***Ministry of Health of Ukraine***

***Zaporizhzhia State Medical University***

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# OTORHINOLARYNGOLOGY

*Course of lectures for English medical of 4 courses of educational and qualification level "Master" of speciailty 222 "Medicine"*

***Zaporizhzhia***

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The manual contains three lectures in otolaryngology. The lectures are ded-icated to the aspects anatomy, physiology, methods of investigation ENT-organs, etiopathogenetic, clinic and treatment diseases of the upper respiratory tract, acoustic and statocinetic analyzers. Otogenic and rhinogenic intra-cranial complications are described to.

Навчальний посібник містить три лекції по отоларингології, які розкривають питання анатомії, фізіології, методів дослідження ЛОР-органів, етіопатогенезу, клініки та лікування захворювань верхніх дихальних шляхів, слухового і вестибулярного аналізаторів. Розглянуто питання отогенних і риногенних ускладнень.

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# INTRODUCTION

Otorhinolaryngology is a complex and multifactorial discipline, that unites four clinical areas, as well as the clinical and physiological aspects of audiology, phoniatrics, vestibulology. The latest achievements of science and technology are reflected in modern methods of diagnosis and treatment of ENT pathology (otoacoustic emission, computed tomography, magnetic resonance imaging, electron cochlea, functional endoscopic surgery of the middle ear, nose, paranasal sinuses, etc.). In connection with the foregoing, it is extremelu important to update existing educational material by preparing and giving new lectures for students. The proposed selected lectures on otorhinolaryngology allows to study students more deeply and fully in the speciality with which the doctor will meet daily at an appointment or at the patient's bed. This publication includes 3 lectures on general and private otorhinolaryngology and highlights issues that are most difficult for students to master.

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# INTRODUCTION

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**Lecture №1**

***The concept of otorhinolaryngology, its tasks and place among other medical disciplines.***

***Clinical anatomy and physiology of the nose, paranasal sinuses. Acute and chronic rhinitis. Acute and chronic sinusitis.***

***Clinical anatomy and physiology of the pharynx. Acute and chronic tonsillitis.***

Otorhinolaryngology is a clinical discipline that studies the morphology, physiology and pathology of the ear, upper respiratory tract and adjacent areas. Its name comes from four Greek words: otos - ear, rhinos - nose, Laringos - larynx and logos - science. However, this does not fully address the range of issues that otolaryngology develops. Modern otorhinolaryngology is engaged in diagnostics and treatment, including surgical, such diseases as meningitis, brain abscesses, thrombosis of the brain sinuses, foreign bodies of the esophagus, perforations of the esophagus, plastic of the larynx, trachea, face, cysts, tumors, phlegmons of the neck.

All this applies directly to the ENT clinic of our medical university, which for forty-five years has been the center of providing urgent care to patients with oto- and rhinogenic intracranial complications, foreign bodies of the esophagus and their complications, patients with malignant tumors of the organs, and Center for reconstructive surgery of ENT organs.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.

***Clinical anatomy, physiology and methods of investigation***.

***External nose*** *(nasus externus).* There are the external nose, the nasal cavity and the paranasal sinuses. The skeleton of the external nose is formed by bones and cartilages. The bony part of the nose is formed by paired nasal bones and by the frontal processed of the maxilla. The free ends of these bones form a piriform aperture. The cartilaginous framework of the nose includes triangular cartilage, paired ala cartilage, and the accessory cartilage. The skin on the external nose has many sebaceous and sweat glands. The upper narrow part of the nose is called the root. The lateral movable parts of the nose (ala) slightly protrude outside to form the nostrils, which, together with the nasal septum, form the entrance (vestibule) to the nasal cavity. The inner part of the nostrils (about 4-5 mm) is covered with fine hairs (cilia) and sebaceous glands.

The external nose is supplied with blood via branches of the ophthalmic artery. The blood outflows through the anterior facial and angular veins into the su-

perior ophthalmic vein which communicates with the cavernous sinus. The external nose is innervated by the fifth and seventh pairs of the cranial nerves.

***Nasal cavity*** *(cavum nasi).* The nasal cavity is divided by the septum into the right and left parts. The anterior part of the nasal cavity opens with a piriform sinus (anteriorly) and choanae (posteriori). The nasal cavity has four walls, namely, the superior, inferior, internal, and external walls. The inferior wall (the floor) of the nasal cavity is the hard (bony) palate. The superior wall (the roof) of the nasal cavity includes the bones of the nose anteriorly, the cribriform plate of the ethmoid bone in the middle (the greater part of the roof) and the anterior wall of the sphenoidal sinus. The fibbers of the olfactory nerve and the branches of the ethmoidal artery and the veins pass through the perforations of the cribriform plate. The medial (internal) wall, or the septum, consists of the anterior cartilaginous and posterior bony parts. The bony part of the septum is formed by the perpendicular plate of the ethmoid and the vomer. The lateral (external) wall of the nasal cavity has a more complex structure. Three nasal conchaes extend from the external wall toward the nasal septum: the superior, middle and inferior conch. Three nasal meatuses are distinguished accordingly: the superior, middle, and inferior meatuses. The space between the nasal conchae and the septum, extending from the floor to the roof of the nasal cavity, is called the common nasal meats. A nasolacrimal duct opens into the anterior part of the inferior nasal meats. The middle meats contains a crescent-shaped semilunar hiatus where the maxillary and frontal sinuses, and also the anterior and middle cells of the ethmoidal labyrinth open. The posterior cells of the ethmoidal labyrinth and sphenoid sinus open into the superior nasal meats.

The nasal cavity is lined with the mucous which is continuous with the mucous of the paranasal sinuses, the pharynx, and the middle ear. The nasal cavity can be divided into three parts: the anterior (vestibule), respiratory, and the olfactory. The respiratory part of the nasal cavity extends from the floor to the inferior border of the middle conch. The mucous lining this cavity consists of multilayered columnar ciliated epithelium rich in goblet cells, which produce mucus, and serous glands producing serous or seromucous secretion. The mucous of the conch overlies the cavernous tissue which can become engorged instantaneously, thus narrowing the nasal meatuses or, on the contrary, become contracted.

The olfactory part of the nose is found in the superior regions of the nasal cavity; it extends from the inferior border of the middle conch to the roof. The mucous of this part of the nasal cavity is lined with olfactory cells. The axons of these bipolar cells run up through the openings of the cribriform plate of the nasal roof to the olfactory bulb in the cranial cavity, then it continues into tracts olfactorius, septum pelucidum and ends into the cortex centers (gyres hippocampus, gyres dentate, sulks olfactorius) The nasal cavity, is supplied with blood via the branches of the

external carotid arteries (a.sphenopalatina) and internal carotid artery (aa. ethmoidales anterior and posterior, the branches of a.ophthalmica). The outflow of the blood is through the anterior facial and ophthalmic veins. The veins of the posterior parts of the conch empty into the pharyngeal veins. The anterior part of the nasal septum has an area (Kiesselbach's area) which is usually covered with a small vascular varicosity. It is often called the bleeding area, because it is a common locus of nasal bleeding.

Four types of innervation are distinguished in the nasal cavity: the olfactory, sensory, motor and secretory. The olfactory fibbers (about 20) originate from highly differentiated cells and pass to the olfactory bulb through the cribriform plate. The sensory innervation of the nasal cavity is accomplished by the first and second branches of the trigeminal nerve. The motor innervation of the external nose is accomplished by facial nerve. The secretory innervation of the nasal cavity is represented by the sympathetic nervous system. The fibbers of the sympathetic nerve pass from the pterygopalatine ganglion. They serve to communicate with the sympathetic nerves of the thoracic, abdominal, and endocrine organs. All this establishes reflex connection between the nasal cavity and other organs and systems.

***Paranasal sinuses.*** The paranasal sinuses are located by sides of the nasal cavity and communicate with it. There are four paired air cavities, namely, the maxillary, cells of the ethmoidal labyrinth, frontal, and sphenoid.

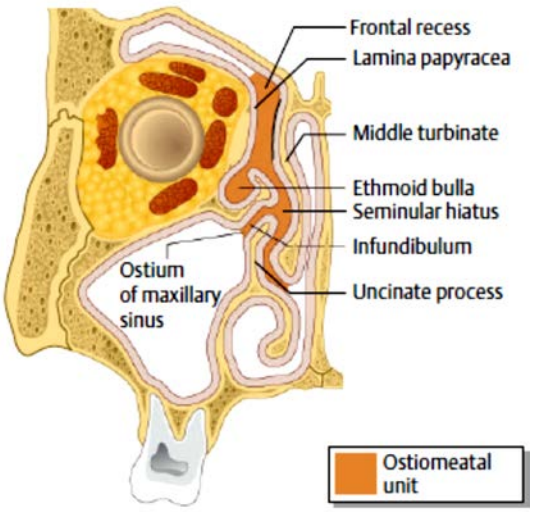
The *maxillary sinuses* are located inside the maxilla; these are the largest paranasal sinuses. The anterior surface of the maxillary sinus has a depression which is known as the canine fosse. The medial wall of the maxillary sinus, or the lateral wall of the nasal cavity, has opening at the level of the middle nasal meats, through which the sinus communicates with the nasal cavity. The upper wall of the maxillary sinus is at the same time the inferior wall of the orbit. The alveolar process of the maxilla forms the lower wall (the floor) of the sinus. In most adults, the floor of the sinus is found below the floor of the nasal cavity. The posterior wall of the sinus is thick; it is formed by the maxillary tuberosity.

The *ethmoidal sinuses* (ethmoidal labyrinth) consist of air cells of the ethmoid which is located between the frontal and the sphenoid sinuses. Anterior, middle, and posterior cells of the labyrinth are distinguished (6-7 cells of each type on either side). In healthy man the cells are filled with air.

The *frontal sinuses* are found in the squama of the frontal bone. Each sinus has four walls: the anterior (facial); the posterior, which borders with the cranial fosse; the inferior, which in most cases is the superior wall of the orbit and borders with the cells of the ethmoid and the nasal cavity over a small area; and the internal wall (the septum).

The *sphenoid sinuses* are found in the body of the sphenoid bone. The septum separating the sinuses extends anteriorly to the nasal septum. The roof is formed by the bone underlying the optic chiasm, the clinoid processes, and the cella turcica with the pituitary gland. The posterior wall is formed by the solid bone of the basissphenoid. The lateral wall is in relation to the optic foramen and nerve, the cavernous sinus and the internal carotid artery. The floor is the roof of the nasopharynx. In the anterior wall is the natural orifice which opens into superior nasal meats.

A neonate has only ethmoidal; these sinuses are only in their initial stage of development. The maxillary, frontal and sphenoid sinuses are absent in neonates. All of the sinuses normally continue to grow during childhood and reach their final size at about the age of puberty. The maxillary sinus begins to take shape from the fifth to fourteenth year during the constant teething. The topography of the paranasal sinuses approaches its final development by the age of 20.




**Anatomy of the ostiomeatal unit**

The term “ostiomeatal unit” describes the area on the lateral nasal wall where the ostia of the paranasal sinuses (except for the sphenoid sinus) open into the nasal cavity in a duct-like fashion. Even minor changes (e.g., anatomical variants, mucosal swelling) can hamper ventilation in this region, leading to pathologic sequelae in the paranasal sinuses (see below). The functionally significant anatomic structures of the ostiomeatal unit are the uncinate process, the semilunar hiatus, the frontal recess, the ethmoid bulla, the ethmoid infundibulum, and the maxillary sinus ostium (a coronal section is shown at right).

The **frontal sinus** is connected to the ostiomeatal unit via the frontal recess, which has an hourglass-like shape. The **uncinate process** is a thin fibrous or bony process of ethmoid bone on the lateral nasal wall that arises slightly behind the anterior border of the middle turbinate and may narrow the passage from the nasal cavity to the ostiomeatal complex, depending on its degree of development. Located between the posterior border of the uncinate process and the first ethmoid cell (the **ethmoid bulla**) is another slitlike passage within the ostiomeatal complex, known as the **semilunar hiatus**. The space between the uncinate process, ethmoid bulla, and lamina papyracea of the ethmoid bone is called the **ethmoid infundibulum**. The ostiomeatal unit is bounded medially (toward the nasal cavity) by the middle turbinate and laterally by the lamina papyracea. The main *clinical signifi-*

*cance* of this region relates to the sites of narrowing in the ostiomeatal unit. For example, hyperemia and swelling of the mucosa in the setting of a common cold can obstruct the narrow passages in the ostiomeatal unit, preventing adequate ventilation of the dependent paranasal sinus system and setting the stage for a rhinogenic inflammation of the paranasal sinuses (sinusitis).

***Clinical physiology***

Nasal respiration is very important because, in addition to the respiratory function, the nose also performs the protective, resonating, and olfactory functions.

The *respiratory function* of the nose is part of the entire respiratory function in man. During inspiration, which is due to creation of negative pressure in the chest, air enters both parts of the nasal cavity mostly through the respiratory part of the nose. The inspired air passes upwards and then descends by the superior and middle meatuses and passes posteriori to the choanae. The pressure of the air on the nasal mucous excites the inspiratory reflex. If a subject breathes through his mouth, the inspiration becomes shallow and the amount of the air oxygen intake decreases; this in turn can cause a pathological effect on the nervous, vascular, circulatory, and other systems of man (especially in children).

The *protective function* of the nose consists in warming the inspired air, its moistening and filtering. Cold air stimulates a rapid expansion of the cavernous sinuses and their filling with blood. The volume of the conch thus increases significantly; their surfaces become enlarged as well, and the nasal passages are narrowed accordingly. The inspired air is moistened by the wet mucous. As the air passes through the vestibule of the nose, large dust particles are retained by thick hairs. Fine dust and air-borne microbes, that pass first filter, are precipitated on the nasal mucous moistened with mucous secretion. Dust is also retained because the nasal passages are narrow and curved. About 40-60 per cent of dust particles and microbes inspired with air are retained in the nose and then removed from it with mucus. This function is performed by ciliated epithelium. Lysozyme, contained in the nasal mucus and secretion of the lachrymal glands, has a marked disinfecting property. The sneezing and lachrymal reflexes are also important protective mechanisms. Dust particles, cold, chemical, mechanical, and other factors can stimulate these reflexes. The olfactory, trigeminal, and facial nerves are involved in the reflex arc to stimulate contraction of the muscles of the face, trunk, and the limbs.

The *olfactory function* in man is provided by the olfactory mucous that contains the neuro-epithelial fusiform olfactory cells, which are chemoreceptors. The molecules of gases, vapor, mist, dust, or smoke stimulate the olfactory receptors. It should be noted that man can also perceive odor of some substances (e. g. spirit of ammonia that act on the endings of the trigeminal nerve).

The *resonating function* of the nose accounts for the special timbre of the human voice. Pathological changes in the nasal cavity or in the nasopharynx (polyps, hypertrophy of the conchae, inflammation of the nasal mucous, tumor, adenoids, and other changes) cause rhinolalia clause (nasal speech). If the nasal cavity has unusually large communication with the nasopharynx (e.g. due to the absence of the soft palate or its paralysis), the patient develops rhinolalia aperta.

***Methods of examination***

The external nose should be palpated. Palpation should also be used to examine the anterior and inferior walls of the frontal sinuses, the anterior walls of the maxillary sinuses, and also the cervical regional lymph nodes.

The respiratory function of the nose should be examined separately on each side. To that end, the wing of the one nostril is pressed to the nasal and the patient is asked to breathe air quietly in and out; a small piece of cotton wool held close to that will show if the passage is free. A special rhinopneumometer is used for a more accurate assessment of the nasal breathing function.

The olfactory function of each side of the nose is tested separately using odoriferous substances from a special olfactometric set, or using a special instrument called olfactometer. The technique for testing the olfactory function is the same as that described for testing the respiratory function. Olfaction can be normal (normosmia), decreased (hyposmia), perverted (cacosmia), or it can be absent (anosmia).

Rhinoscopy can be anterior, middle, and posterior. Anterior rhinoscopy should be carried out on both sides of the nose.The normal color of the nasal mucous is pink; its surface is smooth; the normal position of the septum is central. The other side of the nose should be examined in a similar way.

Inspection of the posterior parts of the nose is called posterior rhinoscopy (epipharyngoscopy). The posterior parts of the nasal cavity are inspected by slightly turning the speculum to the required side. The posterior ends of the nasal conchae, the nasal meatuses, and the vomer can thus be inspected. The nasopharynx can be examined in a similar way.

***Examination of the paranasal sinuses.*** According to the contemporary achievements of science and thechnics, basic methods of investigation are: endoscopic, roentgenological ones, compjuter X-ray tomography, magneto-resonance tomography, radio nuclide scintigraphy and such additional methods as ultrasonar biolocation, distance infra-red thermography, SHF-radiometry.

*Roentgenography* and clinical analyse of rentgenological signs is one of the main methods of investigation of PNS. The next special projections are used for the best observation of sinuses: naso-frontal, naso-mental, mento-parietal, lateral and semi-axial ones. Every type of pathology is characterized by the certain structural

shadings, changes of bone walls.The typical signs of the inflammatory diseases are: near-wall thickening of mucous membrane, liquid level by the exudative forms, "spotty" shading by polyposis. Osteo-destructive changes of the walls, dilation of sinuses, the presence of tissues of high intensity with the clear contours are character for the volume formations (tumours, cysts). Layer investigation - tomography in the certain depth, contrast investigation by jodolipol of the injuried sinus are used to specify the pathological process.

*Compjuter X-ray tomography* (CT). By CT the picture is got not in the X-ray film but is synthetized with the help of electronic compjuter (EC). X-rays, coming from the tube in differents directions (the set of irradiation is turned around the patient), are perceived by semi-conducting detectors, where the quanta make flashes. The flashes are calculated, turned into the figures with the help of analogue-figure transformer and they come to the EC, where the layer is reconstructed in the form of tomogram. CT lets to see bones and soft tissues of paranasal sinuses and nasal cavity in the same time and measure their X-ray density. So, with the help of CT we can carry out differential diagnose of inflammatory processes and tumours of PNS, determine the presence of osteo-destructive changes. The scale of density, expressed by relative Haunsfild's (H) units. Water density is accepted as 0 H, bone density - +1000 H, air density - -1000 H

*Nucleo-magnetic resonance.* Diagnostic picture, got by magneto-resonance tomography (MRT) (such investigation is called MR-tomography). MRT reflects two-dimensional distribution of water protons' density (water makes 60-99% of our organism). Protons of hydrogen become excited and then, coming to the initial condition, they irradiate got power. This signal is registered, and EC reconstructs the picture of organ's layer on the base of it. Air, bones, calcifications almost don't give MR-signal. MRT-investigation lets to carry out differentiation between inflammatory processes and tumours, determine their localization, dimensions and spread, contours, invasion of the neighbour anatomical structures.

*Radio-nuclide scintigraphy.* Radio-isotope diagnostics is based on the registration and mesuring of irradiations form the radio-pharmaceutical matters (RPM), introduced to the organism. Scanning and scintigraphy are intended to get gamma-topographical picture of ENT-organs and parts of body, concentrating RPM. The character of pathological process is estimated by the degree of RPM fixing in the injuried organ. Higher degree is evidence of tumour, lower degree is evidence of inflammation.

*Unionizing methods.* Last times such methods of ray diagnostics, as thermography, SHF-radiometry, ultrasonar biolocation, became widespread. They are absolutely undangerous and unharmful, cheap. That's why they can be used during

professional observations, in children's otorinolaryngology, in pregnant women. But these methods don't give enough information about tumours of PNS, inflammatory processes in the back sinuses (middle and back cells of ethmoidal labyrinth, sphenoidal sinus).However, they can be used as the methods of preclinical express-diagnostics, and for the undangerous and repeated control of the dynamic during the treatment.

*Infrared thermography.* It is based on the registration of heat irradiation from the surface of human body. It is intended for the measure of temperature by the diseases of paranasal sinuses and nasal cavity. During the estimation they determine the presence of asymmetrical heat picture and the difference between the investigated regions to within 0.1C. Compjuter lets to find and calculate the parts of thermogram, measure the coefficient of asymmetry.

*SHF-radiometry.* This method is based on the receiving of human irradiation, provoked by the heat movement of electrons in tissues in radio-frequental diapason. Penetrating ability of SHF-radiometry 2-3 times more than thermometry. The intensity of registered irradiation is directly proportional to the temperature of investigated region. The depth of irradiating layer increases togehter with the length of wave.

*Ultrasonar biolocation.* Ultrasound (2-3 mHz) can penetrate through the tissue, be absorbed and reflected in the border between different tissues. US-screening is recommended for the diagnostics of pathology in PNS. Scanning investigation is carried out with a help of medical sound generator. The depth of US-location is 40-80 mm. Reflected signal gets receiver and provokes the lighting of corresponding photodiode. The lighting of every photodiode corresponds to 2.5-5 mm (depending on the generator's conditions

***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 im-

paired 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 electrophoresis 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 bilat-

eral 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 mucopurulent 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.

***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 for-

mations 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 tis-

sue. 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 phar-

ynx 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 sur-

faces 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 autoinfection 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-necrotic.

***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 funcions.

*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 paient 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 antibacterial 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.

**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 leu-

cocyte 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.

***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).

*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 decompensatory form should be treated surgically, but can also be treated conservatively (1-2 courses). If treatment is not sufficiently effective, tonsillectomy is indicated. 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 in the absence of effect from conservative treatment.

2. Decompensatory form chronic tonsillitis.

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 im-

portant for prevention of acute and chronic tonsillitis. Health education of population is another important measure.

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.

**Lecture №2**

**Acute inflammation of the middle ear. Acute otitis media in children. Mastoiditis.**

**Chronic suppurative otitis media. Sensorineural hearing loss. Methods of treatment.**

***Acoustic analysator*** is of importance in process cognition of surrounding world, is assist to forming speech function.

Diseases of the ear, the breach acoustic function are one of the most frequent pathology; the fall of ear and deafness are reflected on the capacity for work, on its condition.

Inflammatory diseases of the ear can be the reason of the heavy lively dangerous intracranial complications. The knowledge of the theme can be used for studying pathology of external, middle and internal ear, intracranial otogenic complication by the students and also of otorhinolaryngologist, otoneurologist, neuropatholologist, neurosurgeon, ophthalmologist in practical work.

***External and middle ear***

Organ of hearing in anatomical relations is divided into three parts: external, middle and internal ear; functionally into - sound conducting and sound apprehensive apparatus. The auricle, external auditory tube passage, which gather sound waves, tympanic membrane, chain of ossicle bones and perilympha of internal ear belong to the sound conduction apparatus. The external ear comprises the pinna, or auricle, and the external auditory meatus. The shell-shaped *pinna* is composed of a skin-covered cartilaginous lamella whose posterior surface is evenly convex and smooth, while its anterior surface is concave, with semilunar folds and hollows between them. The skin on the anterior surface of the pinna adheres directly to the perichondrium; on the posterior surface, however, it may form folds owing to the presence of a small layer of loose cellular tissue. The free anteroexternal margin of the pinna is known as the *helix;* towards the bottom the pinna gradually turns into the *lobe* devoid of cartilage and consisting of well-developed fat and cellular tissue with a small number of vessels and nerves. The small protuberance of cartilage projecting over the external auditory meatus is named the *tragus.* In front of the helix and parallel to it is a ridge known as the *anthelix,* with the *antitragus* at its posterior end.

The *external auditory meatus* extends from the funnel-shaped hollow *(cavum conchae)* on the outer surface of the pinna to the tympanic membrane or drum. It is a canal directed horizontally inwards and a little forward. Its average length from the tragus top to the drum edge is 3.5 cm. The drum at the end of the canal separates the external and the middle ears. The outer third of the auditory canal consists of cartilage and membranous tissue, and both inner portions of bone.

The external auditory meatus is curved in the horizontal and frontal planes. The cartilaginous and bony portions of the meatus form an obtuse angle opening forward and downwards. Therefore, when examining the drum, the pinna must be pulled backwards and upwards, in order to straighten out the meatus. The oval lumen of the external auditory meatus has a longitudinal diameter of 1 cm. Its width varies with age and in different individuals. Its narrowest part is the isthmus, where the cartilaginous and bony portions form a junction and where foreign bodies are most likely to lodge. The walls of the auditory meatus are lined with skin which in the bony portion gradually becomes thinner, loses its subcutaneous tissue and accretes closely with the periosteum. The skin covering the cartilaginous portion abounds in hair, sebaceous glands and ceruminous glands which secrete the earwax, or cerumen. The skin of the bony portion has neither hair, nor glands.

The external bony meatus has four walls: the *superior* wall formed by the squamous portion of the temporal bone, its internal part bordering on the floor of the middle cranial fossa; the *posterior* wall serving as the front wall of the mastoid process; the *anterior* and *inferior* walls whose inner parts are formed by the tympanic portion of the temporal bone. The external third of the anteroinferior wall is made up of cartilage with two vertical fissures through which an inflammatory process in the external auditory meatus can spread to the connective tissue surrounding the parotid gland, and vice versa. The anterior wall adjoins the articular head of the mandible, which explains why it is painful to open the mouth and chew in cases of inflammation of the anterior wall of the external auditory meatus. Injury to the lower jaw, a fall, or an upward blow to the chin may cause a fracture in the anterior wall of the auditory meatus with the articular head of the mandible pushed backwards and upwards.

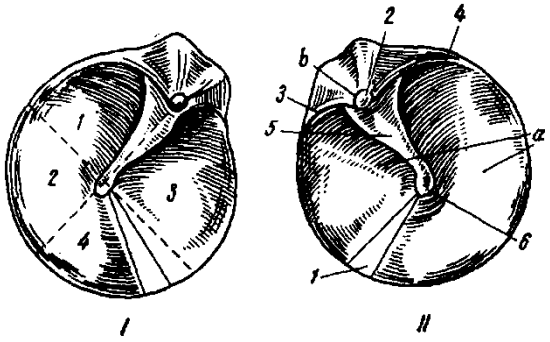
In the newborn, there is neither bony auditory meatus, nor mastoid process, and in place of the former there is a bony ring or *annulus,* which is deficient in a small upper section, and is directly connected with the membranocartilaginous auditory meatus. By the end of the third year the external auditory meatus is fully developed.

The external ear is supplied with blood by branches of the external carotid artery. It is innervated, in addition to the trigeminal branches, by the auricular nerve *(ramus auricularis n. vagi)* in the posterior wall of the auditory meatus. Mechanical irritation of the latter wall, as in wax removal, often causes reflex cough. The lymph from the walls of the auditory meatus drains into the nearest lymph nodes located in front of the auricle, on the mastoid process, and under the inferior wall of the auditory meatus. Inflammations in the external auditory meatus are often accompanied by swelling and pain in these lymph nodes.

The ***tympanic membrane*** or drum is a thin semitransparent elliptical disc

situated between the external and middle ear. The greater part of the drum fitted into the bony furrow of the tympanic ring is taut, and is called the *pars tensa;* the other, smaller part of the drum facing forward and upwards and directly attached to the incisure in the squamosa known as the notch of Rivinus *(incisura Riuini) is* lax, and is called the pars *flaccida* or *Shrapnell's membrane.*

The drum consists of three layers: an outer or epidermal layer continuous with that of the auditory meatus, a middle layer of radiating and circular connective tissue fibres, and an inner layer of mucosa continuous with the mucous membrane of the tympanic cavity. Shrapnell's membrane or pars flaccida consists only of two layers and lacks the middle stratum of fibrous tissue.




Normal Drum. Right and Left Sides

I—right drum is divided Into tour quadrants: 1) posterosuperlor; 2) postero-in-lerior; 3) anteroauperior; 4) anterolnferior.

II—left drum: a) pare tensa; b) pars flaccida or Shrapnell's membrane; t) light reflex (cone); 2) short process of malleus; 3), 4) anterior and posterior holds; 5)handle of malleus; 6) umbo

In early childhood, the drum is comparatively thick owing to the presence of a loose submucous layer. It grows compact with time and in old age becomes quite thin.

The drum is placed obliquely and not perpendicularly to the long axis of the auditory meatus, so that it faces forward, downwards and inwards. In the newborns and breast-fed babies, the drum is almost horizontal.

Examination of the drum through the auditory meatus reveals a funnel-shaped concavity in its centre with an eminence called the *umbo* in its deepest place. The handle of the malleus embedded in the fibrous layer of the drum starts from the umbo and goes forward and upwards to end above in a tiny knob the size of a pin-head—the short process. The two folds stretching anteriorly and posteriorly from the short process separate the upper lax membrana flaccida from the lower taut membrana tensa.

The ***middle ear*** comprises the tympanic cavity, the mastoid process with its cellular system and the Eustachian tube, all directly interconnected.

The *tympanic cavity* is a small chamber, about 1 cub cm in size, lying in the

depth of the temporal bone, between the tympanic membrane and the internal ear. In front, through the Eustachian tube, the tympanic cavity communicates with the nasopharynx; behind, through the entrance into the mastoid antrum *(aditus ad antrum mastoideum),* it communicates with the latter and the cells of the mastoid process. The tympanic cavity, similar to the cells of the mastoid process, contains air coming through the Eustachian tube.

It is customary to divide the tympanic cavity into three parts: the middle and biggest part, *mesotympanum,* corresponding to the pars tensa of the drum; the upper part, *epitympanum,* lying above the former and also known as the epitympanic recess or attic; the lower part, *hypotympanum,* lying below the drum level.

The tympanic cavity has six walls.The *roof* of the tympanic cavity is a thin plate of bone separating the tympanic cavity from the middle cranial fossa where the temporal lobe is situated. This plate often has congenital fissures through which vessels pass from the middle cranial fossa. These anatomical features may account for the meningeal symptoms frequently observed in young children with acute otitis media.The *inferior wall* or floor of the tympanic cavity is separated from the jugular bulb by a fairly thick bony plate. Bone fissures in this wall are rarely found. The Eustachian tube begins with an opening in the *anterior wall* separating the tympanic cavity from the internal carotid canal.An opening in the upper part of the *posterior wall* leads to the mastoid antrum *(aditus ad antrum mastotdeum).* The *internal wall* separates the tympanic cavity from the internal ear. It is marked by a gentle eminence, the promontory *(promontorium),* corresponding to the basal turn of the cochlea. Above and behind the promontory is an oval window or the fenestra vestibuli which leads into the vestibule and is closed by the foot plate of the stapes. Behind and below the promontory in a niche is a round window or the fenestra cochlea which leads into the cochlea, and is filled with a thin membrane, the secondary tympanic membrane. On the internal wall above the oval window is a bony torus-the horizontal part of the facial nerve canal. On reaching the entrance to the antrum, the facial nerve canal turns downwards to form a descending knee, then passes behind the posterior wall of the auditory meatus and through the stylomastoid foramen to the base of the skull. The walls of this bony canal may be eroded; in such cases, the middle ear mucosa may come through fissures into direct contact with the sheath of the facial nerve. This sometimes causes the development of facial paresis and paralysis in suppurative otitis media. Somewhat behind and above the facial nerve canal, on the inner wall of the aditus ad antrum, lies the peak of the horizontal semicircular canal the clear contour of which serves for orientation in operations on the middle ear. The *external wall* of the tympanic cavity is formed by the tympanic membrane, and above the drum-by the external bony wall of the epitympanic recess or attic.

The tympanic cavity contains the three auditory ossicles-the malleus, the incus and the stapes which are interconnected by joints and ligaments to form a continuous and rather flexible chain between the drum and the oval window. The handle of the malleus is woven into the fibrous layer of the tympanic membrane, and the foot plate of the stapes is fixed in the oval window by means of an annular ligament. The incus lies between the malleus and the stapes. The whole system is kept in place by ligaments fastening the malleus and incus to the walls of the tympanic cavity.

The *tympanic muscles.* There are two muscles in the tympanic cavity: The *tensor tympani* muscle which stretches the tympanic membrane. It lies in the bony canal above the Eustachian tube, and is attached to the handle of the malleus. The *stapedius* muscle which arises from the posterior wall of the tympanic cavity and is attached to the head of the stapes by a slender tendon. The tensor tympani is innervated by a branch of the trigeminal nerve, and the stapedius muscle by a branch of the facial nerve.

The *Eustachian* or *auditory tube* which is about 3.5 cm in length connects the tympanic cavity with the nasopharynx. The upper third of this tube, adjoining the tympanic cavity, has bony walls, while the remaining lower portion leading into the nasopharynx is made up of membrane and cartilage. The movement of the cilia of the ciliated epithelium lining the Eustachian tube is towards the nasopharynx. At rest, the Eustachian tube is in a collapsed state, but with each swallowing movement it opens by contraction of the soft palatal muscles attached to it, to let air into the tympanic cavity.

The *mastoid process* located just behind the external auditory meatus is a bony structure protruding downwards with the sternocleidomastoid muscle attached to it. In young children, the mastoid process is not fully developed and represents a bony tubercle behind the osseous tympanic ring.

The upper border of the process is the temporal line *(linea temporalis),* a bony torus which is a backward extension of the zygomatic process. The floor of the middle cranial fossa usually lies on a level with this line.The *anterior wall* of the mastoid process is the posterior bony wall of the external auditory meatus. Behind the spot where the superior wall of the auditory meatus merges with the posterior wall, there is a small bony peak or the suprameatal spine *(spina suprameatum)* lying above the external auditory meatus. Behind the spine there is a smooth depression, the mastoid fossa *(fossa mastoidea).* The suprameatal spine and the temporal line are important landmarks in surgical operations; the mastoid antrum *(antrum mastoideum)* lies on the projection of the mastoid fossa *(fossa mastoidea)* in the depth of the mastoid process.The *internal wall* of the mastoid process abuts upon the labyrinth, and more posteriorly is bordered by the post-cranial fossa. On

the surface facing the post-cranial fossa there is a rather wide S-shaped groove, the sigmoid sulcus, containing part of the sigmoid sinus of the dura mater. The central part of the mastoid process is the antrum lying just behind the epitympanic recess. The antrum communicates with the tympanic cavity and the airfilled cells of the mastoid process. The superior wall or roof of the antrum separates it from the middle cranial fossa.

The following types of structure are to be found in the mastoid process: the pneumatic or large-celled, the diploic and the compact or "sclerotic". In the case of pneumatic structures, the cavity of the mastoid process is divided by thin bony partitions into a lattice of larger and smaller cells. The diploic structure has tiny cells resembling a diploetic bone; the most frequent is the mixed form of mastoid structure where smaller cells are to be found alongside bigger ones. In compact structures the bone is indurated and the cells are very few; this structure frequently occurs as a result of chronic suppurative otitis media.

Cellular system of mastoid process:

- periantral, apical, threshould- under cortical layer of bone, perisinous, perifacial, perilabyrinth, peridural, angular - along the edge of pyramid up to the angle, zygomatic

The walls of the tympanic cavity, antrum and mastoid cells are lined with a continuous thin mucosa devoid of mucous glands. The mucous membrane of the Eustachian tube and of the adjoining part of the tympanic cavity floor is covered with ciliated columnar epithelium; the mucosa of the cartilaginous part of the Eustachian tube contains mucous glands which are absent in the mucosa of the other parts of the middle ear.

The middle ear is supplied with blood mainly by branches of the external carotid artery. Venous blood drainage from the middle ear is maintained by the veins of the dura mater, the venous sinuses and the venous plexuses round the carotid artery. Lymph drainage is carried out in two ways: through the lymphatic vessels of the Eustachian tube to the retropharyngeal lymph nodes and further to the deep cervical glands; through the lymphatic vessels across the tympanic cavity to the lymphatic ducts of the external auditory meatus and the lymph nodes in front of and behind the auricle. The nerve supply of the middle ear is through branches of the glossopharyngeal, facial and sympathetic nerves.

The ***internal ear*** consists of membranous and bony labyrinths, the latter surrounding the former like a capsule. The membranous labyrinth is filled with fluid known as endolymph, while around it and separating it from the bony shell is the spinal fluid known as perilymph. The bony labyrinth is made up of the vestibule, three semicircular canals and the cochlea. The *vestibule (vestibulum)* lies in the center of the bony labyrinth on whose external wall is the oval window; on the op-

posite, internal wall, there are two recesses for the two membranous sacs of the vestibule. The front sac known as the saccule *(sacculus)* communicates with the membranous cochlea lying before the vestibule, while the rear sac or utricle *(utriculus)* is connected with the three membranous semicircular canals passing behind and above the vestibule.

The *cochlea* is a bony tube which describes two-and-a-half turns around a central pillar called the modiolus and resembles a snail-shell in apperance. The cochlea connected with back skulls pit with the help of acoustic internal passage and waterpipe of the snail, which connect perilymphatic space of labyrinth with liquor system. An osseo-membranous lamina leading from the modiolus to the external wall and also turning round the former, divides the tube lumen into two directions, the upper or scala vestibuli and the lower or scala tympani which communicate at the apex of the cochlea through a small opening known as the helicotrema. Both channels are filled with perilymph. The scala vestibuli communicates with the vestibule, while the scala tympani borders on the tympanic cavity through the round window covered by the secondary tympanic membrane. The scala vestibuli of the cochlea contains the thin Reissner's membrane which extends from the osseous spiral lamina to cut off a small membranous canal of triangular section filled with endolymph and known as the cochlear duct or *ductus cochlearis.*

The organ of Corti, a complex receptive structure of the auditory analyzor, rests on the basilar membrane (*membrana basilaris),* the lower wall of the ductus cochlearis. The basilar membrane is an arrangement of elastic fibres of different lengths strung from the edge of the bony spiral lamina to the opposite, outer wall of the cochlea. The organ of Corti has a very complex histological structure containing external and internal hair cells, column cells and supporting cells. The sensory cells covered with hairs are situated in small groups between the supporting cells. The cells are covered with a membrane called the tectorial membrane *(membrana tectoria).* At the foundation hair cells there are nerve plexi which are formed snail part of VIII nerve. Its contact with cells by means of synapses.

Bloodsupplie is carry out from internal auditive artery. This is the branch of basilar artery (a.vertebralis) enters to inside acoustic passage together with VIII and VII cerebro-medullar nerves. Snail branch penetrate into modiolus and give on first spiral branch in every curl of snail. As, bloodsupplying of snail realise by one artery, any breach of bloodsupply in system this artery can bring to the defeat of organ Corty, which is very sensible to deficiency of oxygen.

***Auditory neural pathways and their nuclei.*** Hair cells are innervated by dendrites of bipolar cells of spiral ganglion which is situated in Rosenthal's canal. Axons of these bipolar cells end in cochlear nuclei, the dorsal and ventral, on each side of medulla. Further course of auditory pathways is complex. From cochlear

nuclei the main nuclear centres in the ascending auditory pathways, sequentially, from below upwards, are: superior olivary complex, nucleus of lateral lemniscus, inferior colliculus, medial geniculate body, auditory cortex. The auditory fibres travel via the ipsilateral and contra lateral routes. Thus each ear is represented in both cerebral hemispheres. The area of cortex concerned with hearing is situated in superior temporal gyros.

***Auditory Function***

The auditory function of the ear consists in the conduction of sounds through the external and middle ears or cranial bones and their reception by the spiral organ of Corti, the receptor of the auditory analyzor. The external and middle ears make up the sound-conducting apparatus, whereas the internal ear, specifically, the organ of Corti, belongs to the sound-perceiving apparatus.

The external auditory meatus conducts sound waves from the outer medium to the tympanic membrane. The meatal diameter has nothing to do with hearing acuity. Its atresia, however, as well as its complete obstruction, as occurs in earwax impaction, hinders the passage of sound waves and considerably impairs the hearing.

Sound waves striking the tympanic membrane set it into vibration. The drum being connected to the handle of the malleus, these vibrations are transmitted to the ossicular chain; and the foot plate of the stapes, closing the oval window of the labyrinth, rocks in and out of the oval window according to the phase of sound vibrations. The vibration of the foot plate of the stapes in the oval window sets up vibrations in the perilymph. These vibrations are transmitted to the basilar membrane and the organ of Corti which it supports. The vibration of the basilar membrane causes the hair cells of the spiral organ of Corti to get in touch with the overhanging tectorial membrane. At the same time, the mechanical energy of vibrations changes into the physiological process of nervous excitation which is conveyed to the most delicate receptors of the auditory nerve to be passed further to its nuclei in the medulla oblongata and through appropriate canals to the cortical auditory centres in the temporal brain lobes where nervous impulses are interpreted as sounds heard.

Normal hearing depends on the normal condition of the apparatus for sound perception and conduction. The tension of the drum and the ossicular chain necessary for normal sound conduction is maintained by the combined action of the tympanic muscles. For normal vibration the tympanic membrane requires a constant equilibrium between air pressure in the middle ear cavity and in the outer air, that is on both sides of the drum. This is maintained by the passage of air through the Eustachian tube during swallowing. Disturbance of air supply to the middle ear through the Eustachian tube causes air in the middle ear to be sucked in and the

drum to be indrawn, which is followed by deterioration of hearing. The normal condition of the sound-conducting apparatus is extremely important for the transmission of low tones to the labyrinth, that is, sounds with a low frequency of vibrations per second.

There are two ways of conducting sound waves to the labyrinth: air conduction (through the external auditory meatus, the tympanic membrane and the chain of ossicles), and bone conduction (directly through the cranial bones and the stapes).

High tones, i.e. sounds of a high vibration frequency per second, are easily conducted to the labyrinth not only through the tympanic membrane and the ossicular chain, but through the cranial bones and the stapes as well.

Two mechanisms of conduction of sound exist:

1. Transmissional mechanism.

Sound cave is double oscillation, in which there are phases of decrease and increase of pressure. They enter into the external acoustic meatus, reach the tympanic membrane and cause its vibration. During this the whole chain of auditory ossicles moves, which displaces the perilympha of vestibule. As a result of this displacement, oscillations of basal and Reisner membrane do arise. Delivery of sound into the internal ear is basically done by air path. Another path - bony conduction. This mechanism possesses double character: from one side - compressive, from the other side - inert.

2. Transformational mechanism.

Tympanic membrane and auditory ossicles increase force of sound-wave oscillations due to changes of their amplitude. Since the area of base of stapedus (3 square mm) is less than the area of tympanic membrane (about 55 square mm) , as well as the result of lever type of joints of auditory ossicles, pressure on the surface of window of vestibule makes about 20 times more than that on the tympanic membrane. In such way, air oscillations of big amplitudes and relatively of small force of tympanic membrane and auditory ossicles are transformed into oscillation of perilymph with relatively small amplitude with big pressure.

Man can hear external sounds with a frequency of 16 to 20,000 cycles per second. Speeching diapason of hearing is from 500 to 4000 Hz. Velocity of sound is different in different media. In the air, at 20°C at sea level, sound travels 344 metres per second; and is faster in liquid and still more fast in a solid medium. It is the strength of sound which determines its loudness. It is usually measured in decibles. At a distance of one metre, intensity of whisper = 30 dB, normal conversation = 60dB, shout = 90 dB, discomfort of the ear = 120 dB.

The human ear can differentiate between sounds of different, pitch intensity and timbre. There are a number of theories which seek to explain the essence of

hearing and the ability of the ear to differentiate between sounds. The oldest and most widespread among them is the resonance theory advanced by Helmholtz in 1863 and based on the physical phenomenon of sympathetic vibration. According to this theory the fibres of the basilar membrane vibrate in unison with sounds, similar to the action of strings in certain musical instruments, such as the piano or the harp. The short, thin and tighter fibres of the basilar membrane which lie in the basal turn of the cochlea vibrate in unison, i.e. resonate when stimulated by a high tone, whereas the longer, thicker and less taut fibres in the apical turn of the cochlea resonate in response to low tones.

There are a number of serious objections to the resonance theory as it oversimplifies the essence of hearing as a physiological process by describing it from the mechanical aspect alone, and fails to give a picture of the physiological properties of the auditory analyzor as a whole. It should be noted, however, that the localization of perception of high and low tones in the basal and apical cochlear turns respectively, on which the resonance theory is based, has been confirmed by experiments and clinical observations.

In opposition to the resonance theory so-called telephonic theory of hearing asserts that the basilar membrane vibrates all over like a telephone membrane. It denies any analysis of sound being made in the peripheral receptor contained in the cochlea. This concept has been disproved by clinical practice and experimental research. The mechanism of spreading of acoustic wave inside ear have been taught by experimental work of Hungarian scientists Bekeshi, who called the cochlea as hydrodynamic organ. Bekeshi elaborates the theory of “running wave” which explain the mechanism of differential perception of sounding frequencies in snail. According to Bekeshi date space perception of tones different pitch in snail is tightly connected with the wave’s length: by the influence of the sound, the global area of the main membrana is being into vibration movement.

The first to prove beyond doubt that sounds of different pitch are perceived in different parts of the cochlea was L.A. Andreyev by experiments with conditioned reflexes made in I.P. Pavlov's laboratory. The experiments were made on dogs which developed conditioned reflexes of salivation to tones of low, moderate and high frequency. After the reflex had been firmly established, the cochlea on one side was completely destroyed, and the animal retained its conditioned reflex. This was followed by a selective destruction of different parts of the cochlea. Destruction of the cochlear apex with a thin drill caused disappearance of the conditioned reflex to low-pitched sounds, whereas destruction of the cochlear base was followed by disappearance of the reflex to high-pitched sounds. These experiments have proved that an injury to the apical turn of the cochlea causes loss of low tone perception, whereas an injury to the basal turn of the cochlea is accompanied by

loss of high tone perception.

Thus, according to the teachings of I.P. Pavlov and his followers, the peripheral receptor of the auditory analyzer makes a primary analysis of sound by converting the latter's mechanical energy into the physiological process of nervous excitation. This, in turn, is conveyed through nerve canals to appropriate centres in the brain cortex where the nerve impulses are finally interpreted as sounds heard. I.P. Pavlov's teaching gives a clear idea of the functions of each part of the auditory analyser, thus presenting the latter's entire activity as a single physiological process.

The starting moment of mechanism of sound perception in cortiev organ is the difference stereocily of receptor cells in results of movement endolymph and cover membrana under the mechanic influence of acoustic wave. The most popular mechanic-electric theory of Davis, which is explain the essence of process transformation mechanical energy sound vibrations in process nervous excitement. And more popular theory is cytochemical one of Vinnikov and Titova. In the works of T.V.Gershun and V.F.Undric are learnt electric appearances, which had happened in snail.

The faculty of locating the origin of sounds, the so-called *ototopia* depends upon binaural hearing. It is largely lost in people with unilateral hearing, who have to turn their heads in various directions to locate the origin of sound. People with two healthy ears can easily determine the direction of sounds without turning their heads.The ability to find the direction of sounds is a function of the central nervous system. If a sound comes from one side, it arrives at the ear on the other side with an insignificant delay of 0.0006 sec. This delay makes it possible to determine the direction of sound.

***Methods of examination.***

*Hearing test (whispered and spoken voice tests).*

1. The patient is at a distance of 6 metres from the examiner, with the examined ear toward the physician, while the assistant closes the other ear by pressing the tragus tightly with the forefinger. In order to produce a slight masking noise, the assistant should rub the forefinger against the thumb.

2. The patient is asked to repeat loudly the words uttered by the physician. In order to prevent visual hearing (lipreading), the patient should not look at the physician.

3. The physician exhales normally, and then whispers words with low vowels, e. g. "hawl, raw", etc., and then with high vowels, such as "feet, cheese", etc.

4. If the patient cannot hear at a distance of 6 metres, the physician should approach the patient to a distance of 5 metres, and examine the patient again. The

distance should thus be shortened by 1 metre each time until the patient repeats correctly all the words pronounced by the physician.

5. The results of the test are expressed in metres at which the examinee hears the whispered words.

6. The patient can be tested for hearing spoken voice using the same technique as in the whispered voice testing.

***Tuning-fork tests.*** *Test for air conduction.* A set of tuning forks (Ci28, C512, C2048) is used for the purpose. The test begins with the lower frequency (C128). The tuning fork is tapped on the palm. A vibrating tuning fork should be held by its stem with two fingers and brought to the external acoustic meatus of the examinee to a distance of 0.5 cm. The time during which the examinee hears the tuning fork is measured by a stopwatch. After the patient does not perceive the sound of the fork, it should be moved away from the patient's ear and then brought close to it again (without reactivation). As a rule, the patient can now hear the tuning fork again for a short time. The stopwatch is read finally by the last answer of the patient.

*Test for bone conduction.* This is performed with a C128 tuning fork, because higher frequencies can be perceived by the ear through air conduction, while lower frequencies are perceived by the skin.

The vibrating tuning fork is placed perpendicularly to the mastoid. The time during which the patient perceives the vibrations is measured by a stopwatch.

*Weber's test.* A vibrating fork (C128) is placed on the vertex of the patient's head so that the stem of the fork is in the midline of the head. The prongs should vibrate in the frontal plane, i. e. from one patient's ear to the other. Normally the patient hears the tuning fork in the middle of the head, i. e. by both ears. If the sound is heard better by the affected ear, the conduction system is probably damaged. If the sound is better heard by the normal ear, this is probably due to disease of the auditory apparatus.

*Rinne's test* (comparison of air and bone conduction).

A vibrating tuning fork (C128) is placed with its stem on the mastoid. After the patient reports discontinuation of sound perception, the fork (without reactivation) is put to the external acoustic meatus. If the patient hears the fork sound through air, the Rinne test is considered positive (+). If the patient does not hear the fork through the external acoustic meatus, the result is negative (-).

*Schwabach's test* (determining hearing length with bone conduction).

A vibrating tuning fork is placed on the vertex of the patient's head and held there until the patient hears it no longer. The examiner, whose hearing power is normal, puts the vibrating fork (without reactivation) on his own head. If he can hear the ringing fork, the result is expressed as "Schwabach shortened or dimin-

ished".

*Gelle's test.* A vibrating tuning fork is placed on the head vertex; the pressure in the external acoustic meatus is increased using a rubber bulb. If hearing is normal, the sound perception decreases with increasing pressure, which is due to impaired articulation in the ossicular chain (the stapes is impressed into the fenestra ovalis). If the stapes is fixed, as in otosclerosis, the increasing pressure in the external acoustic meatus will not reduce the sound perception. If the auditory apparatus is affected, increased air pressure inside the external acoustic meatus will cause the same reduction of the sound perception as in normal persons.

*Bing's test.* This test is used to determine relative and absolute bone conduction. A C128 tuning fork is used for the purpose. A vibrating tuning fork is applied to the mastoid process and the external acoustic meatus is then alternately closed (by pressing the tragus against the auricle) and left open. If the auditory apparatus is normal and if the ossicular chain is flexible, exclusion of the air conduction (by closing the acoustic meatus) prolongs bone conduction. In the presence of otosclerosis bone conduction remains the same with the external acoustic meatus open and closed.

*Federici's test.* C128 tuning forks are used. A vibrating fork is applied to the mastoid process. As the patient hears it no longer, the fork is placed on the tragus. Persons with normal hearing function perceive the vibrations of the fork longer if it is held to the tragus rather than to the mastoid process. The picture is reverse in the presence of otosclerosis.

***Audiometric tests***

1. *Pure tone audiometry*

If the investigation by speech and turning forks not always come enough for definition the character of the defeat the ear, its degree and line others peculiarity this defeat and also don’t give enough complete information for solve question about the deposition to operation, which improved the ear, ear prosthesis, that the modern audiology is disposes the methods so-called electro-acoustic investigation of the ear.

The term “audiometry” means the methods of investigation the ear with the help of electroacoustic apparatus – audiometer. An audiometer is an electronic device which produces pure tones, the intensity of which can be increased or decreased in 5 dB steps. Usually air conduction thresholds are measured for tones of 125, 250, 500, 1000, 2000, 4000 and 8000 Hz and bone conduction thresholds for 250, 500, 1000 and 2000 and 4000 Hz. The amount of intensity that has to be raised above the normal level is a measure of degree of hearing impairment at that frequency. Maximum intensification of the sound by investigation ear conductivity 60-80 DB. The investigation is accompanied in special soundsolate chamber.

It is charted in the form a graph called *audiogram.* On scale of audiometer the level of normal (according international standart) ear correspond to line 0 Db, that is loss of ear on this level is 0. The threshold of bone conduction is a measure of cochlear function. The difference in the thresholds of air and bone conduction is a measure of degree of conductive deafness.

Pure tone audiogram is a measure of threshold of hearing by air and bone conduction and thus the degree and type of hearing loss.

The basis sign of the defeat soundperceiving apparatus is the lowering level of bone conductivity, the most expressed, as a rule , on high frequency. The level of earconductivity coincide with the level of bone conductivity.

At the defeat of soundconducting apparatus is typically fall the level of ear conductivity only, primary of low and middle frequency, at the normal level of bone conductivity: under curves of the ear and bone conductivity has distance, called “bone-ear break”.

The presence on the audiogramm bone-ear break always testify about the defeat soundconducting apparatus which can go with the defeat soundperceiving apparatus, so-called combine or mixed hard of hearing.

*2. Speech audiometry*

In this test the patient's ability to hear and understand speech is measured. A set of spondee words is delivered to each ear through the headphone of an audiometer. Each ear is tested separately. Patient repeats the words. Intensity at which the words are delivered is varied in 5dB steps. We measure speech reception threshold (SRT) and discrimination score (DS). SRT is the intensity at which 50% of the words are heard correctly. Usually the average of thresholds of pure tones of speech frequencies (512,1000 and 2000 HZ) and SRT are almost equal. Poor agreement between the two is a sign of non-organic hearing loss. A person with normal hearing or conductive deafness will hear 95-100% of the words correctly. DS falls markedly in sensoneural deafness, particularly of the neural type. Poor discrimination score (below 80%) will affect the ability to understand speech, which is more marked in the presence of noise.

*3. Impedance audiometry*

It is an objective test widely used in clinical practice and particularly useful in children.Its uses are:

1. To differentiate ossicular fixation from ossicular dislocation in cases of conductive deafness.

2.To find fluid in middle ear in serous otitis media.

3.To assess function of eustachian tube.

4.By eliciting stapedial reflex, it can be used to localise lesions of facial nerve and find prognosis of facial paralyis, find recruitment, detect malingerers and

test hearing in infants.

When a sound signal (tone of 220 Hz) is introduced into a sealed external canal, it strikes the tympanic membrane. Some of it is absorbed and the rest reflected. More sound energy is reflected when the tympanic membrane is stiff and less of it when it is compliant. Optimal compliance of tympanic membrane is seen when air pressure in the external canal and middle ear is the same. The measurement of compliance of tympanic membrane and ossicular system conditions of positive, normal or negative pressures is called *tympanometry.*

Compliance of tympano-ossicular system can be charted against pressure changes in the form of a graph called *tympanogram.*

Acoustic reflex. It is based on the fact that contraction of stapedial muscle causes stiffening of tympano-ossicular system and change in compliance. A tone of 70-100 dB will cause stapedial muscle to contract.

Presence of reflex at 60 dB HL indicates that the ear is recruiting and the loss may be of cochlear type. If a tone of 70-100 dB HL fails to elicit reflex in a case of facial paralysis, the lesion of facial nerve will lie proximal to the nerve to stapedius. A person who feigns total deafness and does not give any response on pure tone audiometer but shows presence of acoustic reflex is a malingerer. Similarly a rough estimate of hearing can be made in infants and young children, who are otherwise difficult to test by pure tone audiometry.

*Types of tympanogram*:

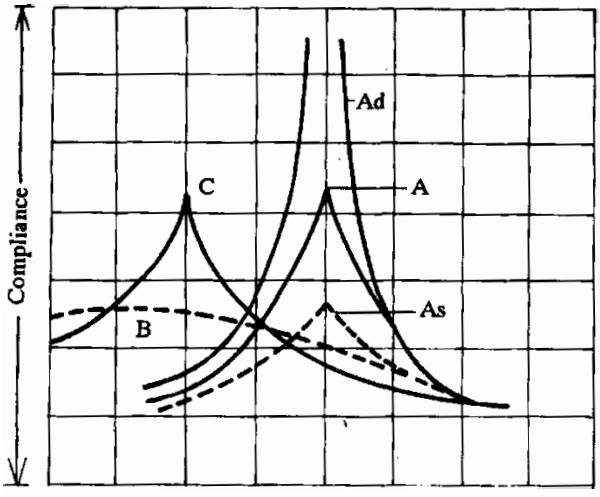
A - Normal

As - Reduced compliance at ambient pressure (otosclerosis)

Ad - Increased compliance at ambient pressure (ossicular discontinuity)

B - Flat or dome-shaped (fluid in middle ear)

C - negalive pressure in middle ear




*Evoked response audiometry*

It is a n objective test which measures electrical activity in the auditory pathways in response to auditory stimuli. It requires special equipment with averaging computer. There are sever-

al components of evoked electric response but only two have gained clinical acceptance. They are:

*Electrocochleography* It measures electrical potentials arising in the cochlea and CN VIII in response to auditory stimuli. The response is in the form of cochlear micro-phonics, summating potentials and action potential of VIIIth nerve. The recording electrode is usually a thin needle passed through the tympanic membrane onto promontory. In adults it can be done under local anaesthesia but in children or anxious persons sedation or general anaesthesia is required. Sedation has no effect on these response.

*Auditory brain stem responses.* It is a non-invasive technique to find integrity of centra] auditory pathways through the VIIIth nerve, pons and midbrain. In this method, electrical potentials are generated in response to several click stimuli and picked up from the vertex by surface electrodes. In a normal person seven waves are produced in the first 10 milliseconds. The first, third and fifth waves are most stable and used in measurements. The waves are studied for absolute latency, inter-wave latency (usually between wave I and V)and amplitude. The exact anatomic site of origin of waves is still disputed but they are thought to arise from the following parts.

Wave I: VIII-th nerve
Wave II: Cochlear nuclei (pons)
Wave III: Superior olivary complex (pons)
Wave IV: Lateral lemniscus (pons)
Wave V: Inferior colliculus (mid brain)
Wave VI: Medial geniculate body (thalamus)
Wave VII: Auditory radiations (thalamo-cortical)

Brain stem evoked audiometry is of great value to find threshold of hearing in infants, particularly the high risk group, and in the diagnosis of retrocochlear pathology.

***Vestibular apparatus***

Study of vestibular apparatus structure, investigation of it's functions importance, as vestibular apparatus controls position of the body in space and secures body's balance during movement and immobility.

Examination of vestibular apparatus is importance in clinic for determination of character of disease; in examination during selection of candidates for work with hard vestibular loading (pilots, sailors, astronauts).

Vestibulometry is necessary for examination of mechanism of development internal ear damage after infectional disease, after dose of ototoxical antibiotics; during force of production noise, vibrations and chemical agents. Symptoms of ir-

ritation and oppression of vestibular analysator give knowledge for discovery of different intracranial complications. So vestibular dysfunction is one of the widespread form of pathology in clinical practice with which otolaryngologist, neurologist and therapeutists have to deal with.

Works of scientists-otolaryngologists have great importance in elaboration of questions on physiology, methods of investigation of vestibular analisator. They are S.S. Stein, N.T. Simanovsky, V.I. Voyachek, K.L. Khilov. They are founders of vestibulometry.

Experience of long standing of such scientists physicians, vestibulogists, physiologists as V.F. Undric, S.N. Khechinashvili, I.B. Soldatov, A.H. Minkovsky, A.E. Kurashvili was put in basis of new branch of medicine. Works of scientists-otolaryngologists have great importance in elaboration of questions on physiology, methods of investigation of vestibular analisator. They are S.S. Stein, N.T. Simanovsky, V.I. Voyachek, K.L. Khilov. They are founders of vestibulometry.

Labyrinth on whose external wall is the oval window; on the opposite, internal wall, there are two recesses for the two membranous sacs of the vestibule. The front sac known as the saccule *(sacculus)* communicates with the membranous cochlea (through ductus reunions) lying before the vestibule, while the rear sac or utricle *(utriculus)* is connected with the three membranous semicircular canals passing behind and above the vestibule.

The intercommunicating sacs (through utriculosaccular ducts ending endolymphatic ducts and endolymphatic sacs on the posterior side of the pyramid of temporal bone, that is posterior cranial fosse) of the vestibule contain the statokinetic receptors or *maculae acusticae,* otolithic organs made up of a highly-differentiated specific neuroepithelium covered with a membrane containing granules of carbonate and phosphorate of lime, i.e. the otoliths. Mechanism of irritation in sacculus of vestibulum is determined by displacement of otolith by irritation of neuroepitelium. Besides, otolithic apparatus is exposed by continuous irritation of gravitave acceleration. During the irritation arising impulses cause changing of body and limbs muscles' tone. These changing of muscular tone give possibility for stable position of head and body and also for conservation of this position.

The semicircular canals are set at right angles to each other and represent the three planes of space. They are three in number: the external or horizontal, the superior or frontal, and the posterior or sagittal. One end of each canal opens out into a larger space known as ampulla, the other end is even. The frontal and sagittal canals have a common even stem (*crus commune).* The ampulla of each membranous canal contains a ridge, the criste *ampullaris,* which is a receptor, i.e. a nerve ending consisting of a highly-differentiated neuroepithelium or hair and supporting cells.

The free surface of the hair cells is covered with hairs which respond to the

slightest displacement or pressure of the endolymph. Mechanism of irritation of vestibular analisator (ampular receptor) is determined by deviation of endolymph, displacement of cupulae, irritation of neuroepitelium in cristae ampullaris.

The receptors of the vestibule and semicircular canals are the peripheral nerve endings of the vestibular analyser.

The first neuron of vestibular tract is situated in ganglion Scarpe in internal acoustic meats. Vestibular nuclear four in number: the superior, medial, lateral and inferior are situated on the bottom of IV ventricle of brain. *Afferent* to these nuclei come from peripheral vestibular receptors (semicircular canals, utricle and saccule), cerebellum, reticular formation, spinal cord, contralateral vestibular nuclei. Thus information received from labyrinthine receptors is integrated with information from other somato-sensory systems.

*Efferent* from vestibular nuclei go to:

I. Nuclei of III, IV, VI cranial brain nerves via medial longitudinal bundle. It is the pathway for vestibulo-ocular reflexes and explains genesis of nystagmus (it slow component).

II. Motor part of spinal cord (vestibulo-spinal fibers). This coordinates the movements of head, neck and body in maintenance of balance.

III. Cerebellum (vestibulo-cerebellar fibers). It helps to coordinate input information to maintain body balance.

IV. Autonomic nervous system (reticular formation). This explains nausea, vomiting, palpitation, sweating and pallor seen in vestibular disorders.

V. Cerebral cortex (temporal lobe). This is responsible for subjective awareness of motion and fast component of nystagmus.

Nuclears of vestibular analisator in bottom of rhombic fosse (superior angular Bechterev's nuclear, lateral Deiters'es nuclear, medial triangular Swalbe's nuclear, inferior Roller's nuclear) communicate with different parts of central nervous system. So, there is possibility for many complexes of various reactions. We select three types of reactions:

1. Sensitive reactions, which can express weakly (like as motion sense of the body); and sensitive reactions which can express powerfully (like as dizziness).

2. Somatic reactions: from eye's muscles (nystagmus), from upper extremities (miss), from the body (deviation of the body, changing of walk)

3. Visceral reactions, which express changing in deepness and rhythm of breathing, functions of cardio-vascular system, gastro-intestinal tracts

*Vestibular Function*

Orientation of the body and its individual parts in space is made possible by cooperation of many receptors. Apart from eye-sight, the location of the body and its parts is identified through nerve endings lying in the skin, as well as in the mus-

cles, joints and tendons, which are called proprioceptors.

In addition to the above-mentioned receptors, the cerebellum and, above all, the vestibular apparatus perform an important function in body orientation and in maintaining equilibrium at rest and in motion. The vestibular apparatus consists of the vestibule containing the otolith system and the semicircular canals with their ampullae containing the nerve endings of the vestibular analyzer.

The accelerations imparted to the body during its movement in space are adequate or specific stimulants for the nerve endings of the vestibular analyzer. Movements along a straight line cause displacement of the otoliths and stimulate the receptors of the otolith, or statolith, structure contained in the vestibular sacs. Angular or rotator motions are followed by displacement of the endolymph in the semicircular canals and stimulation of receptors in the ampoule.

Stimulation of the receptors of the vestibular analyzer produces a number of reflex reactions which cause a change in the tonus of some muscle bundles of the torso, extremities, neck and eyes. This, in turn, causes the whole body to change position and maintain balance.

Angular acceleration is equivalent irritation for semicircular canals; threshold of stimulation is angular acceleration 2-3$^{o}$/sec$^{2}$. Equivalent irritant for otolith apparatus are rectilinear acceleration, changing of head and body position, centrifugal acceleration, acceleration of gravitation force threshold (cut-off) of stimulation is - 0,01g.

One of the unconditioned reflexes observed in stimulation of the semicircular canals is nystagmus which consists in a rhythmic movement of the eyes in a certain direction and back, such as lateral and vertical nystagmus. Nystagmus may be observed in different positions of the eyeball, for example, in gazing straight ahead and in an extremely side-long glance. The observation of nystagmus is used to assess the reaction of a stimulated vestibular apparatus.

Examination of ampular apparatus is based on laws of Evald**:**

1) flatness (plane) of nystagmus corresponds to plane of semicircular canal, in which current of endolymph occurs, so irritation one of the canal causes nystagmus in the plane of canal;

2) ampulopental current of endolymph prevails over ampulofugal in horizontal semicircular canal. In vertical semicircular canals all is just the opposite. So it determines direction of nystagmus. That is to say, that ampulopental current causes more powerful irritation in horizontal semicircular canal, than ampulofugal current. All is just the opposite in vertical canals.

3) nystagmus deviates always to the side of more irritated ear.

Voyachheks' laws:

1) Nystagmus always occurs in the plane of rotation or plane of nystagmus coincides with plane of rotation.

2) Join contraction muscles of eye causes slow component of nystagmus; and slow component of nystagmus is directed to the side of movement current of endolymph in semicircular canal. Direction of nystagmus is determined by his fast component. So we have 2 law: nystagmus is always opposite to the direction of endolimph's displacement. Slow component of nystagmus and protective movements coincide always with endolymph's current.

The role of the vestibular apparatus becomes particularly apparent during an acute disturbance or cessation of its function, which occurs in some diseases. The patients suffer from severe static and dynamic disorders: they are unable to stand, walk and sit; they cannot coordinate their movements, develop spontaneous nystagmus, etc. This is accompanied by vertigo, nausea and vomiting. Three to four weeks later these symptoms subside due to compensation from the central systems. A more or less intensive reaction of the vestibular apparatus to adequate, i.e. specific, stimulation depends on the state of the central nervous system, its higher division, the brain cortex, in particular.

***Methods of examination of the vestibular apparatus.***

Plan of vestibulometry examination of sick concludes such stages:

1. Scrutiny of complaints and anamnesis of disease.
2. Determination general state of sick.
3. Examination of nose, ears, larynx, pharynx.
4. Registration spontaneous vestibular reactions.
5. Employment of experimental vestibular tests.
6. Appreciation of results and conclusion for function of vestibular apparatus.

The patient is asked about his complaints: the feeling as if the surrounding objects or the patient himself move about (systemic vertigo), uncertain gait, falling to one or other side; it is important to know if the patient has fits of nausea and vomiting, if vertigo intensifies as the patient changes the position of his head. The anamnesis of the disease should be collected.

*Romberg's test.* The patient stands upright with his feet close together and the arms stretched at the level of the chest and the fingers set apart. The patient's eyes are closed. (The assistant physician must see to it that the patient does not fall.) In labyrinthine dysfunction, the patient falls on the side opposite to the nystagmus. The patient's head is turned 90° to the left: if the labyrinth is affected, the patient falls to the other side. The situation is the same if the patient turns the head to the right.

*Example.* The patient develops nystagmus to the right. His head is turned 90° to the left. The direction of nystagmus remains the same but the orientation

relative to the trunk changes: the slow component is directed backward, and the patient falls to the side of the slow component, i.e. backward in this particular case.

If the cerebellum is affected, the change in the position of the head does not change the direction of the fall. The patient falls only in the direction of the affected side.

*Straight and sideways walking*. 1. Straight walking. The patient closes his eyes and makes five steps forward, then five paces back without turning. If the vestibular apparatus is affected, the patient deviates from rectilinear movement to the side opposite to nystagmus. If the cerebellum is involved, the deviation is in the direction of the affected side.

2. Sideways walking. The patient moves his right leg one step aside, and then brings the left leg to the right one. Thus he makes five steps to the right and then back to the left.

If the vestibular apparatus is affected, the patient performs this test adequately in either direction. In case of cerebellar affection, the patient cannot perform this test and falls.

*Pointing test.* The physician sits facing the patient and stretches his arms toward him at the level of his chest. The forefingers are straightened, while the other fingers are closed in fists. The patient's arms rest on his laps, with the fingers in the same position.

The patient must raise his arms and touch the forefingers of the physician.

First the patient performs this manipulation three times with his eyes open, and then with closed eyes. If the labyrinth is normal, the patient is successful in accomplishing this test. If the labyrinth is affected, the patient misses the physician's fingers, the movement of his both arms deviating in the direction opposite to nystagmus. If the cerebellum is affected, the patient cannot touch the physician's finger with one arm on the involved side (deviation in the direction of the involved side).

*Adiadochokinesia* (specific symptom of cerebellar affection). The patient assumes the Romberg station and performs supination and pronation with both hands. If the cerebellar function is disturbed, the movement of the hand on the involved side is markedly delayed.

*Spontaneous nystagmus.* The physician sits facing the patient, and sets his index finger vertically at the level of the patient's eyes, at a distance of 60-70 cm and to the right, and asks the patient to look at his finger. It is necessary that the patient had not moved his eyes to the right to a great extent, since the ocular muscles will be overstrained and the eyeballs will jerk. The presence or absence of nystagmus is determined in this position. If there is spontaneous nystagmus, its characteristics are determined (with respect to plane, direction, degree, amplitude, and

speed). The test is repeated with the finger set in front of the patient and to the left of him.

For example, spontaneous horizontal nystagmus to the right, second degree, small amplitude, fast. It is necessary to remember that congenital spontaneous nystagmus occurs in rare cases. It is characterized by uniformity of oscillations, the absence of the slow and fast components, and independence of the direction of the sight.

*Caloric test.* The physician must inquire the patient if he had diseases of the middle ear and then carry out otoscopy. If the tympanic membrane is not perforated, a caloric test can be carried out. The patient sits with his head tilted back at an angle of 60°. A Janet's syringe is used for the test. The temperature of water is 19°C.

1. Right ear. The right external acoustic meats is douched with 100 ml of water at 19°C within ten seconds, directing the jet from the syringe on the posterosuperior wall of the meats. The time from the end of douching to the onset of nystagmus is determined (the latent period). Normally this is 25-30 seconds.

The patient fixes his eyes at the index finger of the physician which is first set at a distance of 60-70 cm and to the left of the patient's eyes; then the eyes are set straight and to the right.

The nystagmus with respect to the plane, direction degree, amplitude, and speed is first determined for each eye position; then the sight of the patient is transferred in the direction, of the fast component and the duration of nystagmus is determined. The duration of nystagmus is determined after assessing its degree, when the eyes are directed to the side of the fast component; normal length of experimental nystagmus after the described calorization is 30-70 seconds.

2. Left ear. The caloric test for nystagmus in the left ear is carried out in the same way, except that the sight to the right (i. e. in the direction of the expected nystagmus fast component) is first analyzed.

3. Caloric test with hot (45°C) water is carried out in the same way as with cold water. In irrigation with cold water, the nystagmus (its fast component) is directed to the side opposite to the tested ear. When hot water is used, nystagmus develops toward the side of the irrigated ear.

*Rotation test.* The patient sits in a revolving (Barany's) chair with his back tightly pressed against the back of the chair, feet placed on the foot support, and arms on the elbow-rests. His head should be tilted 30° forward and downward and his eyes should be closed. The chair should then be turned at a uniform speed, 10 turns clockwise over 20 seconds, and then stopped abruptly.

After the chair is stopped, the endolymph will continue moving in the lateral semicircular canals. The slow component of nystagmus will be directed to the right

as well, while the direction of nystagmus (fast component) will occur to the left. The patient must raise his head immediately after rotation is discontinued and fix his eyes on physician's finger which should be held at a distance of 60-70 cm to the left of the patient's eyes.

The physician determines the direction of nystagmus (right, left, upward, downward), its plane (horizontal, rotary, vertical), degree (I-III), amplitude (low, moderate, and high), speed (brisk, slow) and duration (normal, 20-30 seconds).

To determine the degree of nystagmus, the physician's index finger, at which the patient fixes his eyes, should be held at the side of the expected nystagmus component. As nystagmus develops, the patient's sight changes from lateral to straight; disappearance of nystagmus indicates degree I. If nystagmus persists with a straight look, the nystagmus is degree II, but only on the condition that nystagmus disappears as the sight is moved toward the slow component side. In the latter case, if nystagmus persists when the patient moves his eyes in the direction of the slow component, the nystagmus is degree III.

The somatic reactions to the test (inclination of the head and the trunk) are as follows: degree I, weak, deviation through 0 to 5°; degree II, moderate, deviation through 5 to 30°; and degree III, strong, the patient's sense of balance is lost and he falls.

The vegetative reactions are also classified as degree I (weak), characterized by pallor and fall of pulse; degree II (moderate), attended with cold sweat and nausea; and degree III (strong), characterized by vomiting, a neurogenic shock and syncope.

*Pneumatic test.* The physician sits facing the patient, who fixes his sight at the left ear of the physician. The physician creates excessive pressure inside the meats by pressing the tragus with the left index finger (or using a rubber bulb). If the labyrinth is intact, no nystagmus appears. If there is a fistula in the lateral semicircular canal, nystagmus occurs in the direction of the affected side (to the right). On decompression a nystagmus to the opposite side (to the left) develops.

The left ear is tested in a similar way. The body inclines in the direction opposite to nystagmus.

*Testing the function of the otolithic apparatus.* The patient sits in a revolving chair (Barany's chair), closes his eyes, and tilts his head and trunk 90° forward. The physician turns the chair (5 turns within the course of 10 seconds) and then stops the rotation suddenly. Possible motor reactions are recorded. In 5 seconds the patient is allowed to open his eyes and to straighten.

The deviation of the head and trunk from the median line in the direction of rotation and the vegetative reactions are evaluated; the function of the otolithic apparatus is thus assessed.

*Electronystagmography.* It is a method of detecting and recording of nystagmus which is spontaneous or induced by caloric, positional, rotational or optokinetic stimulus. The test depends on the presence of corneo-retinal potentials which are recorded by placing electrodes at suitable places round the eyes. The test is also useful to detect nystagmus which is not seen with the naked eye. It also permits to keep a permanent record of nystagmus.

*Optokinetic test.* Patien is asked to follow a series of vertical stripes on a movingdrum first from right to left and then left to right. Normaly it produces nystagmus with slow component in the direction of moving stripes and fast component in the opposite direction. Optokinetic abnormalities are seen in brain stem and cerebral hemisphere lesion. Thus this is useful to diagnose a central lesion.

*Galvanic test.* It is the only vestibular test which helps to differentiate an end organ lession from that of vestibular nerve. Patient stands with his feet together, eyes closed and arms autstreched and a current of 1 mA is passed to one ear. Normaly person sways towards the side of anodal current.

***Acute otitis***

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.

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 exam-

ple, 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 gastrointestinal 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, phenolglycerol ear drops should be used.

Rp.: Acidi carbolici 0,5
Cocaini 0,3
Glycerini 10,0
MDS. A warm dose of three drops to be instilled in the ear tree time daily for ten minutes.

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 nys-

tatin 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 syringed 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 administration).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 ($39.5^0$-$40^0$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 in 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 pro- | Painless | Painful |

| | | |
|---|---|---|
| cess | | |

***Differentiative symptoms of mastoiditis and furuncul 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:

endolymphatic sac surgery

decompression of facial nerve

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 from the 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 $38^0$-$39^0$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 purulent middle otitis is the most frequent disease of the ear and you can meet it in 20-25 per cents of cases among the all pathology of ENT organs. But unsymptomatically taking chronic otitis, especially epitympanitis, can suddenly causes the hard intracranial complications (meningitis, sepsis, brains abscesses, etc).

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

***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 occu-

pying 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 |
|---|---|---|
| Pathomorph o 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.<br>Caries of the walls of tympanic caviity and acoustic bones. | Purulent excretions with annoying smell;<br>Progressive hardness of hearing;<br>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.

***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.

***Otogenic intracranial complications.*** 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).

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 pro-

nounced 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 car-

diovascular 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 intracranial 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, electroencephalography, 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 al-

most 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.

| Characteristic of liquor | Norm | Otogenic purulent meningitis | Epidemic cerebrospinal meningitis | Tuberculous (serous) meningitis | Virus (serous) meningitis |
|---|---|---|---|---|---|
| Pressure | 100-250 mm | High | High | Increased | Increased |

| | | | | | |
|---|---|---|---|---|---|
| Trans-parence | It is transpa-rent like water | From opal-escen ce to darkness | The same, may be xantho-chrome | Transparent | At influence can be haemor-rhage |
| Cytosis | 0-3-5in 1 mkl | Thousands (80-90% of ne utrophils) | Increases quiqly. At first it is neutro-phil's one, then it is lymph. one. | Hundreds of cells (mainly these are lym-pho cytes) | 200-300 ctlls in 1 mkl (lymphocytes) |
| Albu-men | 150-450 mg\l 0,15-0,45 g\l 0,2-0,3 % | increased | In great number | Its maintenance is early in-creased | There is a small number |
| Globuli n's reaction | negative | Sharply positive | Sharply positive | Always positive | Positive |
| Chlo-rides | 118-132 mol\l 720-730 mg\l | Lowered or norm | lowered | Small lowering in compare with meningitis of other aetiol-ogy | In norm |
| Sugar | 2,5-4,2 mm\l 0,5-0,75g\l | Norm or lowered | lowered | Sharply lowered | Norm or lowered |
| Bacterio logic research | - | Streptococ cus staphyloco ccus | Meningo-coccus is sowed in the first days of dis-ease | 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 | Epidemic | Tuberculous | Serous (virus) |
|---|---|---|---|---|

| | meningitis | cerebrospinal | meningitis | 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 | Big-sweeping, horizontal. Quick and slow components aren't clearly distinguished. Turning off nystagmus is character to the injured side. |

| | | |
|---|---|---|
| | of giddiness, its force shanges and de-pends on position of a head. At tur-ning off labyrinth it is di-rected to the health side and quickly is exhausted | |
| Spontaneous miss | Two-sides, always in di-rection 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 cerebel-lum. This concurs with direction of nystagmus. It doesn't depend on the head's position. It is ob-served for a long time. |
| Deflection at walking | A patient deflects to the side of the slow compo-nent 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. |
| adiadochokinesi s | Is absent | Is observed. There is lag of a hand on the injured side. |
| Giddiness | Is very intensive and be-comes 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, gradual-ly 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 de-pending on the form of labyrinthitis | Doesn't change |
| Experimental | There is increased excita- | There is normoreflexia of vestibu- |

| | | |
|---|---|---|
| irritation of labyrinth | bility or absence of vestibular excitability | lar analizer if abscess is unlabyrinthogenic. |
| Stagnant optic teat | There is no | Can be. |

***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.

## Lecture №3

## Clinical anatomy and physiology of the larynx. Stenosis of the larynx. Chronic laryngitis. Methods of treatment. The formation of ENT organs.

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 relationships in the larynx leads to different pathological processes, firstly discovered by the development of the nose , larynx and voice dysfunction. 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 occurring 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 symptoms of infectious granulomas of the upper respiratory tract and ear for mating the differential diagnosis.

***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 cri-

coid 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 (rima 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 mem-

brane 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)*:vocal 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 $40^0$-$50^0$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.

***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.

***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 mu-

cosa which is usually symmetrical on both sides of the larynx and in the interarytenoid 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 tis-

sues. 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 extralaryngeal 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 ac-

companied 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 simultaneously.

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 per-

spires; 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. Laryngostomy 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 intramuscularly, 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.

***Formations of the upper respiratory tract.***

***Morphologic classification of the upper respiratory tract tumors***.

I type – Higher differentiated neoplasms:

*1 group* – Benign: osteoma, chondroma, fibroma, angiofibroma, angioma, neurofibroma, neurinoma, adenoma.

*2 group* – Terminal: chondroma of soft tissues, mixed neoplasm, cylindroma, ectodermal plasmocytoma, soft papilloma, epithelioma, hemangioma, cementoma.

II type –Differentiated neoplasms (malignant tumors):

*1 group* – Connective: various sarcomas, except the reticulosarcomas, lymphosarcomas.

*2 group* – Neurogenal (neuroectodermal): melanoblastoma, esthesioblastoma.

*3 group* – Epithelial: various cancers, except the transitional cell carcinoma.

III type – Non-differentiated (tonsillary): lymphoepithelioma, reticulocytoma, transitional epithelial cancer, cytoblastoma, embrioocytoma.

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 carci-

noma. 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) 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;

b) 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.

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

d) 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 postcricoidcarci-

noma – 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 – in-

to 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.

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