars often adhere to the free surface of the tonsils. The crypts become a most convinient place for the retention and propagation of virulent streptococci and staphylococci whose vital activity keeps up the inflammatory process in the tonsils. In unfavourable conditions, like chilling or reduced body resistance, etc., these bacteria may cause exacerbations, such as acute tonsillitis, peritonsillar abscess and a number of general complications, for example, infectious polyarthritis, rheumatic heart, nephritis, etc.

Symptoms and clinical classification of chronic tonsillitis. Frequently recurring acute tonsillitis in the anamnesis is the most reliable evidence of chronic tonsillitis. According to various authors, chronic tonsillitis can develop without preceding acute tonsillitis in about 2-4 per cent of cases. The diagnosis should be based on the assessment of all symptoms taken together because each separate sign can be caused by some other disease of the pharynx, teeth, jaws, nose, etc. Chronic tonsillitis cannot be diagnosed during exacerbation because all pharyngoscopic symptoms will characterize acute rather than chronic tonsillitis. Only 2-4 weeks after exacerbation it is possible to assess the objective signs of chronic inflammation of the palatine tonsils.

Chronic tonsillitis would be usually exacerbated 2 or 3 times a year, but acute tonsillitis can also occur 5 and 6 times during one year. In some patients chronic tonsillitis is exacerbated once or twice in the course of 3 or 4 years, but this recurrence should also be considered frequent.

The complaints of the patients are frequently recurring acute inflammation of the tonsils,

unpleasant breath, discomfort and feeling of a foreign body in the throat during swallowing, dryness and prickling. The patient often complains of fatigue, flaccidity, headache, decreased working capacity, the temperature is often subfebrile. For many patients, sore throat in the anamnesis is the only complaint.

Inspection of the tonsils and the surrounding tissues reveals ridge-like thickening in the margins of the anterior and posterior palatine arches, their oedema, especially of the upper parts, hyperaemic margins of the palatine arches, often their adhesion to the tonsils and the triangular fold.

The tonsils of most adults with chronic tonsillitis are small, in children they are enlarged, but hyperplasia of the lymphoid tissue of the pharynx (of the palatine tonsils included) is considered normal for children. The surface of chronically inflamed tonsils can be loose, especially in children; but in most cases the tonsils remain smooth. The presence of fetid caseous matter or purulent plugs in the tonsillar lacunae is an important and most common sign of chronic tonsillitis. The lacunar contents are usually taken for diagnostic studies by expressing with a spatula. A common local sign of chronic tonsillitis is enlargement of the regional lymph nodes: upper deep cervical, those located by the anterior edge of the sternocleidomastoid muscle.

The classification of chronical tonsillitis was accepted by 7-th Conference of Specialist in USSR in 1975 and recommended by Health Ministry of USSR in 1979. Classification of tonsillitis of Academic E.B.Soldatov tracts them as following form. In first compensatory form there are only local symptoms of chronic inflammation of tonsils. General reaction of organism doesn't occurs due to sufficient barrier of tonsils and resistance of human body. Second decompensatory form is characterized by disturbance of tosilla function in form of residual tonsillitis, paratonsillitis, paratonsillary abscess, different pathological reactions, diseases of other organs and systems. In the formulation of diagnosis in decompensation condition precise form of decompensation are indicated. Examples of formulating diagnosis: chronic tonsillitis, compensatory type; chronic tonsillitis, decompensatory type (residual tonsillitis, rheumatism).

Classification of Preobrazensky-Palchun: The *simple form* of chronic tonsillitis is characterized by the above described symptoms in the absence of toxaemia or allergic reaction of the body associated with the chronic process in the palatine tonsils. Chronic tonsillitis in its simple form does not impair the general condition of the patient between exacerbations. The *toxicoallergic form, first stage* is diagnosed by the same criteria as the simple form, and also by the symptoms of toxaemia and allergization: periodical elevation of temperature during acute tonsillitis, increasing fatigue and decreasing working capacity, periodic pain in the joints and the heart, functional disorders of the nervous, renal and other systems. The concomitance diseases may occur. Chronic tonsillitis often concurs with diseases of different aetiology which are, however, related to it through common reaction of the body. Essential hypertension, hyperthyroidism or diabetes mellitus can concur with chronic tonsillitis. In the presence of a concomitance disease, chronic tonsillitis can be simple or toxico-allergic, first stage. The *second stage* is characterized by organic changes of internal organs and system and their conjugate diseases. Conjugate of systemic diseases with chronic tonsillitis is established by the presence of the same aetiology, including the aetiology of exacerbations. For example, streptococcus or other microbe is known to be an aetiological factor for chronic tonsillitis and rheumatism (as well as for nephritis, infectious polyarthritis, etc.). In this concomitance, the connection between the diseases is manifested by periodic or constant direct effect of one disease on the other, especially during exacerbations. This phenomenon determines the physician's tactics in the treatment of chronic tonsillitis in the presence of conjugate chronic infections.

The course of conjugate diseases is aggravated by the presence of chronic infectious foci in the tonsils, but the pathogenetic connections here are realized through the general reaction of the body.

Treatment. Treatment of chronic tonsillitis depends on its form. Simple chronic tonsillitis is as a rule managed conservatively, and only if this treatment proves ineffective in 3-4 courses, the tonsils should be removed.

The toxico-allergic form should be treated surgically, but the first degree of this disease can also be treated conservatively (1-2 courses). If treatment is not sufficiently effective, tonsillectomy is indicated. Toxico-allergic symptoms of the second degree are direct indications for tonsillectomy. If this operation is contraindicated (e.g. in the presence of haemophilia), cryotherapy with liquid nitrogen should be recommended. In 1972 in ENT department professor V.D.Dragomiretsky practically introduced cryosurgical method of treatment of chronic tonsillitis by using autonomal cryoapparatus (KAO-01 $ KAO-02). Clinical and immunological investigation shoved that extreme cold not only leads to remove pathological changes of parts of palatine tonsills but have stimulating effect on organism of type tissue therapy Academic V.P.Filatov. It has hyposensibilising action and possesses immunocoregulator property. Cryoaction doesen't accompany general and local reaction of organism and these gives us to using cryosurgical method ambullatory to the patient to whom surgical method are contraindicated with high degree of risk. Cryosurgical method has the following advantages: cryodestruction is less painfull and in most cases is performed without anesthesia; there is no blood loss and method is usefull for the patients with high blood pressure and problems with blood coagulation; this can be used for serious somatic patients.

Methods of conservative treatment are quite varied. Irrigation of the lacunae with various antiseptic solutions (furacin, boric acid, ethacridine lactate, potassium permanganate) and also mineral alkaline water, peloidin and interferon is effective. A special syringe with a long curved cannula is used for the purpose. Among physiotherapeutic methods are UV rays, electromagnetic UHF and SHF oscillations, and ultrasound.

Indications for tonsillectomy are the following:

1. Chronic tonsillitis, simple and toxico-allergic (the first degree), in the absence of effect from conservative treatment.
2. Toxico-allergic chronic tonsillitis of the second degree.
3. Chronic tonsillitis complicated with peritonsillitis.
4. Tonsillogenic sepsis.

Tonsillectomy is absolutely contraindicated in the presence of severe systemic diseases of the cardiovascular system with circulatory insufficiency of the second and third degrees, renal failure with threatening uraemia, severe diabetes mellitus with threatening coma, severe hypertension with possible vascular crises, haemophilia (haemorrhagic diatheses), and other diseases of the blood and the circulatory system (chromocytopaenic purpura, Osler-Rendu syndrome) that are attended with haemorrhage and resist any therapy, acute systemic diseases, exacerbations of chronic systemic diseases. Dental caries, inflammation of the gums, pyogenic diseases, menstruation, and last weeks of pregnancy are temporary contraindications for tonsillectomy.

Pre-operative management is carried out in out-patient conditions. In the majority of cases the operation is performed under local anaesthesia with the patient in the sitting position. Whenever necessary, tonsillectomy is performed under inhalation intubation anaesthesia.

The most common complication of tonsillectomy is bleeding from the tonsillar fossa. During the first day after the operation, the discharge from the mouth should be constantly controlled. It is necessary to remember that blood can pass into the oesophagus. In suspected bleeding, the patient's pharynx should immediately be inspected and blood clots, if any, should be removed and examined thoroughly. The bleeding sites should be clamped and ligated with catgut after preliminary anaesthesia. Pulse and pressure should be taken.

As distinct from vascular bleeding, parenchymatous bleeding is usually not profuse. It can be managed by haemostatics, such as vitamin K (vicasol) parenterally, a 10 per cent calcium chloride (or calcium gluconate) solution intravenously. The tonsillar fossa should be packed with a tampon soaked with haemostatics. If a tampon has to be held in place for a long time, the palatine arches can be ligated above it. If bleeding is profuse and all measures to arrest it fail, the external carotid artery is ligated on the involved side. In rare cases bleeding occurs at later terms: in 7-10 days after the operation. It should be arrested as described above. The patient should be

hospitalized.

Prophylactic measures against chronic tonsillitis are substantially the same as against acute tonsillitis. There exist individual and social aspects in prevention of tonsillitis. *Individual prophylaxis* includes invigorating measures which strengthen the patient's resistance to infection and unfavorable environmental conditions. Acute tonsillitis is often preceded by local or general chilling. Hence the importance of general and local hardening of the body: regular exercises and sports, air baths, and sponging with water (with gradually lowering temperature). But all these measures should be taken gradually and regularly.

Social prophylactic measures include control of microbial and other kinds of contamination of the environment, including improvement of working and living conditions. Treatment of infectious foci in the mouth and nose is also very important for prevention of acute and chronic tonsillitis. Health education of population is another important measure.

HYPERTROPHY OF LYMPHOID TISSUE OF THE PHARYNX

The volume of lymphoid tissue of the pharynx can vary significantly depending on its functional activity. But hypertrophy of the pharyngeal lymphoid tissue can sometimes be persistent. Hypertrophy may be so significant that respiration not only through the nose but also through the mouth becomes difficult; food is swallowed with difficulty and speech is impaired. The function of the auditory tubes is affected as well. In the overwhelming majority of cases, the palatine tonsils are hypertrophied significantly only before the onset of sexual maturation. Less frequently they are enlarged in persons aged under 30.

Hypertrophy of the pharyngeal tonsil (adenoids). Adenoids usually grow at the age from 3 to 15, but they also occur in younger patients and in adults.

Adenoids are lodged in the posterior part of the naso-pharyngeal vault, but they can also grow over its entire dome and involve the lateral walls, downwards to the pharyngeal openings of the auditory tubes. They are usually attached to the underlying tissue by their wider base. Adenoids are irregular rounded formations divided by a deep cleft along the median sagittal line. Each half is, in turn, divided into two or three lobes.

The main *symptoms* of adenoids are upset respiration through the nose, constant serous nasal discharge, dysfunction of the auditory tubes, and recurrent inflammation of the nasopharynx and the nasal cavity.

Three degrees of adenoid growth are distinguished: degree I-adenoids cover to one third of the vomer; degree II-about half of the vomer is covered; degree III-the vomer is covered to two thirds or almost completely. Degree I adenoids do not impair significantly respiration through the nose in child. If a child is ill for a long time, the face bones become distorted: the dropping jaw becomes narrow and long, while the hard palate undergoes malformations: it becomes high and narrow; incorrectly growing teeth cause malocclusion. These changes give a specific dull expression to the face of children with adenoid growths (adenoid facies).

Children with hypertrophy of the pharyngeal tonsil can develop pigeon chest. The size of the blind spot on the fundus of the eye can increase. Children with adenoids are usually flaccid; they are absent-minded, their advance at school is slow; they often complain of headache. Palpation of the nasopharynx confirm the diagnosis.

Treatment of adenoids is commonly surgical. Conservative treatment is helpful only if hypertrophy is insignificant or there are contraindications for the operation. Antihistaminics and calcium gluconate help in some cases.

The surgical removal of the adenoids (adenoidectomy) is performed in cases where the enlarged tonsil impedes respiration through the nose. The operation is usually performed at the age from 5 to 7, but infants and adults can also be operated on if nasal breathing is pathologically impeded, the hearing function is impaired, or other diseases concur. Children can be operated under out-patient conditions, while adults only in hospital. Children should not take breakfast on the day of the operation. The operation can be performed either without anaesthesia, or after instilling 5 drops of a 10 per cent lidocaine solution into each side of the nose. Contraindications for adenoidectomy are diseases of the blood, severe diseases of the cardiovascular system, and

infectious diseases (the patient may be operated on only in 1-2 months after the disease). An important pre-operative measure is immobilization of the child. The nurse sits on a stool or in a surgical chair facing the surgeon and holds the child in her laps so that his legs are fixed between the nurse's knees; the right arm is used to hold the child's arms and the trunk, while the left arm holds the child's head. A sterile cloth should cover both the nurse and the child The operated child is placed on his side in bed on a low pillow for 25-30 minutes. Hot or spicy foods should not be given, since bleeding can resume for 4-5 days. Physical strain, active movements and chilling should be avoided.

Hypertrophy of the palatine tonsils occurs mostly in children. Hypertrophied tonsils can interfere with normal respiration through the mouth, speech, and swallowing of food. If hypertrophy of the palatine tonsils concurs with adenoids, the respiratory function is severely upset. The child suffers from paroxysmal asphyxia during sleep, he is tortured by cough and frequently wakes up during sleep. These factors cause neurasthenia and other disorders.

Diagnosis is made during pharyhgoscopy. Simple hypertrophy of the palatine tonsils should be differentiated from chronic tonsillitis which is characterized by recurring acute tonsillitis in the anamnesis and pharyngoscopic signs of chronic inflammation.

Treatment of pronounced forms of the disease is surgical. The tonsils are partly removed (the parts protruding beyond the palatine arches are excised). The pre- and postoperative treatment is the same as in adenoidectomy. If hypertrophy is insignificant, it can be left without treatment.

Adenoiditis (inflammation of the nasopharyngeal tonsil). Acute adenoiditis occurs mostly in children because the adenoid tissue of the nasopharynx grows during childhood. The aetiological and pathological processes in adenoiditis are substantially the same as in acute inflammation of the other tonsils.

Symptoms of acute adenoiditis in older children and in adults are slight indisposition, subfebrile temperature, local burning in the nasopharynx, which is later attended by acute rhinitis. Respiration through the nose is difficult. Watery, mucous, and then purulent discharge from the nose is characteristic. The patient complains of pain in the ears and nasal speech. Acute otitis media concurs in some cases. The regional lymph nodes are enlarged. Acute adenoiditis in infants begins with elevation of body temperature to 40 °C and general symptoms of toxaemia, such as vomiting, liquid stools, and meningeal irritation. Severe cough usually indicates penetration of the mucous discharge into the larynx and the trachea which can cause tracheobronchitis or bronchopneumonia. The possible dangerous complications are catarrhal or purulent otitis media, retropharyngeal abscess, suppuration of the regional lymph nodes, and also general infectious complications.

Treatment (local and general) is the same as for other acute tonsillites, acute rhinitis, and catarrh of the upper respiratory tract. Nursing babies should be given vasoconstrictors (into the nose),protargol, collargol before each feeding; the nasal discharge should regularly be removed by suction.

Nasopharyngeal fibroma. The nasopharyngeal fibroma is a special type of tumour which occurs almost exclusively in males between the age of 8 to 13 years and in full puberty, i.e. from 20 to 25 years, when the tumour, if still present, begins to shrink. As the tumour is predominantly seen in adolescent males in the second decade of life it is thought to be testosterone dependent.

The site of origin of the tumour is still a matter of dispute. Earlier it was thought to arise from the roof of nasopharynx or the anterior wall of sphenoid bone but now it is believed to arise from the posterior part of nasal cavity close to the margin of sphenopalatine foramen. From here the tumour grows into the nasal cavity, nasopharynx and behind the posterior wall of maxillary sinus which is pushed forward as the tumour grows. Laterally it extends into pterygomaxillary fossa and thence to infratemporal fossa and cheek.

The essential element of a nasopharyngeal fibroma is dense connective tissue containing a great number of elastic fibres and blood capillaries. The tumour is histologically benign, but for

its clinical course marked as it is by irresistible growth and destruction of the surrounding tissue, postoperative relapses and frequent copious hemorrhages endangering the patient's life, it may sooner be classified as a malignant neoplasm.

It may extend into :

1. Nasal cavity causing nasal obstruction, epistaxis and nasal discharge.
2. Paranasal sinuses. Maxillary, sphenoid and ethmoid sinuses can all be invaded.
3. Pterygomaxillary fossa, infratemporal fossa and cheek.
4. Orbits giving rise to proptosis and "frog-face deformity". It enters through the inferior orbital fissure and also destroys apex of the orbit. It can also enter the orbit through superior orbital fissure.
5. Cranial cavity. Middle cranial fossa is the more common.

Symptoms. The initial clinical symptom is unilateral nasal obstruction. After four to six months full nasal obstruction occurs as well as more or less marked complications in the ear. In advanced cases with a rapid growth of the tumour it causes the eye, as well as the soft and hard palates to bulge, swells out the nose, etc. Repeated nasal hemorrhages at the very onset of the disease weaken the patient and aggravate his condition still further. Other clinical features like broadening of nasal bridge, proptosis, swelling of cheek, infratemporal fossa or involvement of IInd, IIIrd, IVth, VIth, cranial nerves will depend on the extent of tumour.

Investigations

Soft tissue lateral film of nasopharynx shovs soft tissue mass in the nasopharynx. X-rays of paranasal sinuses and base of skull may show displacement of nasal septum, opacification of sinuses, anterior bowing of posterior wall of maxillary sinus, destruction of medial antral wall, erosion of greater wing of sphenoid or pterygoid plates, widening of lower lateral margin of superior orbital fissure. C.T. scan with enhancement is a non-invasive technique and essential to evaluate the extent of tumour. It is particularly useful for intracranial extension. Carotid angiography shows extension of tumour, its vascularity and feeding vessels. Embolisation of feeding vessels can be done, if desired, before surgery.

Treatment. The. treatment is by surgery. The operation is rather difficult because of the deep position of the tumour, its firm adherence to the basal tissue and severe hemorrhage. Small tumours located in the nasopharynx are removed via the nose or mouth.

In neglected cases where the tumour invades the zygomatic area and paranasal sinuses the removal is performed only after a preliminary operation has been undertaken to provide access to the tumour proper. This access may be obtained through the sinus maxillaris with complete removal of the lateral nasal wall. There may be about 2 litres of blood loss dunng surgery. Therefore attempts are made, pre-operatively, to reduce the vascularity of tumour. A course of oestrogen therapy may reduce vascularity of tumour. Preoperative radiation also helps to reduce vascularity but is not generally.

ANATOMY OF LARYNX

The larynx lies in front of the hypopharynx opposite the third to sixth cervical vertebrae. It moves vertically and in anteroposterior direction during swallowing and phonation. The larynx of an infant differs from that of an adult in being smaller, funnel-shaped and of a narrower lumen. Cartilages are also softer and collapse easily. Infant's larynx contains more of submucosal tissue which makes it more liable to become oedematous in response to trauma or inflammation. Until puberty the larynx of male differs little from that of the female. After puberty the larynx of male grows rapidly with increase in length of rima glottidis and change in character of the voice while the larynx of female changes little.

There are 3 unpaired and 3 paired cartilages.

1. Thyroid. It is the largest of all the cartilages. Its two alae meet anteriorly forming an angle of 90° in males and 120° in females.

2. Cricoid. It is the only cartilage forming a complete ring. Its posterior part is expanded to form a lamina while anteriorly it is narrow forming an arch.

3. Epiglottis. It is a leaf-like yellow elastic cartilage forming anterior wall of laryngeal

inlet.

4.Arytenoid cartilages. They are paired. Each arytenoid cartilage is pyramidal in shape. It has a *base* which articulates with cricoid cartilage; a *muscular process,* directed laterally to give attachment to intrinsic laryngeal muscles; a *vocal process* directed anteriorly giving attachment to vocal cord; and an *apex* which supports the corniculate cartilage.

5. Corniculate cartilage (of Santorini).They are also paired. Each articulates with the apex of arytenoid cartilage.

6. Cuneiform cartilages (of Wrisberg). Each is situated in aryepiglottic fold.

Extrinsic laryngeal membranes

Thyrohyoid membrane. Connects thyroid cartilage to hyoid bone.

Cricothyroid membrane. Connects thyroid cartilage to cricoid cartilage.

Cricotracheal membrane.Connects cricoid cartilage to the first trachea

1 ring.

Intrinsic laryngeal membranes:

Cricovocal membrane. It is a triangular fibroelastic membrane. Its upper border is free and stretches between middle of thyroid angle to the vocal process of arytenoid and forms the vocal ligament. Its lower border attaches to the arch of cricoid cartilage. From its lower attachment the membrane proceeds upwards and medially and thus, with its fellow of opposite side, forms conus elasticus.

Quadrangular membrane**.** It lies deep to mucosa of aryepiglottic folds and is not well defined. It stretches between the epiglottic and arytenoid cartilages. Its lower border forms the vestibular ligament which lies in the false cord.

They are of two types muscles, namely intrinsic, which attach laryngeal cartilages to each other, and extrinsic, which attach larynx to the surrounding structures.

Intrinsic muscles. *Acting on vocal cords*

Abductors : Posterior cricoarytenoid

Adductors : Lateral cricoarytenoid, Inlcrarytcnoid (transverse arytcniod) Thyroarytenoid (external part)

Tensors : Cricothyroid, Vocalis (internal part of thyroarytenoid)

Acting on laryngeal inlet: Openers of laryngeal inlet: Thyroepiglottic (part of thyroarytenoi'l); Closers of laryngeal inlet: Interarytenoid (oblique part), Aryepiglottic (posterior oblique part of interarytenoids)

Extrinsic muscles. They connect the larynx to neighbouring structures and are divided into elevators or depressors of larynx. They include sternohyoid, sternothyroid and omohyoid.

Laryngeal cavity starts at the laryngeal inlet where it communicates with the pharynx and ends at the lower border of cricoid cartilage where it is continuous with the lumen of trachea. Two pairs of folds - vestibular and vocal - divide the cavity into three parts, namely the vestibule, the glottic and the subglottic space.

Vestibule extends from laryngeal inlet to vestibular folds. Glottic is a deep elliptical space between vestibular and vocal folds and also extending a short distance above and lateral to vestibular fold. The saccule is a diverticulum of mucous membrane Subglottic space (infraglottic larynx) extends from vocal cords to lower border of cricoid cartilage. Vestibular folds (false vocal cords). Two in number; each is a fold of mucous membrane extending antero-posteriorly across the laryngeal cavity. Vocal folds (true vocal cords). They are two pearly-white sharp bands extending from thyroid angle to the vocal processes of arytenoids. Each vocal cord consists of a vocal ligament which is the true upper edge of cricovocal membrane covered by closely bound mucous membrane with scanty subepithelial connective tissue. Glottis (rirna glottidis). It is the elongated space between vocal cords anteriorly and vocal processes and base of arytenoids posteriorly. Antero-posteriroly it is about 24 mm in men and 16 mm in women.

Supra glottic larynx above the vocal cords is drained by lymphatics which pierce the thyrohyoid membrane and go to upper deep cervical.*Infraglottic larynx* below the vocal cords is

drained by lymphatics which pierce cricothyroid membrane and go to prelaryngeal and pretracheal nodes and thence to lower deep cervical and mediastinal nodes.

The larynx performs the following important functions:

1. Protection of lower airways
2. Phonation
3. Respiration

Phylogenetically this is the earliest function to develop; voice production is secondary. The larynx protects the lower air passages in three different ways: sphincteric closure of laryngeal opening, cessation of respiration, cough reflex.

Larynx is like a wind instrument. Voice is produced by the following mechanism *(aerodynamic myoelastic theory of voice production):v*ocal cords are kept adducted, infraglottic air pressure is generated by the exhaled air from the lungs due to contraction of thoracic and abdominal muscles, the air forces open the cords and is released as small puffs which vibrate the vocal cords and produce sound which is amplified by mouth, pharynx, nose and chest. This sound is converted into speech by the modulatory action of lips, tongue palate pharynx, and teeth.

Nerve supply of larynx

Motor. All the muscles which move the vocal cord (abductors, adductors or tensors) are supplied by the recurrent laryngeal nerve except the cricothyroid muscle. Right recurrent laryngeal nerve arises from the

vagus at the level of subclavian artery, hooks round it and then ascends between Ihe trachea and oesophagus. The left recurrent laryngeal nerve arises from the vagus in the mediastinum at the level of arch of aorta, loops round it and then ascends into the neck in the tracheo-oesophageal groove. Thus, left recurrent laryngeal nerve has a much longer course which makes it more prone to paralysis compared to the right one.

Sensory. Mucous is supplied by superior laryngeal nerve. It arises from inferior ganglion of the vagus. *Laryngeal* reflexogenic zones are mostly located on the laryngeal surface of the epiglottis, the true vocal folds, arytenoid cartilages and in the interarytenoid space and also in the rima vestibuli.

METHODS OF EXAMINATION

Laryngoscopy is visual inspection of the larynx interior. Direct and indirect laryngoscopy are distinguished. Direct laryngoscopy is used in cases where inspection with a speculum is infeasible (in infants) or if inspection is not sufficiently informative. Direct laryngoscopy is also used when specimens of live tissue have to be taken (biopsy) for histological studies, or if a newgrowth should be removed. At the present time direct laryngoscopy precedes the intubation of the airways under anaesthesia and is the first step in tracheobronchoscopy. Direct laryngoscopy in children can be performed without anaesthesia.

Indirect laryngoscopy is carried out using a laryngeal speculum. The mirror is fixed in the handle, warmed in hot water (to 40-50 °C) for 2-3 seconds and dried up with a piece of cloth. The patient is asked to open the mouth, produce the tongue and breathe through the mouth. The tip of the tongue should be held between the first and the third fingers of the left hand using a piece of gauze, with the second finger placed on the upper lip. The laryngeal speculum is held in the right hand as a writing pen and introduced into the mouth with the mirror down. The speculum should be moved parallel to the tongue without touching its root or the posterior wall of the pharynx as far as the soft palate; then the mirror is positioned at an angle of 45° to the middle axis of the pharynx. The patient is asked to utter a long sound 'ah' and take a deep breath. During phonation, and then during inspiration the inner surfaces of the larynx become visible in two phases of the physiological activity.

The image reflected in the mirror differs from the natural view of the larynx: the anterior parts of the larynx are seen below and therefore appear as if they are located posteriorly, while the posterior parts are seen in the upper part of the image and appear anteriorly. The left and right sides in the mirror reflection and in reality are the same. The root of the tongue with the

lingual tonsil are first of all seen in the mirror, then viewed is the leaf-shaped epiglottis. The mucosa of the epiglottis is normally pale-pink or slightly yellowish. Two valeculae are seen between the epiglottis and the tongue root; they are bounded by the lateral and middle glosso-epiglottic folds. During phonation and deep inspiration, the vocal (true vocal) folds are well seen. Normally they are pearl-white. Their anterior ends, at the point of their origination from the thyroid cartilage, form an acute angle, the anterior commissure. Pink vestibular folds (false vocal cords) can be seen above the true vocal cords. Laryngeal ventricles are found between the vocal and vestibular folds. Smooth pink aryepiglottic folds extend from the arytenoid cartilages to the epiglottis. The piriform recesses are located laterally to the aryepiglottic folds; their mucosa is smooth and pink.

Roentgenotomography is an important paraclinical method of examination of the larynx.

The **trachea** is the continuation of the larynx, with which it is connected through the cricotracheal ligament. The trachea is a long tube, about 11-13 cm long, beginning at the level of the seventh cervical vertebra; at the level of the fourth and fifth thoracic vertebrae it divides into two main bronchi. The point of division is called the bifurcation. In neonates, the lower end of the trachea is found at the level of the third-fourth thoracic vertebrae. The tracheal wall consists of 16-20 U-shaped hyaline cartilages, whose free ends are directed posteriorly. The cartilages are connected by a membranous wall and annular ligaments owing to which the tracheal lumen broadens during inspiration and narrows during expiration. The tracheal lumen varies between 15 and 22 mm in men and between 13 and 18 mm in women. In infants, the lumen is 6-7 mm. The trachea is lined with mucous membrane with columnar ciliated epithelium. The cilia move in the upward direction. The posterior membranous part of the tracheal wall is adjacent to the anterior wall of the oesophagus. A semilunar projection, known as the carina of the trachea, is found at the point of bifurcation. One of the main bronchi, the right one, is wider and shorter than the left. The right bronchus is 3 cm long and the left one, 5 cm long. Foreign bodies usually get into the right bronchus.

The blood to the trachea is supplied by the inferior thyroid and the bronchial arteries. The veins of the trachea and the bronchi empty into the thyroid veins.

The trachea and the bronchi are innervated by the recurrent laryngeal branch of the vagus nerve and its tracheal branches which form a plexus in the inferior portion of the airways. The muscular fibres of the bronchi are innervated by the fibres of the vagus nerve and the nerves of the sympathetic trunk.

The oesophagus is continuous with the pharynx at the level of the inferior edge of the cricoid cartilage (the sixth cervical vertebra). This is a 24-25-cm long muscular tube with a shorter anteroposterior diameter. The oesophagus opens into the stomach at the eleventh thoracic vertebra, at the point where the 7th costal cartilage is attached to the sternum. In adults, the total length of the part of digestive tract from the incisor teeth through the mouth, the pharynx, and the oesophagus to the stomach is 40-42 cm. The oesophagus has the following three anatomical narrowings: the proximal end of the oesophagus, known as the mouth of the oesophagus; the bronchial narrowing, at the level of the tracheal bifurcation; and the diaphragmatic narrowing in the oesophageal opening of the diaphragm. These anatomical constrictions play an important role in the pathology of the oesophagus.

The cervical portion of the oesophagus is supplied with blood from the oesophageal arteries and from the inferior thyroid artery. The thoracic part of the oesophagus is supplied from- the branches of the thoracic aorta; and the abdominal part, from the diaphragmatic artery and the left gastric artery.

The veins of the cervical portion of the oesophagus are emptied into the inferior thyroid veins; the blood from the veins of the thoracic part of the oesophagus drains into the hemiazygos and azygos veins, and the veins of the abdominal part into the branches of the portal vein.

The oesophagus is innervated by the branches of the vagus nerve and sympathetic nerves. The main function of the oesophagus is to propel food actively into the stomach. At the moment of swallowing, the mouth of the oesophagus opens and food is propelled by the pharyngeal

muscles into the proximal end of the oesophagus. The oesophageal muscles then contract peristaltically to move food further to the stomach.

The **diagnostic inspection** of the trachea and the bronchi is indicated for respiratory dysfunction in the presence of newgrowths, tracheo-oesophageal fistula, atelectasis of any location, etc. Therapeutic *tracheobronchoscopy* is indicated mainly for the presence of foreign bodies, scleroma, and infiltrations in the infraglottic compartment. Lower and upper tracheobronchoscopy are distinguished, depending on the level to which the tube is introduced. In upper bronchoscopy the tube is passed through the mouth, the pharynx and the larynx, while in lower tracheobronchoscopy, through a preliminarily formed tracheostomic opening, the tracheostoma. Lower tracheobronchoscopy is used mostly in infants under 3 years of age and in persons that have undergone tracheotomy. At the present time general anesthesia would be normally preferred, the more so that special breathing systems (Friedel) are available. Inspection of the trachea and the bronchi in children is carried out only under general anesthesia. The patient lies on his back with the head tilted backwards. After tracheobronchoscopy, the patient should be kept under physician's observation for at least two hours because edema of the larynx and its stenosis are likely to occur during this period.

The oesophagus is examined for various pathological conditions, such as foreign bodies, diverticula, and cicatricial constrictions caused by burns, tumours, infectious granuloma, etc. Two objective methods of examination are now used: X-ray (rhoentgenoscopy and rhoentgenography) and endoscopy (oesophagoscopy).

The apparatuses used for oesophagoscopy are the same as those used for tracheobronchoscopy. Their designs are quite varied. Some of them can be used under local anesthesia to the pharyngeal and oesophageal mucosa (fibrooesophagoscopy, Friedel, Brunnings systems), while others can be used only under general anesthesia.

Acute catarrhal laryngitis. Acute inflammation of the laryngeal mucosa is usually extension of catarrhal inflammation of nasal and pharyngeal mucosa, e.g. in measles, pertussis, influenza, typhus, rheumatism, and some other diseases.

Symptoms. The disease is characterized by hoarse voice, tickling and dryness in the throat. The body temperature is usually normal and less frequently it rises to subfebrile. Simultaneously with the subjective signs, develops also dry cough, which later turns into wet cough. Voice production disturbances are characterized by various degrees of dysphonia to complete aphonia. Respiration is sometimes difficult because of accumulation of mucopurulent crusts and swelling of the mucosa.

Treatment. The larynx should first of all be spared. The patient is not allowed to talk until acute inflammation subsides. Spicy or cold food, alcoholic drinks and smoking are prohibited. A warming compress should be applied to the neck. Medicamentous therapy is directed at eliminating inflammation in the larynx and preventing complications. In some cases it is recommended to add of hydrocortisone suspension to the above mentioned mixture. Antibiotics can also be given by inhalation, but in all cases the patient's sensitivity to the drug should be tested. Counter attracting hot foot baths, mustard plasters on the calves, and inhalation of humidified oxygen are recommended to children. Air in the room where a sick child is treated should be moist.

Subglottic laryngitis (false croup) is a variety of acute catarrhal laryngitis which develops in the infraglottic space. It occurs in children ageing from 2 to 5 and is associated with the anatomy of their larynx (narrow lumen and loose connective tissue in the infraglottic space). The onset of the disease is as a rule connected with acute inflammation of the mucosa of the nose or the pharynx. False croup occurs mostly in children who tend to develop laryngospasm and suffer from diathesis. The onset of the disease is sudden: an attack of barking cough occurs during night sleep. The child wakes up and tosses in his bed. Breathing becomes very difficult and whistling; inspiratory dyspnoea is pronounced. The nails and the visible mucosa become cyanotic. The child is frightened and this intensifies coughing. Inspection of the child reveals retraction of the soft tissues of the jugular fossa, supra- and subclavicular spaces, and the

epigastric region. This condition lasts from a few minutes to half an hour; the child then sweats excessively and his respiration becomes almost normal. The The laryngoscopic picture in subglottic laryngitis is characterized by ridge-like swelling of hyperaemic mucosa in the infraglottic compartment.

Treatment includes common hygienic measures, ventilation in the room, and therapeutic measures. The child is given warm milk and mineral water. Poultice and mustard plasters should be applied to the neck. Hot foot baths are also effective. The attack of asphyxia can be aborted by touching the posterior wall of the pharynx with a spatula thus stimulating the vomiting reflex.

Haemorrhagic laryngitis develops mostly as a complication of toxic influenza. Morphologically the process is characterized by haemorrhage into the thickness of the laryngeal mucosa, especially into the vocal and aryepiglottic folds (in the form of petechiae and large maculae). A permanent symptom is dry cough, usually in the morning. Later streaks of blood appear in the sputum during expectoration of crusts; scarlet blood is expectorated less frequently. Haemorrhage can develop in some cases with subsequent asphyxia.

Treatment. A 10 per cent calcium chloride solution, vitamin K (a tablet of 0.015 g 2 times a day), and an expectorant are quite effective to arrest small haemorrhage. The subsequent treatment is the same as for acute laryngitis.

Submucous laryngitis (angina laryngea). This is an acute inflammation of the lymphoid tissue of the larynx. These are substantially the same as in inflammation of the palatine tonsils. The patient complains of painful swallowing, painful turning of the head, and dry throat. The voice is changed in some cases; the larynx can be stenosed significantly to impede respiration. The body temperature is often 37.5-38^{0}C. Palpation of the neck reveals enlarged and very tender lymph nodes, usually on one side. Laryngoscopy shows hyperaemia and infiltration of the laryngeal mucosa on one side or over a circumscribed area. Separate follicles with punctate patches can sometimes be seen. If the disease runs a prolonged course, an abscess can develop on the tongue surface of the epiglottis.

Treatment is the same as for acute catarrhal laryngitis, but antibacterial preparations should be given in bigger doses. Tracheostomy is indicated for significant stenosis.

Phlegmonous laryngitis is a suppurative inflammation of the submucous layer, possibly of the muscles, tendons, and the laryngeal perichondrium. Its aetiological factor is infection (staphylococcus, streptococcus, etc.). The disease occurs mostly in males ageing from 20 to 35. The affection can be circumscribed and diffuse.The patient complains of severe pain on swallowing, especially if the phlegmona is located on the tongue surface of the epiglottis and the arytenoid cartilages. If the glottis tissues are affected, the first symptom is hoarse barking cough and respiratory distress (to asphyxia). The body temperature is high. Examination reveals inflammation of the regional lymph nodes. Laryngoscopy reveals hyperaemic and infiltrated laryngeal mucosa with sites of necrosis. The formation of an abscess is characterized by circumscribed swelling; pus can be seen through the thinned mucosa. Mobility of some laryngeal structures is strongly restricted.

Treatment. The patient must be taken to hospital. Tracheostomy is indicated for increasing stenosis. Local and general antibacterial and anti-inflammatory therapy is started at the early period of the disease. If an abscess is present, it should be opened surgically. If the phlegmona spreads onto the soft tissues of the neck, external incisions are made to ensure adequate drainage of suppurative cavities.

Chronic inflammatory diseases of the larynx is in the majority of cases secondary to acute inflammations. It may follow incompletely resolved acute simple laryngitis. Presence of chronic infection in paranasal sinuses, teeth and tonsils and chronic chest infections, occupational factors, e.g. exposure to dust, fumes and other chemical components, smoking, alcohol, vocal abuse are important contributory causes. Three forms of chronic inflammatory diseases of the larynx and the trachea are now distinguished: catarrhal, hyperplastic, and atrophic.

Chronic catarrhal laryngitis is in most cases secondary to acute laryngitis. The main aetiological role of this pathology in singers, actors, lecturers, etc. is the occupational overload on the vocal apparatus. Laryngoscopy reveals congestive hyperaemia of the laryngeal mucosa, which is more pronounced in the region of the vocal folds; blood vessels are often dilated.

Treatment is aimed at eliminating the aetiological factor. The patient must rest his voice. Local therapy includes instillation of an antibiotic solution containing hydrocortisone suspension (5 ml of isotonic sodium chloride solution, 50000 U of streptomycin, and 30 mg of hydrocortisone suspension). This solution is instilled into the larynx once a day in a dose of 1.5-2 ml. The same mixture should also be given by inhalation 2 times a day. The course includes 10 sessions.

This course can be followed by inhalations of oil solution. The use of only oil and alkaline-oil inhalations should be limited, because these preparations have an adverse effect on the ciliated epithelium (inhibiting its function).

Chronic hyperplastic laryngitis is characterized by hyperplasia of the laryngeal mucosa. Local and diffuse forms of the disease are distinguished by the extent of involvement. The main complaint of the patients is hoarseness and even aphonia, which are usually due to uneven thickening of the vocal folds and paresis of the vocal muscles. Direct and indirect laryngoscopy reveal hypertrophy of the mucosa which is usually symmetrical on both sides of the larynx and in the interaryte-noid notch. This hyperplasia can however be malignant and the diagnosis of chronic hyperplastic laryngitis should be established not only by observing the clinical signs of the disease but also by the histologic and cytologic findings.

Treatment is, in the first instance, directed at removing the causative factors; talking must be prohibited. Exacerbations are treated like acute catarrhal laryngitis. If mucosal hyperplasia is significant, a 1-2 per cent silver nitrate solution is applied every other day during the course of 2 weeks.

Pachydermia laryngis is characterised by heaping up of epithelium in the interarytenoid region and vocal processes of aryttnoids. Exact aetiology is not known but disease mainly affects males who indulge in excessive smoking and alcohol. When changes are confined to the vocal processes, disease is termed as "contact pachydermia" or "contact ulcer". Hoarseness or huskiness of voice is the main presenting feature and is due to faulty approximation of cords. Hawking, i.e. constant desire to clear the throat. This is because mucus keeps sticking in the interarytenoid region. Examination shows heaping up of epithelium in interarytenoid region which may extend to vocal processes and sometimes arytenoids. On phonation, it stands out like a "cock's comb". Biopsy is essential to exclude tuberculosis or carcinoma.

Treatment is generally unsatisfactory. Surgical removal of hypertrophic tissue under operating microscope, sometimes in several sessions, may be required.

Leukoplakia or keratosis are also a localised form of epithelial hyperplasia involving upper surface of one or both vocal cords. It appears as a white plaque or a warty growth on the cord without affecting its mobility. It is regarded as a precancerous condition because “carcinoma in situ” frequently supervenes. Hoarseness is the common presenting symptom. Treatment is stripping of vocal cords and subjecting the tissues to histology for any malignant change.

Polypoid degeneration of vocal cords (Reinke’s oedema). It is bilateral symmetrical swelling of the whole of membranous part of the vocal cords, most often seen in middle aged men and women. This is due to oedema of the subepithelial space (Reinke 's space) of the vocal cords. Hoarseness is the common symptom. Patient uses false cords for voice production and this gives him low-pitched and rough voice. Vocal cords show pale, translucent fusiform swellings. Ventricular bands may appear hyperaemic and hypertrophic and may hide view of the true cords.Treatment: Decortication of the vocal cords, i.e. removal of strip of epithelium, is done first on one side and 3-4 weeks later on the other. Voice rest. Speech therapy for proper voice production.

Chronic atrophic laryngitis. Atrophic laryngitis is usually connected aetiologically and pathogenetically with atrophy of the nasal and pharyngeal mucosa. Pollution of air with dust or gases, smoking and abuse of alcohol are among the provoking factors. Patients complain of dryness, tickling and the feeling of a foreign body in the throat, and progressing dysphonia. In the early period of the disease laryngoscopy reveals bright hyperaemia of the mucosa which looks lustrous. Hyperaemia subsides at later stages and tenacious secretion appears, which thickens into dark-green crusts in the larynx. On coughing-up streaks of blood can be seen in the expectorated sputum due to destruction of the laryngeal epithelium during cough.

Treatment. The patient must not smoke or take irritating food; he should rest his voice. Preparations thinning sputum and facilitating its expectoration should be given. Throat irrigation and inhalations of an isotonic sodium chloride solution should be performed (200 ml of isotonic solution, 5 drops of a 10 per cent iodine tincture). The irrigations and inhalations are performed 2 times a day using 30-50 ml of the solution for a session. The course lasts 5-6 weeks. The procedures can be done at home in the morning and in the evening. Oil-alkaline inhalations are carried out for 3-5 days only in the presence of tenacious mucus and crusts in the larynx. A 1-2 per cent oil solution of menthol should be inhaled daily during 10 days. This preparation can also be instilled into the larynx (menthol has weak irritating and disinfecting properties and therefore the patient's sensitivity to the drug should be checked). Concurring atrophic process in the larynx and the pharynx can be effectively treated with submucous injection (into the lateral portions of the posterior wall of the pharynx) of a novocain and aloe solution. In order to stimulate the action of the glandular apparatus of the mucosa, 8 drops of a 30 per cent potassium iodide solution should be given per os 3 times a day during two weeks.

Chondroperichondritis of larynx is associated with spreading of the inflammation from the soft tissues onto the cartilage. Acute and chronic processes are distinguished.

Symptoms. These mainly depend on the location of the focus. Indurated soft tissues usually circumscribe the inflamed part of the cartilage; external and internal purulent fistulae are periodically formed. Laryngoscopy reveals indurated and oedematous areas of the mucosa, which narrow the lumen of the larynx. The disease is usually long-standing; it can persist for several months and even years.

Treatment of acute chondroperichondritis includes administration of big doses of antibiotics and sulpha drugs which eliminate inflammation. Physiotherapy should be prescribed depending on the character of the inflammation: UV light, UHF- and SHF-therapy, ion-galvanization of the larynx with calcium chloride, chymotrypsin, and potassium iodide; warming compresses are effective. The patient with chondroperichondritis should be given pasty non-irritating food. Tube feeding is not recommended, because the gastric tube can irritate the laryngeal tissues. The general reactivity of the body can be increased by biological stimulants (aloe, vitreous body, etc.). Surgical intervention is indicated for an abscess which should be emptied to remove the necrotized tissues. The presence of fistulae is also an indication for surgery, by which the fistula is opened and necrotized tissue removed.

DISORDERS OF LARYNGEAL NERVES. Sensory and motor disorders of the laryngeal nervous apparatus are distinguished.

Disorders in the sensibility can be central and peripheral. Central disorders cause bilateral affections. The only exception is hysteria. The sensory disorders are anaesthesia, hyperaesthesia and paraesthesia.

Anaesthesia usually occurs in injuries to the larynx and the superior laryngeal nerve. Surgical intervention on the organs of the neck can also cause anaesthesia. Anaesthesia usually causes an insignificant subjective feeling. But in some cases it can be dangerous because food and liquid can pass into the airways.

Hyperaesthesia can be of various intensity. In some cases it can take the course of neuralgia. If sensitivity increases, perverted sensations may appear (paraesthesia). Hyperaesthesia is usually caused by the systemic nervous diseases (neurasthenia, hysteria) or

changes in the peripheral nerves of the mucosa. The disorder is characterized by the tingling sensation when breathing and talking; sometimes the patient feels an urge to cough-up mucus.

Paraesthesiae can be manifested by various sensations such as burning, tingling, foreign body in the throat, spasm, and the like.

Treatment. This includes measures acting on the nervous system, such as immersion and pine sedative baths, vitamin therapy, aloe, rational labour and leisure, etc. Novocain block is effective when administered into the ganglion or the conduction routes.Physiotherapy of peripheral affections includes intra- or extra-laryngeal galvanization, diathermia, and the like.

Motor disorders. A weakening, or paralysis, of the laryngeal muscles may be associated with their lesions or disturbed nerve supply.

Distinction should be made between functional paralyses, which in most cases are caused by affections of the constrictor muscles of the glottis, and organic paralyses which are due to lesions of the laryngeal nerves, above all of the dilator muscles of the glottis.

Disturbances of the laryngeal motor function may originate both in the central and the peripheral nervous systems. The cause of *central paralysis* may be syringomyelia, tabes, hysteria, as well as gummas, tumours, hemorrhages in the cerebral cortex, bridge of Varolius, medulla oblongata, and sometimes in other parts of the brain stem. Sometimes, inferior laryngeal nerve paralysis of central origin is accompanied by simultaneous lesions of other neighbouring cranio-cerebral nerves, viz., the 9th, 10th, llth and 12th.

Peripheral paralysis follows an injury to the recurrent laryngeal nerve which on its relatively long path may be compressed by mediastinal tumours, aortic aneurysms, goitre and carcinoma of the esophagus, or it may result from affection of the nerve itself, such as alcoholic and syphilitic neurites in tabes, and neuritis of rheumatic origin. Lesions of the inferior laryngeal nerve are frequently caused by excision of the goitre. Laryngeal examination reveals that the vocal cord on the paralyzed side, instead of being abducted, lies half-way between the position during respiration and during phonation, that is, in the intermediate position, otherwise known as the cadaveric position.

The clinical symptoms of unilateral paralysis of the recurrent nerve are slight. The affection of the vocal cords is relatively mild with slight hoarseness, quick vocal fatigue and free respiration. Bilateral paralysis, however, endangers the patient's life and often requires tracheotomy, since both cords lie so close to the median line as to narrow the glottis to the point of asphyxia.

Apart from neuropathic or organic paralyses of the larynx there are frequent *myopathic, functional paralyses* caused by all kinds of inflammations in the larynx or vocal abuse by public speakers, singers, teachers, etc. The lesion more often affects the vocal muscles.

Paresis of both vocal cords prevents their full approximation in phonation, and the glottis in such cases is a long and oval chink pointed at both ends. The voice becomes hoarse, in some cases there may be complete aphonia. It should be noted that myopathic and neuropathic paralyses are clinically very much alike and offer completely identical signs in laryngoscopy. It should also be borne in mind that paralysis of the inferior nerve is a symptom of constitutional, and perhaps very serious disturbance.

Treatment. The primary measure is to remove the causes of the disease. Prolonged vocal rest, treatment of chronic inflammation and the wide use of electrotherapy with galvanic and faradic currents may be recommended to hasten cure. At the onset of the disease, these measures are usually effective. The chances of recovery from neuropathic paralysis of the laryngeal muscles are strictly contingent on the outcome of the basic disease.

STENOSIS OF THE LARYNX is the narrowing of its lumen interfering with normal passage of air to the dependent airways.

Acute stenosis occurs suddenly or develops within a comparatively short period of time. The main pathophysiological factors that should be assessed immediately in acute stenosis of the larynx are the following: (1) the degree of external respiratory insufficiency; (2) the body reaction to oxygen deficit.

The body reserves cannot be realized during acute development of stenosis. The adaptation reactions of the body are respiratory, haemodynamic, blood and tissue reactions. The respiratory reaction is manifested by dyspnoea which increases ventilation of the lungs due to deeper breathing and higher respiratory rate. The haemodynamic compensatory reactions are characterized by tachycardia and increased vascular tone, which increase the minute blood volume 4 or 5 times. These mechanisms can to a certain degree lessen hypoxia and hypercapnia; insufficient lung ventilation can be compensated for on the condition that a certain minimum volume (individual for each particular patient) of air is inhaled. In these conditions, increasing stenosis induces severe pathological reactions.

Acute stenosis of the larynx can be caused by local inflammatory diseases such as the laryngeal oedema, acute infiltrative or abscessing laryngitis, chondroperichondritis of the larynx or submucous laryngitis, local non-inflammatory processes, various injuries, foreign bodies, etc., acute infectious diseases such as measles, scarlet fever, diphtheria and the like, systemic diseases of the body such as diseases of the heart and vessels, of the lungs, the kidneys, etc. Depending on the degree of stenosis, stridor develops. Examination reveals retraction of the supraclavicular fossae and the intercostal spaces; respiratory rhythm becomes upset. All these symptoms are associated with increasing negative pressure in the mediastinum. A patient with pronounced stenosis develops fear and motor excitation (the patient tosses in his bed and tries to run). The face is pale , the patient perspires; the heart activity and the secretory function of the stomach and the excretory function of the kidneys are upset. If stenosis persists, the pulse is accelerated, the lips, the nose and the nails become cyanotic due to accumulation of carbon dioxide and the oxygen deficit and decentration of blood circulation. Inspiratory dyspnoea develops si-multaneously.

The following stages classified in the *clinical course* of stenosis: stage I, compensation; stage II, subcompensation; stage III, insufficiency or decompensation; and stage IV, asphyxia.

At the stage of compensation the patient does not develop respiratory distress at rest, but tachypnoea develops during walking; the width of the glottis is 6-7 mm.

At the stage of subcompensation the patient develops inspiratory dyspnoea at rest, with involvement of the accessory muscles in the respiratory act; the intercostal spaces, soft tissues of the jugular and the supraclavicular fossae are retracted; stridor, pallor and restlessness are characteristic. The glottis is 4-6 mm.

The insufficiency stage is characterized by shallow and accelerated respiration; the patient assumes a forced position (half-sitting in his bed and holding fast on the headrest or some other object). The larynx moves to maximum possible distance up and down. The face is pale and cyanotic; the patient is frightened, he perspires; his lips, the nose tip and the terminal phalanges are cyanotic; the pulse is fast. The glottis is 2-3 mm wide.

At the stage of asphyxia, respiration is hardly possible and discontinues at any moment. The width of the glottis is about 1 mm. The heart activity is distressed, the pulse is fast and thready, the skin is grey and pallid. In severe cases the patient is unconscious; exophthalmos is characteristic; the patient urinates and defaecates involuntarily; death ensues quickly.

Treatment depends on the cause and stage of acute stenosis. Emergency care in stenosis caused by oedema and inflammation of larinx: antiinflammatory therapy ; use of corticosteroids (3-5 mg. per kg. mass). Gglycocorticoids give antiinflammatory, as well as antiallergic affect; use of lytic mixture, consisting of 2% solution of papaverine, 1% dimedrol solution; 2.5% solution of aminasine , in clinical conditions. This mixture is injected intramusculary. Simultaneous intravenous injection of 20% solution of glucose, hydrocortisone, 2.4% solution of euphillini, 10% solution of Ca gluconate, 5% solution of ascorbinic acid; inhalation of antiedemic mixture : ephedrine hydrochloride 5% -1; adrenaline hydrochloride 0.1% - 1.0; pipolfen 2.5 – 1.0; humid oxigen, hot bath .

Decompensation (stage III) should be treated surgically: immediate tracheostomy or intubation are indicated. The patient can be intubated with elastic tubes used for intratracheal

anaesthesia in intensive therapy departments. Asphyxia (stage IV) requires urgent coniotomy and then tracheostomy.

Chronic stenosis arises due to persistent morphological changes in the larynx and the adjacent organs and tissues. As a rule, chronic stenosis develops slowly and gradually. Causes of chronic stenosis of the larynx are quite varied. Common causative factors are (1) chondroperichondritis (traumatic, infectious, radiation); (2) disturbed mobility of the cricoarytenoid joint; (3) dysfunction of the inferior laryngeal nerves due to toxic neuritis, following strumectomy, compression by a tumour, and the like; (4) tumour, tuberculosis, syphilis, or scleroma.

Patients with chronic stenosis of the larynx often develop bronchitis and emphysema due to long-standing hypoxia; bronchopneumo-nia is frequent in children. The heart is enlarged and the myocardium hypertrophied. These affections narrow the tracheal lumen and are therefore very dangerous.

Treatment of chronic stenosis is often very difficult and in some cases the lumen of the larynx is restored to normal size only after a prolonged treatment. Special dilators are used for regular artificial dilatation of the stenosed larynx. La-ryngostomy and prolonged (for some months) dilatation of the larynx by T-tubes (better plastic) give more reliable results.

Tracheotomy may be superior or inferior depending on whether the trachea is opened above or below the isthmus of the thyroid gland. The patient is placed on the operating table with his shoulders propped high on a round bolster and his head tilted far back. The skin and superficial cervical fascia are incised strictly in the midline of the neck, and the incision is carried from the lower edge of the thyroid cartilage some 6 cm downwards. The front surface of the cricoid cartilage is then exposed with blunt instruments strictly in the midline, a transverse incision made in the capsule of the thyroid isthmus lying below, and the isthmus pushed down to expose the first tracheal rings. Following the arrest of bleeding, two or three tracheal rings are cut with a sharp scalpel for insertion of the tracheotomy tube. This consists of two connected metal tubes which slide one within another. The insertion of the tube is followed by a vigorous expectoration of sputum and then by quiet respiration. The tube is fastened with a bandage applied to the neck, while the incision is sutured with one or two stitches above and below the tube. The operation is commonly performed under local anesthesia but in the event of asphyxia where time is a factor of overriding importance no anesthesia is applied.

A too big incision of the trachea and complete stitching of the skin cut may give rise to subcutaneous emphysema, which is provoked by violent cough. This condition is identified by a markedly swollen neck and characteristic cracking sounds produced by the movement and bursting of air bubbles when the affected areas are being palpated. In such cases, the stitches of the wound must be loosened.

Tuberculosis of the larynx is the most frequently occurring tuberculous affection of the airways. The larynx is infected with tuberculosis mycobacteria mainly by three routes. The most common of them is contact infection with sputum expectorated from the lungs of patients with pulmonary tuberculosis. The other route of ingress is with blood (haematogenic route). The third way of infection spreading is by lymphatics. Three stages are distinguished in the development of a tuberculous process in the larynx: the first stage is infiltration; the second is characterized by formation of ulcers; and the third stage is associated with affection of the cartilages and perichondrium. The vocal function is upset only in cases when the vocal or vestibular folds and the interarytenoid notch are involved. Development of the pain syndrome is associated with infiltration in the epiglottis, posterior surface of the arytenoid cartilages and the ary-epiglottic folds.

The laryngoscopic picture corresponds to the stages of the pathological process. But the most common sites of infection residence should be remembered. These are the interarytenoid space, arytenoid cartilages and the adjacent parts of the vocal cords. Tuberculosis of the larynx progresses slowly.

Treatment. This should first of all be aimed at elimination of the main disease (usually pulmonary tuberculosis). Streptomycin should be administered intramus-cularly, 2 times a day. Not less than 60-80 g of streptomycin should be given in one course. **PASA,** phthivazid and other antituberculous preparations are also used. The combined local use of these preparations is believed to have the best effect. Ulcerated surfaces should be cauterized with trichloroacetic acid after preliminary anaesthesia of the larynx with a 5 per cent cocaine solution and a 0.5 per cent citral solution. Anaesthetics should be used to prevent or relieve pain during swallowing.

Tuberculosis of the pharynx occurs comparatively rarely. The clinical *symptoms* are characterized mainly by severe pain during swallowing of both solid food and liquids. As a rule, joining secondary infection accounts for the fetid breath. These symptoms are associated with formation of ulcers mainly on the palatine arches and the mucous membrane of the posterior wall of the pharynx.

At later stages, the diagnosis is established by the clinical picture, Pirquet's test, microscopic study of granulation taken from the region of the ulcers, and by general examination.

Tuberculosis of the nose. The patho-morphological substrate is the infiltration which is accumulation of tubercles in the submucous layer. Destruction or curd-like degeneration of these tubercles causes ulcers. Crusts are formed in the involved side of the nose. When the crusts are removed, accumulations of translucent tubercles can be seen in the mucous membrane.

Tuberculous otitis arises when infection is spread by the bloodstream from any distant primary focus, usually from the lung. The morphological changes are characterized by formation of specific tubercles which later undergo caseous degeneration. Soft tissues undergo purulent disintegration and the tuberculous granulations rapidly proliferate.If the tympanic membrane is affected, isolated tubercular foci develop in it which decompose and cause multiple perforations. Extension of the process to the bone dissolves the osseous tissue under the action of invading granulations. Tuberculous otitis usually runs a chronic course.

Treatment should begin with active general anti-tuberculosis therapy. Radical operation on the ear is indicated for carious-granulation process in the middle ear together with the general anti-tuberculosis therapy.

Scleroma (rhinoscleroma) is an endemic disease occurring in the middle East, Eastern Europe and Central and South America, in the Western regions of Ukraine and Belorussia. The disease attacks the young. The causative agent is believed to be Friesch-Volkovich bacillus *(Klebsiella rhinoscleromatis)*. The pathomorphological substrate is infiltrate consisting of fibrous connective tissue rich in plasma cells and vessels, among which are specific for scleroma Mikulicz's cells, Friesch-Volkovich capsule cells included into the Mikulicz's cell vacuole. The infiltrate also contains hyaline globules (Russel's bodies).

Three stages are differentiated in the course of the disease: the first stage is nodular-infiltrative; the second stage is diffuse-infiltrative or specific; and the third stage is regression (scarring). The so-called atrophic form sometimes occurs which is characterized by atrophy of the mucosa. The main signs of scleroma infiltrates is the absence of ulceration. The incubation period of the disease is very long. The onset of the disease is characterized by atrophy of the mucous membrane which is attended with formation of thick crusts of tenacious mucus. Separate infiltrates can be seen. Scleroma affects mostly the nasal mucosa and the patient complains of dryness in the nose in the early stage of the disease. Rhinoscopy reveals infiltrates in the form of flat or tubercular pale-pink patches; these are absolutely painless to palpation. The infiltrates narrow the lumen of the nasal cavity, the vestibule of the nose, choanae, the nasopharynx, and the larynx. In other words, scleroma infiltrates arise mainly in physiologically narrow areas. Firm scars are later formed at the site of infiltrates. The scars stretch the surrounding tissues and stenose various parts of the respiratory tract.

Treatment can be conservative and surgical. Conservative treatment includes intravenous administration of embichin with 20 ml of a 40 per cent glucose solution. The dose should be gradually increased from 1-2 mg to 4 mg. Streptomycin should also be injected intramuscularly,

twice a day, and by instillation into the trachea once a day. Radiation therapy (a total dose of 3000-4000 R) is sometimes effective. Surgical treatment includes excision of the infiltrates and scars, their elimination by cryosurgery (liquid nitrogen).

Syphilis of the pharynx. All stages of syphilis can occur in the pharynx. Hard chancre develops as an erythematous, erosive, and ulcerous lesions. The process is usually unilateral; it can last several months. The disease is associated with unilateral regional lymphadenitis. A hard chancre can develop on the lip, buccal mucosa, tongue, soft palate, or a palatine tonsil. Secondary changes in the pharynx occur 6-8 weeks following the appearance of a hard chancre. Simultaneously similar lesions on the skin develop (roseolas and papules). Roseolas appear on the palatine arches and the tonsils. The specific process in the palatine tonsils differs from acute tonsillitis by normal body temperature and painless swallowing. Pharyngoscopy during the secondary stage is characterized by a copper-hued diffused hyperaemia which extends to the palatine arches, the mucosa of the soft and hard palate. The secondary-stage papules are greyish-white circular eruptions elevated over the surrounding tissue and circumscribed by a red margin. These lesions (plaques) often ulcerate, and can be seen on the tip and sides of the tongue, on the mucosa of the cheek and the hard palate. *Treponema pallidum* can be found in great quantity in the discharge from the ulcers. The oozing plaques become enlarged and form a large condyloma sometimes crowned with papillar formations. This stage of the disease is characterized by polyadenitis affecting the cervical, occipital, supratrochlear and other lymph nodes.

Diagnosis of the secondary syphilis is established by the positive Wasserman reaction, by discovering *Treponema pallidum* in the papular contents, and by inspection of the pharynx.

Treatment is general and specific. Local treatment includes gargling with weak disinfectant solutions (hydrogen peroxide, camomile tea, and the like).

Syphilis of the nose occurs as a primary sclerosis of the secondary and tertiary stages of the disease. A hard ulcer (chancre) occurs rarely. Development of the syphilitic process in the nose causes the reaction on the part of the occipital and submandibular lymph nodes. They swell but their palpation is painless. Inspection reveals a smooth painless erosion in the vestibule of the nose. The erosion is red and is 0.2-0.3 cm in size. The margins of the erosion have a ridge-like thickening. An infiltrate, whose consistency resembles that of a cartilage, can be palpated under the erosion. Secondary syphilides appear as erythema and papules. Such discharge in neonates or nursing infants suggests examination of the baby for possible specific disease. The tertiary stage of syphilis occurs more frequently than the two former stages. This is characterized by diffuse infiltrates or decomposing gummas. A gumma can be located on the mucosa, the bone, periosteum and cartilage. Bony tissue undergoes necrosis and sequestration.

Syphilis of the larynx manifests itself as a systemic disease. Hard chancre in the larynx occurs in extremely rare cases. The secondary stage is manifested by erythema simulating catarrhal laryngitis with involvement of the mucous membrane of the vocal folds, arytenoid cartilages and epiglottis, and also by papules and large condylomas. The tertiary stage of syphilis of the larynx occurs mainly in males ageing from 30 to 50. Gummas are located mainly in the epiglottis, and less frequently in the interarytenoid notch and on the vestibular folds. When located in the infraglottic space, a gumma appears in the form of a symmetric infiltrate.

Syphilis of the ear. Secondary syphilis (roseola, papule) affects the skin of the external ear simultaneously with similar affections of the other parts of the skin. Affections of the internal ear are most important. Congenital and acquired forms are distinguished. In congenital form, the affections of the internal ear become evident at the age of 10-20. Hutchinson's triad is pathognomonic: Hutchinson's teeth, parenchymatous keratitis, and cochlear neuritis. Hearing disorder is the leading symptom; it is always bilateral. *Treatment* is specifically antisyphilitic.

Tumours of the upper respiratory tract.

Morphologic classification of the upper respiratory tract tumors.

I type. Highly differentiated tumors - benign (osteoma, chondroma, lipoma, fibroma, angioma, adenoma, neurinoma, etc)

II type. Malignant tumors - Cancer

1 grade. Highly differentiated cancer;
2 grade. Cancer of the moderate degree of maturity;
3 grade. Low-differentiate cancer.
4 grade. Undifferentiated cancer

International classification of cancer

The concept about the stage of the process in ENT oncology: the stage of the process can be I, II, III, IV; it is determined in the form the combinations of symbols T (tumor) (1-4), N (nodulus) (0-3), M (metastasis) (0,1).

Neoplasms of the upper respiratory tract average 3-4 % of all tumours localization. Tumours of larynx average more than half neoplasms of upper respiratory tract, tumours of pharynx are on the second place, tumours of nose and paranasal sinuses are on the third place. Neoplasms of ear are met much rarely.

Benign neoplasms are characterized by high degree of differentiation, not infiltrative and not destroying growth (even during rapid growth); they don't give metastasis, don't relapse and are resistant to radial therapy.

Benign tumours of upper respiratory tract and ear have different structure, because they may develop from all tissues forming these organs. It may be epithelium, soft tissues, osseous, cartilaginous, nervous tissues. Papillomas, hemangiomas and fibromas are the most frequently occurred benign tumours of nose , pharynx and larynx. In the paranasal sinuses which are affected by benign tumours more rarely than nasal cavity, osteoma is usually found. Osteoma usually becomes localized in frontal sinus, rare in the ethmoid sinus. Benign odontogenus tumours, such as cementoma are usually occurred in maxillary sinus.

Papilloma is on the first place among the most frequently occurred benign tumours of upper respiratory tract. This tumour develops from flat and transitional epithelium. Depending on quantity of connective tissue in tumours stroma, tumour may be soft or hard. The most often papilloma becomes localized in larynx, and may occur at any age. Children are effected by papilloma between a year and half and five years old. Boys are affected twice more often than girls, men are affected a four times more often than women.

Multiple papillomas are found on vocal cords, extend down to subglottic space and trachea, restrict gap of larynx and cause difficult breathing. During first five years of child's life papilloma grow fast, often relapse in spite of therapy, but almost are never malignant. During puberty papillomas may disappear spontaneous. At adult papilloma of larynx is solitary formation on vocal cord with slowly growth. Hard papilloma with proliferous crawling growth is found in every fourth case. Such growth causes transformation papilloma to flat (squamous) cell carcinoma. It is found in 15-20% and gives grounds to regard papilloma of larynx at adult as obligatory precarcinoma.

Vascular tumour among benign tumours of upper respiratory tract and ear are the second (take second place). It is usually hemangioma. Angiomas are distinguished in capillary (of arterial vessels), cavernous (of venous vessels) and also there are lymphangiomas. Hemangioma usually becomes localized in nose and pharynx, more rarely in larynx and ear . As a rule it has wide base ,especially in pharynx. It we want establish a cause of nasal bleeding, we should remember about vascular tumour and thoroughly carry out rhynoscopy after control of bleeding. Such benign tumour as fibroma is found mainly in larynx and nasopharynx. In larynx fibroma proceeds benignly, it is usually solitary tumour, like millet or no bigger than a pea. It settles down on free side of vocal cord. Fibroma of larynx manifests by violation of voice, sometimes cough and very rarely hard breathing (when the tumours is big like cherry). Fibroma is removed by endolaryangeal access during laryngoscopy with laryngeal forceps.

Fibroma of nasopharynx is the most often tumour of this localization. It is also called angiofibroma or fibroma of skull base.

Tumour is occurred at boys and youths; it is found in of nasopharynx, often penetrates in nasal cavity through choanas. This tumour with expansive growth causes atrophy of osseous walls (in consequence of compression) and can grows in cavity of skull. Fibroma of nasopharynx

grows rapid and often relapses even after radical removal of tumour. Both these circumstances let us fall youth angiofibroma under the category of border tumours. Clinic of nasopharynx fibroma is enough typical: increasing difficulty of nasal breathing , then impossibility of nasal breathing through one nasal passage (then through both passage), stuffiness in the ear, relapsing nasal bleeding. During posterior rhinoscopy tumour of purple colour is determined and during palpation we can find that the tumour is solid and uneven. Owing to superficial arrangement of vascular vessels investigation of pharynx quite often is accompanied by bleeding.

Let's pay attention to another tumour. It is tumour of drum glomus in the region of vena jugularis bulb and it called tympanojugular paraganglioma. At onset of the disease the tumour is showed itself by stuffiness in the ear and by subjective noises in it. During otoscopy we can find pink and bulging ear drum. As tumour grows and destroys bones the patient takes note of reduction hearing, dull pain in the ear, bleeding from the ear, paresis of facial nerve, dizziness, symptoms of damages of 9th,10th,11th,12th cranial nerves. It is difficulty to diagnose tympanojugular paraganglioma. Usually we can give diagnosis in several year after beginning of tumour's growth. Main treatment is surgical.

Malignant tumour. Frequency of damages of different parts of upper respiratory tract and ear by malignant tumours is equal: larynx is affected in 67%, pharynx in 18 %, nose and paranasal sinuses are affected in 14%,ear in 1% of observations. Frequency of damages by tumours differs at children: nose and paranasal sinuses are affected in 35%, nasopharynx in 30%, oropharynx in 19%, meddle ear in 16% of cases, cancer of larynx at children occur very rarely.

The most often malignant tumours are found in larynx at adult, and almost always it is flat (squamous) cell carcinoma, rarely it is basal cell carcinoma or sarcoma. Cancer of larynx is on the fourth place among all cancers at men. It is not as frequent as cancer of stomach, lungs and esophagus. At women cancer of larynx is on one of the last places among other cancerous diseases. Many patients with cancer of larynx are admitted for treatment on last stage of disease. Clinic of cancer of larynx in beginning depends on localization of tumour. Patient's complaints are the very usual, occurring in many disorders of larynx. So, when the tumour is found on epiglottis, patient complains to sensation of discomfort on swallowing, a feeling of a foreign body in the throat. Pain in the throat (spontaneous or on swallowing) disturbs the patient as tumour continues to grow and ulcerate, also the pain radiates to the ear. Small nodular tumour of pale-pink or grey colour is found during laryngoscopy, quite often with ulceration areas covered by coat. It is difficult to find tumour on endophytic growth of tumour, especially in the region of epiglottis's base. That is why in questionable cases it is necessary to perform larynogoscopy with retraction of epiglottis after anesthesia. The beginning of cancer of larynx's upper floor (cord of vestibule, ventricle of larynx) doesn't accompany lonely by subjective symptoms, excepting such light symptoms: changing voice's trimbre, weakness. Laryngoscopy reveals thickening of vestibule cord, more marked in its front region. Vocal cord may be covered by enlarged cord of vestibule or by infiltrated mucous membrane of ventricles of larynx.

It takes place when exophytic growth of tumor is observed. In case the growth of tumor is endophytic the vocal fold is pink, has diffusive **intumescence,** sometimes it may have spindle shaped form. The mobility of the affected fold can be limited. The unilateral affection is a very important diagnostic symptom of the initial stage of the disease. Unilateral localization makes it possible to exclude the inflammatory process and it is necessary to make a differential diagnosis with such infectious granulomas as tuberculosis and syphilis. The final diagnosis is made after carrying out biopsy.

At the initial stages of the affection of the lower part of the larynx the symptoms are very scanty and vague. Large tumor causes the breach of vocal and then of respiratory functions such as the muffled voice, slight dyspnea, hoarseness and increasing difficult breathing. The tumor which grows exophytic may be discovered with the help of the indirect laryngoscopy.

The symptoms which appear with the further growth of tumor very little depend upon the region of the initial localization. They become common for cancer of larynx (hoarseness or aphonia, cough, sanguinolent sputum, pain on swallowing which irradiates in the ear, increasing

difficult breathing). The tumor sprouts in cartilages of the larynx, causing chondroperichondritis. The further growth of the tumor leads to decompensated stage of laryngostenosis; patients lose weight because of cancerous intoxication, there is an erosive bleeding that often causes death. Metastatic spreading is carried out in the regional lymphatic apparatus of the neck, distal metastases are found very seldom and lately.

The choice of the method of treatment depends upon the stage of cancer of larynx, its localization and character of tumoral growth. The treatment is combined or even complex. It is better to use the combined treatment together with radiotherapy at the first stage when there is limited spreading of the tumor. If a patient undergoes half of the course of radiotherapy and the tumor becomes smaller than half as much, then radiotherapy is continued, if there is no effect, the surgical treatment is recommended to the patient. When you prescribe the radiotherapy for your patient you should take into consideration that cancer of the middle part of the larynx is more radiosensitive, cancer of the vestibule of the larynx is less radiosensitive and the cancer of the lower part is radioresistant. In case of spreading tumors of the first part the surgical treatment is carried out. There are various surgical interventions as to the cancer of the larynx depending on the spreading of the tumor:

a) at the initial stages of cancer of the larynx middle part it is possible to carry out endolaryngeal removal of the tumor;

b) in case of the thyreotomy or laryngofissure when there is limited affection of the middle part of the larynx, the external access of tumor removal is used. This treatment is also possible in case of pharyngotomy (suprahyoid, infrahyoid or lateral) and affection of vestibular part of the larynx and lower part of pharynx;

c) when there are limited affections of larynx with the tumor, larynx resection is used (horizontal, diagonal, frontal, sagital).

This operation is kind of saving of organ.

d) Laryngectomy or extirpation of the larynx is the removal of the whole organ; it is used when it is impossible to preserve the organ.

e)Dilated laryngectomy is the removal of the larynx with the of the tongue.

Comminuted treatment consists of use of the surge and radial methods. Including this fact there are possible the next variants as so:

a) operation with following radiotherapy of the regional metastasing zone as a prophylaxis;

b) radiotherapy at the first stage and if there is no an excessive effect after the half doses affection, then the surgical operation is indicated;

c) the “sandwich”- radiation: at first- the gamma-therapy half doses, then operation and the second doses of the gamma-therapy on to the metastasing region.

Chemotherapy is usually used as a supplemented method to the basic one – radial or surgical.

Results of treatment of a cancer of larynx are estimated by the fifth-years survival rate all observations report that in all stages of diseases the most effective is a combined treatment as this – operation with following irradiation of the regional lymphatic outflow region.

MALIGNANT AND HIGHER MALIGNANT TUMORS OF THROAT.

By the rate of morbid affection cancer is of the first place, but tonsillary tumors occupy the second ones. The differentiated malignant connective and especially neuroectodermal tumors of pharynx are rarely registrated . All these neoplasms, more oftenly, develop in rhinopharynx – 53%, some rarely in oropharynx – 30%, some more rarely – 17% of observations in laryngopharynx.

In pharynx the most frequents is an endophytic carcinoma – the tuberous infiltrate with ulceration, more rarer is an exophytic form – the morphologic formation on large base as a cauliflower. Also there is observed the mixed form.

If the tumor localized in rhino-pharynx, then the early signs are the difficult nasal breathing, headache, tinnitus, decreased hearing, but in case of the neoplasm ulcerating there are

a mucous bloody and sanguine purulent nasal discharges. As a consequence, if the tumor fills in the rhinopharyngeal cavity, the clinic features are the changed vocal timbre, rhinolalia clause. Symptoms of the cranial nerves impairment report about a prolonged terminal neoplasm process.

For the oropharyngeal carcinoma at the early stages there are sensation of foreign body, painfulness during swallowing (oftenly accompanied with irradiation into ear. Then the signs apply which are caused with tumoral germination and involving of the chews, root of the tongue and by collateral edema the larynx, too. Decomposition of the tumor and pain increasing during swallowing lead to hemoptysis and cachexia.

Cancer of the laryngopharynx usually develops in the recesses performs, some more rarely on the posterior wall and retrocartigeal region. At first a patients complain of sensibility of foreign body during swallowing and periodical pains in throat. Tumoral germination is accompanied with symptoms of laryngeal affection – hoarseness and difficult breathing. Also, there are a narrowing of the recesses performs and accumulation of saliva inside it, but in case of the postcricoidcarcinoma – moreover there is an edematic arytenoid cartilages and, oftenly, a rotation of larynx around the vertical axis.

Cancers of pharynx have a tendention to frequent metastasing. Regional metastases appear in the lymphonodes of neck - the profound jugular chain and oftenly in the retropharyngeal lymphatic nodes. Distant metastases are in bones, lung, liver and other organs.

The laryngeal cancer diagnoses is based on anamnesis, endoscopic and radial examination. But the biopsy means mainly in this diagnosing. Differential diagnosis should be with the infections granulomas.

Treatment of the cancer of the rhinopharynx is complex: irradiation and chemotherapy. If the tumor is on the posterior wall of pharynx, the cryosurgery method is indicated. For treatment of the cancer of the laryngopharynx is better to use, also the combined, but in another consequence – at first by surgical operation (enlarged extirpation of the laryngopharynx with resection of the cervical part of gullet) with following radiotherapy.

Among the pharyngeal neoplasms, there is special and most malignant group – the lower differentiated (radiosensitive) tonsillary tumors. They developing out of the lymphoid tissue compounds and being a higher radiosensitive, these tumors, moreover, have a supplementary characterize clinic symptoms. Tier clinic signs are:

1. rapid infiltrative germination;
2. early metastasing in to regional lymphatic nodes, besides these metastases, as a rule , enlarged more quickly than the primary tumor;
3. a very excessive tendency to generalization manifesting as a multiple metastases in distant organs.

More often the radiosensitive neoplasms develop out of the palate tonsils, but rarely – the pharyngeal, tubal, lingual ones. Sometimes, the atypical localization of the primary tumors occurs, it develops in the mucous membrane of nose, larynx, trachea, where the neoplasm growths out of lymphoid tissue. At first – there is observed an enlarged one of tonsils. If the tumor locates on the palate tonsil, it usually, wouldn't disturb patient, but more rarely it would cause a sensation of foreign body in throat. Unlike the vulgar hypertrophy, this process is always one-sided. In case of the pharyngeal tonsil tumor, there is a progressing difficulty of nasal breathing, but if there is an affection of the tubar ones there is dull hear on homolateral side. The enlarged tonsil has a densive elastic consistantion during palpation. Then, tumor tends to enlargening and involves a surrounding pharyngeal tissues to the tonsil so, that it oftenly ulcerates and is accompanied with pain. The primary tumor may be enlarged over the pharynx and involves the gingival, root of the tongue, surrounding bones, but in case of affection of the rhinopharynx – into nasal cavity. In that case a chewing and swallowing are difficult. Tumor decay is accompanied with very harsh nasty odor out of oral cavity.

Oftenly, the first patient's complains of is a metastatic enlargening of the lymphonodes. If the primary neoplasm is in the palate tonsils, then the regional metastases develop inside the

retromaxillary lymphatic nodes. But in case of the pharyngeal tonsil tumor, then the regional metastases are in upper lateral cervical lymphatic, usually, in both sides. Some patients have a primary tumor without metastasing, but with tendency to germination and involving of the base of the skull - so called “secondary form” of the tonsillary tumors. Neoplasm of the lingual tonsil manifests with regional metastases in the upper lymphonodes of the profound jugular chain of neck which is on a place of the common carotic artery bifurcation. They are discovered as a densive elastic nodes which tend to quickly enlargening, compressing neural and vascular trunks so, that causing an acute pain and collateral edemas.

At present, the general therapeutic method for the radiosensitive tonsillary tumors is a radiotherapy during an adequate chemotherapy. Relapses of the tonsillary tumors, oftenly, are not on a place of the primary focus, but in region of the regional and other lymphonodes (direct organs). During relapses of the primary neoplasm and regional metastases so, there is indicated a recurrent radiotherapeutic course.

Among malignant tumors of nose and nasal sinuses, the most higher rate of localization is the maxillary sinus (2/3 of all observations), more rarely there is affected the ethmoid sinus (1/5 of all observations) and nasal cavity (1/8 of observations);very rarer localization of malignant tumor is in the frontal sinus. Into the sphenoidal sinus, the tumor germinates usually from the nasal cavity or maxillary sinus.

At the first stages the malignant tumors of nose and nasal sinuses, as a rule, aren’t diagnosed, because of a patients complains of sense of closed nasal breathing and sneezing are accounted for a signs of inflammatory process. Apparently, therefore the most higher rate (60% and over) of mistakes during the primary diagnosis of the upper respiratory tract malignant tumors just occur in neoplasms of nose and nasal sinuses, and besides a some favorable prognosis after treatment is provided by any therapy for fifth-years survival rate no more than in 35% of patients.

More oftenly, in nose and nasal sinuses there are an epithelial neoplasms, that are a various carcinomas, but the connective tumors (sarcomas) are more rarer. Sometimes, there are a lover differentiated tonsillary tumors in nose – they are: reticulosarcoma, lymphoepithelioma, also as a rarer tumors as – melanoblastoma and the specifical for nasal cavity – esthesioneuroblastoma.

Initial symptoms of the nasal malignant tumors are a patient’s complains of the one-sided sneezing, difficult nasal breathing, then there are a purulent and blood-purulent nasal discharges, headache without specific localization, teethache. Neoplasms, with primary lie in the maxillary sinus or spreading to there from nasal cavity, have a clinic features of the stomatologic disease (teethache, edema of the alveolar processus and cheek region), owing to these, oftenly, there are fulfilled an extraction of tooth, cut of mucous membrane of gingiva and other operations. Tumors of nose and nasal sinuses, sometimes, at first manifestate with late symptoms: displacement of the eyeball, exophthalmos, diplopia, partial ophthalmoplegia (limited internal mobility of the eyeball), edematic internal angle of eye, hyperlacrimation, depraved vision, neuralgia. These sins are a patient’s course of seeing a doctor who should to suspect a secondary affection of eyeball and send a patient to an otorhinolaryngologist.

Diagnosing is fulfilled with account of the have above-mentioned symptoms, also on a base of data of the anterior and posterior rhinoscopy with gives possibility to see an tumoral formation on a large base and has a grey-pink or reddish colour (but melanoblastoma is dark greyish-brown), tuberous, bleeded during palpation. The radiologic examination has an important part in diagnosing. It includes of the surveying and contrasted roentgenography, tomography, angiography, radioisotopic visualization - there are osteal destructions and focus higher concentration of the tumorotrophic radiopharmopreparation on the gamma-scintigramm that reports about tumoral genesis of the process.

Differential diagnosis of the malignant tumors of nose and nasal sinuses should be not only with benign neoplasm’s and rhinosinusitis, but the infections granulomas: syphilis, tuberculosis, scleroma must be differentially excepted. There may be helpful a specific serologic reactions,

dermal syphilitic manifestations, tuberculosis foci in other organs, examination of the nasal discharging microflora and so on.

Treatment of the malignant tumors of nose and nasal sinuses should be comminuted, including of surgical and radial therapy. Oftenly, both these general methods are confirmed by chemotherapy (general and regional).

Surgical operation, as a rule, may have a large volume, but more frequently with the external approach – the Moure's, Preucing's operations and other modifications of the rhinotomy. If it's necessary the rhinotomy is supplemented with the exenteration of orbit, enucleation, maxillary resection. After this operation formed large defect of tissues and morphologic elements in the maxillofacial region now is removed with use of complex prothesis and synthetic materials.

Malignant tumors of an ear are registrated in 0,04% of all neoplasms cases and in 0,5-1% of the upper respiratory tumors ones. Inspite of its lower rate, we need to describe them, because of they, for all that, are observed in adults and infants, are very aggressive and very lower therapeutic effect. That is enough, that the fifth-years survival rate of patients isn't over 8-10%.

If the all ear's malignant tumors compose 100%, then 85% - are tumors of the auricle, 10% - external acoustic meatus and 5% - middle ear. In this localization, the most often neoplasm are a cancer, sarcoma and melanoblastoma.

Cancer of the external acoustic meatus is as a warty nodes or flat ulcer with legibly limited infiltrated borders. By its growing the carcinoma occupies the floor of the auricle in whole, it may involve a lateral surface of head and neck. Germinating into the external acoustic meatus it causes an acute headache like as in the furuncle. The following growing of the external ear carcinoma is accompanied by infiltration and necrosis of basal and surrounding tissues, with large defects formation.

Carcinoma oftenly develops as a weeping eczema or pale granulations on thick base, cowered with easy desquamated crust, if the tumor located in the external acoustic meatus. Patients complain of itch, but then there is a progressively increased pain of the floor of the auricle and external acoustic meatus. At least a carcinoma of the external acoustic meatus is a dermal cancer, but its prognosis (despite to the dermal cancer of any other localization) is unfavorable and poor, even if we use a combined therapy. At first, there is used a radiotherapy, then an extended surgical operation. Inspite of the operative radicalism, these patients live only 1-2 years after treatment.

Cancer of the middle ear, usually, develops during the chronic purulent otitis and its clinic current an early stages has no specific features and doesn't differ from the purulent inflammatory process of the middle ear.

How do we may suspect a malignant process of the middle ear? It may be suspected on a base of frequent and rapid relapsing of granulations (which are really a tumoral tissue), infiltration in the osteal part of the external acoustic meatus, concentrically narrowing its lumen, rough paresis or paralysis of the facial nerve, limited mobility of mandible, enlarged retromaxillary lymphonodes.

To discover a malignant tumor of the middle ear in relatively early terms, then the extracted from ear tissues must be always histologically examined. Besides, it should be multiply conducted!

Treatment of the middle ear cancer as in the external ones is combined : the preoperative gamma-therapy, in II-III weeks - the intended radical operation of ear, then – the postoperative gamma-therapy. If it is possible (in case of there is no bleeding, presence of isotopics), then a doctor introduce a radioactive preparations into the operative wound.

All these methods of treatment have a some effective results. That is localization of tumor in depth and layer of osteal tissue connected with an important vital organs (large arterial and venous vessels, labyrinth, brain) leads to less using possibility of radial and surgical operation. Therefore, the malignant tumors of ear are one of the most poor chapters of the otorhinolaryngo-oncology.

Foreign bodies of ear. *Non-living.* Children may insert a variety of foreign bodies in the ear, the common ones often seen are : a piece of paper or sponge, grain seeds (rice, wheat, maize), slate pencil, piece of chalk or metallic ball bearings. An adult may present with a broken end of match stick used for scratching the ear or an overlooked cotton. Vegetable foreign bodies tend to swell up with time and get tightly impacted in the ear canal or may even suppurate. Methods to remove a foreign body include: syringing, suction, microscopic removal with special instruments (hook), postaural approach.

Most of the seed grains and smooth objects can be removed with syringing. Smooth and hard objects like steel ball-bearing should not be grasped with a forceps as they tend to move inwards and may injure the tympanic membrane. In all impacted foreign bodies or those where earlier attempts have been made, it is preferable to use general anaesthetic and an operating microscope. Occasionally postaural approach is used to remove foreign bodies impacted in deep meatus or those which have been pushed into the middle ear. Unskilled attempts at removal of foreign bodies may lacerate the meatal lining, damage the tympanic membrane or middle ear ossicles.

Living. Flying or crawling insects may enter the ear canal and cause intense irritation and pain. No attempt should be made to catch them alive, howsoever tempting it may be. First the insect should be killed by instilling oil (a household remedy), spirit or chloroform water. Once killed, the insect can be removed by any of the methods described above.

Foreign Bodies of Air Passages. A foreign body aspirated into air passages can lodge in the larynx, trachea, or bronchi. Site of lodgement would depend on the size and nature of a foreign body. A large foreign body unable to pass through the glottis will lodge in the supra glottic area while the smaller one will pass down through the larynx into the trachea or bronchi. Foreign bodies with sharp points, e.g. pins, needles, fish bones, etc. can stick anywhere in the larynx, or tracheobronchial tree.

Children are more often affected; more than half of them are below 4 years. Accidents occur when they suddenly inspire during play or fight while having something in the mouth. In adults, foreign bodies are aspirated during coma, deep sleep or alcoholic intoxication. Loose teeth or dentures may be aspirated during anaesthesia.

Symptomatology of foreign body is divided into 3 stages: 1. Initial period of choking, gagging and wheezing. This lasts for a short time. Foreign body may be coughed out or it may lodge in the larynx or tracheobronchial tree. 2.Symptomless interval. The respiratory mucosa accommodates the foreign body and initial symptoms disappear. Symptomless interval will vary with the size and nature of the foreign body. 3. Late symptoms. They are caused by obstruction to the airway, inflammation or trauma induced by the foreign body and would depend on the site of its lodgement.

Laryngeal foreign body. A large foreign body may totally obstruct the airway leading to sudden death unless resuscitative measures are taken urgently. A partially obstruction will cause discomfort or pain in the throat, hoarseness of voice, croupy cough, aphonia, dyspnoea, wheezing and haemoptysis.

Tracheal foreign body. A sharp foreign body will only produce cough and haemoptysis. A loose foreign body like seed may move up and down the trachea between carina and undersurface of vocal cords causing "audible slap " and "palpatory thud". Asthmatoid wheeze may also be present. It is best heard at patient's open mouth.

Bronchial foreign body. Most foreign bodies enter the right bronchus because it is wider and more in line with the tracheal lumen. A foreign body may totally obstruct a lobar or segmental bronchus causing atelectasis or it may produce a check valve obstruction - allowing only ingress of air but, not egress, leading to obstructive emphysema. Emphysematous bulla may rupture causing spontaneous pneumothorax. A foreign body may also shift from one side to the other causing change in physical signs. A retained foreign body in the lung may later give rise to pneumonitis, bronchiectasis or lung abscess.

Diagnosis can be made by detailed history of the foreign body "ingestion", physical

examination of the neck and chest and radiographs. Soft tissue X-ray and lateral view of the neck in its extended position. This can show radio-opaque and sometimes even the radiolucent foreign bodies in the larynx and trachea. Posteroanterior and lateral view of the chest. X-ray chest at the end of inspiration and expiration. Atelectasis and obstructive emphysema can be seen. They also give indirect evidence of radiolucent foreign bodies. Bronchograms. To delineate radiolucent foreign bodies or to evaluate bronchiectasis.

Management. Laryngeal foreign body. A large bolus of food obstructed above the cords may make the patient totally aphonic, unable to cry for help. He may die of asphyxia unless immediate first aid measures are taken. The measures consist of pounding on the back, turning the patient upside down. Cricothyrotomy or emergency tracheostomy should be done. Once acute respiratory emergency is over foreign body can be removed by direct laryngoscopy or by laryngofissure approach, if impacted.

Tracheal and bronchial foreign bodies can be removed by bronchoscopy with full preparation and under general anaesthesia.

Foreign Bodies of Food Passage may lodge in the tonsil, the base of tongue/vallecula, the pyriform fossa, the oesophagus.

Tonsil. Usually it is a sharp fish bone or a needle in one of the tonsillar crypts. It can be easily observed by oropharyngeal examination and removed.

Base of tongue or valeculla. Here again it is usually the fish bone or a needle. It can be observed by mirror examination. It can be removed as an office procedure by asking the patient to hold his own tongue while examiner holds a large laryngeal mirror in one hand and a curved forceps in the other.

Pyriform fossa. Fish bone, chicken bone, needle or a denture may lodge in the pyriform fossa. Small foreign bodies can be removed under local anaesthetic with a curved forceps as described above. Large impacted foreign bodies or those in children should be removed by endoscopy under general anaesthesia.

Oesophagus. Usual foreign bodies lodged in the oesophagus are a coin, piece of meat, chicken bone, denture, safety pin, or a marble. Children are more often affected. They have a tendency to put anything in the mouth. Playing while eating is another factor. Use of upper denture prevents tactile sensation and a foreign body is swallowed undetected. Loss of consciousness, epileptic seizures, deep sleep or alcoholic intoxication are other factors. Pieces of food may be held up in cases of oesophageal stricture or carcinoma. The first symptom of carcinoma oesophagus may be sudden obstruction from a foreign body such as a piece of meat, fruit or vegetable. Foreign body may be swallowed with an attempt to commit suicide.

Site of lodgement of foreign body

By far the commonest site is at or just below the cricopharyngeal sphincter. Foreign bodies which pass the sphincter can be held up at the next narrowing at broncho-aortic constriction or the cardiac end. Sharp or pointed objects impact anywhere in the oesophagus.

Clinical features Symptoms. *History* of initial choking or gagging. *Discomfort or pain* located just above the clavicle on the right or left of trachea. Discomfort increases on attempts to swallow. *Dysphagia.* Obstruction to swallowing may be partial or total. Partial obstruction becomes total with time due to oedema. *Drooling of saliva.* It is seen in cases of total obstruction. Saliva may be aspirated causing pneumonitis. *Substernal* or *epigastric pain* may occur due to oesophageal spasm or incipient perforation. Tenderness in the lower part of neck on the right or left of trachea. Pooling of secretions in the pyriform fossa on indirect laryngoscopy. They do not disappear on swallowing.

Plain x-rays. Soft tissue lateral view of neck, posteroanterior and lateral view of chest may show the presence and location of a radio-opaque foreign body. Radiolucent foreign bodies can be diagnosed on fluoroscopy when the patient is given a piece of cotton soaked in barium or barium-filled capsule to swallow and its passage observed through the oesophagus.

Management. **Oesophagoscopic removal.** Most of the foreign bodies in oesophagus can be removed by esophagoscopy under general anaesthesia. **Cervical oesophagotomy.** Impacted

foreign bodies or those with sharp hooks such as partial dentures located above thoracic inlet may require removal through an incision in the neck and opening of cervical oesophagus.

Transthoracic oesophagotomy. For impacted foreign bodies of thoracic oesophagus, chest is opened at the appropriate level. A foreign body which has reached the stomach may pass through the rest of gastro-intestinal tract without difficulty; stools should be carefully examined everyday.

Complications of oesophageal foreign body. *Respiratory obstruction.* This is due to tracheal compression by the FB in the oesophagus, or laryngeal oedema especially in infants and children. *Perioesophagea! cellulitis* and *abscess* in the neck. *Perforation.* Sharp objects may perforate the oesophageal wall, setting up mediastinitis, pericarditis or empyema. They may perforate the aorta and prove fatal. *Tracheo-oesophageal fistula.* Rare. *Ulceration and stricture.*

Burns of the pharynx and the oesophagus can be thermal, chemical, electrical and those induced by radiation. Thermal burns of the mouth, pharynx and the oesophagus occur mainly during swallowing of hot food (usually liquid). Inhalation of hot air, gas or vapour can also cause thermal burns. Chemical burns of the pharynx and the oesophagus are usually more severe. Corrosive liquids (strong acids or alkalies) ingested by mistake or intentionally (a suicidal attempt) cause severe burns of the pharynx and the oesophagus. Among commonly ingested poisons is concentrated acetic acid. Less frequently ingested are caustic soda, spirit of ammonia, concentrated sulfuric, nitric, hydrochloric, chromic and other acids.

If a large amount of a concentrated poison is ingested, the patient usually dies in several days because of intoxication and affection of the parenchymatous organs, and perforation of the stomach or the oesophagus, and due to deep necrosis of the walls of these organs. Three clinical degrees of pathological changes in the affected tissues in thermal and chemical burns are distinguished: degree I-erythema; degree II-blistering; and degree III-necrosis.

Weak acids or alkalies and slight thermal effect cause usually catarrhal inflammation (erythema) of the pharyngeal and oesophageal mucosa; this affection usually ends in complete recovery without leaving any cicatrices. If the muscle layer of the pharynx or oesophagus is necrotized, deep scars occur throughout the entire thickness of the organ wall. Elasticity and distensibility of the oesophageal and pharyngeal walls decrease considerably. Severe tubular stenosis occurs in such cases.

The first morphological stage is that of ***necrosis.*** It lasts 1 week. After the rejection of the necrotic masses the second stage that of ***ulcers*** begins. It lasts near 1 week. Ulcers are covered with succulent granualations. They become dense, newly formed connective tissue wrinkles, becomes scarring, tightens the esophageal walls and does its lumens less. So gradually develops the III stage that of ***granulations.*** . It lasts several weeks The fourth stage (***scarring)*** is that of formation of stenosis

There are three clinical stages of the pathology under discussion.

1. An acute period. After the admittion of the caustic substance an sharp pain appears in the pharynx, it may lead to the lost of consiocness. A bloody vomiting appears. In the first hours the symptoms of intoxication develops. First they are connected with resobtive action of the caustic substance later intoxication are aggravating because of absorption of the products of the damaged tissues dissociation. The body temperature increases and may be 39 - 40^0 C. Under objective examination on the mucosal membrane of the mouth, pharynx, esophagus they observe hyperemia, edema or necrotic patches as well as hypersalivation. In the acute stage they observe leukocytosis, partial decay of erythrocytes, high ESR, C-reactive protein, increases indices of dyphenil- amino test. All these indices allow to judge about the severity of esophageal burn and efficacy of the antiinflammatory treatment

2. Latent period or a period of sham prosperity. The patient's self - feeling is of amean severity, symptoms of intoxication are not promoted distinctly. At this period patients feel well and insist on their being discharged from the hospital. This period corresponds to the pathologoanatomical stage of granulations.

3. Period of esophagus stenosis. The patient has difficulties under eating first dense then porridge - like food. The patient has to chew the food for a long time, take some water after it. These difficulties are of constant and progressive character, but without acute pains like in the first stage of the disease. An eructation and vomiting develops after meals and gradually an esophageal obstruction may develop.

Complications of oesophageal burns are oedema of the larynx, tracheobronchitis, perioesophagitis, oesophagotracheal fistula, pneumonia, sepsis, and cachexia. First- and second-degree burns in children cause extensive reactive processes in the form of oedema of the pharynx and the larynx and hypersecretion of sputum, which seriously impair respiration because of stenosis in the pharynx and the larynx.

Treatment should begin as soon as possible. In chemical bums, the poisonous substance should be neutralized during the first 6 hours. If the poison is an alkali, the patient should be given a weak solution of acetic, tartaric, or citric acid. If it is an acid, magnesia or chalk should be given. If the antidotes are not available, warm boiled water should be given, adding half of the volume of milk or egg white. If a gastric tube cannot be passed into the stomach, the patient should drink 5-6 glasses of washing liquid and then the tongue root should be pressed down with the fingers or a spatula to induce vomiting. The stomach should be lavaged several times with 3-4 litres of liquid.

In addition, antishock and detoxicating measures should be taken in cases of second- and third-degree burns. Pantopon or morphine should be injected subcutaneously. Treat shock and acid-base imbalance by I/V fluids and electrolytes. Monitor urine output for renal failure. A 5 per cent glucose solution, haemodez, blood plasma should be infused intravenously. Parenteral antibiotics should be started immediately and continued for 3-6 weeks depending on the degree of burns.) Steroids should be started within 48-96 hours and continued for 4-6 weeks to prevent stricture. Cardiac and antibacterial preparations should also be given. If the patient can swallow, a sparing diet and much liquid should be prescribed. If the patient is unable to swallow, parenteral and rectal nutrition is indicated.

Oedema of the larynx is an indication for elimination of stenosis with diprazine, prednisolone, and calcium chloride. Severe stenosis occurs in some cases and tracheostomy is then required.

Follow the patient with oesophagogram and oesophagoscopy every two weeks till healing is complete, for the development of any stricture. If stricture develops it can be treated by : 1. Oesophagoscopy and prograde dilatations, if permeable, 2. Gastrostomy and retrograde dilatation, if impermeable, Esophageal reconstruction or by-pass, if dilatations are impossible. Patients of corrosive injuries of oesophagus may require life-long follow up.

Professional selection, professional consultations and examination.

The understanding of professional selection includes definition of condition of healthy people. Possibility of carrying out definite productive functions and to undergo the other productive activities. This medicinal selection includes all the workers, including the teenager which admit to work in factories, mills or in professional technical college.

Professional consultation concludes in formation and giving recommendation to anybody in choosing one's job with consideration about the conditions of health, partly the conditions of ENT (ear, nose and throat) organs.

Professional selection and professional consultation depend upon character of work in a period of a year may be repeated many times. For example, drivers of different types of transports must undergo two or three times of medical check up within a year. In certain cases medical check up is carried out with regularity mainly according to the type of jobs – people which are under extreme change of barometric pressure (pilots, divers, workers in deep sea beds and etc.). As a rule questions regarding the suitableness of people in certain working conditions are solved by doctors of different specializations, the participation of otorhinolaryngologist and doctors of productive hygiene is a must. The main documents which are indispensable guide to

professional selections turn up to be the orders from health ministry in Ukraine, list of professional diseases and instructions about the use of this list.

In every concrete case during professional selection must consider not only the conditions of certain organs and system. But also their functions. Such as disturbance of the function of upper respiratory tracts can temporarily or constant character. For example, difficulty in nose breathing in different chronic inflammated process which leads to breathing through the mouth. In conditions of particle production (job in which small particles are frequently inhaled when working), works, related to some other harmful chemicals, shutting off or decreasing the protection function of the nose can lead to various complications from the lower respiratory tracts: trachea, bronchus and lungs. For certain professions the nose breathing must be normal – in pilots, divers, musicians, players of wind instruments and workers of food industries and etc.

People which are suffering from chronic destructive processes of mucous membrane of nose (atrophic rhinitis, ozena, chronic polypous sinusitis and etc.), in allergic and vasomotor rhinitis are forbidden to work in chemical, food and perfume industries. During selection of people in jobs related to huge functional burden in vocals apparatus, they are absolutely forbidden to work if they suffer from ozena, serious forms of atrophic rhinitis, atrophic pharyngitis, chronic laryngitis, paresis and paralysis of vocal muscles and tumors.

Special attention is required in selection of professional with different disturbance of hearing and vestibular function of internal year. However one most consider those requirements towards different professions are not the same. Normal hearing must present in pilots, drivers of transports, chauffeurs, workers of communications (radio operators, telephone operators, telegraph operators), examinator of motors and etc. Normal functioning of vestibular apparatus must be in person whose job is related or may be related to different burdens on this apparatus (pilots, marines, mountaineers, persons working with rotating subjects or minute machines, and others).

Certain attentions during job selections must be given to possible affects (influences) on ear by industrial noise and vibrations. It is prohibited to take people in work when there are chances of decreasing the hearing functions and disturbances of spatial analyzer. Those people with chronic inflammatory process of middle ear are not allowed to work in the fields of transport professional examination of persons working in harmful conditions must carry out regulary with the aim of timely discovery of professional sickness.

In this way correctly and rightly carries out check up which enable early discovery and prevention of pathology processes related to professions, it enables keeping up the fitness of labourers and increase the production rates.

Examination. Examination (check up) is used in social insurance, during job arrangements, in military and judicial medicine. In social insurance, medical experts related to labour activity (committee of medical experts in labour) CMEL and medical consultation committee (MCC). The work of CMEL includes definition the degree of working capabilities and rational job arrangement. To limit the working capability of person is included in the assignment of MCC – examination in temporarily loss of working capabilities.

At present time temporarily and constantly incapabilities are defined. Temporal working incapability is characterized by disturbance of health which is related to reverse process. Constant or steady (firm) working incapability is characterized by irreversible disturbances of not only organs but also his anatomical structures. Temporarily working incapability is frequently related to aggravation of chronic and acute inflammatory sickness of ENT–organs, traumata (injures), intoxications and etc.

Examination of working incapability starts from treating doctors which give sick certificate (sick leave), its extension is carried out by MCC and further if necessary CMEL solves the question about the degree of steady (firm) working incapability. In definition the degree of working incapabilities, CMEL directs the results of medical investigation of persons which are sent to examination. Those details are included in special cards, form which is verified by minister of health. In examination functional disturbance of organs and degree of their

compensation are considered. Other important methods in the work of CMEL remains the definition of relations arise due to disturbance of functions of organs related of production. In other words, CMEL establishes professional character of sickness, for example affection of sound analyzer in textile workers or vibration sickness in miners. In these (group of disabled workers) or give recommendation for change of job. In firm (constant) disableness (working incapability) as a rule in defining group of disabled depend upon the character of affected organs. In otorhinolaryngology firm disableness may be due to (cancer) malignant tumor affection of ENT organs, several affection of vestibular and hearing analyzer and etc.

Military medical examination provides definition the health condition of persons and their fitness toward service in army in different types of forces. The main document which governs the military medical commission remains the orders given by the defence ministry in Ukraine.

Judicial medicinal examination provides check up of living persons, dead bodies, studying material proofs and other materials which indispensables for the organization of investigations and judicial processes. In otorhinolaryngology practical several cases happen to meet upon are simulation, malingering, dismalingering (dissimulation). Simulation – knowingly and deliberately forms unexisting sickness, malingering – exaggerates symptoms of existing sickness, dismalingering – attempts to cover up or to lessen the existing sickness.

Materials on the activation of students of higher education during the lecture:

A 17-year-old patient complains of severe pain in the throat, which worsens when swallowing, an increase in body temperature, general weakness, headache, lack of appetite. She fell ill three days ago after hypothermia.

Objectively: the skin is moist. Body temperature is 38.80C. Pulse 88 beats. per minute, rhythmic. Bright hyperemia of the mucous membrane of the palatal arches, tonsils and the back wall of the pharynx; on the surface of the tonsils - a white coating, which is easily removed. Submandibular lymph nodes are enlarged, painful on palpation. Other ENT organs are unchanged. What is the patient's diagnosis? How to treat a patient?

A 3-year-old child was brought to the ENT hospital by ambulance with complaints of difficulty breathing, barking cough, minor fainting. The child has been ill for 4 days, when he developed a runny nose, cough, and increased body temperature. Breathing worsened during sleep. The child is excited, the skin is pale, the auxiliary muscles are involved in the act of breathing. During direct laryngoscopy, the mucous membrane of the larynx is reddened, red ridges are defined in the subfolded space, the glottis is sharply narrowed. During coughing, a significant amount of purulent sputum was released. Establish a preliminary diagnosis.

A 14-year-old patient, who was treated for a long time for chronic hypertrophic rhinitis, developed frequent nosebleeds (3-4 times a week), hemoptysis. During posterior rhinoscopy, a lumpy tumor is visible, occupying the erector spinae and the lateral walls of the nasopharynx, the choanae are closed in the upper and middle parts. What is your previous diagnosis?

General material and teaching-methodical support of the lecture: Methodical aid of the lecture, multimedia presentation, mock-ups, tables, sets of tools, sets of radiographs and tomograms, simulators.

Questions for self-control

1. Classification of the tonsillitis.
2. Acute primary tonsillitis, ethiology and contributing factors.
3. Clinical forms, pathomorphological changes in tonsills.
4. Clinical picture of the various forms of tonsillitis.
5. Objective data at tonsillitis.
6. Differential diagnostics of tonsillitis with secondary inflammation of tonsils.
7. Treatment of acute tonsillitis.
8. Complication of tonsillitis, reason, way of distribution of an infection.
9. Peritonsillitis, ethiology, classification, principles of treatment, probable outcomes.
10. Retropharyngeal, peripharyngeal abscesses,neck lymphadenitis, phlegmon of a neck. Ethiology, clinic, treatment.

11. Tonsillogenic sepsis, clinic, treatment.
12. Simanovsky-Vensan's tonsillitist (ulcero-membranous).
13. Acute secondary tonsillitis at infectious diseases (diphteria, scarlet fever, infectious mononucleosis, celiac typhus)
14. Acute secondary tonsillitis at diseases of blood (agranulocytosys, alimentary toxic aleukia, leukosis), principles of treatment.
15. What are the differences between acute and chronic laryngostenosis?
16. Name the causes of acute laryngostenosis.
17. What of the diseases of larynx and pharynx may cause laryngostenosis?
18. Name the causes of chronic laryngeal constriction.
19. Name the specific diseases of larynx, which cause chronic stenosis.
20. Name the stages of laryngostenosis and describe them.
21. Pathogenetic mechanism of laryngostenosis.
22. With which diseases should laryngostenosis be differentiated.
23. Name the contents of the antiedemic inhalatory mixtures and their doses.
24. Specify the daily dose of corticosteroids.
25. What does "medicinal tracheotomy" mean?
26. Indications for tracheotomy in laryngostenosis.
27. What will you do in presence of foreign bodies in respiratory tract?
28. Name the types of tracheotomy.
29. Name the complications of tracheotomy.
30. Indications for tracheotomy in acute laryngostenosis.
31. Which stage of stenosis is characterized by white asphyxia.
32. Acute laryngitis. Name the causes and main symptoms.
33. Acute laryngotracheitis in children. Enumerate key symptoms. Draw a laryngoscopic picture.
34. Chronic laryngitis, the main clinical forms, treatment.
35. The signs of malignant tumors.
36. Morphologic classification of the tumors of the upper respiratory tract.
37. Methods of therapy of malignant tumors of ENT organs.

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7. K.S.Helfer, E.L.Bartlett, A.N.Popper, R.R.Fay. Aging and Hearing: Causes and Consequences// Hardback< 2020. – 326p.
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Electronic information resources

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2. European Regional Office of the World Health Organization. URL: www.euro.who.int.
3. www.ama-assn.org – American Medical Association
4. www.dec.gov.ua/mtd/home/ - State Expert Center of the Ministry of Health of Ukraine
5. http://bma.org.uk– British Medical Association
6. www.gmc-uk.org- *General Medical Council* (*GMC*)
7. www.bundesaerztekammer.de – German Medical Association