DEVELOPMENT OF OTORHINOLARYNGOLOGY

Giving the otorhinolaryngological aid began simultaneously with the appearance of medical aid generally, that means in primitive society. You have already studied the history of medicine and you are well aware of such written documents of ancient medicine as papyri of the Ancient Egypt (1550 – 1560 B.C.), papyri of Eberot, works of Hippocrates (460 – 377 B. C.), Cornelius Cels (I century B.C), Halen (I-II century A.D.). A lot of ENT-diseases and methods of conservative and surgical treatment were described in those works.

In Kyivskaya Russ different herbal methods of treatment of ear, throat and nose were described. Patients were treated by quacks and not always rationally. For instance, in ear diseases they advised to apply sparrow bile. Noble people were treated by foreign doctors. As it is known, doctors and bone-setters for army were prepared by Moscow medical school of Pharmacy Department since 1654. No textbooks of that school have been kept because education in it was of mediocre character. In XVIII century in Moscow Pharmacy Department there were two "masters of larynx problems" - Ivashko Gubin and his son Vlas Gubin.

In 1707 by order of Peter I Moscow hospital based Sosnovsky medical-surgical school was organized and it was the beginning of systematic higher medical education. Surgeons for Russian army were prepared in that school. The founder and chief of that school was professor N.L. Bidloo (1707-1735), a physician from Holland.

Latin manuscript by N.L. Bidloo "Directions for those who study surgery in anatomic theater", dated January,3, 1710 is being kept in Military-medical Academy named after S.M.Kirov fundamental library. This manuscript was translated into Russian in Lvov medical Institute, and in1976 it was published by"Medicine" publishing house .The contents of the book permits to judge about the first program of scientific surgery teaching in Russia and also gives a notion about the level of surgery at the beginning of XVIII century.

In the "Directions" some ENT-diseases systematology and methods of treatment were described

Considerable changes in the development of scientific and practical medicine took place in Russia in the second half of XVIII century. At that period the following medical institutes were founded: medical faculty of Moscow University (1764) and Petersburg medical-surgical academy (1798). Special therapeutic, surgical and obstetric-gynecologic clinics were founded rather quickly. There was no strict specialization. Patients with diseases of ear, throat and nose were treated by therapists and surgeons (diseases of ear with a formation of abscesses needed surgical treatment).

That's why first works on otolaryngology were issued by other specialists. Anatomist and obstetrician N.M. Ambodik issued «The medical-surgical dictionary» (1780) and «Anatomy-physiology dictionary» (1783). In those works the terminology of otorhinolaryngology was gathered for the first time in the Russian literature

A surgeon of Petersburg medical-surgical academy professor T.F. Bush (1771-1843) played an important role in the development of native and foreign otorhinolaryngology. His three volume «Manual to teaching surgery» contained a lot of original observations and statements, which were connected with otiatria, rhinology and in less degree with laryngology. Chapters dealing with our specialty had more than 100 pages. Some historians consider those chapters to be the first native guidance on otorhinolaryngology.

Questions of nonsurgical diseases of nose and throat are dealt with in detail in the works of professor of Petersburg medical-surgical academy K.F. Uden (1754-1823) «The academic readings». Much attention in this book was paid to the pharynx pathology. The classification of tonsillitis is very interesting. Uden distinguished serous, catarrhal, phlegmonous, purulent, polypous and, the most interesting, symptomatic tonsillitis. The "consumption of respiratory throat" is especially interesting.

Valuable topographic data for otolaryngology development were given in the works of M.I. Pirogov (1810-1881). Having used his original methods of three different layer cuts of frozen corpses , he made a lot of specifications in the field of the normal and topographic anatomy, including nose, pharynx larynx. M.I. Pirogov also described some surgical operations of ear, nose and throat.

Investigations of ear, throat and nose were complicated by the fact that these organs are situated in the depth and can not be examined with the help of usual methods: palpation, auscultation, percussion, which

were at the disposal of the physicians at those times. That is why the middle of XIX century, when the first endoscope methods of examination of ENT-organs were suggested, is considered to be the beginning of otolaryngology development.

At first Kramer (Berlin) proposed ear funnel. In 1841 an ordinary German doctor Gofman proposed a primitive reflector for examination of ENT-organs in depth. In 1854 London professor of the vocal Manuel Garsia for the first time carried out an indirect laryngoscopy on himself. In a few years that method became widespread in the diagnostics of the larynx diseases. In Russia that method was first used by a famous anatomist and pediatrician K. Rauchfus (Petersburg) and therapist I.O. Zaborovskiy (Moscow). The fact that a doctoral thesis " The larynx mirror and its application to diagnose larynx diseases" was defended by Ivan Turino in 1861 in Petersburg medical-surgical academy shows how important that method was at that time .The thesis described a laryngoscopic picture of 12 patients.

The founders of our largest native schools S.P. Botkin in Petersburg and G.A. Zacharin in Moscow played an important role in the development of our specialty ,especially laryngoscopy. They quickly understood the importance of the new, just born specialty and they assigned independent assistants to master and put in practice the new endoscopic methods. They, as therapists, were naturally interested in laryngology and in rhinology in particular. That is explained by the fact that patients with throat diseases with signs of hoarse cough who didn't need an operation were placed in therapeutic department.

Outstanding professors of Petersburg medical-surgical academy insisted on the necessity to teach otolaryngology. In 1866 the position of private-docent for the optional course of ear diseases was established, though it was not obligatory for students.

A new position at first was given to one of S.P. Botkin's pupils – V.T. Petrovskiy, who delivered lectures on ear and nose diseases. A course of laryngology was delivered by another Botkin's assistant – D.I. Koshlakov. Since 1870 the course of otiatria was delivered by doctor of medicine O.F. Prussak (1839 – 1897), who also used to be Botkin's pupil.

The founder of native otolaryngology was M.P. Symanovskiy (1854 –1922). He was Botkin's intern and by his suggestion he studied laryngology under the guidance of D.I. Koshlakov. M.P. Symanovskiy worked closely with I.P. Pavlov. At firsts he was a private-docent and then, when Koshlakov retired, he became a docent on the staff. In 1885 he was elected to be an adjunct-professor on laryngology.

In 1892 after O.F. Prussak retirement, H.N. Symanovskiy organized the united clinics of ear, nose and throat diseases. Later on he was elected to the academy. In 1893 M.P. Symanovskiy insisted on making teaching otorhinolaryngology in academy obligatory for students. His pupil and successor in the department V.I. Voyachek wrote: "In this sense the academy passed ahead of all the cultural countries of the West", where separate clinics of ear diseases and separate clinics of nose and throat diseases existed for many years.

M.P. Symanovskiy supplied the new clinic with the equipment modern for those times and raised its level to the level of other clinical disciplines. This clinic had played the basic role in teaching and advanced training of doctors in the field of otorhinolaryngology.

M.P. Symanovskiy developed the problem of larynx enervation, stroboscopy methods, studied mutational disorders of voice, introduced the method of larynx tuberculosis treatment with para- and ortochlorphenol, described a new form of ulcerous affection of pharynx mucous membrane (the Tonsilitis of Symanovskiy-Vensan). He used the methods of conditioned reflexes by I.P. Pavlov in objective examination of hearing. In 1903 Symanovskiy organized Petersburg ear, nose and throat diseases scientific society. In 1909 he became the founder of the journal «The bulletin of ear, nose and throat diseases». He wrote monographs about nose diseases and affections of lymphadenoid pharyngeal cycle. M.P. Symanovskiy built a clinic in his home town, Saratov, which was led by one of his pupils –M.F. Cytovitch M.P.

Symanovskiy founded a great school of otorhinolaryngologists, the most famous of those were: V.G. Voyachek, M.F. Cytovitch, P.P. Shevaljov, N.V. Bilogolovov, N.M. Aspisov, Ya.B. Kaplan and others.

Among Moscow otorhinolaryngologists we should mention E.M. Stepanov and S.F. Shtein, who initiated the building of ear,nose and throat diseases at the expenses of their patient Bazanova.

Before the 1917 in Russia there were only 5 otolaryngology clinics, the study of this specialty was not obligatory. That's why most students were not educated in the field of ear, throat and nose diseases diagnostics and

treatment or were not familiar with these special problems. Due to this fact physicians couldn't give the proper medical help to the population.

There were only about 400 doctors-otolaryngologists in the whole Russia. They worked mainly in capitals and large towns , the majority of them had private practice that sharply decreased the availability of getting aid. Meanwhile, otorhinolaryngology has expressed prophylactic character.

In 1922 the historical event for our specialty took place – otorhinolaryngology was introduced into the curriculum of medical institutes as an obligatory subject. Since that time every native medical student has got basic theoretical information and practical skills on this specialty. Departments of ear, throat and nose diseases were organized in all institutes.

Now there are many ENT—departments of medical institutes and institutes of doctors' advanced training, which together with Ukrainian researchl institutes of otorhinolaryngology solve all the scientific and practical problems of otorhinolaryngology, and teach doctors- otorhinolaryngologists. There are ENT-departments in the clinics of all cities, district centers; ENT consulting rooms in all polyclinics.

Due to the great success of the modern otorhinolaryngology, it has expanded and deepened its knowledge in such a way that the following parts have separated from it:

1. Phoniatria –the science of physiology and pathology of the speech apparatus.
2. Surdology, i.e the diagnostics and treatment of people hard of hearing and deaf-and -dumb.
3. Otoneurology – doctors- otorhinolaryngologists working in neurosurgical departments are engaged in a diagnostics of the diseases of VIII pair of skull-cerebral nerves.
4. ENT-professional pathology
5. Phthisiolaryngology.
6. ENT-diseases of childhood.
7. ENT-oncology.

We should always follow the example of the best representatives of medical science, who devoted all their life to serve the people. After professor M.P. Symanovskiy had retired , professor V.G. Voyachek (1876-1971) who was the full member of the Academy of Medical Science of the USSR, Honoured Scientist, lieutenant-general of medical service and an outstanding scientist headed the ENT department from 1917 to 1962, . He is said to be the only person in the world who served in Field Forces for more than 75 years. He died at the age of 95 , working as a scientific consultant of that department. The scientific activity of V.G. Voyachek was versatile, but his most fundamental researches were dedicated to the internal ear and military otorhinolaryngology. He was the first to invent the tables of Russian words for hearing examination, tests to determine the functional condition of the otolith apparatus, methods of sparing operations on ear and nose septum, operations on the larynx, new instruments for the operations on ear, more than 300 scientifical articles and monographs by him have joined the golden fund of the world otorhinolaryngology. He created the whole school of modern otorhinolaryngologists, many of them were the professors and heads of departments: V.F. Undrits, K.L. Hilov, I.P. Kutepov, K.A. Zasosov, N.A. Drennova, A.A. Arjukov, F.A. Popov, V.V. Shapurov, I.A. Lopotko.

Voyachek was succeeded by his follower K.I. Hilov, who made a lot of investigations of vestibular analyzer physiology and pathology; his monograph «The organ of balance» received the International award named after Purkinie.

A valuable contribution to scientific and practical otorhinolaryngology was made by professors A.F. Ivanov, L.I. Svizjevskiy, B.S. Preobrazjenskiy, O.G. Lihachev, M.F. Cytovich, L.T. Levin, S.M. Kompaniets, I.P. Aspisov, V.G. Ermolaev, O.I. O.I. Kolomijchenko, who organized Kiev Research Institute of otorhinolaryngology in 1960.

First ENT-department in Odessa was organized by doctor I.S. Geshelin in 1895 on the basis of the first city hospital. In 1911 Odessa ENT-society was organized. But otorhinolaryngology course wasn't studied at the medical faculty of Novorosiijskiy University, only private docent course was delivered by assistant of the department of normal anatomy Kalachov and then – by the head of the department of operative surgery and topographic anatomy professor I.F. Samarin.

In 1918 the chief of Odessa military hospital doctor of medicine O.M. Puchkovskiy organized some clinics. There were 10 beds for ENT-patients, who were treated by him, on the basis of faculty surgical clinic; he also delivered private-docent course of otorhinolaryngology.

In 1920 the department of otorhinolaryngology became independent in accordance with the resolution of professors' Council of Odessa medical institute. Professor O.M. Puchkovskiy became the head of that department and created an independent ENT-department for 25 patients. The department staff consisted of the professor and 6 assistants; the educational basis was also organized (tables, plaster casts, macro- and micropreparats and so on).

In 1922 professor O.M. Puchkovskiy was elected the head of the department in Kiev medical institute, he also organized of the ENT-department of Kiev medical institute. Doctor of medicine A.I. Geshelin headed the department in Odessa medical institute

In 1927 the clinic moved to the premises the former dental clinic. Hospital for 30 beds and outpatients' clinic were organised. Lessons took place in the clinic and the outpatient department.

In 1930 the clinic moved to another city clinical hospital (now the 11 city clinical hospital). In this clinic ENT-department was organized in 1902, before 1930 it was headed by Ilijashenko, who was a perfect clinical physitian and surgeon.

Prof. A.T. Geshelin was the chief of the clinic from 1922 till 1952.

From 1952 till 1955 the head of the department was docent M.T. Garshin.

From the February 1955 till the January 1963 the Department was headed by the pupil of A.M. Puchkovskiiy prof. L. A. Zaritskiy, who had headed the ENT-department of Kiev dental institute before. Prof. Zaritskiiy was a perfect clinical physician, surgeon, good teacher and fruitful scientific worker, he wrote more than 120 scientific articles, among them several monographs and a text book on ear, throat and nose diseases, which has been republished there times. In 1961 L. A. Zaritskiiy was awarded the title of Honored scientist of the USSR. In 1963 prof. L.A. Zaritskiiy moved to Kiev, where he worked as a deputy director of Kiev research institute of otolaryngology till 1974.

From 1974 till 1990 the department was headed by professor V.D. Dragomiretsky, an outstanding figure of Ukrainian science and engineering. He paid much attention to tonsilar problem. His doctor dissertation was dedicated to clinical -experimental study of peritonsillitis. Since 1968 he has been elaborating the methods of cryosurgical treatment of ENT-diseases.

From 1990 till 1997 the Department was headed by docent G.M.Penkovsky, from 1997 to 2000 - by professor O.V.Dumin . Now the head of the ENT Department of ONMedU is professor Pukhlik S.M.

What scientific problems is the Department working at?

For the last 25 years the department has been studying the problems of working out and perfection of hearing-improving operations in cases of otosclerosis and purulent middle otitis. Some works are devoted to ENT-oncology, because ENT-oncologic patients can get surgical, X-ray and chemotherapy treatment in full volume only at that department.

Much attention is paid to allergology, purulent diseases of the ENT organs, professional pathology. Our ENT clinic has two ENT departments: ENT of general structure and ENT- oncology for 90-100 patients. Besides the work at hospital, the doctors of the Department and hospital provide advisory help to the medical institutions of our city and district. Patients with heavy ENT pathology, with combined pathology are sent for consultation from polyclinics of the city, from ophthalmology clinics, dental, neurosurgery and other specialized institutions.

In our clinic a wide volume of medical aid is given to the patients with a various purulent pathology of ENT, ENT-oncology patients. The plastic and sanative operations on middle ear, nasal cavity, paranasal sinuses, throat, larynx are performed. We shall try to do our best to acquaint you with various kinds of ENT pathology.

There is a scientific students circle at our Department. In the circle you can master practical otorhinolaryngology, make abstract reports, carry out master practical otorhinolaryngology, carry out research work.

CLINIC ANATOMY, PHYSIOLOGY AND METHODS OF EXAMINATION OF ENT - ORGANS

Clinic Anatomy of the External and Middle Ear

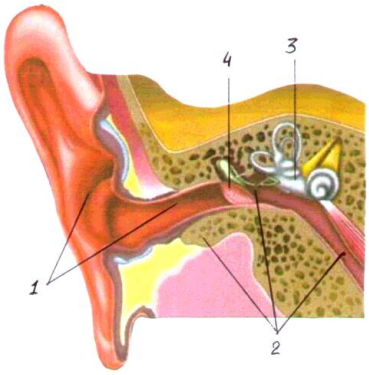


Fig.1. Ear

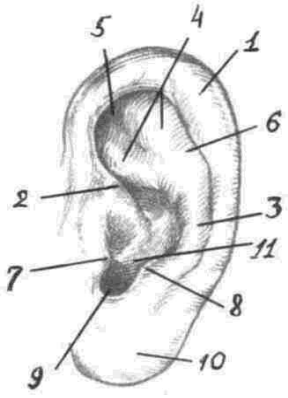


Fig.2. Pinna

Organ of hearing (fig.1) in anatomical relations is divided into three parts: external (1), middle (2,4) and internal (3) ear; functionally into - sound conducting and sound apprehensive apparatus. The auricle, external auditory tube passage, which gather sound waves, tympanic membrane, chain of ear bones and perilympha of internal ear belong to the sound conduction apparatus. The external ear comprises the pinna, or auricle, and the external auditory canal. The shell-shaped *pinna* (fig.2) 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 margin of the pinna is known as the *helix* (1)*;* towards the bottom the pinna gradually turns into the *lobe* (10) 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 canal is named the *tragus* (7). In front of the helix and parallel to it is a ridge known as the *anthelix* (3), with the *antitragus* (8) at its posterior end.

The *external auditory* canal 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 auditory canal consists of outer cartilage and membranous tissue and inner portions of bone.

The external auditory canal is curved in the horizontal and frontal planes. The cartilaginous and bony portions of the canal 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 canal. The oval lumen of the external auditory canal 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 canal 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 canal 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 too third of the anteroinferior wall is made up of cartilage with two vertical fissures through which an inflammatory process in the external auditory canal 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 canal. 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 canal with the articular head of the mandible pushed backwards and upwards.

In the newborn, there is neither bony auditory canal, 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 canal. By the end of the third year the external auditory canal 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 canal. Mechanical irritation of the latter wall, as in wax removal, often causes reflex cough. The lymph from the walls of the auditory canal drains into the nearest lymph nodes located in front of the auricle, on the mastoid process, and under the inferior wall of the auditory canal. Inflammations in the external auditory canal are often accompanied by swelling and pain in these lymph nodes.

Tympanic Membrane

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 incisures in the squama known as the notch of Rivinus *(incisures 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 canal, a middle layer of radiating and circular connective tissue fibers, 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.

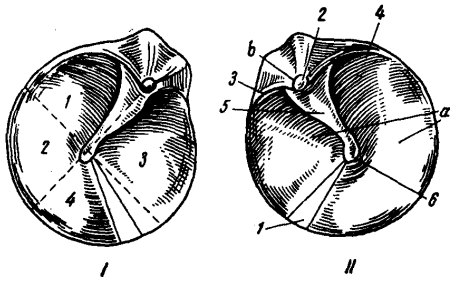


Fig.3. Normal drum

I—right drum is divided into four quadrants: (1) posterosuperior; (2) posteroinferior; (3) anterosuperior; (4) anteroinferior.

II—left drum: (a) pars 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 canal, so that it faces forward, downwards and inwards. In the newborns and breast-fed babies, the drum is almost horizontal.

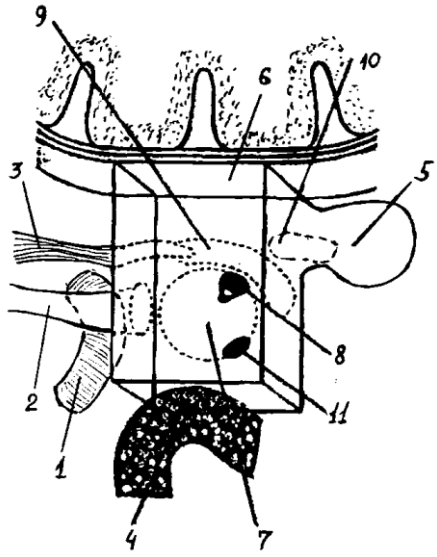


Fig.4. Left tympanic cavity

Examination of the drum through the auditory canal 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 flaccid membrane from the lower taut tense membrane.

Middle Ear

The middle ear comprises the tympanic cavity (fig.4), the mastoid process with its cellular system and the Eustachian tube, all directly interconnected.

The *tympanic cavity* is a small chamber, about 1 cm^3 in size, lying in the depth of the temporal bone, between the tympanic

membrane and the internal ear. In front, through the Eustachian tube(2), the tympanic cavity communicates with the nasopharynx; behind, through the entrance into the mastoid antrum *(aditus ad antrum mastoideum),* it communicates with the cells of the mastoid process (5). The tympanic cavity, similar to the cells of the mastoid process, contains air coming through the Eustachian tube.

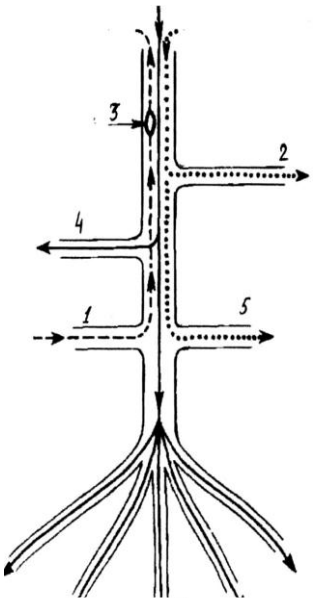


Fig.5. Scheme of the facial nerve
1- n.intermdius
2- n.petrosus major
3- ganglion geniculi
4- n.stapedius
5- chorda tympany
6- pes anserinus major

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 (6) 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 middle otitis. The *inferior wall* or floor of the tympanic cavity is separated from the jugular bulb (4) 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 (1). An opening in the upper part of the *posterior wall* leads to the mastoid antrum *(aditus ad antrum mastoideum).* 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 (7). Above and behind the promontory is an oval window or the vestibular fenestra (8) 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 cochlear fenestra (11) 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 (9). On reaching the entrance to the antrum, the facial nerve canal (fig.5) turns downwards to form a descending knee, then passes behind the posterior wall of the auditory canal 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 purulent middle otitis. 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 (10) 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.

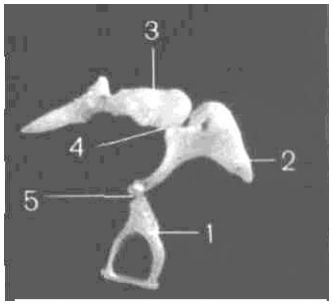


Fig.6. Auditory bones

The tympanic cavity contains the three auditory bones (fig.6) —the malleus (3), the incus (2) and the stapes (1) which are interconnected by joints (4,5) 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 *stapes* 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 stapes 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 canal 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 canal. Behind the spot where the superior wall of the auditory canal merges with the posterior wall, there is a small bony peak or the suprameatal spine *(spina suprameatum)* lying above the external auditory canal. 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 posterior cranial fossa. On the surface facing the posterior 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 air-filled 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 diploes 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 diploes structure has tiny cells resembling a diploes 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, threshold-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 canal 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.

Clinic Anatomy of the Internal Ear or Labyrinth

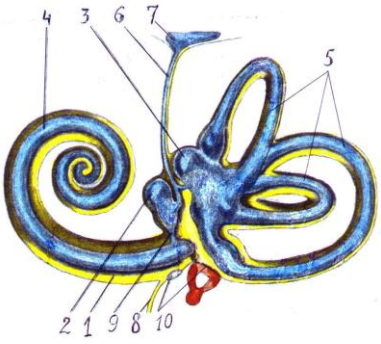


Fig.7. Internal ear

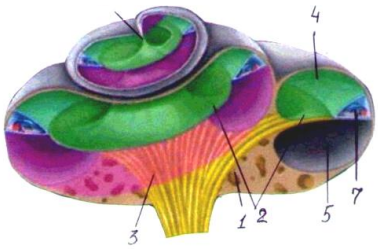


Fig.8. Cochlea

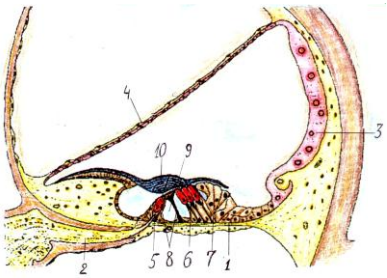


Fig.9. Cochlear duct

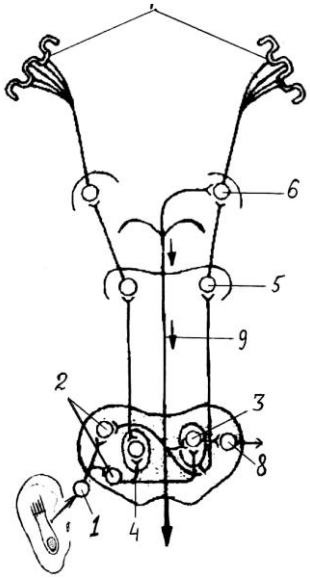


Fig.10. Auditory neural pathways

The internal ear (fig.7) consists of membranous and bony labyrinths, the latter surrounding the former like a capsule. The membranous labyrinth (1) 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 (10); on the opposite, internal wall, there are two recesses for the two membranous sacs of the vestibule. The front sac (2) known as the saccule *(sacculus)* communicates with the membranous cochlea lying before the vestibule, while the rear sac (3) or utricle *(utriculus)* is connected with the three membranous semicircular canals (5) passing behind and above the vestibule.

The *cochlea* (4) is a bony tube which describes two-and-a-half turns around a central pillar called the modiolus and resembles a snail-shell in appearance. The cochlea connected with back skulls pit with the help of acoustic internal passage and water pipe of the snail, which connect perilymphatic space of labyrinth with liquor system (8). An osseomembranous lamina (fig.8) (2) leading from the modiolus (3) to the external wall and also turning round the former, divides the tube lumen into two directions, the upper or scala vestibuli (4) and the lower or scala tympani (5) which communicate at the apex of the cochlea through a small opening known as the helicotrema (6). 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 (7) or *ductus cochlearis.*

The organ of Corti (fig.9), a complex receptive structure of the auditory analyzer, rests on the basilar membrane (*membrana basilaris)* (1), the lower wall of the ductus cochlearis. The basilar membrane is an arrangement of elastic fibers of different lengths strung from the edge of the bony spiral lamina (2) to the opposite, outer wall of the cochlea. The organ of Corti has a very complex histological structure containing external (6) and internal (5) hair cells, column cells (8) and supporting cells. The sensory cells covered with hairs are situated in small groups between the supporting cells (7). The cells are covered with a membrane called the tectorial membrane (10) *(membrana tectoria).* At the foundation hair cells there are nerve plexus which are formed snail part of VIII nerve. Its contact with cells by means of synapses.

Blood supply is carrying out from internal auditive artery. This is the branch of basilar artery (a. vertebralis) enters to inside acoustic passage together with VIII and VII cerebromedullar nerves. Snail branch penetrate into modiolus and give on first spiral branch in every curl of snail.

Auditory neural pathways and their nuclei (fig.10)**.** Hair cells are innervated by dendrites of bipolar cells of spiral ganglion (1) which is situated in Rosenthal's canal. Axons of these bipolar cells end in cochlear nuclei (2), the dorsal and ventral, on each side of medulla. Further course

of auditory pathways is complex. From cochlear nuclei the main nuclear centers in the ascending auditory pathways, sequentially, from below upwards, are: superior olivary complex (3), nucleus of lateral lemniscus, inferior colliculus (5), medial geniculate body (6), auditory cortex (7). The auditory fibers travel via the ipsilateral and contralateral 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 analyzer. 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 canal conducts sound waves from the outer medium to the tympanic membrane. The canal 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 centers 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 canal, the tympanic membrane and the chain of bones), and bone conduction (directly through the cranial bones and the stapes).

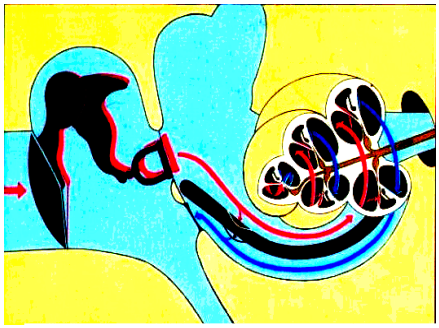


Fig.11. Transmission of the sounds

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 (fig.11).

Sound cave is double oscillation, in which there are phases of decrease and increase of pressure. They enter into the external acoustic canal, reach the tympanic membrane and cause its vibration. During this the whole chain of auditory bones 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 bones increase force of sound-wave oscillations due to changes of their amplitude. Since the area of base of stapes ($3mm^2$) is less than the area of tympanic membrane (about 55 mm^2) , as well as the result of lever type of joints of auditory bones, 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 bones are transformed into oscillation of perilymph with relatively small amplitude with big pressure.

Man can hear external sounds with a frequency of 16 to 20000 cycles per second. Specking 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 is 344 meters 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 decibels (dB). At a distance of one meter, 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 fibers 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 fibers 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 fibers 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 analyzer 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

centers 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 analyzer, thus presenting the latter's entire activity as a single physiological process.

The starting moment of mechanism of sound perception in organ of Corti 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 Titov. 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 Hearing Examination. Hearing Passport

AD (auris dextra)				**Tests**	**AS** (auris sinistra)			
IV	III	II	I		I	II	III	IV
+	-	-	-	**SN**	-	+	+	+
1 m	6 m	6 m	6 m	**WT**	6 m	2 m	ad conch.	0 m
4 m	60 m	60 m	60 m	**ST**	60 m	5 m	3 m	1m
	←		↓	**W**	↓	→		→
-	+	+	+	**R**	+	-	+	-
short.	N	N	N	**Sch**	N	N	short.	short.
-	+	+	+	**G**	+	+	+	-
-	+	+	+	**B**	+	-	+	-

I – normal hearing*; II – left side conductive hearing loss; III – left side sensoneural hearing loss;*
IV – both side mixed hearing loss (otosclerosis); *SN – subjective noise; WT – whispered tests;*
ST – spoken voice tests; W – Weber's test; R – Rinne's test; Sch – Schwabach's test;
G – Gelle's test; B - Bing's test

Hearing test (whispered and spoken voice tests).

1. The patient is at a distance of 6 meters 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 (lip-reading), the patient should not look at the physician.

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

4. If the patient cannot hear at a distance of 6 meters, the physician should approach the patient to a distance of 5 meters, and examine the patient again. The distance should thus be shortened by 1 meter each time until the patient repeats correctly all the words pronounced by the physician.

5. The results of the test are expressed in meters 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.

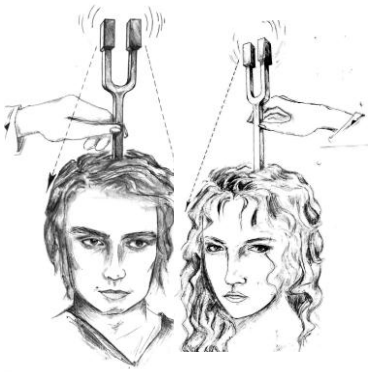


Fig.12. Weber's test.

Fig.13. Rinne's test

Tuning-fork Tests. *Test for air conduction.* A set of tuning forks (C128, 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 canal 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. (fig.12) 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) (fig.13). 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 canal. 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 canal, the result is negative (—).

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

A vibrating tuning fork is placed on the mastoid processes of the patient 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 mastoid processes. If he can hear the ringing fork, the result is expressed as "Schwabach shortened or diminished".

Gelle's test. A vibrating tuning fork is placed on the head vertex; the pressure in the external acoustic canal 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 oval fenestra). If the stapes is fixed, as in otosclerosis, the increasing pressure in the external acoustic canal will not reduce the sound perception. If the auditory apparatus is affected, increased air pressure inside the external acoustic canal 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 canal 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 canal) prolongs bone conduction. In the presence of otosclerosis bone conduction remains the same with the external acoustic canal open and closed.

Audiometric Tests

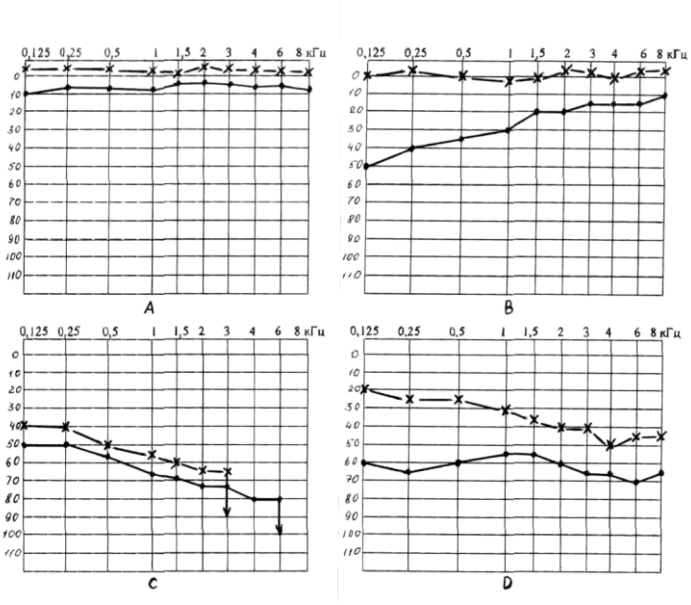


Fig.14. Types of the pure tone audiogramm

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, 6000 and 8000 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 sound isolate chamber.

It is charted in the form a graph called *audiogram* (fig.14). On scale of audiometer the level of normal (according international standard) ear correspond to line 0 dB. 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 sound perceiving apparatus is the lowering level of bone conductivity, the most expressed, as a rule, on high frequency. The level of ear conductivity coincide with the level of bone conductivity.

At the defeat of sound conducting 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.

Speech Audiometry

In this test the patient's ability to hear and understand speech is measured (fig.15). 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 5 dB 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.

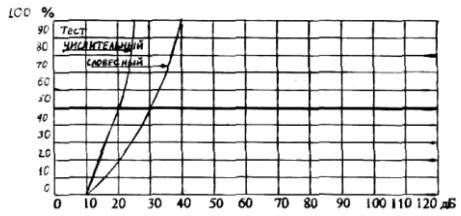


Fig.15.Speech audiometry

Impedance Audiometry

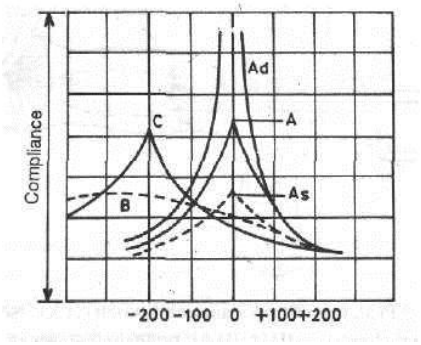
It is an objective test widely used in clinical practice and particularly useful in children. Its uses are: 1. To differentiate ossicular fixation from ossicular dislocation in cases of conductive deafness. 2.To find fluid in middle ear in serous otitis media. 3.To assess function of Eustachian tube. 4.By eliciting stapedial reflex, it can be used to localize lesions of facial nerve and find prognosis of facial paralysis, 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 *tympanogramm.*

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 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 stapedial nerve. 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 Tympanogramm:

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 - Negative pressure in middle ear

Evoked Response Audiometry

It is an objective test which measures electrical activity in the auditory pathways in response to auditory stimuli. It requires special equipment with averaging computer. There are several 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 VIII nerve. The recording electrode is usually a thin needle passed through the tympanic membrane onto promontory. In adults it can be done under local anesthesia but in children or anxious persons sedation or general anesthesia is required. Sedation has no effect on this response.

Auditory brain stem responses. It is a non-invasive technique to find integrity of central auditory pathways through the VIII 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 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.

Control tests

1. Name anatomic formations of the external ear, except one:
   A. membraneous-cartilaginous part of the external meatus
   B. bony part of the external meatus
   C. tragus
   +D. Eustachian tube
   E. auricle
2. What is not related to the stapes parts?
   +A. handle
   B. anterior leg
   C. posterior leg
   D. head
   E. basis
3. What anatomic formation does the lateral wall of the tympanum border on?
   A. canal of the internal carotid
   B. median cranial fossa
   +C. labyrinth
   D. eardrum
   E. bulb of the jugular vein
4. What types of structures of the mastoid process exist, except one:
   +A. connective tissue
   B. sclerous
   C. diploetic
   D. pneumatic
   E. mixed
5. In formation of the tympanic plexus of Yacobson the following structure takes part:
   A. III, VIII, X pairs of the craniocereberal nerves
   B. III, VI, IX pairs of the craniocereberal nerves
   +C. V, VII, IX pairs of the craniocereberal nerves
   D VI, VIII, IX pairs of the craniocereberal nerves
   E. X, XI, XII pairs of the craniocereberal nerves
6. In innervation of the external meatus the following structure takes part:
   A. auricular branch of the glossopharyngeal nerve
   B. auricular branch of the abducent nerve
   C. auricular branch of the hypoglossal nerve
   D. auricular branch of the accessory nerve
   +E. auricular branch of the vagus nerve
7. What of the structures is not included in the transformation system of the ear?
   A. auricle and external meatus
   B. eardrum and hammer (malleus)
   C. incus
   D. stapes
   +E. Eustachian tube

8. What does the bony part of the posterior wall of the external meatus border on?

A. median cranial fossa

B. bulb of the jugular vein

C. eardrum

+D. mastoid process

E. joint of the mandible

9. Peridural cells of the mastoid process are located:

+A. adjacent to the cranial fossa

B. adjacent to the sigmoid sinus

C. adjacent to the cortical layer of the platform of the mastoid process

D. at the apex of the mastoid process

E. around of the canal of the facial nerve

10. The auditive tube is lined with:

A. single-layer flat nonkeratinized epithelium

B. multilayered flat keratinized epithelium

C. cylindrical epithelium

+D. cubic and ciliary epithelium

E. multilayer flat nonkeratinized epithelium

11. A patient aged 27 complains of stuffiness feeling in the ear, diminished hearing, tinnitus, periodic shooting pain. Against the background of developing catarrhal otitis media there was a painful perception of loud sounds - hyperacusis which was evidence of dysfunction of the strapedius muscle of the tympanum. Affection of what nerve can cause hyperacusis?

A. trigeminal

+B. facial

C. glossopharyngeal

D. vagus

E. abducens

12. A 3-month's baby suddenly developed raised temperature, there were restlessness, nausea, vomiting, dyspeptic disorders, moderate rigidity of the occipital muscles. On examination the ENT doctor diagnosed preperforated stage of acute purulent otitis media. What could cause the listed symptoms in the child?

A. signs of general intoxication

B. development of purulent meningitis and irritation of the dura mater membrane

+C. presence of fissures in the upper wall of the tympanum and irritation of the dura mater membrane

D. gastrointestinal disorders

E. presence of fissures in the medial wall of the tympanum and irritation of the labyrinth

13. In microsurgical operative interventions in the middle ear it is necessary to observe extreme care while manipulating in the area of the lower wall of the tympanum as injury is possible of:

+A. internal jugular vein

B. canal of the facial nerve

C. sigmoid sinus

D. internal carotid artery

E. dura mater membrane

14. A patient with chronic middle purulent otitis accompanied by destruction of the bone tissue (epitympanitis) was performed sanation operation in the middle ear which was accompanied by trepanation of the bone of the mastoid process. It is necessary to take into consideration that in this case most often there is the following type of the mastoid process structure:

A. diploetic

+B. sclerotic

C. pneumatic

D. cartilaginous

E. mixed

15. A patient developed liquorrhea from the ear after the craniocereberal trauma, accompanied by fracture of the skull basis in the area of the temporal bone. Trauma of what part of the ear should we think of?
+A. upper wall of the bone part of the external meatus and/or tympanum
B. anterior wall of the bone part of the external meatus and/or tympanum
C. posterior wall of the bone part of the external meatus and/or tympanum
D. lower wall of the bone part of the external meatus and/or tympanum
E. upper wall of the membraneous-cartilaginous part of the external meatus
16. Where are Santorini's fissures located in the external meatus?
+A. in the cartilage of the anterior-lower wall of the membraneous-cartilaginous part
B. in the cartilage of the posterior wall of the membraneous-cartilaginous part
C. in the cartilage of the upper wall of the membraneous-cartilaginous part
D. in the anterior-lower wall of the bony part
E. in the back wall of the bony part
17. What muscles of the tympanum do you know?
A. stapedius and incudal
B. mallear and stapedius
C. tensor tympani
+D. stapedius and tensor tympani
E. mallear and tensor tympani
18. What anatomic formation does the posterior wall of the tympanum border on?
A. canal of the internal carotid
B. median cranial fossa
C. labyrinth
+D. mastoid process
E. bulb of the jugular vein
19. What are features of the Eustachian tube structure of a newborn in comparison with an adult person, except one:
A. wider
+B. more twisting
C. shorter
D. gapes
E. located more horizontally
20. What branches do not diverge from the facial nerve?
A. petrosus superficialis major
B. stapedius
C. chorda tympany
D. chorda tympany, stapedius
+E. petrosus minor
21. There are the following groups of cells of the mastoid process, except:
A. periantral
B. perifascial
C. perilabyrinthine
D. angular
+E. intracranial
22. What of the listed structures are not related to transmission system of the ear?
A. auricle and external meatus
B. eardrum and hammer
+C. chorda tympany
D. incus
E. stape
23. Where is tympanic aperture of the Eustachian tube located?
A. on the lower wall of the tympanum

+B. on the anterior wall of the tympanum
C. on the medial wall of the tympanum
D. on the posterior wall of the tympanum
E. on the upper wall of the tympanum

24. Perisinusal cells of the mastoid process are located:
A. adjacent to the median cranial fossa
+B. adjacent to the sigmoid sinus
C. adjacent to the cortical layer of the mastoid process platform
D. at the apex of the mastoid process
E. around the canal of the facial nerve

25. The auditory tube consists of the following parts:
A. dermal and membraneous-cartilaginous
B. mucous and bony
C. pharyngeal and tympanic
+D. bony and membraneous-cartilaginous
E. dermal and bony

26. Against the background of mastoiditis - inflammation of the cellular system of the mastoid process - a patient had dysfunction of the mimic muscles of the same half of the face, taste sensitivity of the anterior 2/3 of the tongue of the same side, dryness of the eye. At what level was there affection of the facial nerve?
+A. up to the first knee of the facial nerve
B. between the first and second knee of the facial nerve
C. lower the second knee
D. in the area of soft tissues of the neck
E. central type of affection

27. A patient had blood-purulent discharge from the ear against the background of exacerbation of chronic middle purulent otitis. On examination by the otolaryngologist there were revealed granulations coming from the attic against the background of total perforation of the eardrum. What part of the tympanum were these granulations coming from?
A. anterior
B. posterior
C. lower
+D. upper
E. anterior-lower

28. In microsurgical operative interventions in the middle ear it is necessary to observe extreme care while manipulating in the area of the superior wall of the tympanum as injury is possible of:
A. internal jugular vein
B. canal of the facial nerve
C. sigmoid sinus
D. internal carotid artery
+E. dura mater

29. After feeding the baby was laid in bed, regurgitation of the food mass has arisen in the horizontal position. In some hours the child developed acute purulent otitis media. What is possible mechanism of development of acute otitis media?
A. ARVI
B. ingress of food masses in the external meatus
C. gastro intestinal disorder, intoxication of an organism
D. as a result of a narrow, twisting auditory tube drainage function of the middle ear is bad in the newborn
+E. there was ingress of food masses through the wide, horizontally located auditory tube in the baby

30. While performing otoscopy to a patient with total perforation of the eardrum which resulted in chronic purulent otitis media, the doctor has found a polyp localized in the promontorim. What wall of the tympanum was the polyp localized on?

A. upper
B. lower
C. lateral
+D. medial
E. posterior

31. Name anatomic formations of the auricle without the cartilage:

A. helix
B. anthelix
+C. earlobe
D. tragus
E. triangular fossa

32. What nerve is the stapes muscle innervated with?

A. glossopharyngeal
+B. facial
C. vagus
D. abductor
E. cochlear-vestibular

33. Where is the entrance into the mastoid antrum localized?

A. on the lateral wall of the tympanum
B. on the medial wall of the tympanum
C. the anterior wall of the tympanum
+D. on the posterior wall of the tympanum
E. on the lower wall of the tympanum

34. What are parts of the Eustachian tube?

A. only cartilaginous
B. only bony
+C. membraneous–cartilaginous and bony
D. pulled
E. loose

35. What is function of the chorda tympany?

A. innervation of the mimic muscles of the same side
B. painful sensitivity of the same side of the face
C. taste sensitivity of the posterior third of the tongue
D. motor innervation of the pharyngeal muscles
+E. taste sensitivity of the anterior 2/3 of the tongue of the same side

36. Where is the sigmoid sinus located?

A. on the upper side of the pyramid of the temporal bone
B. on the anterior side of the pyramid of the temporal bone
+C. on the posterior side of the pyramid of the temporal bone
D. on the internal side of the pyramid of the temporal bone
E. on lateral margin of the pyramid of the temporal bone

37. What is transmission of sound?

A. protection of the labyrinth against excessively strong sounds
B. transformation - amplification of sound vibrations
C. accommodation of hearing in low sounds
+D. conduction of sound vibrations to neuroepithelium
E. distortion of sound vibrations

38. Where are round and oval windows of the labyrinth located?

A. on the lower wall of the tympanum
B. on the anterior wall of the tympanum
+C. on the medial wall of a the tympanum
D. on the posterior wall of the tympanum
E. on the upper wall of the tympanum

39. Threshold cells of the mastoid process are located:
A. adjacent to the median cranial fossa
B. adjacent to the sigmoid sinus
+C. adjacent to the cortical layer of the mastoid process platform
D. at the apex of the mastoid process
E. around the canal of the facial nerve

40. What is not related to functions of the external ear?
A. catching of sound
B. protective
+C. perception of sound
D. transformation of sound
E. transmission of sound

41. A round foreign body of the external meatus in a 7 –year-old child got stuck in the area of its isthmus. Where is this foreign body located?
A.at the entrance into the external meatus
B. in the middle of the external meatus
C. on the border of external 1/3 of the meatus
+D. on the border of internal 1/3 of the meatus
E. at the eardrum

42. A patient with chronic purulent epitympanitis was found to have bone destruction on palpation by Voyachek probe in the area of entrance into the antrum. In what area of the tympanum wall was the destructive process localized?
A. anterior
B. posterior
+C. lower
D. upper
E. medial

43. In microsurgical operative interventions in the middle ear it is necessary to observe extreme care while manipulating in the area of the anterior wall of the tympanum as injury is possible of:
A. internal jugular vein
B. canal of the facial nerve
C. sigmoid sinus
+D. internal carotid artery
E. dura mater membrane

44. The doctor makes otoscopy in a newborn with acute purulent otitis media. For qualitative examination of the eardrum it is necessary to pull of the auricle:
A. upward and backward
+B. downward and backward
C. forward and upward
D. forward and downward
E. upward

45. A patient had a cicatricial narrowing of the external meatus after the craniocereberal trauma, accompanied by fracture of the mandible. Trauma of what part of the ear should we think of?
A. upper wall of the bony part of the external meatus
+B. anterior wall of the bony part of the external meatus
C. posterior wall of the bony part of the external meatus

D. lower wall of the bony part of the external meatus
E. upper wall of the membraneous-cartilaginous part of the external meatus

46. What identification points are not related to the auricle?
+A. handle of the hammer
B. helix
C. tragus
D. triangular fossa
E. antihelix

47. What anatomic formation does the anterior wall of the tympanum border on?
A. bulb of the jugular vein
B. median cranial fossa
C. posterior cranial fossa
+D. canal of the internal carotid artery
E. entrance to the antrum

48. Name identification points of the eardrum, except one:
A. promontary
B. umbo
C. handle of the malleus
D. tense part
E. flaccid part

49. What is located on the border of the membraneous-cartilaginous and bony parts of the external meatus?
A. earwax glands
B. Santorini's fissures
+C. isthmus - the narrowest part of the meatus
D. isthmus - the widest department of the meatus
E. eardrum

50. What is the function of the first branch of the facial nerve - the n.petrosus superficialis major?
A. innervation of the mimic muscles of the same side
B. painful sensitivity of the same side of the face
C. taste sensitivity of the anterior 2/3 of the tongue of the same side
+D. secretory innervation of the lacrimal gland of the same side
E. taste sensitivity of the posterior third of the tongue

51. What does the sigmoid sinus come from?
A. sagittal sinus
+B. transversal sinus
C. cavernous sinus
D. internal jugular vein
E. external jugular vein

52. What is the transformation of sounds?
A. protection of the labyrinth against excessively strong sounds
+B. amplification of sound vibrations
C. accommodation of hearing to low sounds
D. conduction of sound vibrations to the neuroepithelium
E. distortion of sound vibrations

53. On what wall of the tympanic cavity is the tympanic portion of the facial nerve (Fallopian canal) located?
A. lower wall of the tympanum
B. anterior wall of the tympanum
+C. medial wall of the tympanum
D. posterior wall of the tympanum
E. upper wall of the tympanum

54. Angular cells of the mastoid process are located:

A. adjacent to the medial cranial fossa

+C. on the border of the medial and posterior cranial fossa

D. at the apex of the mastoid process

E. around of the canal of the facial nerve

55. What is the acoustic impedance?

+A. acoustic resistance of media of the external, middle and internal ear

B. conduction of the sound through the external, middle and internal ear

C. transformation of the sound in the external, middle and internal ear

D. perception of the sound in the external, middle and internal ear

E. transmission and transformation of sounds in the ear

56. The patient complains of earache, growing worse on chewing, pressing on the tragus. On examination the otolaryngologist has found hyperemia, infiltration of the skin in the membraneous-cartilaginous part of the meatus and diagnosed external diffuse otitis. Choose what part of the external meatus makes the membraneous-cartilaginous part:

A. 1/3

B. 1/2

+C. 2/3

D. 1/4

E. 3/4

57. Against the background of mastoiditis a patient had dysfunction of the mimic muscles of the same half of face, taste sensitivity on the anterior 2/3 of the tongue of the same side, hyperacusis (painful perception of sounds), lacrimation from the eye. At what level was there affection of the facial nerve?

A. up to the first knee of the facial nerve

+B. between the first and second knee of the facial nerve

C. lower the second knee

D. in the areas of soft tissues of the neck

E. central type of affection

58. In microsurgical operative interventions in the middle ear it is necessary to observe extreme care while manipulating in the area of the middle wall of the tympanum as injury is possible of:

A. internal jugular vein

+B. canal of the facial nerve

C. sigmoid sinus

D. internal carotid artery

E. dura mater membrane

59. Against the background of exacerbation of chronic purulent otitis media a patient had hectic temperature, tenderness on palpation, edema of the soft tissues and expansion of the venous pattern in the field of the posterior surface of the mastoid process and neck along the course of m.sternocleidomastoideus. What affection of the cerebral sinus can we think of, taking into consideration extension of the inflammatory process onto the jugular vein?

A. lower sagittal sinus

B. cavernous sinus

C. transversal sinus

D. upper sagittal sinus

+E. sigmoid sinus

60. A patient developed parotid phlegmon as a result of furuncule of the external meatus complicated by abscess. What is most probable way of distribution of infection in this case?

A. hematogenic

B. lymphogenic

C. through bony part of the external meatus

+D. through Santorini's fissures of the external meatus

E. through the mandibular joint

61. After the flu a patient developed sensoneural relative deafness. The following represents the basic parts of pathways of the auditory analyzer except:

A. spiral ganglium
B. dorsal and ventral nuclei
+C. Bekhterev's nucleus
D. upper olives, posterior tubers of the quadrigeminal plate
E. Geshle's gyrus

62. A unit of oscillation frequency of a sound wave is:

A. DeciBell
B. Ampere
+C. Hertz
D. Tesla
E. Second

63. A unit of loudness of a sound is:

A. Watt
B. Hertz
C. Tesla
+D. DeciBell
E. Microbar

64. The human ear perceives a strip of sound with frequencies of:

A. 6-16000 Hz
B. 10-18000 Hz
C. 16-16000 Hz
+D. 16-20000 Hz
E. 22-24000 Hz

65. The author of the resonant theory of hearing is:

A. Politcer
B. Bekeshi
C. Simanovsky
+D. Helmgolts
E. Pavlov

66. Basic theses of the resonant theory are:

A. Resonance of certain sites of Reissner's membrane
B. Resonance of certain sites of the integumentary membrane
+C. Resonance of certain sites of the basic membrane
D. Resonance of certain groups of Corti's cells
E. Resonance of certain sites of the membranous labyrinth

67. While examining hearing a patient is noted to have sharp increase of thresholds of bone conduction of sounds in frequencies above 8000Hz. In what part of the cochlea the focus of affection is located?

A. apex of the cochlea
B. middle third
+C. the basic helix of the cochlea
D. can be in any part of the cochlea
E. the focus is located outside the cochlea, in spiral ganglia

68. Fibres of the auditory nerve represent:

A. axons of afferent cells
+B. neurons of the first order
C. neurons of the second order
D. axons of the dorsal nucleus
E. axons of the ventral nucleus

69. The cortical part of the auditory analyzer is:

A. precentral gyrus
B. a frontal lobe of the brain
+C. Geshle's gyrus
D. postcentral gyrus
E. occipital lobe of the brain

70. A patient has dysfunction of the mimic muscles, sensoneural hearing loss, taste sensitivity on the anterior 2/3 of the tongue on the same side, dryness of the eye. Where pathological process is localized:
+A. cerebellipontine
B. precentral gyrus
C. posterior central sulcus
D. Geshle's gyrus
E. basic sulcus

71. Where is Corti's organ located:
A. in horizontal semicircular canal
B. in vertical semicircular canal
C. in sagittal semicircular canal
D. in labyrinthine vestibule
+E. in the cochlea

72. As a result of the craniocerebral trauma a patient had a transversal fracture of the temporal bone pyramid at the level of the internal meatus. What craniocerebral nerves pass through it?
A. VI and VII pairs
+B. VII and VIII pairs
C. VIII and IX pairs
D. IX and X pairs
E. X and XI pairs

73. For differential diagnosis of the affection level of the sound perception apparatus it is necessary to know its pathways. The following represents a nuclear zone of the vestibular and auditory analyzer. Name nuclei of the cochlear analyzer:
A. Bekhterev's upper nucleus
+B. dorsal and ventral nuclei
C. lateral Deiters' nucleus
D. medial triangular nucleus of Shvalbe
E. descending Roller's nucleus

74. The following tests are applied to examination of hearing by means of tuning forks, except one:
A. Rinne
B. Weber
+C. Valsalva
D. Shvabah
E. Bing

75. Choose correct sequence of arrangement of the tuning fork in carrying out Rinne experiment:
A. tuning fork is placed at the auricle at first, and then moved to the mastoid process
B. tuning fork is moved from one mastoid process to another
+C. tuning fork is placed on the mastoid process, and then moved to the auricle
D .tuning fork is placed on the tragus, and then on the mastoid process
E. tuning fork is placed on the mastoid process, and then on the tragus

76. While investigating hearing in Weber's experiment the leg of the sounding tuning fork can be placed in the following points, except one:
A. on the top of the head, on the median line
B. on the back of the head, on the median line
C. on the nose bridge, on the glabella point
D. in the projection of the first vertebra body, on the median line
+E. on the chin, on the median line

77. While examination of hearing in Schwabach's test the following is compared:
A. bone and air conductivity of the healthy ear
B. air conductivity of the healthy and sick ear
+C. bone conductivity of the healthy and sick ear
D. bone and air conductivity of the healthy and sick ear
E. bone and air conductivity of the sick ear
76. Time shortening of the tuning fork in Schwabach's test is a sign of:
A. chronic purulent otitis media
B. adhesive otitis
+C. cochlear neuritis
D. exudative otitis
E. foreign body in the auditory canal
77. What impairments can be revealed in the patient by means of the tuning fork examination of hearing:
A. affection of the sound perception apparatus
B. affection of the sound-conducting apparatus
+C. affection of the sound perception and sound-conducting apparatus
D. impairment of articulation of speech
E. affection of the sound perception and sound-conducting apparatus, impairment of articulation of speech
78. The following groups of cells are related to Corti organ, except:
A. external hairs cells
B. internal hairs cells
C. Deiter's cells
D. Gensen's cells
+E. Langgance's cells

Clinic Anatomy of the Vestibular Apparatus

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 (fig.16). The front sac known as the saccule (1) *(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 (3) passing behind and above the vestibule.

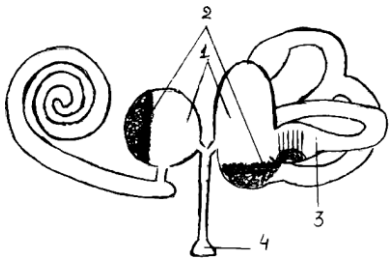


Fig.16. Vestibular apparatus

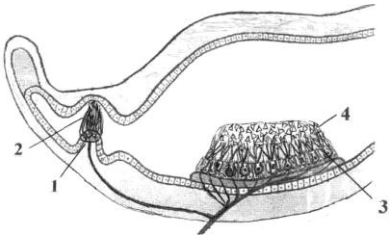


Fig.17. Scheme of vestibular apparatus

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* (2), 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 otolith. Mechanism of irritation in sacculus of vestibulum is determined by displacement of otolith by irritation of neuroepithelium. Besides, otolithic apparatus is exposed by continuous irritation of gravitate 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 sagital. One end of each canal opens out into a larger space known as ampoule, the other end is even. The frontal and sagital canals have a common even stem (*crus commune).* The ampoule of each

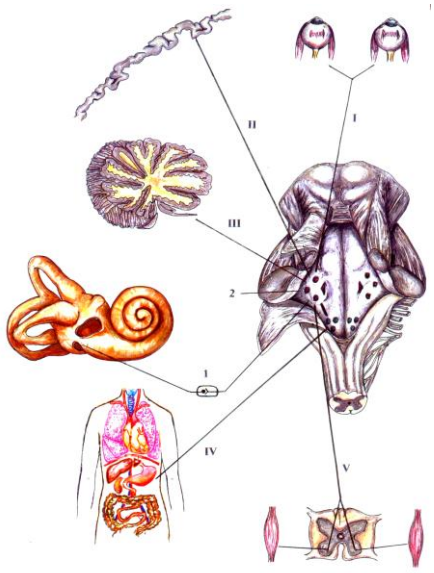


Fig.18. Nervous communications of the vestibular analyzer

membranous canal contains a ridge, the *crista 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 analyzer (ampular receptor) is determined by deviation of endolymph, displacement of cupula, irritation of neuroepithelium in crista ampullaris (fig.17).

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

The first neuron of vestibular tract is situated in Scarpe ganglion 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 (fig.18):

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) 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, n.vagus). This explains nausea, vomiting, palpitation, sweating and pallor seen in vestibular disorders.

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

Nucleus of vestibular analyzer in bottom of rhombic fosse (superior angular Bechterev's nucleus, lateral Deiters'es nucleus, medial triangular Swalbe's nucleus, inferior Roller's nucleus) communicates 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 co-operation 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 muscles, joints and tendons, which are called proprioreceptors.

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 ampoules 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.

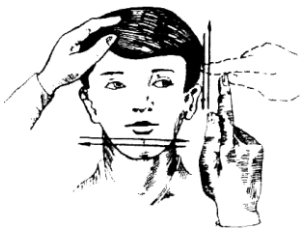


Fig.19. Study of nystagmus

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 (fig.19) is used to assess the reaction of a stimulated vestibular apparatus.

Examination of ampular apparatus is based on ***Laws of Evald*** (fig.20)***:***

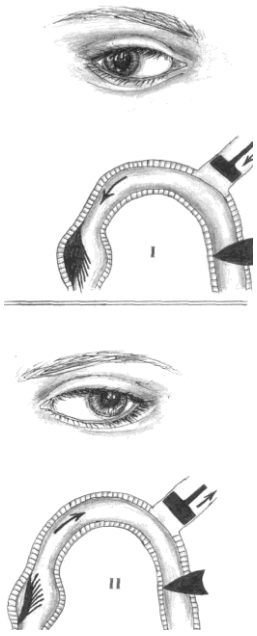


Fig.20. Laws of Evald

1) The plane of nystagmus always correspondent to pane of irritated semicircular canal;

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

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) Direction of nystagmus is always opposite to the direction of endolymph displacement. Direction of nystagmus is determined by his fast component. Slow component of nystagmus and protective movements coincide always with endolymph 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 patient 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.

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.

Statokinetic Tests:

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.

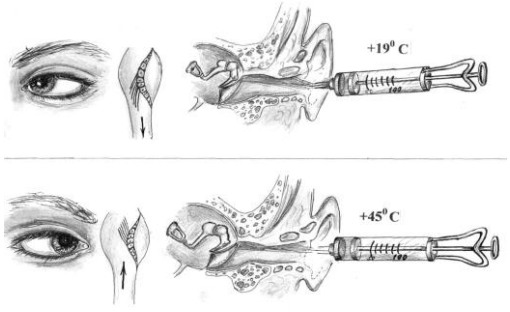


Fig.21. Caloric test

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 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.

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 (fig.21)**.** 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 25°C. The right external acoustic meats is douched with 100 ml of water at 25°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 12-20 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. 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.

Fig.22. Rotation test

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 (fig.22). His head should be tilted 30° forward 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 (fig.23). 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.

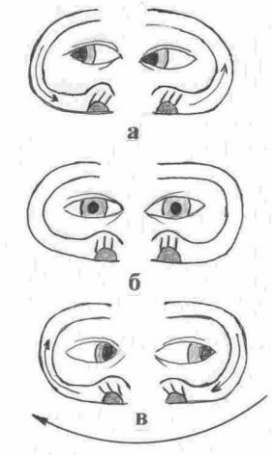


Fig.23. Nystagmus reaction after right side rotation

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 (fig.24)**.** 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 index finger (or using of pneumatic funnel of Zigle or rubber bulb). If the labyrinth is intact, no

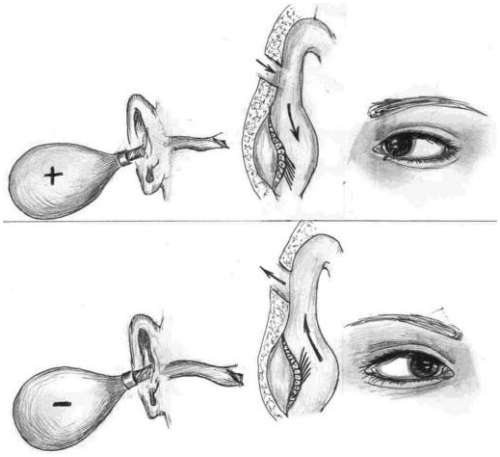


Fig.24. Pneumatic test

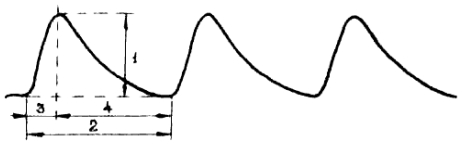


Fig.25. Electronystagmogramm

nystagmus appears. If there is a fistula in the lateral semicircular canal, nystagmus occurs in the direction of the affected side. On decompression a nystagmus to the opposite side develops.

Testing 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 (fig.25). It is a method of detecting and recording of nystagmus which is spontaneous or induced by caloric, positiona l, 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 permits to keep a permanent record of nystagmus.

Control tests

1. Movement of the endolymph from the leg to the ampoule in the lateral semicircular canal causes nystagmus:
   A. does not cause
   +B. to the side of the stimulus
   C. to the opposite side
   D. upward
   E. downward
2. What nystagmus does not occur?
   A. spontaneous
   +B. static
   C. calorific
   D. pressor
   E. postrotational
3. What is not adequate stimulus for the otolith apparatus:
   A. rectilinear acceleration
   B. change in the position of the body
   C. change in the position of the head
   +D. angular acceleration
   E. acceleration of force of gravity
4. Where is the first neuron of the vestibular analyzer located:
   +A. fundus of the internal auditory meatus
   B. diamond-shaped fossa
   C. lower wall of the external auditory meatus
   D. medulla oblongata
   E. trapezoidal body
5. What nerves are located in the internal auditory meatus:
   +A. vestibulocochlear and facial

B. additional
C. abducens and lateral
D. glossopharyngeal
E. trigeminal and olfactory

6. What is related to the vestibular analyzer:
A. antrum
+B. semicircular canals
C. promontorium
D. cochlea
E. Corti's organ

7. Nystagmus, caused by affection of the labyrinth, is characterized by direction:
+A. horizontal
B. diagonal
C. vertical
D. sagittal
E. frontal

8. Name the form of nystagmus, which arises during rotation in Barany chair:
A. congenital
B. adjusting
C. optokinetic
D. spontaneous
+E. experimental

9. What parameters of nystagmus are not determined in visual study:
A. force
B. direction
C. plane
D. amplitude
+E. quality

10. Spontaneous vestibular syndromes, except:
A. vertigo
B. spontaneous nystagmus
C. disequilibrium
D. nausea, vomiting
+E. paresis of the facial nerve

11. What component is the direction of nystagmus determined by:
A. left
B. right
C. fast
D. slow
+E. labyrinthine

12. Where is the endolymphatic sac located:
+A. on the posterior surface of the pyramid of the temporal bone
B. in the internal auditory meatus
C. on the anterior surface of the pyramid of the temporal bone
D. on the surface of the mastoid process
E. on the squama of the temporal bone

13. What is an adequate stimulus for the semicircular canals:
A. change in the position of the body in the space
B. change in the position of the head in the space
+C. angular acceleration
D. rectilinear acceleration

E. acceleration of force of gravity

14. Peripheral nystagmus is not characteristic of:
    A. small amplitude
    +B. large amplitude
    C. frequent rhythm
    D. horizontal plane
    E. binocularity
15. Kind of nystagmus, which is observed in instillation of cold water in the ear:
    A. congenital
    B. adjusting
    C. optokinetic
    D. spontaneous
    +E. experimental
16. Nystagmus is not characterized by:
    A. direction
    B. plane
    +C. reaction of the pupils
    D. amplitude
    E. degree
17. What forms of reactions do not arise in irritation of the semicircular canals:
    A. vertigo
    B. nystagmus
    C. change in the pulse rate
    D. deflection of head to the side of the slow component of nystagmus
    +E. adiachokinesis
18. Movement of the endolymph from the ampoule to the leg in the lateral semicircular canal causes nystagmus:
    A. does not cause
    B. to the side of the stimulus
    +C. to the opposite side
    D. upward
    E. downward

Clinic Anatomy of the Nose and Paranasal Sinuses

External nose *(nasus externus).*

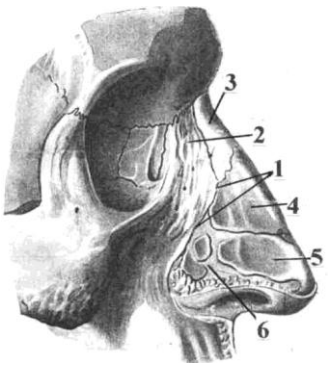


Fig.26. External nose

There are the external nose, the nasal cavity and the paranasal sinuses. The skeleton of the external nose is formed by bones and cartilages (fig.26). The bony part of the nose is formed by paired nasal bones (3) and by the frontal processed of the maxilla (2). The free ends of these bones form a piriform aperture (1). The cartilaginous framework of the nose includes triangular cartilage (4), paired ala cartilage (5), and the accessory cartilage. The skin on the external nose has many sebaceous and sweats 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 superior 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 (posteriorly). 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 (fig.27), or the septum, consists of the anterior cartilaginous (1) and posterior bony parts. The bony part of the septum is formed by the perpendicular plate of the ethmoid (3) and the vomer (2). The lateral (external) wall (fig.28) of the nasal cavity has a more complex structure. Three nasal conchs extend from the external wall toward the nasal septum: the superior, middle and inferior conch (6-8). Three nasal meatuses are distinguished accordingly: the superior, middle, and inferior meatuses. The space between the nasal conch 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 meat 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 (5) open into the superior nasal meat.

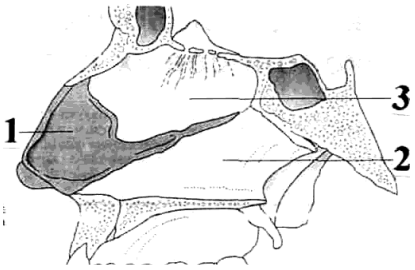


Fig.27. Medial wall of the nasal cavity

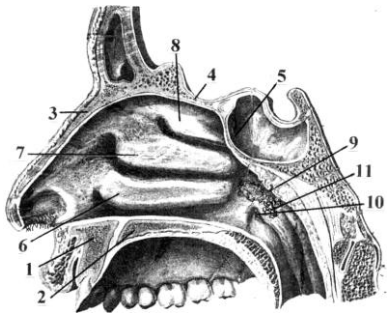


Fig.28. Lateral wall of the nasal cavity

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 olfactory's tracts, pellucid septum and ends into the cortex centers (gyres hippocampus, gyres dentate, olfactory's sulks).

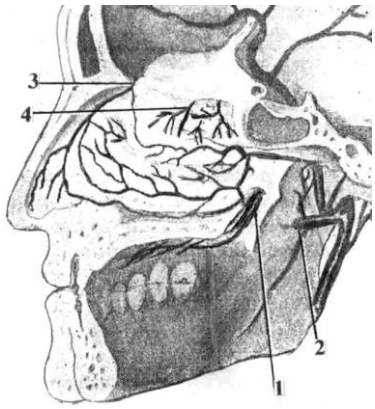


Fig.29. Blood supplied of the nasal cavity

The nasal cavity is supplied with blood (fig.29) via the branches of the external carotid arteries (a.sphenopalatina) (1) and internal carotid artery (aa. ethmoidales anterior and posterior, the branches of a.ophthalmica) (3,4). 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.

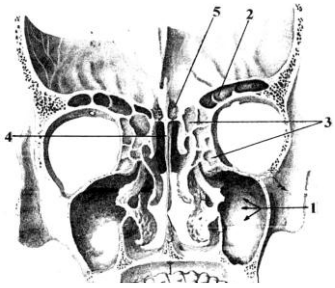


Fig.30. Paranasal sinuses

Paranasal sinuses. The paranasal sinuses (fig.30) 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* (1) 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 tuberoses.

The *ethmoidal sinuses* (ethmoidal labyrinth) consist of air cells of the ethmoid (3) 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* (2) 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 sphenoid basis. 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.

Functions of PNS: air-conductions of the inspired air by the providing large surface area over which the air is humidified and warmed; to provide resonanse to voice; to act as termal isolators to project the orbit and cranium from variations of intranasal temperature; to lighten the scul bones.

Clinic Physiology of the Nose and Paranasal Sinuses

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).

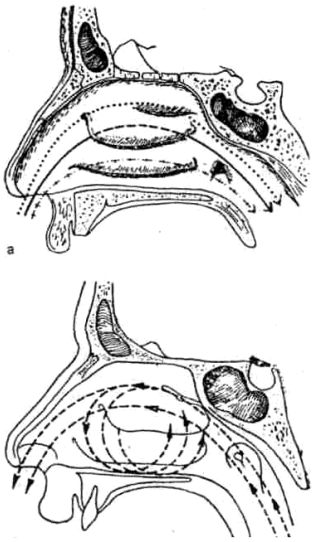


Fig.31. Respiratory function of the nose

The *protective function* (fig.31) 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. Lysocime, 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 neuroepithelial 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 conches, 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 of the Nose and Paranasal Sinuses

Fig.32. Anterior rhinoscopy

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 (fig.32, fig.33). 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 conches, the nasal meatuses, and the vomer can thus be inspected. The nasopharynx can be examined in a similar way.

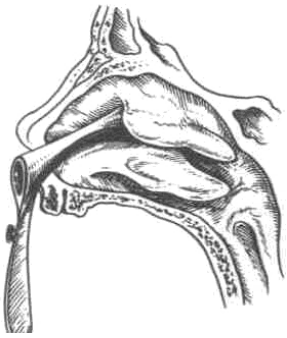
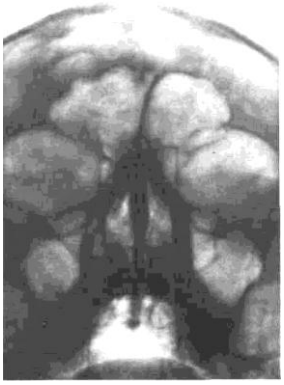


Fig.33. Middle rhinoscopy

Examination of the paranasal sinuses (PNS)**.** Basic methods of examination are: endoscopic, roentgenological ones, computer X-ray tomography, magneto-resonance tomography, radio nuclide scintigraphy and such additional methods as ultrasound biolocation, distance infrared thermography, SHF-radiometry.

Roentgenography and clinical analyze of roentgenological signs is one of the main methods of investigation of PNS (fig.34). 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 (tumors, cysts). Layer examination - tomography in the certain depth, contrast investigation by jodolipol of the affected sinus are used to specify the pathological process.



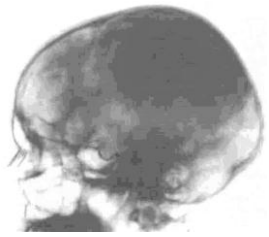


Fig.34. X-ray of PNS

Computer X-ray tomography (CT)**.** By CT the picture (fig.35) is got not in the X-ray film but is synthesized with the help of electronic computer (EC). X-rays, coming from the tube in different 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 tumors 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.

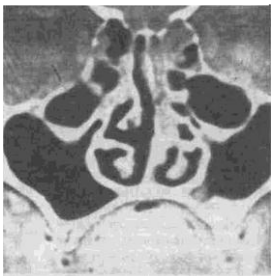


Fig.35.CT of PNS

Nucleo-magnetic resonance (MRT)**.** Diagnostic picture, got by magneto-resonance tomography (fig.36). 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 tumors, determine their localization, dimensions and spread, contours, invasion of the neighbor anatomical structures.

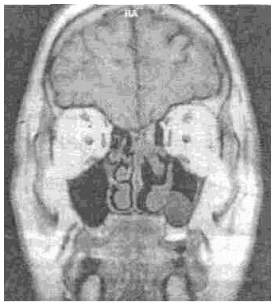


Fig.36. MRT of PNS

Radio-nuclide scintigraphy. Radio-isotope diagnostics is based on the registration and measuring 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 injured organ. Higher degree is evidence of tumor; lower degree is evidence of inflammation.

Unionizing methods. Last times such methods of ray diagnostics, as thermography, SHF-radiometry, ultrasound biolocation, became widespread. They are absolutely undangerous and unharmful, cheap. That's why it can be used during professional observations, in children's otorhinolaryngology, in pregnant women. But these methods don't give enough information about tumors of PNS, inflammatory processes in the posterior sinuses (middle and

posterior cells of ethmoidal labyrinth, sphenoidal sinus). However, they can be used as the methods of pre-clinical express-diagnostics, and for the undangerous and repeated control of the dynamic during the treatment.

Infra-red 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 PNS 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. Computer lets to find and calculate the parts of thermogramm, 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-frequent 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 together with the length of wave.

Ultrasound 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).

Control tests

1. What is opened in the middle nasal passage, except?
   +A. nasolacrimal duct
   B. maxillary sinus
   C. frontal sinus
   D. anterior cells of the ethmoid labyrinth
   E. middle cells of the ethmoid labyrinth
2. How is the secretory innervation of the nose accomplished?
   A. by the vagus nerve
   B. by the upper cervical sympathetic node
   +C. by the Vidian nerve
   D. by the olfactory nerve
   E. none of the enumerated
3. What is done with the air during its passage through the nasal cavity?
   A. warmed, moistened
   B. dried
   C. disinfected
   D. cleaned
   +E. all enumerated
4. A patient aged 28 years old has a furuncle of the nose. What is found on the vestibulum of the nasal cavity, except:
   +A. multiple cylindrical ciliated epithelium
   B. hairs
   C. sebaceous glands
   D. multilayer flat epithelium
   E. cartilages
5. A patient complains of nasal obstruction of one half of the nose. In examination the choanal polyp is diagnosed. What are choanae bounded by, except:
   A. vomer
   B. internal plate of the pterygoid process

+C. quadrangular cartilage
D. horizontal plate of the palatine bone
E. the body of the sphenoid bone

6. There is a patient with the diagnosis: acute sinusitis complicated by periostitis of the orbit. What anatomical structures border with the nasal cavity, except:
A. anterior cranial fossa
+B. posterior cranial fossa
C. orbit
D. oral cavity
E. roof of the teeth

7. An ambulance brought a patient with injury of the nose to the ENT department. What basic anatomical formations does the external nose have, except:
+A. choanas
B. root
C. back
D. tip
E. alas

8. A patient complains of nasal obstruction and discharge from the nose. What are methods of examination of the nasal cavity?
A. anterior rhinoscopy
B. medial rhinoscopy
C. posterior rhinoscopy
D. rhinofibroscopy
+E. all of the enumerated

9. What does the floor of the nasal cavity consist of?
+A. palatine branches of the maxilla, horizontal plates of the palatine bones
B. internal plate of the pterygoid process of the sphenoid bone
C. frontal branches of the maxilla
D. quadrangular cartilage
E. all enumerated

10. What composes of the external wall of the nasal cavity?
A. ethmoidal cells
B. nasal bone
C. the frontal processus of the maxilla
D. lachrymal bone
+E. all enumerated

11. The system of what arteries is the nose and its paranasal sinuses blood supplied from, except:
A. external carotid artery
B. vertebral artery
C. internal carotid artery
+D. external and internal carotid artery
E. all enumerated

12. A canal of what nerve passes in the orbit wall of the maxillary sinus?
A. n.mandibularis
B. n.olfactory
C. n.orbitalis
+D. n.infraorbitalis
E. n.facialis

13. A patient was admitted to the ENT department with complaints on severe pain in the region of the forehead, stuffiness and purulent discharge from the nose. Where is natural aperture of the frontal sinus opened?

A. inferior nasal meatus
+B. middle nasal meatus
C. superior nasal meatus
D. general nasal meatus
E. nasopharynx

14. A patient with acute purulent frontal sinusitis is indicated trepanopuncture. What wall of the frontal sinus is the thickest?
A. posterior
B. lower
+C. anterior
D. middle
E. none of the enumerated

15. A patient with acute purulent frontal sinusitis is indicated trepanopuncture. What wall of the frontal sinus is the thinnest?
A. posterior
+B. lower
C. anterior
D. middle
E. none of the enumerated

16. What age is formation of the frontal sinus finished, as a rule?
+A. 20-25 years
B. by the moment of birth
C. first year of life
D. 6-8 years
E. 12-14 years

17. A patient developed nasal bleeding from the anterior parts of the nasal septum against the background of hypertonic crisis. What is the nasal septum formed by?
+A. perpendicular plate of the ethmoid bone, vomer, quadrangular cartilage
B. perpendicular plate of the palatine bone, quadrangular cartilage
C. frontal process of the maxilla, vomer
D. internal plate of the processus pterygoideus of the sphenoid bone
E. anterior and medial cells of the ethmoid labyrinth

18. A patient is disturbed by purulent discharge from the nose, headache, discharge flows on the posterior wall of the pharynx, it is seen in the upper nasal passage. What is opened in the upper nasal passage?
A. nasolacrimal canal
B. frontal sinus
+C. posterior cells of the ethmoid labyrinth, sphenoid sinus
D. anterior and medial cells of the ethmoid labyrinth
E. maxillar sinus

19. A patient was admitted to the clinic with the diagnosis: furuncle of the nose. On examination: hyperemia and tenderness in the region of the wing of the nose on the right and right cheek. Where does the blood outflow from the external nose?
+A. anterior vein of face, orbital vein, cavernous sinus
B. sphenopalatine vein, sigmoid sinus
C. superficial temporal vein, internal jugular vein
D. internal jugular vein, sigmoid sinus
E. sphenopalatine vein, internal jugular vein

20. A child of 2 years old has been suffering from running nose, discharge from the nose of mucopurulent nature for two weeks. The infiltration in the region of the interior angle of the eye on the right developed two days ago, the body temperature increased. Opacity of the ethmoid labyrinth cells is noted by the X-ray

examination of the paranasal sinuses. What bone is affected during the contact penetration of infection from the ethmoid labyrinth in the orbit?

A. cribriform plate
B. lacrimal bone
C. perpendicular plate of the ethmoid bone
+D. paper plate of the ethmoid bone
E. palatine bone

Clinic 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 esophagus inferiorly, it passes into the esophagus below the sixth cervical vertebra.

From within outwards it consists of four layers: mucous membrane, pharyngeal aponeurosis (pharyngobasilar fascia), muscular coat, buccopharyngeal fascia. Mucous membrane lines the pharyngeal cavity and is continuous with mucous membrane of Eustachian tubes, nasal cavities, mouth, larynx and esophagus. The epithelium is ciliated columnar in the nasopharynx and stratified squamous elsewhere. There are numerous mucous glands scattered in it. Pharyngeal aponeurosis (pharyngobasilar fascia) is a fibrous layer which lines the muscular coat and is particularly thick near the base of skull but is thin and indistinct inferiorly. It fills up the gap left in the muscular coat near the base of skull.

Muscular coat consists of two layers of muscles, with three muscles in each layer:

external layer contains superior, middle and inferior constrictor muscles, internal layer contains stylopharyngeus, salpingopharyngeus and palatopharyngeus muscles. Buccopharyngeal fascia covers outer surface of the constrictor muscles and in the upper part it is also prolonged forwards to cover the buccinators muscles. Above the upper border of superior constrictor, it blends with pharyngeal aponeurosis.

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 fauces opens into the oropharynx; and the inlet of the larynx and the esophagus 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 conches. 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.

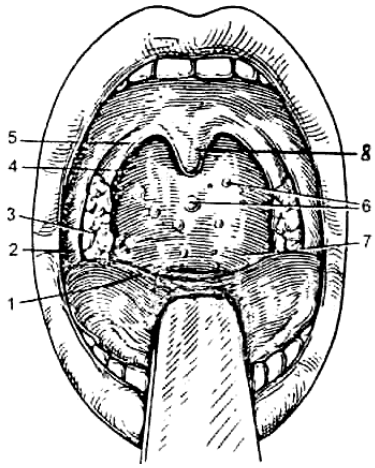


Fig.37. Pharyngoscopy

The ***oropharynx*** (fig.37) 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 (7) borders on the third cervical vertebra. The fauces is confined in the space between the edge of the soft palate (5), the uvula (8), the anterior (2) and posterior (4) palatine arches, and the root of the tongue (1). 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.

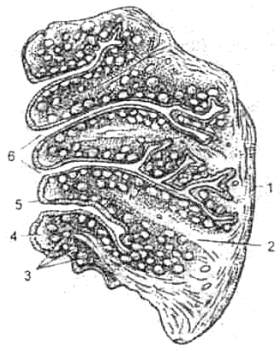


Fig.38a. Palatine onsilla

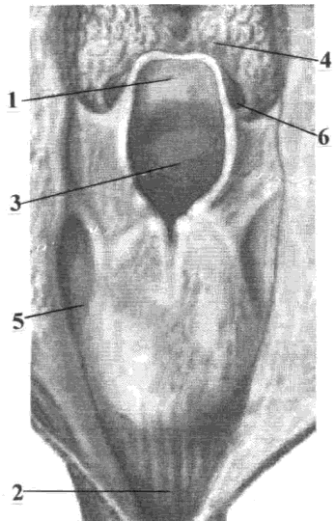


Fig.38b. Laryngopharynx

Palatine tonsils (3) are found in triangular recesses (tonsillar fosses) between the palatoglossal and palatopharyngeal arches (fig.38a). The histological structure of the lymphoid tissue of the pharynx is uniform: a mass of lymphocytes with spherical formations known as follicles is located between connective-tissue fibers (2). 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 (5). The tonsil has 16-18 deep pits known as lacunae, or crypts (6). The total sum of the surface area of the crypt is about 300 sm^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 (1). 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 (3), which are differentiated into: a) so called primary follicles, which are made up only of lymphocytes; b) bigger sized secondary follicles with a germinal centre, surrounded by lymphocytes. In the past years the anatomical unit of the tonsils has been isolated – cryptophon. It is made up of the crypt lumen with its contents, the crypt epithelium, the lymphoepithelial tissue of crypts 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; immunoglobulin 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 defence 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 defence – interferon, and also the lysocime.

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 – streptococcus, which plays a role similar to that of E.coli in the intestines. Streptococcus is saprophytes along with other conditional pathogenic coccus and anaerobic microbes. This function enables the sustainment of the normal microflora. 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 (6) of the pharynx where it is present in the form of small 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 (fig.38b)**.** The superior edge of the epiglottis (1) and the root of the tongue (4) form the border between the oropharynx and the laryngopharynx. The lower end of the laryngopharynx narrows into a funnel and is continuous with the esophagus (2). The entrance to the larynx (3) 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 diverticulitis known as the piriform recesses (5). Food moves to the esophagus by these piriform recesses.

A retropharyngeal space is found between the posterior wall of the pharynx and the prevertebral fascia. The space is a flat slit packed with loose connective tissue. The retropharyngeal space is divided sagital 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 suppurate in children thus causing a retropharyngeal abscess.

The parapharyngeal space is pyramidal in shape with its base at the base of skull and its apex at the hyoid bone. The medial wall is buccopharyngeal fascia covering the constrictor muscles, posterior wall is prevertebral fascia covering prevertebral muscles and transverse processes of cervical vertebrae, lateral wall is medial pterygoid muscle, mandible and deep surface of parotid gland. Through this space pass the carotid artery, jugular vein, IXth, Xth, Xlth, Xllth cranial nerves and sympathetic trunk. It also contains upper deep cervical nodes. Parapharyngeal space communicates with other spaces, viz. retropharyngeal, submandibular, parotid, carotid and visceral. Because infection of parapharyngeal space can come from pharynx (acute and chronic infections o tonsil and adenoid, bursting of peritonsillar abscess), teeth (dental infection usually comes from the lower last molar tooth), ear (apex mastoiditis), other spaces (infections of parotid, retropharyngeal and submaxillar spaces), external trauma (penetrating injuries of neck, injection of local anesthetic in tonsillectomy or mandibular nerve block).

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.

Clinic 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 esophagus, is accomplished by a complicated and well coordinated swallowing reflex. The muscles of the tongue, pharynx and the larynx contract in a specific sequence.

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

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

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

Inspired air is first warmed in the nose and then in the pharynx, where it is also cleaned from dust which sticks to the mucous lining of the pharyngeal walls. The physiology of the palatine tonsils is not

autonomous. It is part of the function of the entire lymphatic system of the body. It is believed that the lymphoid apparatus of the pharynx, in particular, the palatine tonsils (like similar accumulations of lymphoid tissue in the small intestine) protect the body from the ingress of micro-organisms. During the first years of life, the lymphoid structures of the pharynx attain maximum growth, but during sexual maturation (at the age of 14-15) they undergo partial and gradual back development.

Methods of Examination of the Pharynx

Palpation. The regional lymph nodes of the pharynx are palpated: the submandibular nodes, the nodes in the retromandibular fosse, 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.

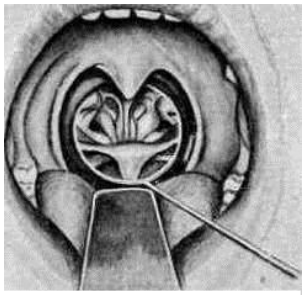


Fig.39. Epipharyngoscopy

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 (fig.39). A warmed nasopharyngeal speculum and a spatula are used for this purpose. The superior parts of the nasopharynx, the choanae and 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 conches) 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 postero-superior wall of the nasopharynx without reaching the superior edge of the vomer and the choanae.

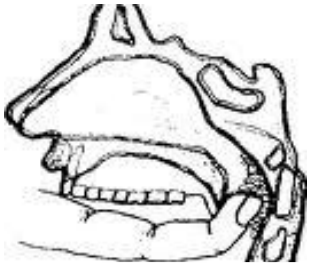


Fig.40. Palpation of the nasopharynx

Palpation of the nasopharynx (fig.40). 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.

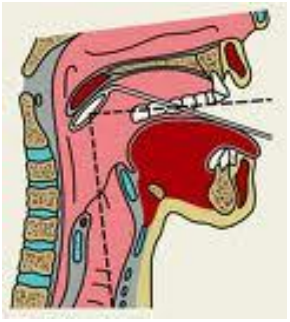


Fig.41. Hypopharyngoscopy

Hypopharyngoscopy (fig.41). The lower portions of the pharynx should be inspected using indirect laryngoscopy with a warmed laryngeal speculum. The tip of the tongue should be wrapped in a piece of gauze and held by the fingers of the left hand so that the thumb is on the superior and the middle finger on the inferior surfaces of the tongue, the index finger lifting the upper lip. The tongue is pulled slightly forward and downward.

The laryngeal speculum should be held by its handle in the right hand and moved into the mouth without touching the root of the tongue and the posterior wall of the pharynx. The mirror surface should be directed downwards. The patient is asked to utter the sound 'ee' and gently breathe in.

Clinic Anatomy of the Larynx

The larynx (fig.42) lies in front of the hypopharynx opposite the third to sixth cervical vertebrae. It moves vertically and in antero-posterior 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 edematous 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 glottides 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 alas 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 (8). It is a leaf-like yellow elastic cartilage forming anterior wall of laryngeal inlet.
4. Arytenoid cartilages (12). 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 ring.

Intrinsic laryngeal membranes:

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

Quadrangular membrane**.** It lies deep to mucosa of aryepiglottic folds and is not well defined. It stretches between the epiglottis 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, Interarytenoid (transverse aryteniod),Thyroarytenoid (external part)

Tensors : Cricothyroid, Vocalis (internal part of thyroarytenoid)

Acting on laryngeal inlet:

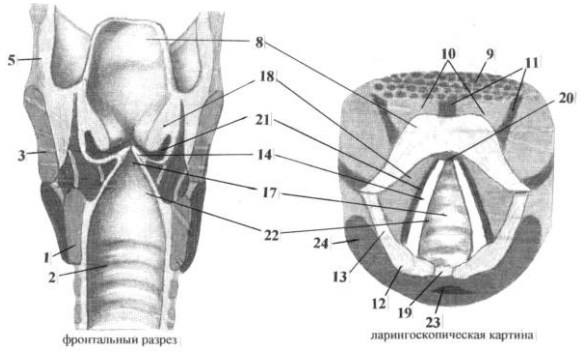


Fig.42. Anatomy of the larynx

Openers of laryngeal inlet: Thyroepiglottic (part of thyroarytenoid;

Closers of laryngeal inlet: Interarytenoid (oblique part), Aryepiglottic

(posterior oblique part of interarytenoids)

Extrinsic muscles. They connect the larynx to neighboring 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 (18) and vocal (14) - 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 (22) extends from vocal cords to lower border of cricoid cartilage (1).

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 subepithelial connective tissue.

Glottis (rima glottides) (17). 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 is drained by lymph to upper deep cervical lymphnodes. Infraglottic larynx is drained to prelaryngeal and pretracheal lymphnodes 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: sphincter closure of laryngeal opening, cessation of respiration, cough reflex.

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

Nerve supply of larynx.

Motor. All the muscles which move the vocal cord are supplied by the recurrent laryngeal nerve except the cricothyroid muscle. Right recurrent laryngeal nerve arises from the n.vagus at the level of subclavia artery, hooks round it and then ascends between the trachea and esophagus. The left recurrent laryngeal nerve arises from the n.vagus in the mediastinum at the level of arch of aorta, loops round it and then ascends into the neck in the tracheo-esophageal groove.

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

Methods of Examination of the Larynx

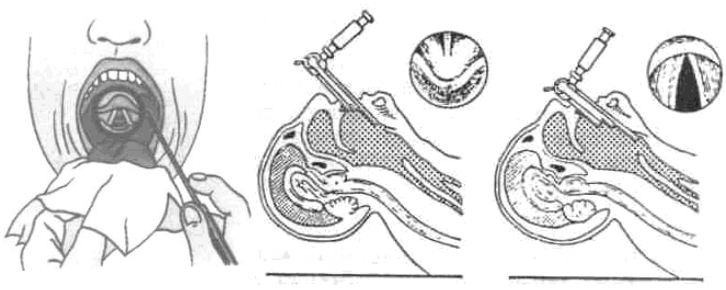


Fig.43. Direct and indirect laryngoscopy

Laryngoscopy (fig.43) is visual inspection of the larynx. 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 tumor should be removed. At the present time direct laryngoscopy precedes the intubation of the airways for anesthesia

and is the first step in tracheobronchoscopy. Direct laryngoscopy in children can be performed without anesthesia.

Indirect laryngoscopy is carried out using a laryngeal speculum. The mirror is fixed in the handle, warmed with fire to 40-50°C 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 valeculas are seen between the epiglottis and the tongue root; they are bounded by the lateral and middle glossoepiglottic 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.

X-ray and CT is an important methods of examination of the larynx.

Clinic Anatomy and Physiology of trachea

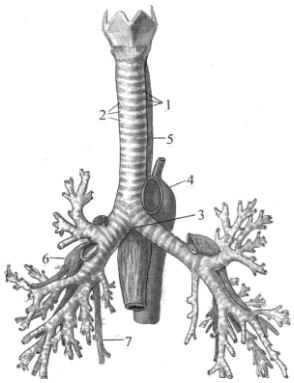


Fig.44. Tracheobroncheal tree

The trachea (fig.44) is the continuation of the larynx, with which it is connected through the cricotracheal ligament. The trachea is a long tube, about 11-13 cm long, beginning at the level of the seventh cervical vertebra; at the level of the fourth and fifth thoracic vertebrae it divides into two main bronchi. The point of division is called the bifurcation. In neonates, the lower end of the trachea is found at the level of the third-fourth thoracic vertebrae. The tracheal wall consists of 16-20 U-shaped hyaline cartilages, whose free ends are directed posteriorly. The cartilages are connected by a membranous wall and annular ligaments owing to which the tracheal lumen broadens during inspiration and narrows during expiration. The tracheal lumen varies between 15 and 22 mm in men and between 13 and 18 mm in women. In infants, the lumen is 6-7 mm. The trachea is lined with mucous membrane with columnar ciliated epithelium. The cilia move in the upward direction. The posterior membranous part of the tracheal wall is adjacent to the anterior wall of the oesophagus. A semilunar projection, known as the carina of the trachea, is found at the point of bifurcation. One of the main bronchi, the right one, is wider and shorter than the left. The right bronchus is 3 cm long and the left one, 5 cm long. Foreign bodies usually get into the right bronchus.

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

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

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

Clinical Anatomy and Physiology of Esophagus

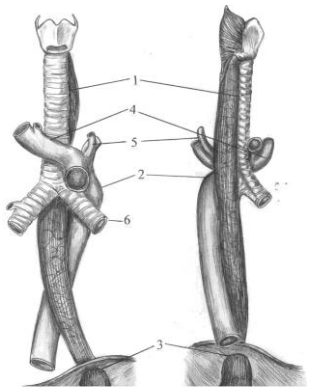


Fig.45.Esophagus

It is a fibromuscular tube about 25 cm long in an adult. It extends from the lower end of pharynx (C6) to the cardiac end of stomach (T11). It runs vertically but inclines to the left from its origin to thoracic inlet and again from T7 to esophageal opening in the diaphragm. It shows three normal constrictions and it is important to know their location at esophagoscopy (fig.45). They are:

1. At pharyngo-esophageal junction (C6)—15 cm from the upper incisors.
2. At crossing of arch of aorta and left main bronchus (T4)—25 cm from upper incisors.
3. Where it pierces the diaphragm (T10)—40 cm from upper incisors.

Foreign bodies in the esophagus can be held up at these constrictions.

The *wall* of esophagus consists of four layers. They are:

- mucosa, which is lined by stratified squamous epithelium;
- submucosa, which connects mucosa to muscular layer;
- muscular layer, which has inner circular and outer longitudinal fibers. Circular fibers at the lower end are thickened to form a cardiac sphincter. The upper third of esophagus has striated, the lower third smooth, and the middle third both striated and smooth muscle fibers.
- fibrous layer which forms loose covering of esophagus.

Nerve Supply: parasympathetic fibers come from n.vagus (X) and sympathetic fibers from the sympathetic trunk.

*Lymphatic drainage: t*he cervical, thoracic and abdominal parts drain respectively into deep cervical, posterior mediastinal and gastric nodes.

Physiology of the swallowing. The act of swallowing is divided into three phases: oral or buccal; pharyngeal; esophageal.

Oral or buccal phase. The food which is placed in the mouth is chewed, lubricated with saliva, converted into a bolus and then propelled into the pharynx by elevation of the tongue against the palate.

Pharyngeal phase is initiated when the bolus of food comes into contact with pharyngeal mucosa. A series of reflex actions take place carrying the food past oro- and laryngopharynx into the esophagus. The communications into nasopharynx, oral cavity and larynx are cut off. Closure of nasopharynx: soft palate contracts against the Passavant's ridge on the posterior pharyngeal wall and completely cuts off the nasopharynx from oropharynx. Closure of oropharyngeal isthmus: the entry of food back into oral cavity is prevented by contraction of tongue against the palate and sphincteric action of palatoglossal muscles. Closure of larynx: aspiration into the larynx is prevented by temporary cessation of respiration, closure of laryngeal inlet by contraction of aryepiglottic folds, closure of false and true cords, and rising of larynx under the base of tongue. Contraction of pharyngeal muscles and relaxation of cricopharyngeus: relaxation of cricopharyngeus muscles is so timed and synchronous that food passes from pharynx into the esophagus during contraction of pharyngeal muscles.

Esophageal phase. After food enters the esophagus, the cricopharyngeal sphincter closes and the peristaltic movements of esophagus takes the bolus down the stomach. Gastro-oesophageal sphincter at the lower end of esophagus relaxes well before peristaltic wave reaches and permits fluids to pass. Bolus of food is passed by contraction of peristaltic waves and then the sphincter closes.

Control tests

1. What is necessary to do in digital examination of the nasopharynx in children in order to prevent biting of the personal finger of the examiner?
    A. press down the tongue by spatula
    +B. index finger of the hand presses the cheek of the child between his teeth
    C. shut the nostrils of the examined by the thumb and index finger
    D. put the tampon
    E. general anesthesia
2. What epithelium is the nasopharynx covered with?
    A. multilayer flat
    +B. ciliated cubic
    C. ceratous multiple-line
    D. cylindrical
    E. nonceratous multilayer flat
3. What epithelium is the oropharynx covered with?
    A. ciliated multilayer flat
    B. ciliated cubic
    C. ceratous multilayer
    D. cylindrical
    +E. nonceratous multilayer flat
4. Where is the retropharyngeal space located?
    +A. between the posterior wall of the pharynx and prevertebral fascia
    B. between the muscular layer and the mucous membrane of the pharynx
    C. between the prevertebral fascia and deep muscles of the neck
    D. into the pharynx
    E. between pharynx and esophagus
5. How is the structural unit of the palatine tonsils called?
    A. crypt
    +B. cryptolymphon
    C. lacuna
    D. follicle
    E. connective tissue capsule
6. Who was the first to describe the lymphadenoid pharyngeal ring?
    +A. Simanovskiy
    B. Voyachek
    C. Waldayer
    D. Polittser
    E. Vensan
7. What parts does the pharynx consist of, except?
    A. nasopharynx
    B. oropharynx
    C. laryngopharynx
    D. hypopharynx
    +E. retropharynx
8. What does the wall of the pharynx consist of, except?
    +A. deep cervical muscles

B. mucous membrane
C. muscular layer
D. fibrous layer
E. adventitia

9. A patient has frequent anginas in the anamnesis. What tonsils of the lymphadenoid pharyngeal ring are known to you, except?
A. two palatine
B. one pharyngeal
C. one lingual
D. two tubal
E. two choanal

10. What do piriform recesses pass to?
+A. in the esophagus
B. in the trachea
C. in the pharynx
D. in the larynx
E. in the nasopharynx

11. Where are laryngeal ventricules located?
A. between the vestibular folds
+B. between the vestibular and vocal folds
C. in the interarytenoid space
D. recess between lateral wall of the pharynx and larynx
E. recess between the epiglottis and the root of the tongue

12. What are piriform recesses?
A. recess between the vestibular and vocal folds
+B. recess between lateral wall of the pharynx and larynx
C. recess between the epiglottis and the root of the tongue
D. recess between epiglottis and lingual tonsil
E. fissure between two vocal folds

13. What epithelium are vocal folds covered with?
A. cubic
B. multilayer cylindical
+C. multilayer flat
D. multiserial ciliary
E. multilayer ceratous

14. What muscle widens the larynx and allows a man to make “inhalation”?
A. vocal muscle
B. thyroarytenoid muscle
+C. posterior cricoarytenoid muscle
D. cricothyroid muscle
E. obliqus interarytenoid muscle

15. What function does external muscles of the larynx perform?
A. direct the epiglottis
B. stretch the vocal folds
C. widen the glottis
+D. move upward and downward the larynx
E. constrict the glottis

16. A patient aged 58 complains of dryness, tickling in the throat. On examination the pharyngeal mucous membrane is dry, glistening, thinned. What methods of the endoscopic study of the pharynx are known to you, except:
A. oropharyngoscopy
B. posterior rhinoscopy

C. indirect laryngoscopy
+D. esophagoscopy
E. fibroscopy

17. A child of 4 years old is revealed to have the adenoids of III degree. What does prolonged disturbance of nasal breathing result in, except?
A. disturbance of hearing
B. disturbance of the sense of smell
C. incorrect development of the maxillofacial skeleton
D. frequent inflammatory processes of the upper and lower respiratory tract
+E. vestibular disturbances

18. A patient complains of the sensation of tickling, pain in the throat. What nerves enter the pharyngeal nervous plexus?
A. trigeminal
B. glossopharyngeal
C. vagus, accessory
D. sympathetic
+E. all enumerated

19. A child of 6 years old has hypertrophy of the palatine tonsils of III degree. What functions do the tonsils carry out, except?
A. immune
B. hemopoietic
C. nervous reflex
+D. olfactive
E. informative

DISEASES OF THE EAR

Earwax (Cerumen) Plug (fig.46) is produced as a result of upset function of the glands in the cartilaginous part of the external acoustic meatus. Earwax is the dried up secretion of the ceruminous glands mixed with desquamated epithelium. If the glands function normally, the cerumen dries into small crusts which are then easily removed from the meatus due to movement of its anterior wall during movement of the jaw (in talking and chewing). If the secretion remains in the ear for a long time, the epidermal plug dries into a dense clot that is firmly fixed in the meatus of the ear. The leading symptom of the earwax impaction is impairment of the hearing function, often noise in the ear and autophonia (conduction of own voice into the ear). In this case the acoustic meatus is obturated with ceruminous mass completely. Vertigo, headache, nausea, and cardiac dysfunction develop sometimes as well.

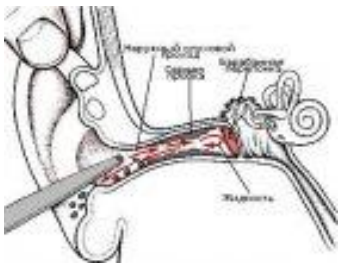


Fig.46 Irrigation of the ear

Treatment. Irrigation of the external acoustic meatus is an effective means. The following rules should be observed when carrying out this manipulation. First, it is necessary to establish if the patient had diseases of the ear in the past, since the tympanic membrane might have been perforated during this disease. Irrigation of the ear with a perforated tympanic membrane is dangerous because water can penetrate into the middle ear, exacerbate the process, and cause pus discharge. Earwax should in such cases be removed using a hook.

Earwax plug can be removed by irrigation only on the condition that obturation of the acoustic meatus is incomplete. Warm (37°C) water should be used for irrigation of the ear. Cold water can irritate the labyrinth with subsequent unpleasant sensation for the patient (vertigo, nausea, vomiting, etc.). Remaining water should be removed from the ear with sterile turunda or dry cotton applicator.

A Janet`s syringe (capacity 100-150 ml) should be used for irrigation of the ear. A jet of water should be directed on the postero-superior wall of the acoustic meatus, which should be straightened by

pulling the pinna up and back. Earwax plug can sometimes be removed by parts soft earwax can be removed by irrigation only, while a solidified mass should first be softened by instilling warm (37°C) drops of sodium bicarbonate in glycerol 3 times a day for 10-15 minutes, during 2-3 days. After instilling drops, the patient should be warned that his hearing can temporarily be impaired as a result of swelling of the earwax plug in his ear. Epidermal plugs should be removed from the ear in the same way.

INFLAMMATORY DISEASES OF THE EXTERNAL EAR

Erysipelas. This can be either primary or secondary. In the latter case the inflammation extends from the skin of the face or the head. The causative factor is the haemolytic streptococcus.

Symptoms. There are marked hyperemia and swelling of the skin of the auricle, including the lobule. The ear is very tender to palpation. The bullous form is characterized by formation of vesicles (bullae) with serous fluid. If the affection is circumscribed, the affected site is separated from intact skin by a ridge of swollen tissue of a different color. The body temperature is high (to 39-40°C); the patient feels chill and burning sensation in the zone of the ear. The erythema and swelling extend outside the ear and the mastoid process (characteristic signs).

Treatment. The intramuscular antibioticotherapy (group of penicillinum) , desensitive therapy are prescribed. Light treatment (UV) is applied to the affected skin (erythema dose) and antiinflammatory ointments should be applied.

Perichondritis is a diffuse inflammation of perichondrium. The skin of the external ear is also involved as a rule. Serous and suppurative perichondritis are distinguished. The latter occurs more frequently.

The first and the main *symptom* is pain in the external ear or the acoustic meatus. Pain can precede reactive infiltration of the skin of the external ear. Soon swelling develops and extends onto the entire pinna except the lobule. Fluctuation can be felt in the swelling because purulent exudate is collected between the perichondrium and the cartilage. Palpation of the pinna is very painful.

Treatment. Local and general antiinflammatory treatment is given during the initial stage of the disease. Burow's solution and alcohol fomentations are effective; the affected part of the pinna is treated with a 5 per cent iodine tincture and a 10 per cent silver nitrate solution. In the absence of contraindications, physiotherapy (UV and UHF) is obligatory.

In the presence of fluctuation, the tissue should be incised parallel to the pinna contours and the necrotized matter removed from the abscess cavity. A tampon soaked in antibiotic solution should then be placed into the empty cavity. The pinna deformation is possible.

Eczema is a frequent disease which develops due to irritation of the skin of the pinna and the external acoustic meatus with the discharge in acute and chronic otitis media, with iodine preparations, and in prolonged exposure to coal or cement dust. Eczema can result from sensitivity to various substances (allergic form) or due to other systemic diseases, such as diabetes mellitus or some forms of metabolic disorders. Exudative diathesis, rickets can cause eczema in children. Eczema of the external ear develops after injuries with infection and impairment of the local resistance.

Acute and chronic forms of the disease are differentiated. Only superficial layers of the skin are involved in acute eczema, while in the chronic disease the deep underlying layers are also involved.

Symptoms. The initial stage of acute eczema is characterized by hyperemic and thickened skin of the pinna and external acoustic meatus due to infiltration. The acoustic meatus is markedly stenosed. The patient complains of constant and severe itching in the ears. The skin is scratched. Small blisters containing serous fluid appear. They open spontaneously releasing the fluid. Weeping develops.

Diagnosis is based on the anamnestic data and clinical findings (erythema, thickened skin, weeping, crusting or desquamation).

Treatment is directed at eliminating the main cause of eczema. The affected areas should be cleaned every day. Crusts should first be softened with vegetable or vaseline oil and then removed and antibiotic and corticosteroid ointment should be applied. Medication should be combined with physiotherapy: UV light (suberythema doses, 5-10-minute exposures) and UHF-therapy. Special attention should be paid to the general condition of the patient, a child in particular.

Furuncle of the External Acoustic Meatus (fig.47) is an acute purulent inflammation of a hair follicle with circumscribed inflammation of the dermis and subcutaneous tissue of the cartilaginous part of the acoustic meatus. The general predisposing factors are metabolic disorders, for example, upset carbohydrate metabolism, malnutrition, avitaminosis, etc.

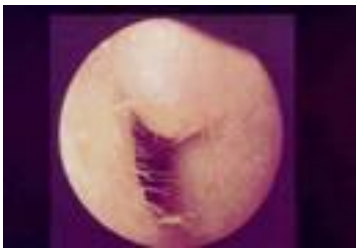


Fig.47. Furuncle of the external acoustic meatus

The leading *symptom* is severe pain. The pain radiates into the eye, teeth, neck, and sometimes becomes diffused in the entire head. Pain intensifies during talking and chewing because the head of the mandible is periodically displaced to press the walls of the external acoustic meatus and hence the inflamed skin.

Severe pain arises when the tragus or the inferior wall of the acoustic meatus is pressed, or when the pinna is pulled. Otoscopy reveals a rounded prominence of hyperaemic and inflamed skin, which narrows the lumen of the meatus. The regional parotid lymph nodes are enlarged, dense, and tender to palpation.

If the furuncle is located on the anterior or inferior wall of the externus acoustic meatus, the infection can extend onto the parotid gland and cause its inflammation. Furuncles often burst spontaneously on ripening and the patient feels alleviation of pain and his condition gradually improves. The length of the disease depends on relapses. On an average, the disease lasts 7-10 days.

The general examination of the patient includes urine and blood tests for sugar (recurrent furuncle is closely associated with diabetes mellitus).

Treatment. Antibacterial preparations should be administered during the first days of the disease. A turunda soaked in boric alcohol should be inserted into the external acoustic meatus. This is an effective local anti-inflammatory means. Sometimes, especially when the process gradually alleviates, a warming compress are used. Antipyretics and analgesics are recommended. Autohaemotherapy is sometimes used. Physiotherapy (UHF and UV) is effective as well.

In cases when the furuncle has ripened and the pain syndrome intensified, the danger of lymph nodes suppuration arises, and the furuncle should be opened surgically.

Diffuse Inflammation of the External Acoustic Meatus. If infection penetrates through minor injuries in the skin (that can be inflicted during manipulations in the ear) diffuse inflammation can develop. Maceration of the skin in chemical and thermal bums also facilitates infection with pyogenic and other microbes. This form of external otitis becomes diffuse and the tympanic membrane is involved. Inflammation spreads into deeper layers of the skin and subcutaneous cellular tissue. This process usually develops in the presence of allergy or metabolic disorders.

Symptoms. The clinical picture of the disease includes the symptoms of weeping eczema and the furuncle of the ear (skin itching, putrefactive discharge, the tragus painful to palpation, etc.). The symptoms are less marked in the chronic form of the disease. The leading symptom is thickened skin in the external acoustic meatus and the tympanic membrane due to inflammatory infiltration.

Treatment. Balanced diet rich in vitamins should be prescribed. Anti-inflammatory therapy should be conducted. In the presence of discharge a warm 2 per cent boric acid solution or furacin (1:5000) should be used for irrigation. After the procedure, the external acoustic meatus should be dried thoroughly and powdered with boric acid. Application of a 2-3 per cent silver nitrate solution or a 1-2 per cent alcohol solution of brilliant green, prednisolone ointment, hydrocortisone emulsion and some other medicines are effective. UHF- and UV-therapy produce a favorable effect too. Desensitizing therapy is indicated for allergic process. The patient should undergo an allergological examination. As a rule, the patient soon recovers.

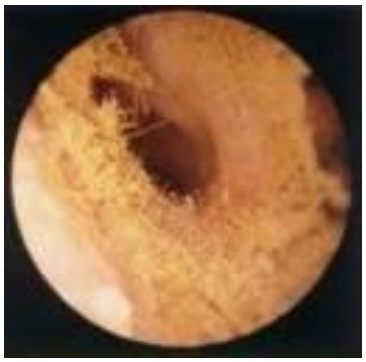


Fig.48. Otomycosis

Otomycosis (fig.48) is a fungal disease characterized by the growth of moulds of the genus *Aspergillus, Penicillin, Rhizopus,* and also yeast-like fungi *Candida* on the walls of the external acoustic meatus. The promoting factors are general and local allergy, metabolic and

neurohormonal disorders, and dysfunction of the ceruminous glands. Fungi grow to form a dense network of mycelium, which causes inflammation of the skin. Fungal infection of the external ear usually begins unnoticed by the patient. The main *symptoms* are constant and severe itching, increased sensi-tivity of the acoustic meatus and the pinna, stuffiness and noise in the ears; in the absence of exacerbation, pain in the ear is mild. Some patients complain of headache on the involved side.

Mould-caused otomycosis is characterized by pathological exudation in the external acoustic meatus, which looks like wet blotting paper. The color of the discharge varies and depends on the color of the mycelium. It can be dark-brown if the ear is affected by *Aspergillus niger,* yellow or greenish in infection with *Aspergillus flavus* and *Aspergillus glaucus,* and grayish-black in affections with *Aspergillus fumigatus.* Otomycosis caused by the fungi of the genus *Candida* is characterized by liquid serous discharge in the external acoustic meatus with soft yellowish-white easily detachable crusts throughout its entire length. The clinical picture of otomycosis caused by yeast-like fungi resembles that of weeping eczema of the external ear. In candidomycosis the process propagates onto the pinna and the retroauricular zone. A person can get infected when using a telephone receiver.

Prophylaxis includes treatment of the pathological processes that can promote the onset of the disease, prevention of inflammation of the external ear, and increasing the protective forces of man. Careful cleaning of the external acoustic meatus in the presence of acute and chronic otitis media without damaging the ear is important. Rational general and local antibiotic therapy (after testing the microbial flora for sensitivity to a particular antibiotic) is an effective prophylactic measure against otomycosis.

Diagnosis should be confirmed by microscopic examination of the discharge from the external acoustic meatus.

Treatment. The specific local antifungal therapy should be prescribed separately for each particular patient after thorough examination of his condition and the clinical picture of the disease, and depending on the particular causative agent. Good curative effect is attained with local use of antifungal preparations: Nitrofungin is effective against *Aspergillus niger* and *Aspergillus glaucus,* Clotrimasol and Nystatin are effective against *Candida;* Terbinafin, Kanesten are effective against *Aspergillus glaucus* and *Candida albicans.* The external acoustic meatus should first be thoroughly cleaned, and then a turunda, soaked in one of the mentioned antifungal preparations, is inserted into the ear and kept there for 20 minutes. The procedure should be repeated two or three times a day for 2-3 weeks.

Desensitizing treatment is indicated for marked general or local allergy. Relapses of otomycosis are possible with all types of treatment. In order to prevent them, the external acoustic meatus should be treated once a day with an antifungal preparation during one month following complete clinical recovery.

INFLAMMATION OF THE MIDDLE EAR

Acute Catarrh of the Eustachian Tube. Inflammations of the nasal and nasopharyngeal mucosa, as in acute rhinitis, influenza and other diseases, are very likely to extend to the mucous membrane of the Eustachian tube.

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

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

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

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

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

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

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

The direct cause of acute otitis media is infection of the middle ear with streptococci, staphylococci, pneumococci, and less frequently other microbes; mixed flora is sometimes responsible for the onset of the disease. Acute otitis is often secondary. It can be a complication or a manifestation of a systemic infection, for example, infection of the upper airways and influenza; scarlet fever, measles, diphtheria and some other diseases provoke acute otitis media in children. It can be due to acute and chronic inflammation of the pharynx and the nose. The main pathological factor is mechanical compression of the pharyngeal orifice of the auditory tube and impairment of its ventilating and draining functions. Among such diseases are hypertrophies rhinitis, adenoids, choanal polyp, hypertrophic 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 (fig.49) 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.

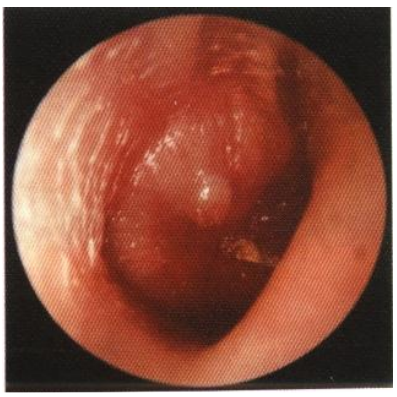


Fig.49. I stage (before-perforate)

The second period is perforation of the tympanic membrane and discharge of pus. All reactions subside. Otopyorrhea 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 patient may complains of hearing loss. In examination the conductive hearing loss is found.

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 adhesions 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 otopyorrhea; 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, hyperemia, protruded	perforation, pulsate reflex	tympanic membrane becomes distinct, appear recognizing 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 or normal	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 to ensure the normal function of the gastro-intestinal tract.

Vasoconstrictors or astringents should be instilled into the nose for restoration or improvement of ventilation and drainage of the auditory tube (naphtyzin, halasolin, sanorin etc.)

In cases of shooting pains and marked redness of the drum, Otipax ear drops should be used. 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 5-7 days. It is necessary to remember that streptomycin, gentamycin, kanamycin and monomycin are contraindicated for local and general use because of their toxic effect on the cochlear and vestibular apparatus. The antibiotic therapy should be combined with nystatin and desensitive drugs.

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 3-hour intervals. A UV-lamp is recommended for warming up the ear.

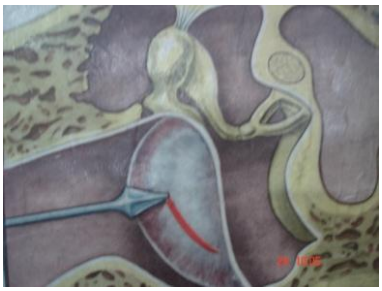


Fig.50. Paracentesis

In 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 (fig.50) 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 paracentesis 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.

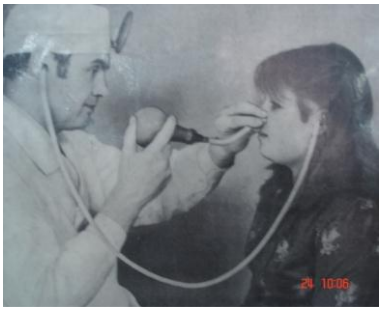


Fig.51. The blowing with balloon of Politcer

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). 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.

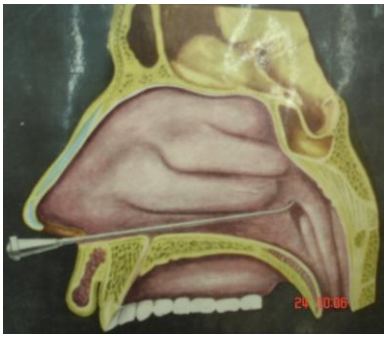


Fig.52. Catheterization of the Eustation tube

The blowing with balloon of Politcer (fig.51), catheterization 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 (fig.52) 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 s hould 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 Middle Otitis 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 morphology of the mucous in the middle ear and the structure of the temporal bone (residues of myxoid tissue, the nutrient medium for infection growth, are present in the tympanic cavity). Inflammation of the middle ear in neonates often develops due to penetration of amniotic fluid into the middle ear through the auditory tube during birth. The infection mechanism in nursing infants is the same, but in addition to infection penetrating from the nose and nasopharynx, food can also pass into the middle ear during regurgitation.

It is more difficult to establish the *diagnosis* of acute otitis media in a nursing infant. But the behavior of a baby with a diseased ear differs substantially from that of a healthy baby. The baby has bouts of inconsolable crying, refuses the breast because of pain during swallowing, rubs his diseased ear against the mother's hand. The main symptoms of the disease are painful palpation of the tragus (because of the absence of the bony part of the acoustic meatus) and high body temperature (39.5-40°C). A baby with otitis media is almost always depressed and sleeps a lot; his gastrointestinal function is upset; vomiting develops and wasting ensues. Meningeal symptoms with dimmed consciousness are possible. As distinct from meningitis, this condition is called meningism and is caused by toxemia (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 Middle Otitis 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 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.

Influenza 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 influenza otitis is characterized by hemorrhagic inflammation which is manifested by a pronounced dilatation of the vessels in the external acoustic meatus and the middle ear with extravasations (hemorrhage) under the epidermis in the bony part of the external acoustic meatus and the tympanic membrane. Extravasations appears as hemorrhagic blisters (bulla) in the mucous membrane of the middle ear.

Influenza 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 unpleasant odor 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 septicemia.

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 hyperemia 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 activated causing subperiostal 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.

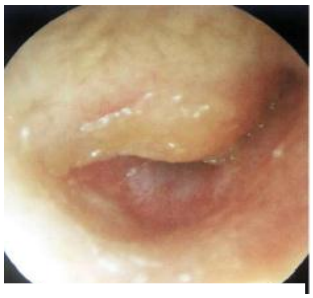


Fig.53. Otoscopy in mastoiditis

The specific otoscopic symptom (fig.53) 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). Otopyorrhea 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 edema 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 sternocleidomastoid 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 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 perisinual abscess.

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

Patient 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 persistent ear discharge with or without deep-seated pain in spite of an adequate cortical or modified radical mastoidectomy also points to petrositis. Fever, headache, vomiting and sometimes neck rigidity may also be associated.

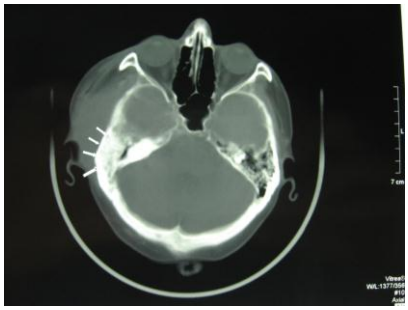


Fig.54. CT in mastoiditis

Diagnosis. Roentgenography (fig.54) 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 (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, warning 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 condition	Improves	In spite of treatment deteriorates
Pain in ear	After perforation decreases	In spite of perforation does not decrease
Noise in ear	Gradually decreases	In spite of treatment does not decrease
Hearing	Improves	Does not improve
Excretion from ear	Stands less, after then disappears. From serous - blood and mucous-purulent stands mucous	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 posterior-superior wall of acoustic meatus
Percussion of mastoid process	Painless	Painful

Differential symptoms of Mastoiditis and Furuncle of external acoustic meatus

Symptoms	**Furuncle**	**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 general anesthesia.

Indication:

1. Acute coalescent mastoiditis.
2. Incompletely resolved acute otitis media with reservoir sign.
3. Masked mastoiditis.
4. As an initial step to perform: endolymphatic sac surgery, decompression of facial nerve, translabyrinthine, 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. The fibers of sternocleidomastoid muscle 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 mastoideum* 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 fibers of posterior belly of digastric. Zygomatic cells, perisinual cells lying between sinus 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.

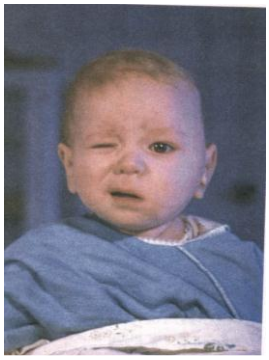


Fig.55. Facial paralysis

Complications:

1. Injury to facial nerve (fig.55).
2. Dislocation of incus.
3. Injury to horizontal semicircular canal (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 favorable 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.

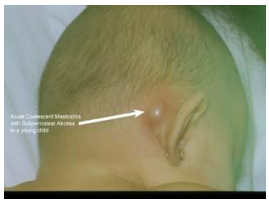


Fig.56. Acute mastoiditis with subperiosteal abscess

Mastoiditis (Antritis) in Children. The mastoid process is under-developed 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; tachypnoe develops. The temperature reaction does not always agree with severity of the condition. Body temperature can be normal, subfebril or be as high as 38-39°C. The blood picture is characterized by neutrophilic leucocytosis; the ESR is accelerated.

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

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

Control tests

1. After short-term pain in the ear a patient began to experience otorrhea. On examination the ear-drum is hyperemic. There is a slit-like perforation in the anterior-inferior quadrant. What is tactic of treatment at this stage of acute otitis media?

A. paracentesis of the ear-drum
B. carbol-glyceric drops in the ear
C. catheterization of the auditory tube
D. dehydratation
+E. cleaning of the ear, antibacterial therapy

2. What are predisposing factors of development of acute otitis media in early children's age, except one:

+A. anatomic peculiarities of the middle ear structure in early children's age and in a newborn
B. infectious diseases with inflammation of the mucous membrane of the upper respiratory tracts
C. pneumatic type of structure of the processus mastoideus
D. hypertrophy of the lymphoid pharyngeal ring
E. horizontal position of a child

3. These symptoms are characteristic of I stage of acute middle otitis, except one:

A. high fever

B. severe pain in the ear

C. loss of hearing, noise in the ear

+D. punctate perforation of the ear-drum in its lower parts

E. hyperemia, infiltration and protrusion of the ear-drum

4. A two –years- old child suddenly developed a severe pain in the ear against the background of ARVI two days ago. The body temperature rose to 39°C. There was single vomiting. On otoscopy: hyperemia, protrusion of the ear-drum, identification points are not expressed. The pain is marked on palpation of the processus mastoideus in the projection of the antrum. What is medical tactics?

+A. paracentesis of the ear-drum

B. antropunction

C. antrotomy

D. analgetics

E. catheterization of the auditory tube

5. A 5 –year- old patient complains of pain in the right ear, diminished hearing, increase of temperature to 37.9°C and general fatigue during 3 days after overcooling. Objectively: hyperemia of the mucous membranes of the nasal and throat. On otoscopy: AD –hyperemia of the ear-drum in the upper parts, smoothed out of contours of the malleus, absence of the light cone. The discharge in the external auditory canal is absent. AS is normal. The right ear hears whisper at the distance of 3 m, left – at 6 m. What is a diagnosis?

+A. I stage of middle acute otitis

B. II stage of acute middle otitis

C. chronic secretory otitis

D. adhesive otitis

E. III stage of acute otitis media

6. A patient had purulent discharge out of the ear after brief pain. On examination there is hyperemia of the ear-drum and slit-like perforation in its anterior-inferior quadrant. What is stage of acute otitis media?

A. I stage of middle acute otitis

+B. II stage of middle acute otitis

C. III stage of middle acute otitis

D. I stage of middle acute otitis, antritis

E. II stage of middle acute otitis, mastoiditis

7. A child aged 7 had acute otitis media. In three weeks otorrhea developed again after brief pain in the ear against the background of ARVI. What character of discharge out of the ear in acute otitis media allows to differentiate this disease with mastoiditis?

+A. discharge gradually becomes serous-blood, mucous, mucopurulent

B. mucopurulent discharge, repeated after ingress of water in the ear

C. purulent, blood-purulent discharge in great amount

D. little discharge with unpleasant smell

E. mucopurulent discharge with admixture of epidermal scales

8. A 8- year- old patient complains of otorrhea, diminished hearing. A disease began with severe pain in the left ear, increased body temperature to 39°C, headache. Otorrhea out of the ear developed 2 days ago. After that the patient's condition improved a little, the body temperature subsided to 37.5°C. There is purulent discharge in the external auditory canal. The ear-drum is hyperemic. The throbbing reflex is present in the anterior-inferior quadrant. The right ear hears whisper at 5 m, left – 1.5 m. What is a diagnosis?

A. initial stage of acute otitis media

+B. acute perforative otitis media

C. secretory otitis

D. mastoiditis

E. tubootitis

9. The patient complains of severe pain in the ear, elevation of the body temperature to 39.2°C, purulent discharges out of the ear and loss of hearing. On examination swelling of the soft tissue in the retroauricular region, protrusion of the auricle are present. Palpation of the processus mastoideus is painful. There is pus, overhanding of the superior-posterior wall of the bony part in the auditory canal, perforation of the ear-drum. Whisper speech: AD-0 m, AS-6m. What is a diagnosis?

A. exacerbation of chronic purulent mesotympanitis
B. acute middle otitis
C. exacerbation of chronic purulent epitympanitis
+D. acute middle otitis, mastoiditis
E. furuncle of the external auditory canal

10. On audiometry diminished hearing is revealed in a patient with acute middle otitis. What will be Rinne test in this case?

A. positive
+B. negative
C. shortened
D. unchanged
E. unchanged or lengthened

11. A patient has been suffering from acute otitis media for three weeks. He was treated by himself. The pain in the retroauricular region, purulent discharge out of the ear, high body temperature developed two days ago. What wall changes of the bony part of the external auditory canal are of primary importance for diagnosis of mastoiditis?

A. superior
B. anterior
C. posterior
D. anterior-superior
+E. posterior-superior

12. A child of 5 years old had acute middle otitis about 3 weeks ago. He was treated at a polyclinic with improvement of the ear condition. Two days ago the body temperature rose again, he had abundant otorrhea, pain in the retroauricular area. On examination protrusion of the auricle, fluctuation and pain on palpation of the processus mastoideus are marked. On otoscopy the overhanding of the superior-posterior wall of the bony part of the external auditory canal, red colour of the ear-drum are revealed. There is thick pus in the auditory canal. What is a diagnosis?

A. acute purulent middle otitis
B. external diffuse otitis, regional lymphadenitis
+C. acute middle otitis, mastoiditis, subperiosteal abscess
D. acute middle otitis, zigomaticitis
E. acute catarrhal middle otitis

13. A 7- month-old baby after ARVI became restless for two days, he often cries, rocks a head, tries to reach his right ear with a hand, refuses to take the breast. The body temperature is 39.2°C. There were short-term convulsions, vomiting, diarrhea. On otoscopy: the right ear-drum is red, protruded. Its contours are not determined. Pressing on the tragus is painful. Rigidity of the occipital muscles is marked. What is a diagnosis?

+ A. acute otitis media, meningism
B. acute otitis media, meningitis
C. exacerbation of chronic purulent otitis, meningitis
D. external diffuse purulent otitis, meningitis
E. otogenic meningitis

14. A patient of 38 years old complains of the pain in the right ear and retroauricular area, otorrhea, diminished hearing, headache, increase of the body temperature to 37.5°C. He has been ill for two weeks. Objectively: external auditory canal is narrowed in the bony part due to overhanging of the posterior-superior wall and filled with mucopurulent discharge. The ear-drum is red, infiltrated. There is a narrow

perforation in the anterior-inferior quadrant with pulsation of pus. Swelling in the area of the processus mastoideus, loss of contours are determined. The auricle is protruded. What is a diagnosis?

+ A. acute purulent middle otitis, mastoiditis
B. acute purulent middle otitis
C. exacerbation of chronic middle otitis
D. diffuse external otitis
E. acute catarrhal middle otitis, mastoiditis

15. A patient with acute middle otitis hearing diminished after closing of perforation of the ear-drum. What are measures of hearing restoration in III stage of acute otitis media?

+ A. catheterization and blowing of the auditory tube by Pulitzers' method
B. paracentesis of the ear-drum
C. antibioticotherapy
D. cleaning of the external meatus
E. operation mastoidotomy

16. A patient complains of pain in the ear after bathing in the basin. The pain increases on mastication. On otoscopy: the skin of the membranous-cartilaginous part of the external auditory canal is infiltrated. The auditory canal is narrowed. The pain is marked on palpation of the tragus. Hearing is normal. What is a diagnosis?

A. acute catarrhal middle otitis
B. acute mastoiditis
+ C. external diffuse otitis
D. purulent parotitis
E. acute purulent middle otitis, mastoiditis

17. A patient complains of low-frequency noise in the ear, autophony, diminished hearing, insignificant pain. These symptoms developed a few hours ago against the background of acute rhinitis. On otoscopy: hyperemia of the ear-drum is marked in the upper parts and around of the handle of the malleus. The light cone is absent. What is the medical tactic?

A. nonspecific antiinflammative drugs
B. vasoconstrictor drops in the nose
C. Otipax drops in the ear
D. antihistaminic medicine
+ E. all of the listed

18. A two- year-old child suddenly developed severe earache, increased temperature to 39°C, once there was vomiting a day ago against the background of ARVI. On otoscopy the eardrum is hyperemic, protruded, identification points are not expressed. There is tenderness on palpation of the mastoid process in the projection of the antrum. What is therapeutic tactics?

+ A. paracentesis of the eardrum
B. antropuncture
C. antrotomy
D. administration of the analgesics
E. catheterization of the Eustachian tube

19. A patient was treated by the otolaryngologist for acute purulent middle otitis. On the fifth day of treatment the suppuration from the ear ceased; however, there are diminished hearing, low-frequency noise in the ear. On otoscopy the eardrum is turbid, hyperemic in the upper part, pulled in. Investigation of hearing determines the conductive type of hearing disorder. What is therapeutic tactics at this stage of the course of acute purulent middle otitis?

A. paracentesis of the eardrum
B. Otipax drops in the ear
C. antibiotic therapy
+ D. blowing of the Eustachian tube by Politzer's method, catheterization of the auditory tube
E. mastoidotomy

20. A patient with acute purulent middle otitis complicated by mastoiditis was admitted to hospital. On X-ray examination of the mastoid process by CT there were noted the darkening of the cellular system on the side of affection, absence of bone trabeculas. What is therapeutic tactics at this stage of mastoiditis?

A. paracentesis of the eardrum, local and general antipyretic therapy

+ B. operation mastoidotomy

C. radical operation in the middle ear

D. tympanoplasty

E. catheterization of the Eustachian tube

21. A 27- year-old woman referred to the ENT- doctor with complaints on diminished hearing in the left ear, ringing in the ear. It is revealed from the anamnesis that she was struck on the ear with opened palm more than two week ago. She did not refer for medical aid, she thought that "it will pass by itself". On examination: there is purulent discharge in the ear, there is a small size perforation in the tense part of the eardrum. What will be your diagnosis?

A. acute purulent middle otitis

B. exacerbation of chronic mesotympanitis

C. chronic mesotympanitis

D. aggravation of chronic epitympanitis

+ E. acute middle purulent otitis

Chronic Purulent Middle Otitis

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 otopyorrhea, and hearing loss of various degrees.

Etiology 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, epitympanitis and epimesotympanitis.

Mesotympanitis (fig.57) occurs in 55 per cent of cases with chronic suppurative otitis media. The mucosa of the middle and lower portions of the tympanic mem brane, 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.

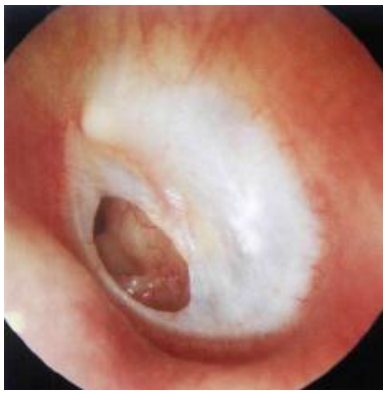


Fig.57. Mesotympanitis

Subjective *symptoms* are indistinct. Patients complain of periodical or constant otopyorrhea 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 or circumscribed external otitis.

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.

Discharge from the ear is mucopurulent; it can be blood-purulent in the presence of granulation and polyps. The discharge is usually odorless. It can be 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. Otopyorrhea 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 anamnesis, 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 odorless discharge. The odor indicates involvement of the bone.

Prognosis is usually favorable, 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 otopyorrhea.

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 boric acid, furacin (1:5000) and antibiotics, after preliminary testing the microflora for sensitivity to them. In the presence of local signs of allergy (edema of the mucosa of the tympanic cavity, watery discharge), 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. 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 Ziegler's speculum or a Politcer's bag can be used for the purpose. The medicine can be administered in catheterization of the auditory tube. Antibiotics should be injected intramuscularly only during exacerbation.

Minor surgical operations are sometimes necessary: treatment of small granulations or polyps with chemical coagulation; removal of large granulation and polyps using an aural snare.

Physiotherapy is also necessary.

Epitympanitis. 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. This form 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.

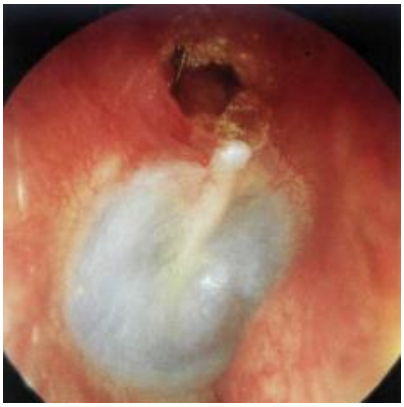


Fig.58. Epitympanitis

The main otoscopic sign of the pathology is persistent marginal perforation (fig.58) 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 odor 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 presence of labyrinthine fistula.

Cholesteatoma causes vast destruction in the temporal bone. Cholesteatoma masses can sometimes be seen during otoscopy

through a perforation in the tympanic membrane. These appear like a white tumor (with a pearly luster) 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 embryonic 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 cholestea toma 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. Suppuration of cholesteatoma can extend to the intracranial tissues to cause intracranial pathology.

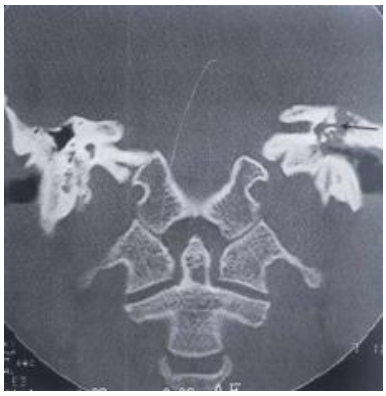


Fig.59. CT in epitympanitis

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 (CN-scan, Schuler, Mayer, Stenvers projection) is a valuable *diagnostic* method (fig.59). The X-ray picture 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.

Differential diagnosis of Mesotympanitis and Epitympanitis

Sings	Mesotympanitis	Epitympanitis
Pathomorpho-logic changes.	Inflammation of mucous membrane of tympanic cavity.	Inflammation of mucous, caries of temporal bone formations.
Ear excretions	Serous – mucous	Purulent with putrid smell
Localization of perforation	Central	Marginal
Test with probe	Negative	Positive

Pathomorphologic sings of epitympanitis and its symptoms

Sings	Symptoms
Obligatory Caries of the walls of tympanic cavity, acoustic bones.	Purulent excretions with annoying smell; Progressive hardness of hearing; Gradual increasing of perforation.
Growth of granulations in the region of attic.	Purulent – blood excretions. Formation of granulations, polyps.
Development of cholesteatoma.	Pus with stinking smell, containing scales of epithelium. Tendency to lowering of hearing.
Destroying of tympanic wall, formation of labyrinth fistula, paresis of facial nerve.	Giddiness, headache, unsteadiness of the step, nystagmus, positive pressure test, lagophtalm, smooth of nasolabial fold, prolapsed mouth angle.

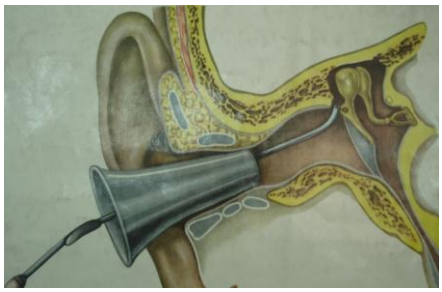


Fig.60. Irrigation of the attic

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 (fig.60) with the following warm solutions: 40% alcohol, 3% alcohol solution of boric acid, 0,25% solution of formaldegide. Conservative treatment is usu ally 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. Atticoantrotomy 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 by otoscopy, X-ray and by other 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 (fig.61).

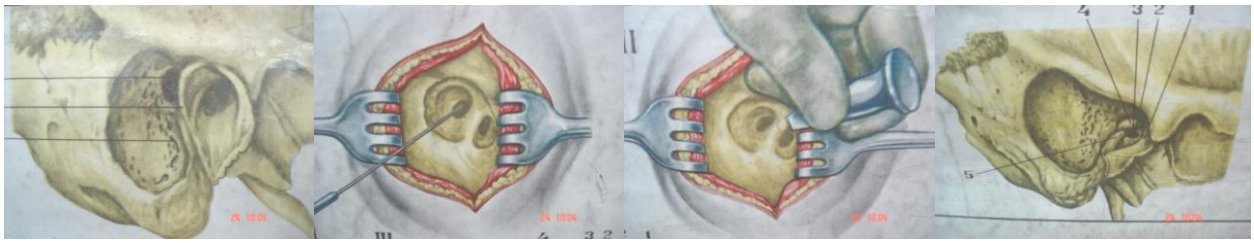


Fig.61. Radical operation

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 bones 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 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.

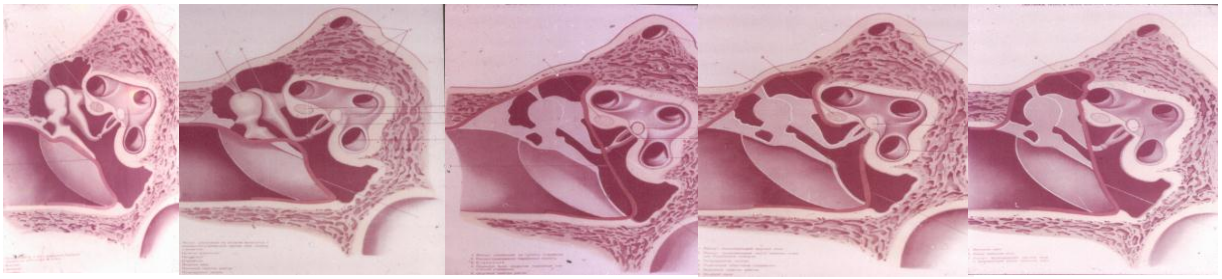
Chronic purulent middle otitis in children differs in some respects from this disease in adults. The differences are associated with the anatomy of the temporal bone in children and the specific reaction of a child's body to various stimuli. Special attention should be paid to children under 3 years 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.

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 antiinflammatory treatment, correct nutrition is also important. Surgical procedures on the temporal bone of a child must be very sparing and preserving the hearing function.



Type I *Type II* *Tepe III* *Type IV* *Type V*

Fig.62. Tympanoplasty

Tympanoplasty (fig.62) 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 dry perforation of the tympanic membrane; disruption of the ossicular chain; 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 Eustachian tube* to provide aeration to the middle ear. A *functioning sensoneural apparatus*, i.e. the cochlea and VIII-th 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 placed 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 myringostapedopexy 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 dura, vein, fascia or cadaver tympanic membrane are also used. Repair can be done by two techniques - the underlay or the overlay. In underlay technique, margins of perforation are freshened and the graft placed medial to perforation supported by gelfoam. In overlay technique, the graft is placed lateral to fibrous layer of the tympanic membrane after carefully removing all squamous epithelium.

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

Repair of ossicular chain can be achieved by the use of autograft incus or cartilage, homograft ossicles, or the prosthetic implants made of ceramics or teflon. The techniques commonly employed in ossicular reconstruction in such cases are the incus transposition or a sculptured ossicle. Most common ossicular fixations are the ankylosis of stapes footplate as in otosclerosis, and the congenital or acquired fixation of head of malleus in the attic. Ankylosis of stapes can be corrected by removal of the fixed stapes and its replacement by a prosthesis while the attic fixation of malleus head entails removal of the head of malleus and entire incus and then establishing contact between handle of malleus and the stapes.

Labyrinthitis

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

The purulent type occurs more often in chronic suppurative otitis, and sometimes in cases of acute scarlet fever and influenza otitis. The destructive effect of cholesteatoma is a particularly frequent cause of labyrinthitis. Purulent labyrinthitis may occur in a localized (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 attack this will suggest an incipient inflammation of the meninges.

Fig.63. MRT of the inner ear

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 (fistula symptom).

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

OTOGENIC INTRACRANIAL COMPLICATIONS

Otogenic intracranial complications arise due to extension of the pathological process from the middle and internal ear into the cranium. The complications include epidural (extradural) and subdural abscesses, sinus thrombosis, intracranial abscesses (temporoparietal and cerebellar), meningitis, and arachnoiditis. Lethal outcome is 24 per cents at otogenic intracranial complications.

Streptococcus and staphylococcus are the main causative agents in the etiology of otogenic intracranial complications. Sometimes inflammatory intracranial processes occur in acute influenza 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; (3) by preformed routes, (4) by labyrinthine 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 vascular 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 meningitis 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.

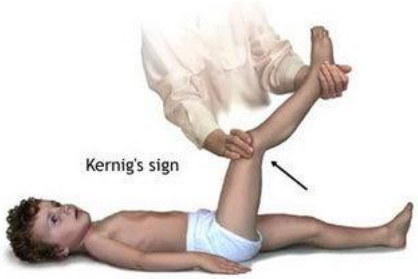


Fig.64. Symptom of Kernig

Symptoms. General symptoms of infectious disease, meningeal signs and sometimes focal symptoms can be differentiated in the clinical picture. T he general symptoms are elevated body temperature, changes in the internal organs (car diovascular, 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 back ground of subfebril 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.

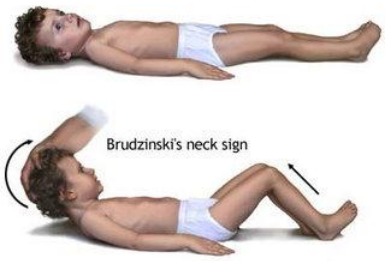


Fig.65. Symptom of Brudzinsky (upper)

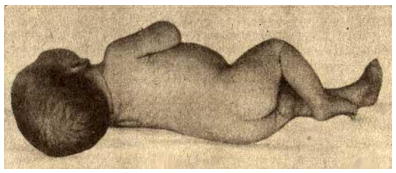


Fig.66. Pose of "gun-dog"

Membrane's or meningeal symptoms:

- rigidity of occipital muscles of 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 (fig.64) - resistance, painfulness at unbending of shank in knee-joint in position lying on the back;
- symptom of Brudzinsky (upper) (fig.65) - bending of legs in coxal and knee-joint in answer on bending of the head to the forward;
- symptom of Brudzinsky (middle) -the same leg's moving at pressure on pubic joint;
- symptom of Brudzinsky (lower) or contralateral-unbending of the led in knee-joints, is accompanied by bending of another led.
- Symptom of "suspending" - there is bending of extremities in copal taking him under hands, Bekhterev's sign (pain inside the head or blepharospasm associated with striking on the zygomatic arch with a hammer), hypertension, and photophobia can be vivid on the very first days of the disease, and become even more pronounced in 2 or 3 days.

In sharply expressed cases you can't take out the patient's head from horizontal position at his paying position. In more sharp form rigidity leads opistotones, 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" (fig.66).

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. Leukocytes counts are as high as 33-34 x 10^9 (usually 10-15 x 10^9) per liter 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. The cerebrospinal fluid (fig.67) pressure is always high: from 300 to 600 mm of the fluid (normal 150-200 mm).

The color 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.

Fig.67. Lumbal punction

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

Etiological 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 toxemia.

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. Etiologi cal 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. Furosemide (2-4 ml/day) should be injected intravenously, 10 ml of a 25 per cent of magnesium sulphate solution intramuscularly, and 7 ml of glycerol per os are prescribed for the same purpose. Depending on the general condition and the state of the cardiovascular system, the patient can be given symptomatic treatment (cardiac glycosides, tonics, analeptics).

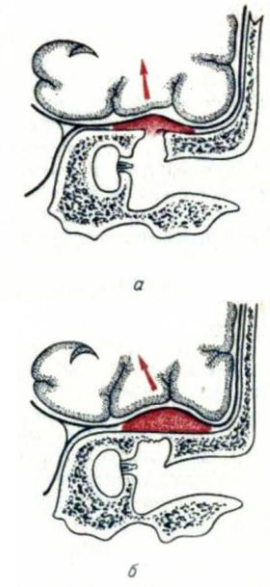


Fig.68. Open and close extradural

Extradural (Epidural) Abscess (fig.68) 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.

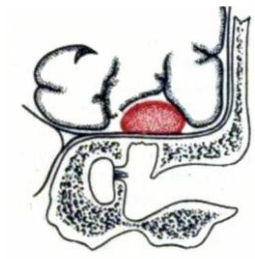


Fig.69. Subdural abscess

Subdural Abscess (fig.69) is a rare intracranial complication. It occurs as a complication of chronic suppurative otitis media, especially cholesteatoma, and less fre quently, 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 (fig.70)**.** 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 is divided into four stages: initial, latent, apparent, and terminal.

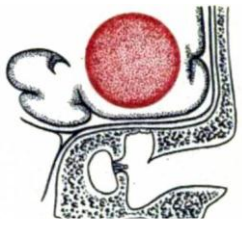


Fig.70. Cerebral abscess

Symptoms differ depending on the stage of the disease. Brain abscess develops through four stages: 1.Stage of invasion (initial encephalitis). 2.Stage of localization (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 hemoglobin content do not usually change in brain abscess. But moderate hypochromic anemia with hemoglobin 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 edema 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 opalescent; 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 amnesic aphasia, the patient fails to name an object but can repeat speech. Spontaneous speech is monosyllabic and poor; verbs prevail and nouns are almost completely absent from speech.

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

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

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

Treatment is surgical. It includes vast radical operation on the ear, finding the abscess and its opening. In addition to a common surgical intervention, a vast radical operation includes exposure of the dura and the middle and posterior cranial fossa. 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 hypoesthesia on the face and a weak corneal reflex. Elevated cerebrospinal fluid pressure causes changes in the ocular bottom. The optic papillae are swollen to a various degree during various stages of the disease and in some cases cause secondary atrophy of the optic nerves. The visual acuity is almost always normal. The composition of the cerebrospinal fluid is often characterized by a slight protein-cell dissociation (0.66 g/1); less frequently the composition is hydrocephalic, with decreased protein content (to 0.26-0.099 g/1).

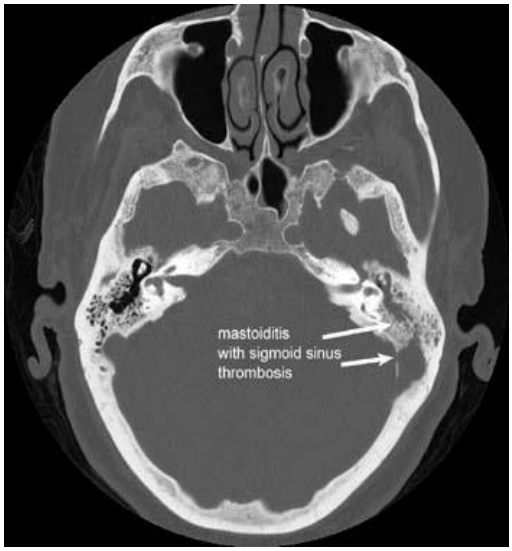


Fig.71.Mastoiditis with the sigmoid sinus thrombosis

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 (fig.71)**.** As a rule, sinus thrombosis is caused by otitis media, with extension of inflammation to the mastoid process. Caries of the petrosal 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.

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 thrombophlebitis process. *Endophlebitis and mural thrombus formation.* Inflammation spreads to inner wall 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 septicemia. *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 sagital sinus or cavernous sinus, and distally into mastoid emissary vein, to jugular vein.

Local symptoms are especially distinct in infected thrombosis. This condition is marked by hyperemia and edema 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. Edema 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 asso ciated 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 ocular bottom, 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.

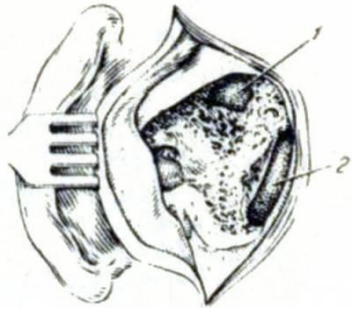


Fig.72. Spreading antromastoidectomy 1- dura mater of the middle cranial fossa:2- siamoid sinus.

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

Treatment is surgical (fig.72). 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.

Data of spinal fluid in normal condition and in different form of meningitis

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

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

Differential diagnosis of otogenic and other meningitis

Symptoms	Otogenic meningitis	Epidemic cerebrospinal	Tuberculosis meningitis	Serous (virus) meningitis
Beginning is	Acute	Acute, sudden	Gradual	Acute
Current is	Quick, hard	Quick, hard	Slow with remission	Quick, but it isn't hard
Presence of centers	Acute or chronic purulent middle otitis, especially epitympanitis	Frequently there was ARVI early	Tuberculosis of lungs, bones	Appears of measles, typhus, epidemic parotitis, influenza
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, hemorrhagic rash on the body, sharply expressed leucocytosis.	Usually in youth. Temperature is not very high, but constant. Rigidity of occipital muscles of and Kernig's symptom grow gradually. Headache is little	Clinics is less hard then at other meningitis. 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 horizontal-rotator, its both components are clearly distinguished. At labyrinth's irritation it is directed to the injured side and	Big-sweeping, horizontal. Quick and slow components aren't clearly distinguished. Turning off nystagmus is character to the injured side.

	showed during bouts of giddiness, its force changes and depends on position of a head. At turning off labyrinth it is directed to the health side and quickly is exhausted	
Spontaneous miss	Two-sides, always in direction which is opposite to nystagmus. It quickly disappears at turning off labyrinth.	There is on the side of center (there is miss only by ill hand on the injured side). It doesn't depend on nystagmus.
Spontaneous fall	Depends on position of a head (changes direction of the fall) and depends on direction of nystagmus (a patient deflects to the side of the slow component of nystagmus)	To the side of injury of cerebellum. This concurs with direction of nystagmus. It doesn't depend on the head's position. It is observed for a long time.
Deflection at walking	A patient deflects to the side of the slow component of nystagmus.	A patient deflects to the side of cerebellum's abscess.
Flanking gait	It is easily fulfilled to the both sides.	A patient deflects to the side of cerebellum's abscess.
Adiadochokinesia	Is absent	Is observed. There is lag of a hand on the injured side.
Giddiness	Is very intensive and becomes stronger at change of head position	Doesn't connect with change of head position
Nausea, vomiting	There are on the height of bout of giddiness, gradually lowers.	Gradually increases
Headache	Is absent	Acute headache in occipital or frontal region
Bradycardia	Is absent	There is frequently
Hearing	Is lowered or is absent depending on the form of labyrinthitis	Doesn't change
Experimental irritation of labyrinth	There is increased excitability or absence of vestibular excitability	There is normoreflexia.

Control tests

1. What does treatment of exacerbation of chronic mesotympanitis consist in before obtaining the answer of microbiological study of discharge out of the ear?

A. treatment is not given
B. cleaning of the ear, restoration of the function of the auditory tube
+ C. cleaning of the ear, restoration of the function of the auditory tube, wide spectrum antibiotics locally
D. cleaning of the ear, restoration of the function of the auditory tube, wide spectrum antibiotics orally
E. dry and wet cleaning of the ear

2. For dry cleaning of the external auditory canal we use:

+ A. ear probe with the cotton
B. bulbous-end probe
C. Voyachek probe
D. Politcer's ballon
E. otoscope

3. While examining a patient with chronic epitympanitis at the polyclinic, the ENT- doctor asked the nurse to give him Voyachek probe. What is the purpose of using this instrument?

A. cleaning of the auditory canal
B. investigation of possibility of the auditory tube
C. determination of perforation size
+ D. attic examination
E. taking the material for microbiological study

4. A 21- year-old man referred to the ENT- doctor with complaints on insignificant pains in the right ear, diminished hearing in this ear, discharge out of it. He is ill the third day. There was the same situation half a year ago, he was treated by himself, instilled the eardrops. On examination: mucopurulent discharge out of the ear, there is a rounded central perforation of average size in the eardrum. What is your diagnosis?

A. acute purulent middle otitis
+ B. exacerbation of chronic mesotympanitis
C. exacerbation of chronic epitympanitis
D. exacerbation of chronic epimesotympanitis
E. chronic catarrh of the middle ear

5. A patient does not present any complaints, he is sent by the ENT –doctor of Military registration for specification of the diagnosis. It is revealed from the anamnesis that previously the ear has disturbed patient in childhood. On examination: the external auditory canal is wide, there is no discharge. In the tense membrane of the eardrum there is a punctate dry central perforation, there is also no discharge in the tympanum, the mucous membrane of the medial wall is pale pink. The auditory tube is passable. CT of the temporal bones did not reveal any bone-destructive manifestations. On the audiogram hearing is within the age norm. What is your diagnosis?

A. adhesive otitis
B. chronic eptympanitis, remission
+ C. chronic mesotympanitis, remission
D chronic epimesotympanitis, remission
E exudative otitis

6. A patient was admitted to the ENT-department with complaints on diminished hearing in the left ear, periodic pyorrhea. It is revealed from the anamnesis that the ear has been disturbing the patient since childhood, periodically (once a year) there is an exacerbation of the process. The last exacerbation was 10 months ago. On examination: there is no discharge in the auditory canal. In the tense membrane of the eardrum there is a central perforation, there is also no discharge in the tympanum, the mucous membrane of the medial wall is pale pink. The auditory tube is passable. CT of the temporal bones did not reveal any bone-destructive manifestations. On the audiogram - uniform increase in the thresholds of the air-conducting sounds by 10-15 dB along the entire tone scale. What is supposed type of surgery?

A. antromastoidotomy
+ B. antroatticotomy
C. myringoplasty
D radical operation
E shunting of the eardrum

7. A patient complains of hearing loss, pyorrhea in the right ear. There is periodically exacerbation of the process since childhood (2-3 times per year). There is mucopurulent discharge in the auditory canal. In the tense membrane of the eardrum there is a central perforation, mucopurulent discharge in the tympanum, the mucous membrane of the medial wall is hyperemic. The auditory tube is slightly passable. CT of the temporal bones did not reveal any bone-destructive manifestations. On the audiogram - uniform increase of the thresholds of the air-conducting sounds by 25-30 dB along the entire tone scale. What is your diagnosis?

A. acute middle otitis in the stage of perforation
B. acute middle otitis in the stage of perforation, mastoiditis
C. exacerbation of chronic epimesotympanitis
+ D. exacerbation of chronic mesotympanitis
E. exacerbation of chronic epitympanitis

8. A patient complains of diminished hearing in the right ear, periodic pyorrhea. The ear has been disturbing the patient since childhood. There is no discharge in the external auditory canal. In the tense

membrane of the eardrum there is a central perforation, there is also no discharge in the tympanum, the mucous membrane of the medial wall is pink. What is your diagnosis?

A. adhesive otitis

B. chronic epitympanitis, remission

+C. chronic mesotympanitis, remission

D. exacerbation of chronic mesotympanitis

E. exacerbation of chronic epitympanitis

9. On admission patient's state was severe, with confusion, severe headache. There was noted the expressed rigidity of muscles of back of the head, and "+" Kernig's symptom. There are purulent discharge in the auditory canal, extensive marginal perforation in the schrapnel membrane, masses of gray colour are seen behind it. What additional method of examination will be decisive to precise diagnosis?

A. blood count

B. R - gram of the temporal bone by Shuller

+ C. data of investigation of the liquor

D. nuclear - magnetic resonance

E. CT of the temporal bone

10. A man complains of moderate pains in the right ear, diminished hearing in this ear, pyorrhea. He is ill the third day. There was the same situation half a year ago, he was treated by himself, instilled the eardrops. There are mucopurulent discharge in the ear, extended central perforation in the eardrum. What is your diagnosis?

A. chronic mesotympanitis

+ B. exacerbation of chronic mesotympanitis

C. chronic epitympanitis

D. exacerbation of chronic epimesotympanitis

E. acute purulent otitis media

11. For treatment of exacerbation of chronic mesotympanitis it is possible to use the following drops, except:

A. Cyprofloxacine

B. Otofy

C. Dioxidine

+ D. Sofradex

E. Normax

12. For local treatment of exacerbation of chronic otitis it is possible to use the following antibacterial preparations, except:

A. Cephalosporins of II generation

B. Cephalosporins of III generation

C. Ftorchinolon

+ D. Aminoglikosides

E. Tetracyclines

13. For washing out of the epitympanic space (attic) in exacerbation of chronic epitympanitis with cholesteatoma it is expedient to use:

A. solution of the boric acid

B. solution of Furacin

C. solution of Levomycetin

+ D. alcoholic solutions of 40°

E. oil solutions

14. For washing out of the auditory canal and tympanum it is expedient to use the following solutions, except:

A. solutions of antibiotics

B. solutions of corticosteroids

+ C. dye solutions
D. solutions of proteolytic enzymes
E. solutions of antiseptics

15. During catheterization of the Eustachian tube it is expedient to use the following medicinal substances, except:
A. Dioxidine
B. Hydrocortisone
C. Adrenaline
D. Chymotrypsin
+ E. Gentamicin

16. A patient complains of diminished hearing in the left ear, pyorrhea. He has been ill for many years. Three years ago he had otogenic meningitis, cured conservatively. Otoscopy: purulent discharge with unpleasant smell in the auditory canal. There is noted marginal perforation in the anterior parts of the schrapnel membrane with visible greyish masses behind it. CT of the temporal bones determines destruction of the bone in the antrum and aditus area. What is your diagnosis?
A. exacerbation of chronic mesotympanitis
B. exacerbation of chronic mesotympanitis, complicated by mastoiditis
C. exacerbation of chronic epitympanitis
+ D. exacerbation of chronic epitympanitis, complicated by cholesteatoma
E. exacerbation of chronic epimesotympanitis

17. A patient complains of diminished hearing in the left ear, pyorrhea. He has been ill for many years. Three years ago he had otogenic meningitis, cured conservatively. Otoscopy: purulent discharge with unpleasant smell in the auditory canal. There is noted marginal perforation in the anterior parts of the schrapnel membrane with visible greyish masses behind it. CT of the temporal bones determines destruction of the bone in the antrum and aditus area on the left. What is the scope of surgical intervention in this patient?
A. antrotomy
B. antromastoidotomy
C. antroatticotomy
D. tympanoplasty
+ E. radical operation

18. A patient's state was severe, with confusion, severe headache. There was noted the expressed rigidity of muscles of back of the head, and "+" Kernig's symptom. On examination: purulent discharge in the left auditory canal, extensive marginal perforation in the schrapnel membrane, masses of gray color are seen behind it. What is your diagnosis?
A. exacerbation of chronic mesotympanitis
B. exacerbation of chronic epitympanitis
+ C. exacerbation of chronic epitympanitis, otogenic meningitis
D. exacerbation of chronic epitympanitis, otogenic sepsis
E. exacerbation of chronic epitympanitis, abscess of the brain

19. A patient's state was severe, with confusion, severe headache. There was noted the expressed rigidity of muscles of back of the head, and "+" Kernig's symptom.On examination: purulent discharge in the left auditory canal, extensive marginal perforation in the schrapnel membrane, masses of gray color are seen behind it. What is the scope of surgical intervention?
A. atticotomy
B. antroatticotomy
C. mastoidotomy
D. radical operation
+ E. extended radical operation

20. A child of 2 year old developed a severe headache and pain in the left ear, temperature increased to 38.0°C after ARVI. His mother gave him a tablet of Analgin and applied hot water bottle to the ear. The child fell asleep. Next morning the state of the child deteriorated. He became agitated, tossed in the bed, rejected food, nausea arose. There was vomiting after drinking milk. Having examined the patient the

district physician directed him to the infectious hospital. The ENT consultant made the CT of the temporal bones and recommended surgical intervention. What surgical intervention is indicated to the patient?

+ A. antrotomy
B. antromastoidotomy
C. tympanoplasty
D. radical operation
E. stapedectomy with stapedoplasty

21. A patient with chronic purulent middle otitis developed abscess of the temporal lobe of the brain. What are directions of infection spread from the middle ear cavities to the contents of the skull?

A. through the Eustachian tube
B. through the lower wall of the tympanum, having opening of the bone plate,
covering the jugular vein bulb
+ C. through the upper wall of the tympanum and antrum of the mastoid process
D. from the internal ear along the aqueduct of the cochlea and vestibule, along the internal auditory passage
E. none of the enumerated

22. Against the background of acute purulent middle otitis a patient had increased temperature, headache, nausea, vomiting, meningeal signs, sensory amnesia, acalculia, agraphia. What diagnostic methods are most informative in this case?

A. radioisotope encephalography
B. lumbar puncture
C. electroencephalography
+ D. Ultrasound EG, CT, MRT
E. rheoencephalography

23. A patient was admitted to hospital with chronic purulent epitympanitis, complicated by cholesteatoma, paresis of the facial nerve. What is the rational urgent tactics of treatment?

A. conservative therapy, washing of the attic by 40° alcohol
B. tympanoplasty
+ C. radical operation in the middle ear
D. paracentesis of the eardrum
E. electrical stimulation of the mimic muscles of the face

24. Against the background of exacerbation of chronic purulent epitympanitis a patient suddenly developed hectic temperature, general cerebral symptoms, tenderness of the posterior edge of the mastoid process and along the movement of the jugular vein on the neck. What is a probable diagnosis?

A. chronic purulent epitympanitis, purulent otogenic meningitis
+B. chronic purulent epitympanitis, sinus thrombosis, otogenic sepsis
C. chronic purulent epitympanitis, otogenic arachnoiditis
D. chronic purulent epitympanitis, mastoiditis
E. chronic purulent epitympanitis, otogenic abscess of the cerebellum

25. A patient with chronic purulent epimesotympanitis and upper lobe pneumonia had been developing increased headache, increased body temperature to 37.8°C , nausea, vomiting for two weeks. Meningeal signs are moderately expressed on examination. Cytosis to 600 cells in 1ml, predominantly lymphocytes is found n the cerebrospinal fluid, sugar is reduced. What is a suppositional diagnosis?

A. chronic purulent epitympanitis, otogenic purulent meningitis
B. chronic purulent epitympanitis, virus serous meningoencephalitis
+C. chronic purulent epitympanitis, tubercular meningitis
D. chronic purulent epitympanitis, sinus thrombosis
E. chronic purulent epitympanitis, otogenic abscess of the brain

26. Against the background of exacerbation of chronic otitis a patient developed a severe headache in the occiput, vertigo. Objectively: consciousness is confused, the skin is pale, the pulse is 58 beats per minute, the body temperature is 38.9°C. Otoscopy: exacerbation of chronic purulent epitympanitis, tenderness of the occipital region to percussion. Large-swinging nystagmus, fall in Romberg's position,

adiadochokinesia disorder, reduction in the muscular tone of the extremities on the side of affection are determined. The direction of fall does not change in a change of the head position. What complication of chronic epitympanitis arose?

A. purulent leptomeningitis
B. abscess of the temporal lobe of the brain
C. sinus thrombosis, otogenic sepsis
+ D. abscess of the cerebellum
E. labyrinthitis

27. A child of 8 years old developed a severe chill, the body temperature increased to 40.2°C against the background of mastoiditis with all typical signs, and in three hours it was lowered to 37.3°C, which was accompanied by intensive perspiration. On examination: infiltration, hyperemia of the soft tissues on the posterior edge of the mastoid process, positive symptoms of Whiting and Foss. What complication should we think of?

A. acute diffuse purulent labyrinthitis
+ B. thrombosis of the sigmoid sinus
C. subdural abscess
D. subperiosteal abscess
E. purulent meningitis

28. A patient of 28 years old has been suffering from pyorrhea out of the left ear since early childhood. She was not regularly treated. A month ago earache and headache developed. At the beginning the temperature was about 38.0°C, and reduced to 37.0°C. Earache disappeared in a week, and headache became only feeble. But 2 days ago earache developed again, headache became unbearable, but analgesics were not effective. The same day radical operation in the left middle ear was performed, in which cholesteatoma and extradural abscess were found. After the operation the patient's condition improved. Within a week headache was insignificant, and then again it was intensified, vomiting and symptom of amnestic aphasia arose. The patient need of urgent repeated operation. What is the assumed diagnosis?

A. abscess of the cerebellum
+ B. otogenic abscess of the temporal lobe of the brain
C. otogenic meningitis
D. extradural abscess
E. subdural abscess

29. The course of acute purulent otitis media in a patient was complicated by abscess of the left temporal lobe of the brain. Name its characteristic features in right-handers?

+ A. lethargy, sleepiness, localized headache, presence of focal symptoms (amnestic aphasia)
B. psycho-motor excitation
C. cerebrospinal fluid is turbid, expressed pleocytosis
D. otoscopic picture corresponds to the clinical picture of acute purulent middle otitis, mastoiditis
E. adiadochokinesia disorder

30. Against the background of chronic purulent epitympanitis a patient developed intracranial complication - abscess of the temporal lobe of the brain. What form of operation is applicable in this case?

A. mastoidotomy
B. extended mastoidotomy
+ C. extended radical operation in the middle ear with puncture of the brain substance and opening of abscess through the trepanation cavity
D. antroatticotomy
E. tympanoplasty

31. Against the background of exacerbation of chronic epitympanitis a patient began to complain of severe vertigo, nausea, vomiting, disorder of gait, headache. What diagnostic symptoms do not allow to differentiate abscess of the cerebellum from labyrinthitis?

A. change in the muscular tone
B. disturbance of motor coordination

C. large-swinging nystagmus in the horizontal plane; rapid and slow components are not distinguished; it is directed to the sick side

D. flank gait is feasible on one side

+ E. otoscopic data

32. Against the background of chronic purulent otitis a patient developed abscess of the cerebellum. What direction of infection spread from the middle ear cavities to the contents of skull is not encountered in this complication?

A. from the cells of the mastoid process, which closely approach the contents of the posterior cranial fossa along the whole length

B. through the internal auditory passage

C. through the aqueduct of the vestibule

D. through the aqueduct of the cochlea

+ E. through the roof of the tympanum

33. A patient was urgently admitted to the clinic with chronic purulent epitympanitis, complicated by subdural abscess. Determine the scope of surgical intervention in this complication:

A. opening of the antrum and evacuation of pus

+ B. extended radical operation in the ear with baring of the dura mater in the region of the medial and posterior (sigmoid sinus and Trautman's triangle) cranial fosses

C. removal of the posterior wall of the auditory passage and lateral wall of the attic

D. baring of the walls of the sigmoid sinus

E. tympanopuncture and paracentesis

34. Against the background of acute purulent middle otitis a patient developed pain and swelling in the region of the mastoid process, headache, hectic temperature, general malaise, intensive pyorrhea out of the ear. Which of the clinical symptoms allows to suspect sinus thrombosis?

+ A. fever, chill, which is alternated by perspiration

B. nausea, vomiting

C. vertigo

D. intensive pyorrhea out of the ear

E. paresis of the facial nerve

35. A patient who had a craniocerebral injury, developed a fracture in the region of the posterior bone wall of the external auditory passage. What symptomatology is characteristic of this state?

A. sharp deafness, bleeding out of the ear

B. pain during mastication, bleeding out of the ear

C. pain during tmastication, diminished hearing, high temperature

+ D. paralysis of the facial nerve, damage of the sigmoid sinus with bleeding

E. liquorrhea

36. Against the background of exacerbation of chronic purulent epitympanitis a patient had a severe local headache, increased body temperature to 39.0°C, nausea, vomiting. Subdural abscess in the temporal region is found by computer tomography of the skull. Determine localization of this abscess.

A. between the sheets of the dura mater

+ B. between the dura mater and arachnoid

C. between the arachnoid and vascular membrane

D. between the bone and dura mater

E. in the brain substance

37. A patient with acute purulent middle otitis began to complain of severe headache, nausea, vomiting, increased body temperature to 39.0°C. There were tenderness of the mastoid process, positive meningeal signs. What are tactical actions of the doctor?

A. extended cavity operation in the ear, lumbar puncture

+ B. extended mastoidotomy, lumbar puncture

C. surgical treatment after the complete examination of the patient

D. paracentesis of the eardrum

E. tympanoplasty

NONPURULENT PATHOLOGY OF THE EAR
SENSONEURAL HEARING LOSS (SNHL)

Sensoneural deafness or neuritis of the vestibulocochlear 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) gyrus.

The etiology 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 sensoneural hearing loss include :

- Infections of labyrinth, viral, bacterial or spirochete. Most common causes of the disease are infectious diseases such as influenza, measles, scarlet fever, typhus or malaria.
- Trauma to labyrinth or VIII-th nerve, e.g. fractures of temporal bone or concussion of labyrinth or ear surgery.
- Noise induced hearing loss (acoustic, vibration, barotrauma)
- Ototoxic drugs or industrial poisoning. . Degenerative changes in the cells of the organ of hearing prevail in toxic neuritis caused by medicament poisoning (streptomycin, monomycin, kanamycin).
- Presbycusis
- Meniere's diseases
- Acoustic neuroma
- Sudden hearing loss (vessel etiology)
- Familial progressive SNHL
- Systemic disorders, e.g. diabetes, cardiovascular pathology, hypothyroidism, kidney disease, autoimmune disorders, multiple sclerosis etc.

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 cytomegalovirus 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). Sensoneural deafness following meningitis is a well known clinical entity.

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

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

OTOTOXICITY

*1. Aminoglycoside antibiotics***.** Streptomycin, gentamycin 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, sizomycin and dihydrostreptomycin are cochleotoxic. They cause selective destruction of outer hair cells, starting at the basal coil and progressing onto the apex of cochlea.

*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 edema and cystic changes in the stria vascularis of the cochlear duct. The effect, in most cases, is reversible but permanent damage may occur.

Salicylates. Symptoms are tinnitus and bilateral sensoneural 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.

*Quinine***.** Ototoxic symptoms due to quinine are tinnitus and sensoneural hearing loss, both of which arc reversible. The symptoms generally appear with prolonged medication but may occur with smaller doses in those who are susceptible. Congenital deafness and hypoplasia 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.

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

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

*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.

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

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

Noise induced hearing loss. 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.

PRESBYACUSIS. Sensoneural hearing loss associated with physiological aging process in the ear is called presbyacusis. It usually manifests at the age of 65 years but may do so early if there is hereditary predisposition, chronic noise exposure or generalized vascular disease.

Patients of presbyacusis 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 presbyacusis 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. SNHL 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 SNHL further progresses, impaired hearing can turn into complete deafness.

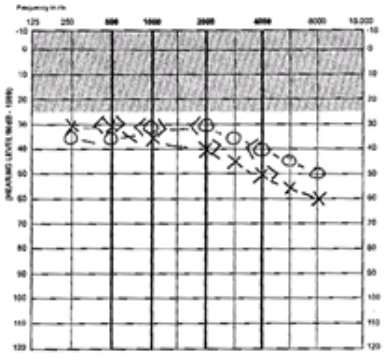


Fig.72. Audiogramm in SNHL

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 SNHL. Tuning-fork and audiometric tests are of leading importance in topical diagnosis.

Hearing disorders associated with neuritis should be differentiated from perceptive disorders due to brain tumor, hemorrhage into the internal ear, and some other affections.

Characteristics of SNHLare:

1. A positive Rinne test, i.e. air conduction better than bone conduction.
2. Weber lateralized 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 (fig. 72) 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. Steroid therapy. Prednisolone, Dexametasone inflammatory and relieve edema. They have been found useful in SNHL 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 (Rheosorbilact, Neohemodes, Rheogluman 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 SNHL of any etiology. 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 parameathal Novocain block (1 per cent Novocain solution is injected 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 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. Unithiol 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, 100 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 (adenosintriphosphoric 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, Trental etc.); remedies, promoting a dissolution of atherosclerotic congestions; remedies, rising a flow of erythrocyte mass through the narrow capillaries. In some cases there is an effect of a treatment of vertebrobasillar 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 SNHL recover spontaneously within 15 days. Chances of recovery are poor after 1 month. Severe hearing loss and that associated with vertigo have poor prognosis. Younger patients below 40 and those with moderate losses have better prognosis.

MENIERE'S DISEASE

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

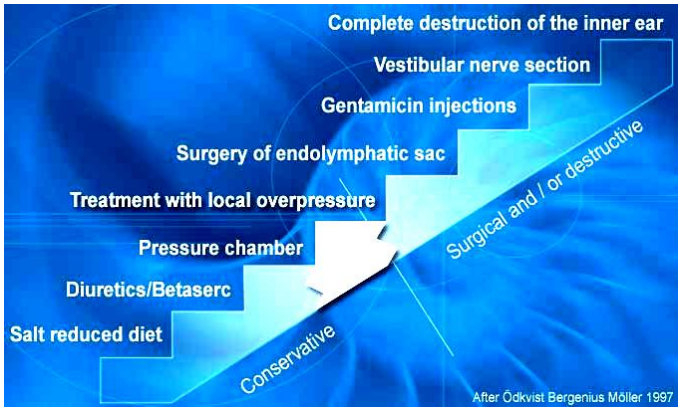


Fig.73. Scheme of treatment of Meniere's disease

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

Fluctuation of hearing is a leading diagnostic sign of the auditory disorder: the hearing can improve considerably between attacks against the background of a gradually progressing deafness. During the initial stage of the disease, the hearing function can be restored completely thus indicating the absence of organic changes in the vestibulocochlear nerve during this period.

Meniere's disease occurs mostly in the young. Its onset is characterized by the noise in the ear which is followed (in a few hours or years) by attacks of systemic vertigo and vegetative disorders. An important point is that the auditory, rather than vestibular, disorders are typical for the onset of the disease. When es tablishing a diagnosis, it is necessary to take into account the periodicity of attacks, their short duration, good subjective condition of the patient during remission, etc.

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

Treatment. The polyetiological origin of the disease accounts for the multitude of methods of treating it. Methods causing reconstruction of the vegetative nervous system are widely used. These are as follows: Betahistin per os 24 mg 2 time a day during 3-6 month; reflex action of Novocain block (intranasal block, the block of the cervical sympathetic trunk); vitamin B, PP**,** A, and E therapy; oxygen therapy and habituation (training with controlled increasing strength of rotation); exposure of the diencephalon (the centre of the vegetative nervous system) and the sympathetic cervical ganglia to X-rays.

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

An acute attack of vertigo is eliminated by subcutaneous injection of 1 ml of a 0.1 per cent atropine sulphate solution, intravenous administration of steroids and 10 ml of a 40 per cent glucose solution. If this measure is not sufficient, 1-2 ml of a 2.5 per cent Aminazine solution should be injected intramuscularly. If the attack fails to be removed completely, administration of Atropine, Aminazine should be repeated in 3-4 hours. The presence of arterial hypotension rules out the use of Aminazine.

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

Surgical treatment is used only when medical treatment fails.

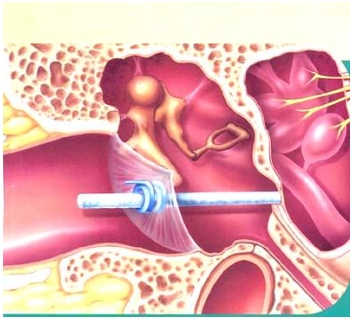


Fig.74. Injection into the tympanic cavity

They are :

Decompression of endolymphatic sac.

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

Succulotomy. It is puncturing the saccule with a needle through stapes footplate. A distended saccule lies close to stapes footplate.

Injection of Gentamycin into the tympanic cavity (fig.74).

Section of vestibular nerve.

Ultrasonic destruction of vestibular labyrinth.

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

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

OTOSCLEROSIS

Otosclerosis is a frequent cause of deafness (it occurs in more than 0.5 per cent of cases). The morphological substrate of otosclerosis is a circumscribed osteodystrophic process manifested by small single foci of newgrowths in the bony walls of the right and left labyrinths. These foci are relatively symmetric in the bony capsules of the internal ear (fig.75). They grow to replace gradually the wall of the labyrinthine capsule by a spongioid or dense bone with a different structure. In most cases the otosclerotic focus is localized anteriorly to the oval window; as it grows, the focus extends to the stapedovestibular junction, the anterior limb of the stapes, which impairs mobility of the stapes thus affecting the hearing function and causing noise in the ear. Hearing is first impaired in one ear; then, following months or years, the other ear is involved. This form of otosclerosis is called clinical. If otosclerotic foci are localized outside the windows of the labyrinth, the form is called histological; it can only be detected during histological examination of pathological material. Otosclerosis is usually associated with dystrophic changes in all tissues of the temporal bones. There are tympanic, cochlear and mixed form of this diseases.

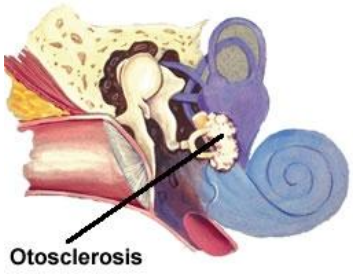


Fig.75.

Otosclerosis occurs mostly in women (in 80-85 per cent of cases). In 70 per cent of cases the disease begins at the age from 20 to 40. Otosclerosis is a hereditary disorder. Various intrinsic and environmental factors can also be important for the onset and the course of the disease.

The main audiological sign of otosclerosis is considerably increasing thresholds of air conduction in both ears. Bone conduction thresholds usually increase to a considerably smaller extent.

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

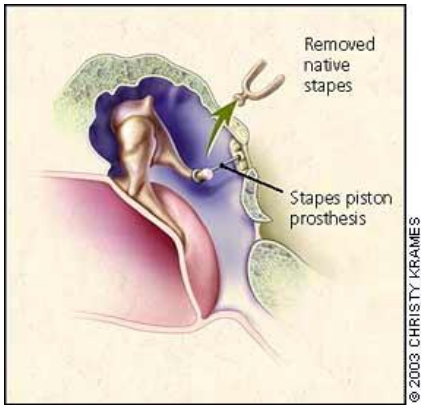


Fig.76. Stapedoplasty by a piston method

Treatment of otosclerosis is surgical. It is actually symptomatic because it does not eliminate the pathogenic factors of the disease and only removes to a lesser or greater extent the sympt om-deafness and tinnitus. The operation is aimed at reconstruction of the sound transmission system, from the ossicles to the perilymph. The mobility of the base of the stapes in the oval window is impaired due to the growth of the otosclerotic focus into the annular ligament and the base of the stapes (usually at its anterior pole).

The following operations aimed at improving the hearing function are now widely used: Stapedoplasty with partial or complete stapedectomy, and Stapedoplasty by a piston method (fig.76).

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

CHRONIC CATARRH OF THE MIDDLE EAR

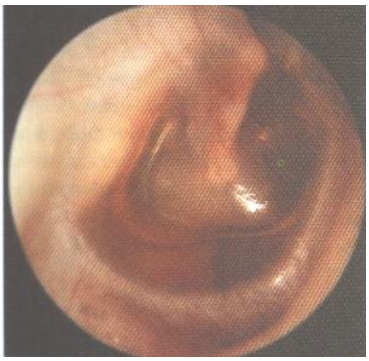


Fig.77. Chronic catarrhal middle otitis

Chronic catarrhal middle otitis (fig.77) is produced by various morbid processes in the nose and nasopharynx which spread up the Eustachian tube and serve to narrow its lumen thereby obstructing ventilation of the middle ear. Repeated acute catarrhs of the middle ear gradually thicken its mucosa and make the drum less elastic. A long-standing obstruction of the Eustachian tube gradually leads to a noticeable and stubborn retraction of the drum followed by ankylosis of the ossicular chain. Frequently lengthwise and crosswise fibers of scar tissue form between the drum and the walls of the tympanic cavity. This condition is known as chronic or adhesive catarrh. The patient complains of progressive deafness and tinnitus. It often happens that the hearing improves at times, particularly in dry weather, and deteriorates when the weather is damp, and in coryza.

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

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

Trial inflation of the tube often improves the hearing immediately.

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

Treatment. The first task is to restore the patency of the Eustachian tube, that is, to eliminate the morbid condition in the nose and nasopharynx. Adenoidectomy is a frequent procedure in such cases, particularly in children, while operations on adults are mostly performed for deformities of the nasal sep-

tum, for removal of hypertrophic posterior ends of the lower nasal conchae, etc. Sometimes, these measures alone are sufficient to remove the tubal obstruction and largely restore the hearing. But should elimination of the nasal disease fail to produce a lasting improvement of hearing, tubal inflation will be required.

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

Tubal inflation may also be performed without the use of water. The patient is directed to call out numbers, and at the count of three an air blast from the bulb is blown into the ear. To check whether inflation has been successful an otoscope is used. In cases where a rubber bulb and composition tip are inadequate equipment for inflation to be properly performed, or if unilateral inflation has to be made, the tube will be inflated through a catheter following a short nasal anesthesia, if necessary. An aural catheter is a slender 15 to 17 cm long metal tube curved like a beak at one end funnel-shaped at the other, basal end. At the base of the catheter, there is a small ring set on edge in the same plane as the beak. Prior to use, the catheter should be sterilized in boiling water. After the catheter has been slipped in along the nasal floor down to the nasopharynx with its beak pointing downwards, the latter is turned to the middle, and the catheter is gently pulled back until the beak has touched the back edge of the vomer. Here, on the lateral wall of the nasopharynx, is the mouth of the Eustachian tube. By turning the beak 180 degrees outwards it is slipped into the mouth of the Eustachian tube. This is followed by inflation . The catheter should be introduced with gentle caution and without any pressure. The beak curvature may be altered, if necessary.

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

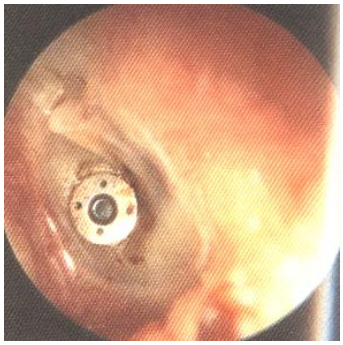


Fig.78. Shunt of eardrum

In severe cases the shunt eardrum should be made (fig.78).

Inflation may improve the hearing for several hours to a few days. Therefore, repeated inflations have to be made every one, two or three days, sometimes up to 5, 10 and 15 times in all. The nasopharynx is simultaneously treated. A pneumatic massage of the drum can also be used in combination with inflation. If a special apparatus is not available, the massage can be made by means of a pneumatic speculum tightly pressed into the auditory canal and compressed with moderate effort up to 60-100 times a minute to produce alternate suction and pressure on the drum membrane. This will make the drum move in and out and set in motion the entire ossicular chain.

Prophylaxis. The best way to avert middle ear catarrh is to ensure normal nasal breathing. The earliest possible treatment of acute catarrh of the upper respiratory tract and timely management of chronic diseases of the nose and nasopharynx will no doubt serve to keep down the rate of severe deafness. The presence of adenoids severely affects the hearing and their timely removal will undoubtedly prevent hearing loss in quite a number of cases.

Differential diagnosis of the nonpurulent diseases of the ear

Anamnesis	Cochlear neuritis	Chronic catarrhal middle otitis	Otosclerosis
Hereditary diathesis	Sometimes – inborn deafness	Absence	Presence of a hardness of hearing at a near relations
The main reason of the disease	Infection diseases, vascular diseases and intoxications, including by antibiotics	The diseases of the nose, paranasal sinuses; inflammation of auditory tube. Tumors in nasopharynx.	Unknown. Disease usually progresses after pregnancy and birth.
The peculiarities of disease's current.	Sudden or gradual lowering of the hearing accompanied by sensation of noise and sometimes – giddiness.	Clicking in the ears at deglutition, the sensation of the noise is unnecessary. The lowering of the hearing has inconstant character.	The sensation of the noise in the ears; slow lowering of the hearing.
The character of noise	High frequency (ringing, whistle).	Mostly absent	Low frequency (A noise of wind, rustle of leaves etc.)

Tuning fork examination in nonpurulent pathology of the ear

Tuning fork's tests	Tympanic type of otosclerosis, exudative otitis	Cochlear neuritis
Tuning fork 128 Hz *Rinne* test of air and osseous conduction	Negative. A patient hears better through the bone of mastoid processes then by the air.	Positive. Perception of tuning fork is low through the air and bone.
Weber test of the sound at a position of the sounding tuning fork at the middle of a scull.	In the side of injured ear, at a breach of a sound conduction from both sides – in a side of less hearing ear.	In a side of a health ear.
Schwabach test – tuning fork's position is on the mastoid processes.	There is no changes.	Perception of tuning fork's sound is shortened through mastoid processes.
Gelle test – degree of perception of a sound through the bone of mastoid processes at a change of the pressure in acoustic duct with the help of Zigle funnel	Variations of pressure in acoustic duct due to stapes ankylosis don't change on perception of tuning fork's sound through the bone.	The tuning fork's sound will be percepted better or less at change of a pressure in acoustic duct.

Tuning fork 512 Hz Federiche test – comparison of perception of tuning fork's sound which is on tragus and bone of mastoid processes	Negative. A patient hears better the sound from a mastoid processes.	Positive. A patient hears better the sound from tragus
Tuning fork 2048 Hz Perception of tuning fork's sound through the air.	It isn't lowered or lowers unimportantly	It lowers considerably.

Differential signs of patient's with nonpurulent pathology of the ear

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

The Hearing Loss and Deaf

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

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

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

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

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

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

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

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

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

Objective audiometry. It includes :

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

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

Impedance audiometry. Stapedial muscle contracts reflex in response to a sound of 70-100 dB and this reflex can be recorded. Elevated intensity levels indicate middle ear or sensoneural hearing loss. Tympanometry can also detect and differentiate causes of conductive deafness.

MANAGEMENT

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

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

1. Parental guidance. It is a great emotional shock for parents to learn of their child being deaf. They should be dealt sympathetically so as to accept the child. They should be told of child's disability and how to care for it. Rehabilitation of the deaf demands a lot from parents : care and periodic replacements of hearing aids, change of ear moulds as child grows, follow up visits for re-evaluation, education at home and selection of vocation.

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

3. Development of speech and language

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

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

Types of hearing aids

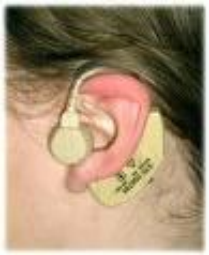


Fig.79. Hearingaid

Air conduction. In this, the amplified sound is transmitted via the ear canal to the tympanic membrane (fig.79).

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

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

1. Body-worn type. Most common type; microphone and amplifier along with battery are in one case worn at the chest level while receiver is a long distance away at ear level.

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

Indications for hearing aid

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

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

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

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

COCHLEAR IMPLANTS

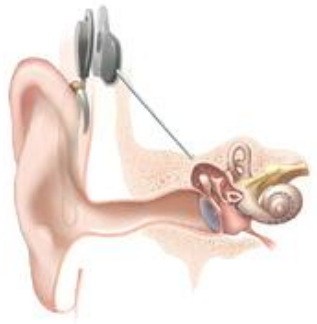


Fig.80. Cochlear implants

Cochlear implants (fig.80) have been developed recently and are still in their devel opmental phase. They are electronic devices which convert sound signals into electrical impulses which thendirectly stimulate the cochlearnerve. Thus they replace the non-functional transducer system of hair cells of the cochlea.

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

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

Selection of patients

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

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

Current status of implants

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

Control tests

1. A patient complains of periodic diminished hearing, tinnitus, changes of hearing depending on the head position. He suffers from allergic rhinosinusopathy. The conductive type of hearing disorder is revealed by audiometry. The eardrum is of yellowish color, turbid. What form of treatment is indicated to the patient?

A. antibiotic therapy
B. tympanoplasty
+C. catheterization of the auditory tubes, shunting of the eardrum
D. alcohol drops in the ears
E. mastoidotomy

2. A patient of 25 years old referred to the ENT doctor for severe hearing disorder, which developed a month after discharge from hospital, where she was treated for pyelonephritis and chronic kidney failure. She was given the course of monomycin in the cumulative dose of 17g. What caused diminished hearing in the patient?

+A. application of ototoxic antibiotic
B. affection of the cardiovascular system
C. application of the diuretics
D. viral infection
E. traumatic injures

3. Parents of the 6-month baby referred to the doctor on suspicion that hearing of their baby is poor. Visible pathology is not revealed on examination of the ENT organs. How is it possible to investigate hearing in the baby?

+A. by registering the auditory caused potentials (ACP)
B. by whisper speech
C. by audiometry
D. by tuning fork methods
E. by tympanometrya

4. A patient presented complaints on diminished hearing in one ear for a month, serous- blood-containg discharge out of the nose, difficulty of nasal breathing. The conductive type of hearing disorder is found on audiometry and salpingootitis. What additional investigation should be conducted, except:

A. X-ray examination of the paranasal sinuses
B. examination of the nasopharynx with pulling aside the soft palate
C. bacterial culture from the nose
D. tympanometry
+E. blowing of the Eustachian tube

5. As a result of frequently recurrent acute middle purulent otitis a patient developed chronic adhesive otitis and conductive type of hearing disorder. What of the enumerated is the most frequent predisposing factors for development of this pathology?

A. use of ototoxic antibiotics
B. frequent ARVI
C. reduction in the imunnologic protection of the organism
+D. disturbance of nasal breathing, frequent inflammatory diseases of the cavity of the nose, nasopharynx
E. injury of the ear

6. A patient had suddenly diminished hearing against the background of taking gentamycin, high-frequency noise appeared. The surdologist diagnosed affection of the sound receptor apparatus. What results of tuning fork tests are characteristic of sensoneural hearing disorder?

+A. lateralization of sound in Weber's experiment in the better hearing ear, Rinne experiment is positive

B. Schwabach experiment is unchanged

C. Schwabach experiment is prolonged

D. Rinne experiment is negative

E. lateralization of the Weber test to the affected ear, Rinne experiment is negative

7. A patient of 29 years old began to note tinnitus, diminished hearing after delivery. Otosclerosis is diagnosed by acumetry. What is most optimum variant of audiogram for the operations for otosclerosis?

A. lowered bone conduction simultaneously with the air, bone-air break does not exceed 10 dB

+B. double-sided bone-air break to 40 dB in insignificant reduction in sound perception

C. bone-air break on either side is not more than 15 dB in significant reduction of bone conduction to 50-60 dB

D. large bone-air break to the right (40 dB) in lateralization of sound in the right ear, in the left - the norm

E. both side sensoneural deafness

8. Against the background of chronic allergic rhinitis a patient began to note
diminished hearing, low-frequency noise, change of hearing depending on change in the head position. The disfunction of the sound-conduction system of the middle ear is revealed on audiometry. What may these symptoms be caused by?

A. change in the intracranial pressure

B. displacement of the auditory bones

C. change in pressure in the cochlea

+D. displacement of transudate in the tympanum

E. acute purulent otitis media

9. After the course of streptomycin a patient began to feel diminished hearing, tinnitus. Objectively: eardrums are unchanged, on the audiogram there is reduction in the bone and air conduction by 40- 50 dB predominantly at the high frequencies. What is the diagnosis?

A. chronic adhesive otitis

B. otosclerosis

C. acute otitis media

+D. sensoneural hearing disorder

E. Meniere's disease

10. A patient complains on suddenly diminished hearing, sensation of pouring liquid in the ear, autophony. The general state is satisfactory. What is a probable otoscopic picture?

A. eardrum is hyperemic, protruded

B. normal

C. central perforation of the eardrum

+D. eardrum is gray, turbid, pulled in

E. eardrum is thinned, the promontary is seen through it

11. A patient presented complaints on diminished hearing, autophony, change of hearing depending on the head position for 4-5 months. On the audiogram: bone conduction is not disturbed, air one is descended, predominantly to low frequencies. The eardrums is turbid, light cone is not expressed. What is a probable diagnosis?

A. acute purulent otitis media

B. otosclerosis

C. sensoneural hearing disorder

+D. chronic catarrh of the middle ear

E. chronic purulent middle otitis

12. A patient complains of periodic attacks of vertigo, which are accompanied by diminished hearing of

the conduction type, tinnitus. Hearing is improved after the attack, but it does not reach the norm. The attack is controlled by diuretic preparations, spasmolytics. What is a probable diagnosis?

A. sensoneural hearing disorder

+B. Meniere's disease

C. otosclerosis

D. chronic catarrh of the middle ear

E. vertebro-basillar insufficiency

13. A patient is troubled by periodically renewed discharge out of the ear of mucous purulent nature, diminshed hearing, tinnitus, sensation of pouring "water" in the ear. On otoscopy the eardrum is somewhat turbid, the level of fluid is determined (in the form of "hair line"). Select the characteristic audiologic features of conduction hearing disorder in secretory otitis:

A. bone conduction is reduced predominantly to low frequencies, there is the bone-air interval

+B. air conduction is reduced predominantly to low frequencies, there is the bone-air interval

C. break of bone conduction to high frequencies

D. bone conduction is reduced predominantly to high frequencies

E. there is no bone-air interval

14. A patient of 38 years old had acute acoustic injury (explosion) at work, after which his hearing was considerably diminished, short-term vertigo developed. What data of the audiogram do not correspond to the characteristic of sensoneural hearing disorder?

A. predominantly worsening in perception of high tones (increased thresholds of perceptibility)

B. gently descending type of the threshold curves in the initial stages

+C. presence of the large bone-air interval

D. absence of bone-air break.

E. steeply descending type of the threshold curves

DISEASES OF THE NOSE AND PARANASAL SINUSES

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

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

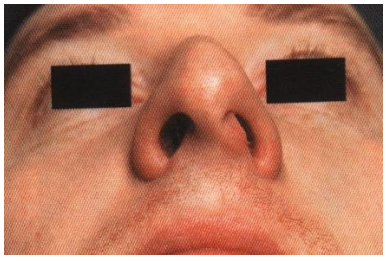


Fig.81. Deformation of the nasal septum

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

Deformation of the nasal septum. This occurs usually due to injury. Physiological deformation is usually the result of disbalanced growth of the septum and the bon y framework into which the septum is included. Three types of deformities are distinguished: deviated septum, septal spur and shelf. The

main complaint of the patient with deviated nasal septum is nasal obstruction. Headache, nasal discharge, occasional pain in the ear, dry throat, etc. also occur in some cases.

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

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

The clinic of acute catarrhal rhinitis includes three stages, which are continuous with one another: the first stage is dry irritation, the second stage is characterized by increased mucous secretion, and the third stage (resolution) is characterized by mucopurulent secretion. Acute rhinitis begins with the feeling of dryness, tension, burning, and itching in the nose and often in the pharynx and the larynx; sneezing is annoying. The patient complains of indisposition, chill, discomfort and headache (mostly pain in the forehead). The body temperature is elevated. Nasal respiration becomes difficult-from insignificant impediment to a complete obstruction due to obturation of the nasal meatuses with swollen mucosa. Olfaction is impaired significantly. The sense of taste is also altered. The speech becomes nasal (rhinolalia clausa). Profuse watery discharge from the nose is characteristic of the first day of acute rhinitis. The amount of mucus in the discharge increases later. This can cause hyperaemia and swelling of the skin at the nose vestibule and of the upper lip. The nasal discharge becomes seropurulent in 4 or 5 days. The amount of nasal discharge decreases gradually during the next few days, swelling of the mucosa subsides, respiration through the nose and olfaction are restored, and the patient recovers in 8-14 days from the onset of acute catarrhal rhinitis.

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

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

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

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

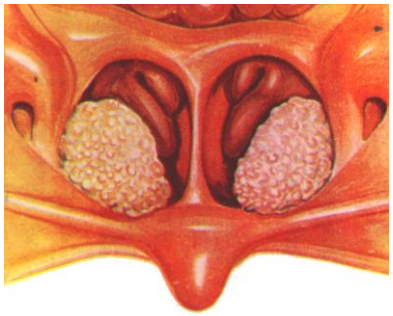


Fig.82. Chronic hypertrophic rhinitis

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 PNS 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 (fig.82).

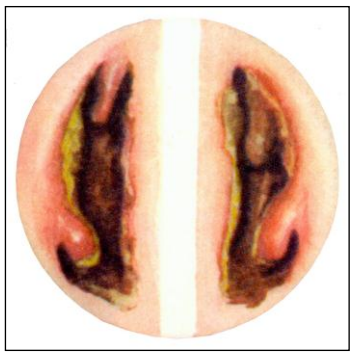


Fig.83. Chronic atrophic rhinitis

Chronic atrophic rhinitis (fig.83)**.** 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 medicament 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 operation vasotomy or 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 (Loratadine, Klaritin, Erius etc.) and hormones (Hydrocortisone, Prednisolone) are used for the purpose. Topical steroids such as Beclomethasone dipropionate, Nasonex and Flixonase 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.

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 (septotomy) is most rational in such cases.

SINUSITIS

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.

Fig.84. Right side hemisinusitis

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 (fig.84), CT 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.

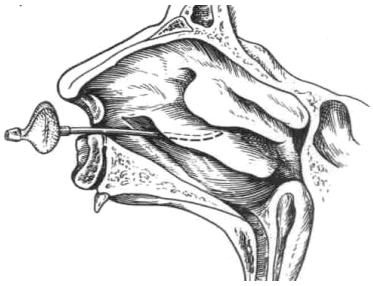


Fig.85. Antral puncture

The Kulikovsky needle is commonly used for antral puncture (fig.85). The sinus wall is punctured by the needle and the sinus contents are aspirated; then, the sinus is irrigated with a disinfectant solution. 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 mucous discharge which is often retained in the nasal cavity, and dries in crusts. The discharge in serous, or allergic maxillary sinusitis accumulates in the sinus and drains in portions when the patient assumes a certain position facilitating drainage of the sinus through the nasal meatus. An unpleasant odour is sometimes the main complaint of the patient who feels the smell himself. In bilateral chronic pathologies in the maxillary sinuses patients always complain of decreased sense of smell. Local or diffuse headache usually develops only during exacerbations or in obstructed drainage of the sinus. During remission, the general objective and subjective condition of the patient is satisfactory. Exacerbation of a chronic process can be attended with elevated temperature, worsening of the patient's condition, painful swelling of the cheek, oedema of the eyelid and local or diffuse headache.

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

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

The pathological discharge from the nose and sinus (taken during antral puncture) is examined in the laboratory for the presence of microflora and for sensitivity to antibiotics. Diagnostic puncture of the maxillary sinus is widely used in older children. Pathology of the maxillary sinus should be differentiated

from frontitis, ethmoiditis, and in rare cases from sphenoiditis. In adults it is necessary to rule out the odontogenic nature of the disease, especially in the presence of a suppurative process in the roots of the upper teeth (4, 5, 6), whose apices are in the immediate vicinity of the floor of the maxillary sinus.
Conservative treatment. Treatment should begin with elimination of causes of the disease. If maxillary sinusitis is odontogenic, the teeth should first of all be treated. It should be noted that radical operations on the sinus will be ineffective if the odontogenic cause remains active. In the presence of adenoids or adenoiditis in children, the tactics should be the same: the nasopharynx should first be treated, and only then should treatment of maxillary sinusitis be started. As a rule, general antibacterial treatment, mycolitics (Fluditec) are administered during exacerbation.

Antral puncture and irrigation of the sinus with a disinfectant solution or enzymes, 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.

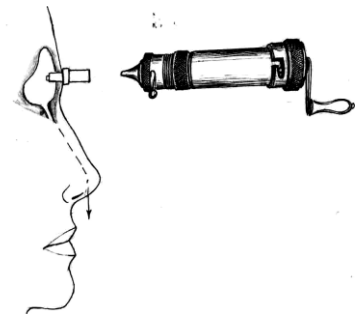


Fig.86. Frontal puncture

Treatment is usually conservative. But if the disease is long-standing 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 (fig.86). Local treatment includes application of preparations causing anaemization of the nasal mucosa: vasoconstrictors drops. 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 conchae, 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 cantus 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 conchae. UHF or SHF on the area of the ethmoidal sinus are indicated. If the body temperature is elevated, antibacterial, mycolitics (Fluditec) preparations are given.

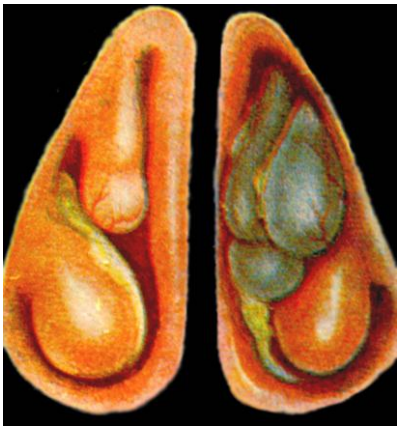


Fig.87.Chronic polipous sinusitis

If a closed empyema or ophthalmic complication develops, the cells of the ethmoidal laby rinth 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 (fig.87) 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.

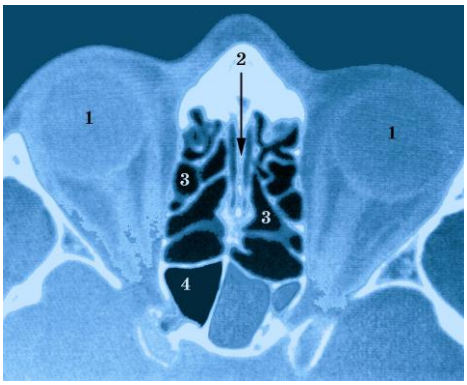


Fig.88. CT: Sphenoiditis

Acute and chronic sphenoid sinusitis. Isolated affection of the sphenoidal si nuses is rare (fig.88). 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 subfebril; 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 sphenoid 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 endonasal. Symptoms of complications (septic, intracranial, ophthalmic) are indications for emergency operation on the sphenoid sinus. Chronic sphenoid sinusitis is provoked by the same conditions as chronic affection of the other paranasal sinuses.

RHINOGENIC COMPLICATIONS

Orbital complications include:

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

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

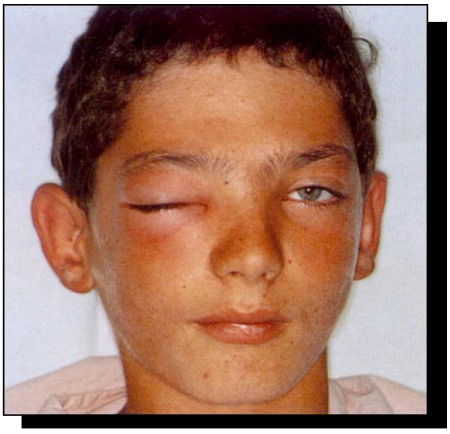


Fig.89. Orbital cellulitis

Orbital cellulitis (fig.89). When pus finds its way into the orbit, it spreads between the orbital fat, extraocular muscles, vessels and nerves. Clinical features will include oedema of lids, exophthalmos, chemosis of conjunctiva and restricted movements of the eye. Vision is affected causing partial or total loss which is sometimes permanent. Patient may run high fever. Orbit cellulitis is potentially dangerous because of the risk of meningitis and cavernous sinus thrombosis.

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

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

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

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

Intracranial complications. Rhinogenic intracranial complications are very dangerous. In 75 per cent of the cases, rhinogenic intracranial complications arise due to chronic inflammation in the sinuses, and in 25 per cent of the cases, they are secondary to acute sinusitis. The infection can spread by the contact, haematogenic and lymphogenic pathways. Frontal, ethmoid and sphenoid sinuses are closely related to anterior cranial fossa and infection from these can cause meningitis and encephalitis, extradural abscess, subdural abscess, brain abscess.

Cavernous sinus thrombosis. Orbital veins have no valves and freely communicate with the cavernous sinus and for this reason infection from the orbit or paranasal sinuses can easily spread to the cavernous sinus. Thrombosis of the cavernous sinus is characterized by pronounced local symptoms which develop due to difficult venous outflow. The affection is characterized by swelling of the eyelids and the adjacent tissues, dilatation of superficial veins and hyperaemia of the orbital veins, cyanosis of the orbit, and exophthalmia. These symptoms are supplemented by papilloedema, oedema and thrombosis of the retinal veins. Focal symptoms are also characteristic. Since the first branch of the trigeminal nerve and the oculomotor nerves (3rd, 4th and 6th pairs) pass along the sinus wall, the patient suffers from neurological pain in the region of innervation of the first branch of the 5th pair (orbital and infraorbital neuralgia), analgesia of this region, including comeal anaesthesia, and decreased or lost corneal reflex. Affection of the oculomotor nerves causes internal and external ophthalmoplegia with paralysis of the eyeball of various type and gravity. Unilateral thrombosis of the cavernous sinus can extend to the other side. Signs of toxaemia, high fever (40°C) with chills, and meningitis are also present. Blood culture should always be done. CT scan is very helpful in diagnosing intracranial abscesses. Treatment includes I/V antibiotics, anticoagulants and drainage of any abscess..

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

Control tests

1. A patient referred to the doctor with complaints on pain in the region of the right cheek, increased temperature, nasal obstruction, purulent discharge from the nose. On anterior rhinoscopy: purulent strips in the right middle nasal passage. X-ray of the paranasal sinuses: opacity of the right maxillary sinus. The diagnosis is: acute right-sided maxillary sinusitis. Through what nasal passage to perform the puncture of the maxillar cavity?

A. middle
B. superior
C. general
+D. inferior
E. through anterior wall of maxillar sinus

2. A patient is disturbed by difficulty of nasal breathing, frequent rhinites. The enlarged lower nasal turbinates are visible on anterior rhinoscopy, their mucous membrane is edematic. What method allows to differentiate the hypertrophic form of rhinitis with the vasomotor one?

A. X-ray examination of the nose
+B. anaemisation of the mucous membrane of the nose

C. study of the function of nasal breathing
D. study of the sense of smell
E. ultrasonic diagnostics

3. 18-year-old patient who has been suffering from vasomotor rhinitis for a long time referred to the doctor. There are no deformations of the nasal septum. Runny nose arose against the background of prolonged application of vasoconstrictor drops. The conservative methods of therapy are not effective. What is most expedient method of treatment?

+A. ultrasonic disintegration or vasotomy
B. lower conchotomy
C. paint of the mucous membrane of the nose with the Lugol solution
D. administration of the vasoconstrictor drops in the nose
E. administration of drops in the nose of astringent action

4. A patient with chronic polypous- purulent maxilloethmoiditis is performed radical operation on the paranasal sinuses. What stages does this operation not provide for?

A. opening of the sinus
B. removal of pathologic contents
C. creation of wide anastomosis between the sinus and nasal cavity
D. expansion of natural aperture for improvement of outflow from the sinus
+E. thorough scraping out of the whole mucous membrane of the sinuses

5. A patient has been complaining of dryness in the nose, crusts, sometimes thick purulent discharge with admixture of the blood for a long time. Sense of smell is not disturbed. X-ray the pathology of the paranasal sinuses is not revealed. Choose a suitable method of treatment of atrophic rhinitis.

A. ultrasonic disintegration
B. lower conchotomy
+C. paint of the mucous membrane of the nose with the Lugol solution
D. administration of the vasoconstrictor drops in the nose
E. nonmedicinal blockades of the nose

6. A patient of 12 years old was admitted to the ENT clinic with complaints on severe headache, purulent rhinitis, difficult nasal breathing. He fell ill 5 days ago, a week after the respiratory disease. The disease began with increasing in the body temperature to 38.5°C. There was swelling of the soft tissues of the forehead and eyelids on both sides. On rhinoscopy the mucous membrane of the nasal cavity was sharply hyperemic, infiltrated, cream-like pus is determined in the middle nasal passages on both sides. There was tenderness to palpation in the region of the projection of the maxillary sinuses and supraorbital region on both sides. Homogeneous density of the maxillar and frontal sinuses is determined by the X-ray examination of the paranasal sinuses. Analysis of the blood: leukocytosis – $19.5x10^9$ /l, ESR - 60 mm/h. How can the patient be treated?

A. puncture of the maxillary sinus
B. trepanopuncture of the frontal sinuses
C. intensive bacterial therapy
D. detoxication treatment
+E. all enumerated

7. A patient has been disturbed by difficulty of nasal breathing, constant serous-mucous discharge from the nose. About 6 months ago he had ARD, since then he daily instills vasoconstrictor preparations in the nose. On PNS X-ray there is no expressed pathology. What is a suppositional diagnosis?

A. chronic sinusitis
+B. chronic vasomotor rhinitis
C. recurrent acute rhinitis
D. chronic atrophic rhinitis
E. ozena

8. A patient complains of pain in the left half of the forehead and left cheek, heaviness in the head, running nose with discharge predominantly from the left half of the nose. He fell ill against the

background of a respiratory disease a week ago. Objectively: on rhinoscopy hyperemia, infiltration of the mucous membrane of the nasal cavity are determined on the left with purulent discharge in the middle nasal passage. On palpation: tenderness in the region of the left maxillary sinus and supraorbital region. What is the diagnosis?

+A. left-sided hemisinuitis

B. left-sided maxillary sinusitis

C. left-sided frontal sinusitis

D. left-sided ethmoiditis

E. pansinusitis

9. A patient referred with complaints on periodic attacks of sneezing, a burning feeling in the nose, difficulty of nasal breathing, abundant serous discharge from the nose, frequent conjunctivites. On anterior rhinoscopy there were noted edema, cyanosis of the mucous membrane of the inferior nasal turbinates with whitish spots. During anaemisation the nasal turbinates considerably decreased in size. What is a suppositional diagnosis?

A. acute rhinitis

B. acute sinusitis

C. chronic hypertrophic rhinitis

D. chronic adenoiditis

+E. chronic allergic rhinitis

10. A patient complains of frequent rhinitis with more discharge from the right half of the nose, which is accompanied by headaches, increase in the temperature, disturbance of his general state. He almost constantly breathes badly through the right half of the nose. He considers himself ill for approximately 4 years. Objectively: the mucous membrane of the nose is moderately hyperemic, infiltrated. The right half of the nose is filled with edematic formations of the oval form, which originate from the middle nasal passage. On palpation tenderness of the right maxillary cavity is determined. Nasal breathing is sharply difficult on the right. There are no deviations from the norm in other organs. What is a suppositional diagnosis?

+A. right-sided polypous-purulent hemisinusitis

B. polypous ethmoiditis

C. purulent frontal sinusitis

D. double-sided maxillary sinusitis

E. chronic allergic rhinitis

11. A patient referred with complaints on dryness in the nose, crusts, sometimes with admixture of the blood. What clinical signs allow to make the differential diagnosis between atrophic rhinitis and ozena?

A. formation of crusts in the nose

B. atrophy of the mucous membrane of the nose

C. difficult nasal respiration

D. purulent discharge from the nose

+E. PNS X-ray- atrophy of the bone tissue of the nasal turbinates

12. A patient with exacerbation of chronic purulent frontoethmoiditis, complicated by subdural abscess in the region of the anterior cranial fossa was admitted to the clinic. What is surgical tactics in this rhinogenous complication?

A. radical operation on the paranasal sinuses, formation of their communication with the nasal cavity

B. denudation of the dura mater of the anterior cranial fossa

+C. frontoethmoidotomy with denudation of the dura mater of the anterior cranial fossa, puncture and opening of abscess through the operating cavity

D. radical operation on the affected sinuses, suturing of the wound

E. puncture of abscess

13. In what diseases of the nose and paranasal cavities cannot intracranial complications be encountered?

A. furuncle and carbuncle of the nose

B. acute or exacerbation of chronic frontal sinusitis, sphenoiditis, ethmoiditis

C. traumatic damages of the paranasal sinuses
D. abscess of the nasal septum, erysipelas inflammation of the nose
+E. nasal septum deviation

14. A month after acute purulent frontal sinusitis a patient developed signs of the expressed stage of abscess of the frontal lobe of the brain. What is rational surgical tactics in this pathology?
A. radical operation on the paranasal sinuses, formation of their communication with the nasal cavity
B. baring of the dura mater of the anterior cranial fossa
C. frontoethmoidotomy with baring of the dura mater of the anterior cranial fossa, puncture and opening of abscess through the operating cavity
+D. radical operation on the affected sinuses, suturing of the wound, removal of abscess by the neurosurgical way
E. puncture of abscess

15. Against the background of acute maxillitis a patient developed phlegmon of the orbit. What therapeutic measures are indicated in this case?
A. radical operation on affected sinuses, opening of purulent foci in the orbit
B. introduction of antibiotics, diuretics
C. application of glucocorticoids, anticoagulants
D. desintoxication
+E. all of the enumerated

16. A patient squeezed out the furuncle of the nose. In several hours his body temperature increased, he developed headache, swelling in the region of the soft tissues of the nose, cheek, medial angle of the orbit, swelling of the eyelids. What symptoms are not evidence of change of phlebitis of the facial veins in thrombosis of the cavernous sinus?
A. exophthalmos
B. chemosis
C. reduction in sight or blindness
D. immobility of the eyeball
+ E. edema of the lower eyelid

17. A patient presents complaints on headache, difficulty of nasal breathing on the left. On examination: there are moderate edema of the nasal mucosa, cariously changed upper premolar on the left. X-ray examination revealed rounded shadow in the left maxillary sinus. The liquid obtained on diagnostic puncture is of amber color. What is the diagnosis?
A. acute catarrhal maxillitis
B. retention cyst of the maxillitis
+C. odontogenic cyst of the maxillary sinus
D. tumor of the maxillary sinus
E. odontogenic chronic purulent maxillitis

18. Against the background of exacerbation of chronic purulent maxillary ethmoiditis a patient suddenly developed increased temperature, pain and infiltration in the region of the lower-medial angle of the orbit, edema of the lower eyelid, limitation of mobility of the eyeball, exophthalmos. What is therapeutic tactics?
A. puncture of the maxillary sinus
B. only conservative treatment
C. operation of maxillary sinusotomy
+D. operation of maxilloethmoidotomy, revision of the cellular tissue of the orbit
E. operation orbitotomy

19. A patient is disturbed by difficulty of nasal breathing, which arose after injury of the nose 5 years ago, he has constant rhinitis. On anterior rhinoscopy there were determined congestive edema of the lower nasal turbinates, the diagonal crest on the nasal septum on the left, which adjoined the nasal turbinate. What is tactics?
A. application of the vasoconstrictor drops in the nose
B. application of the astringent drops in the nose
C. galvanocautery of the lower nasal turbinates

+D. submucous resection of the nasal septum

E. topic corticosteroids into the nose

20. A patient complains of dryness in the nose, formation of crusts, disorder of smell, frequent thick purulent discharge with admixture of the blood. On rhinoscopy the mucous membrane of the nasal cavity is dry, sharply thinned, with dry crusts with unpleasant smell on its surface. Atrophy of the nasal turbinates is noted by X-ray, paranasal sinuses are reduced, their walls are thinned. What is the diagnosis?

A. chronic vasomotor rhinitis

B. chronic atrophic rhinitis

+C. ozena

D. chronic purulent sinusitis

E. chronic atrophic rhinitis

21. A patient suffering from bronchial asthma, is disturbed by difficulty of nasal breathing, profuse mucous discharge from the nose. On anterior rhinoscopy grayish-white mucous formations, originating by the legs from the middle nasal passages are determined in the nose. Darkening of the maxillary sinuses, cells of the ethmoid bone are noted by X-ray. What is the diagnosis?

A. chronic hypertrophic rhinitis

B. chronic vasomotor rhinitis

C. chronic allergic rhinitis

+D. chronic polypous maxilloethmoiditis

E. chronic purulent [maxilloethmoiditis

22. A patient of 46 years old complains of constant dryness in the throat, periodically discomfort becomes intense, there is pain on swallowing. Objectively: the mucous membrane of the nose, posterior wall of the pharynx are dry, its surface is smooth, glistening, it is covered with viscous phlegm. What is the diagnosis?

A. acute rhinopharyngitis

+B. chronic atrophic rhinopharyngitis

C. chronic vasomotor rhinitis

D. ozena

E. chronic atrophic pharyngitis

23. A patient has been complaining of headache, difficulty of nasal breathing, purulent discharge from the nose for 2 weeks. A month ago he filled the upper premolar on the right. Darkening of the right maxillary sinus is determined by X-ray. The large number of thick crumb-like pus with unpleasant smell is obtained in diagnostic puncture. What is a suppositional diagnosis?

A. acute purulent maxillary sinusitis

B. chronic purulent maxillary sinusitis

+C. chronic purulent odontogenic maxillary sinusitis

D. chronic atrophic maxillary sinusitis

E. tumour of the maxillary sinus

24. A patient suffers from difficulty of nasal breathing, frequent rhinitis. On examination the otolaryngologist made the diagnosis of chronic hypertrophic rhinitis. Determine the characteristic rhinoscopic picture.

A. wide nasal passages

+B. nasal turbinates are enlarged, they are not reduced after anaemisation, with the uneven surface

C. pallor and swelling of the mucous membrane of the nasal turbinates, “dove-coloured spots” of Voyachek

D. hyperemia of the nasal turbinates mucous membrane with mucus on it

E. spines of the nasal septum, the mucous membrane is pale

25. Against the background of exacerbation of chronic purulent maxillary sinusitis a patient suddenly developed increased temperature, diffuse headache, nausea, vomiting, rigidity of the occipital muscles, dilatation of the veins of the eye funds. Fetid pus and crumb-like cholesteatoma masses are obtained on puncture of the maxillary sinus. What is therapeutic tactics?

A. puncture of the maxillary sinus, antibacterial therapy

B. only conservative treatment

+C. maxillary sinusotomy, antibacterial, infusion therapy
D. endonasal expansion of the sinus orifice
E. only surgical treatment

26. A patient has been complaining of the periodic pain behind the eyeballs, progressive reduction in sight, difficulty of nasal breathing, flowing in of the mucopurulent discharge on the posterior wall of the pharynx, unpleasant smell in the nose for 3 years. What is a suppositional diagnosis?

A. acute sphenoiditis
+B. chronic purulent sphenoiditis
C. acute rhinitis
D. ozena
E. chronic vasomotor rhinitis

27. A patient has been disturbed by difficulty of nasal breathing, mucopurulent discharge from the nose, headache for three weeks. On anterior rhinoscopy a strip of pus, hyperemia of the mucous membrane of the nose are determined in the middle nasal passages. What diagnostic method should be administered first of all?

A. computer tomography of the skull
B. culture of the nose mucosa
C. puncture of the maxillary sinus
+D. X-ray of the paranasal sinuses
E. anaemization of the nasal mucous membrane

28. A patient is revealed to have increased volume of the left frontal cavity on X-ray of the paranasal sinuses, thinning and displacement of the lower wall downward, increase in the transparency of the sinus, contralateral displacement of the intersinus septum. What is a suppositional diagnosis?

A. acute frontal sinusitis
B. chronic purulent frontal sinusitis
C. mucocele of the frontal sinus
+D. pneumocele of the frontal sinus
E. anatomical peculiarities of the frontal sinus

29. A patient referred to the clinic, who was diagnosed acute purulent hemisinusitis, swollen upper eyelid 2 weeks after influenza. The per oral antibiotic therapy given in polyclinic was not effective. What measure does not correspond to the adequacy of treatment?

A. puncture of the maxillary sinus
B. trepanopuncture of the frontal sinus
C. adequate antibiotic therapy
D. antihistaminic, diuretic preparations
+E. only conservative treatment

30. Against the background of general overcooling a patient began to complain of difficulty of nasal breathing, a feeling of burning, tingling in the nose, increased body temperature. What objective signs are characteristic of the first stage of acute rhinitis?

A. purulent discharge from the nose
B. discharge of the serous nature
C. wide nasal passages
+D. dryness in the nose
E. grayish-white mucous formations in the middle nasal passages

31. Against the background of exacerbation of chronic purulent frontal sinusitis a patient developed a severe headache, nausea, vomiting, high temperatures, meningeal signs. What liquor changes are most characteristic of rhinogenic meningitis?

A. protein- cellular dissociation
B. reduction in sugar in the liquor
C. reduction of the contents of chlorides
+D. high cytosis
E. high contents of protein

INJURIES TO THE NOSE

Common injuries of the skin are contusions, bruises, abrasion or wounds. Injuries to the nose (fig. 90) often concur with concussion of the brain. If such suspicion arises, the nasal bones should be examined with X-rays and the patient should be subjected to neurological examination. Contusion and a bruise do not require special treatment except application of cold immediately after the injury. Crepitation sounds indicate fractures of the ethmoid bone and rupture of the mucous and air passes via the injured tissue to bulge the face skin. The fracture of the cribriform plate is manifested by liquorrhoea from the nose.

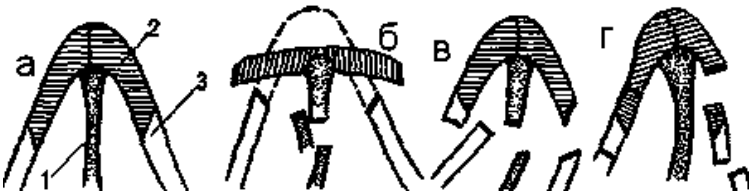


Fig.90. Type of the nasal bones fractures

X-ray examination usually reveals the character and extent of injury to the nasal and facial bones in the presence of severe oedema and infiltrative swelling of the soft tissues.

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

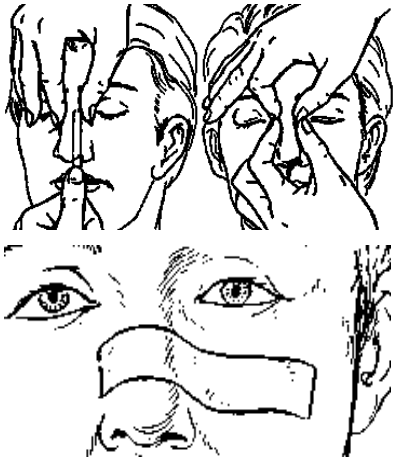


Fig.91. Nasal reposition and fixation

Laterally displaced bone fragments (fig.91) should be repositioned using the thumb of the right hand to correct displacement to the left, and of the left hand to correct the displacement to the right . A significant effort of the finger is required for repositioning of a bone fragment. A specific crunch is heard when the bone fragment is replaced. No anaesthesia is usually required, since the operation lasts only 2-3 seconds. Novocain solution can, however, be injected into the field of injury or the operation can be performed under short-lasting anaesthesia. Posteriorly displaced bone fragments should be repositioned using nasal elevators. In most cases, the replaced bone fragments have to be fixed by anterior tamponade of one or both sides of the nose. If fractures are multiple, a firmer and longer immobilization is required. The fractured nasal bones are usually repositioned on day injury, but if the patient attends doctor at later terms, the operation can also be performed, although the correction be comes more difficult. If the fragments are replaced to the nasal cavity we use endonasal method, which needs the next front tamponade and fixing of fragments. If soft tissues are broken, we carry out a primary surgical treatment and make skin titches. As per to the constructions fixing bandages are different: plasters, universal splints, made of colloidal bandages, stens, gyps. They are put on form 7 to 14 days. Such bandages are formed according to the shape of nose. Their upper part is fixed to the forehead by the bandage, and the lower one covers the nose and is fixed by the splint.

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

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

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

EPISTAXIS

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

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

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

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

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

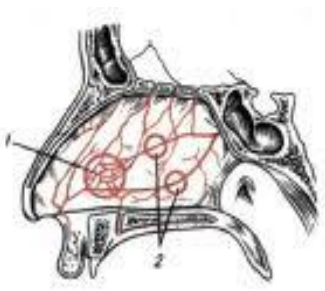


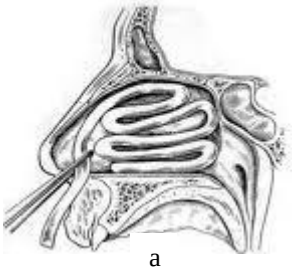
Fig.92. Places of the epistaxis

Mild, moderate, and profuse nasal bleedings are distinguished. Mild nosebleed usually originates from the Kiesselbach area (fig. 92, 1). The bleeding is and only a few millilitres are lost. Such bleedings stop spontaneously. Moderate nasal bleeding is characterized by discharge of larger amount of blood, which, however, does not exceed 200 ml in adults. Measures should be taken in such cases to arrest bleeding rapidly and completely. If blood enters the pharynx and is swallowed, profuse haematemesis can occur with a fall of arterial pressure and tachycardia.

In profuse haemorrhage (from anterior and posterior ethmoidal artery, 2), the blood loss exceeds 200 ml a day. In severe cases one litre and more of blood can be lost. Such haemorrhage is a direct danger to the life of the patient.

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

Insignificant nasal bleeding can in most cases be easily arrested by putting for 15-20 minutes a sterile cotton ball soaked in a 3 per cent hydrogen peroxide solution into the anterior part of the involved side of the nose. The cotton in the nostril should be compressed by the finger against the nasal septum. The patient should be seated upright and ice applied to the nose. If insignificant bleeding from the anterior parts of the nose recurs, the bleeding site should be infiltrated with a 1-2 per cent novocain solution or cauterized with strong trichloroacetic acid, silver nitrate, or chromic acid. Recurrent bleeding from the Kiesselbach area can be managed by separating the mucosa in the area between two incisions. If this measure fails, or if bleeding originates from deeper structures, anterior tamponade is required (fig.93a). A 10 per cent Lidococaine solution can be used (2 or 3 times) for anaesthesia. Anterior tamponade of the nose is performed by means of a 60-70-cm long turunda, nasal forceps, haemostatic paste, or emulsion.



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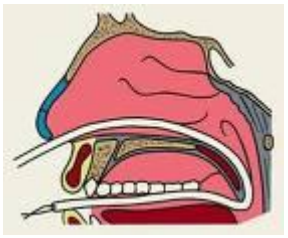

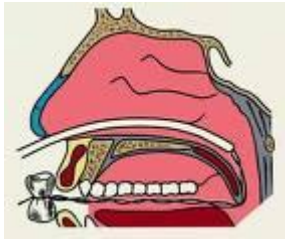

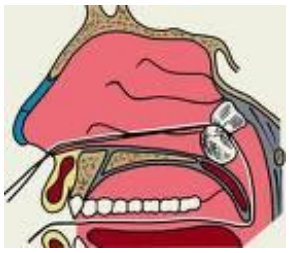

b

Fig.93. Anterior and posterior tamponade of the nasal cavity

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

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

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

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

Posterior tamponade (fig.93b) is done with special sterile tampons. Gauze is folded several times into 3 x 2.5 x 2 cm tampon which is then tied up crosswise with two 20-cm long silk threads. One end is cut off, while the other three ends remain. The posterior tamponade is begun with passing a thin rubber catheter into the bleeding side of the nose until its end enters the nasopharynx to appear in the middle of the pharynx. The end of the catheter is taken with a forceps and pulled outside through the mouth. Two threads of the tampon are tied up to this end of the rubber catheter and pulled back through the nose. The second finger of the right hand should be used to help to seat the tampon behind the soft palate in the nasopharynx and press it tightly to the corresponding choana. The next manoeuvre is to pull the two threads through the nose. The threads should be held strained while the nose is packed with the turunda

and the thread ends are then tied tightly over a gauze pad at the nasal vestibule. The thread in the mouth will be used to withdraw the tampon. Its free end is fixed on the cheek with an adhesive tape.

Posterior tampon is removed in 24 hours. But if bleeding resumes, the tamponade should be repeated and the tampon remains for 3-4 and in some cases for 7-8 days. Antibioticotherapy and antiseptic solution should be used to wet the tampon. It should be remembered that the drainage of the auditory tube is impaired in posterior tamponade and inflammation of the auditory tube and the middle ear can develop.

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

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

DISEASES OF THE PHARYNX

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

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

The exogenic factor attacks the tonsillar mucosa via airborne and alimentary route, and also by direct contact. Three main forms of the development of common acute tonsillitis are distinguished: (1) ocassional acute tonsillitis manifested as auto-infection due to impaired environmental conditions, often as a result of chilling; (2) epidemic form arising as a result of infection from a tonsillitis patient; (3) exacerbation of chronic tonsillitis.

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

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 subfebril; insignificant inflammatory changes in the peripheral blood are found. Pharyngoscopy reveals diffuse hyperaemia of the tonsils and the margins of the palatine arches; the tonsils are somewhat enlarged. The regional lymph nodes are often slightly enlarged. The clinical signs are more pronounced in children. The disease usually lasts 3-5 days. We must differentiate this form with ARVI.

Follicular Tonsillitis. The disease usually begins with elevation of temperature to 38-39° C. The patient feels strong pain during swallowing. The pain radiates into the ear; salivation is often increased. More severe symptoms can develop in children: febrile temperature is often associated with vomiting; signs of meningism develop. The changes in the blood are often pronounced: neutrophilic leucocytes count from 12000 to 15000; moderate shift to the left and eosinophilia are observed; ESR is

often 30-40 mm/h; traces of protein are found in the urine. As a rule, the regional lymph nodes are enlarged; their palpation is painful.

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

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

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

Table of Distinctive Symptoms of Diphtheria and Lacunar Tonsillitis

Symptoms	Tonsillitis	Diphtheria
Swollen tonsils	Less marked than in diphtheria, frequently it is bilateral	More severe, accompanied by oedema of the palate arches, uvula and soft palate. May be unilateral
Patches	Spread within free areas	Extend beyond tonsils to palate arches, soft palate and posterior pharyngeal wall
Colour of patches	Yellowish	White, grey -white, dirty-grey
Adherence of patches	Patches superficial and peel off easily	Patches deep, with necrosis of mucous; in typical cases strip off with difficulty to leave a bleeding surface
Pain on swallowing	Sharp	Not always marked
Regional lymphnodes	Swollen, individual nodes easily palpated and extremely tender	Markedly swollen nodes on both sides from early days of disease, oedema of subcutaneous tissue; flattened out contours of neck
Constitutional disturbance	Less severe than in diphtheria	Increasingly severe in toxic form
Fever	Within 39-40 °C	From subfebrile to 40°C; more stable
Bacteriological examination	Negative (for Loeffler's bacilli)	Positive in most cases

Treatment. Rational treatment includes sparing conditions, local and general therapy. The patient must remain in bed during the first days of the disease and then abstain from physical work. The patient should be separated from the others; he should use separate dishes and other objects. In very severe cases the patient should be hospitalized. Food should be nutritious, rich in vitamins, soft, and not irritating.

Treatment includes also gargling with a warm solution of sodium chloride or hydrocarbonate, furacin, potassium permanganate, calendula or camomile tea. A warming compress should be applied to the neck. Salicylates and antibacte-rial preparations should be used for general treatment.

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

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

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

Necrotic (ulcerous-necrotic) Tonsillitis of Simanovsky-Vensan. 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 underlined 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 leukocyte count moderately increases. The regional lymph nodes are enlarged on the involved side; they are moderately painful to palpation. Swallowing is usually painless. The disease lasts 1 to 3 weeks but can in some cases persist for several months.

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

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

COMPLICATIONS OF TONSILLITIS

Peritonsillitis or Quinsy (fig. 94)**.** Inflammation of the peritonsillar cellular tissue arises due to virulent infection spreading, usually from the palatine tonsil to the peritonsillar cellular tissue in the presence of predisposing local or general factors. In most cases, peritonsillitis is a complication of acute tonsillitis, foreign body or odontogenic aetiology.

Symptoms. The development of the process has three stage: the oedema-infiltrative, purulent and reconvalescent stages. The process is usually unilateral. Tonsillogenic peritonsillitis occur several days

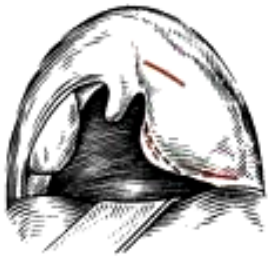

Fig.94. Left side peritonsillitis

following a recurrent exacerbation of chronic or acute tonsillitis. A peritonsillar abscess can be found in the anterior or antero-superior (supratonsillar) part, between the tonsillar capsule and the upper part of the anterior palatine arch. The supratonsillar location of the abscess is most common. Posterior peritonsillitis (developing between the tonsil and the posterior arch) may cause oedema of the larynx. Peritonsillitis can also be inferior, with location of the focus between the inferior pole of the tonsil and the lateral pharyngeal wall, or lateral, occurring between the middle portion of the tonsil and the lateral wall of the pharynx. Lateral abscess runs the most severe course because of difficult spontaneous drainage.

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

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

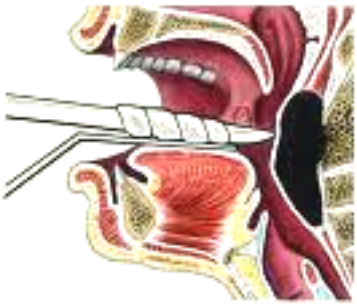

Fig.95. Retropharyngeal abscess

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

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

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

Treatment should be conservative until the abscess develops. Antibiotics and sulpha drugs are prescribed. When an abscess develops, it should immediately be opened (fig. 95); measures should be taken to prevent aspiration of pus. This can be attained by preliminary suction of pus during puncture; the abscess should preferably be opened on a half-lying patient.

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

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

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

AFFECTIONS OF THE PHARYNX IN SYSTEMIC DISEASES

Infectious Mononucleosis. This infectious disease is probably caused by a special lymphotropic virus which occurs together with *Listerella* genus. It is believed that infection occurs by air-borne droplets or by contact; the nasal cavity and the pharynx are the portals of infection. Children and the young usually develop mononucleosis. The disease is characterized by a fever, tonsillitis-like changes in the fauces, adenosplenomegaly, and changes in the blood (high counts of leukocytes and atypical monocytes). The incubation period lasts 4-5 days (sometimes 10 days). At the onset of the disease the body temperature rises to 38-40° C and persists at this level from 5 days to 2-4 weeks (for longer periods in rare cases). The symptoms are sometimes alleviated periodically during this stage. An early and permanent sign of the disease is enlarged lymph nodes, first on the neck and then in the groin, armpits, and the abdomen. The spleen and the liver are also enlarged in most patients. Changes in the fauces usually follow the enlargement of the lymph nodes; they are similar to those occurring in catarrhal, lacunar, fibrinous, and less frequently necrotic tonsillitis.

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

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

Agranulocytosis (Agranulocytic Angina). Affection of the tonsils is the specific symptom of this disease. Agranulocytosis is considered not as an independent nosological disease but as a response of the haemopoetic system to various pathological factors (such as infection, toxicosis, radiant energy) or as a result of altered haemopoesis 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 activating the haemopoetic system and controlling secondary infection. Exception 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 haemorrhages 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 haemorrhage 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 symptoms, 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 antiseptics. Intramuscular antibiotics injections have been used with success.

Control tests

1. In a week after angina a patient suddenly developed one-sided pain in the region of the neck and throat, trismus of the masticatory musculature, infiltration of the soft tissues behind the angle of the mandible, high temperature. What are the pathognomonic clinical symptoms of parapharyngeal abscess?

A. hyperemia of the mucous membrane of the posterior wall of the pharynx with suppurated follicles

B. protrusion, hyperemia of the posterior wall of the pharynx, difficult breathing, swallowing, nasality

+C. swelling, protrusion of the lateral wall of the pharynx and external surface of the neck

D. contraction of opening of the pharynx, difficulty of swallowing, enlarged lymph nodes

E. sharp hyperemia, infiltration, one-sided protrusion of tonsil with the arches

2. A patient of 38 years old, an X-ray technician, complains of a severe sore throat, which is intensified in swallowing, high temperature, pain in the extremities, general malaise. The disease began sharply 2 days ago. The mucous membrane of the pharynx is moderately hyperemic on pharyngoscopy, tonsils are covered with dull gray necrotic coatings. The significant decrease of a quantity of granulocytes is determined in the blood analyses. What is the diagnosis?

A. lacunar angina

B. diphtheria of the pharynx
+C. agranulocytic angina
D. angina of Simanovsky-Vensan
E. infection mononucleosis

3. A patient has been experiencing a sore throat, subfebrile body temperature, general malaise for 3 days. The grayish-white coatings on the palatine tonsils involving the arches are determined on pharyngoscopy. The coatings are removed with difficulty, in this case erosive surface is bared. What is a suppositional diagnosis?

+A. diphtheria of the throat
B. lacunar angina
C. pharyngomycosis
D. angina of Simanovsky-Vensan
E. infection mononucleosis

4. During professional examination a patient is revealed to have cone-shaped whitish "plugs" on the surface of the palatine and lingual tonsils as well as on the posterior wall of the pharynx, which are raised above the surface mucosa, and are removed with difficulty. What is the suppositional diagnosis?

A. chronic hypertrophic pharyngitis
+B. leptotrichosis
C. acute pharyngitis
D. follicular angina
E. follicular angina

5. A patient developed peritonsillar abscess after angina for the first time. On pharyngoscopy displacement of the tonsils to the central line, hyperemia are determined, infiltration of the anterior palatine arch to the right, there is yellowish cleared up area and maximum protrusion in its anterosuperior part. Where is the most rational place of opening of peritonsillar abscess in this case?

A. before the posterior palatine arch
B. through the supratonsillar fossa
C. on the side of the lower pole of the tonsils
+D. at the place of the greatest protrusion
E. through the infratonsillar fossa

6. In acute paratonsillar abscess it is performed:

A. opening of the abscess
B. tonsillectomy
C. antibiotic therapy
D. diuretics
+E. all of the enumerated

7. A patient with the sequential exacerbation of relapsing peritonsillitis was admitted to clinic. He is ill the third day. What is rational tactics of treatment?

A. conservative therapy
B. opening of the peritonsillar abscess
+C. abscesstonsillectomy
D. cryotonsillotomy
E. administration of physiotherapy

8. Clinical symptoms of the retropharyngeal abscess:

A. hyperemia of the mucous membrane of the posterior wall of the pharynx with suppurated follicles
+B. protrusion, hyperemia of the posterior wall of the pharynx, difficult breathing, swallowing, nasality
C. swelling, protrusion of the lateral pharyngeal wall and external surface of the neck
D. contraction of opening of the pharynx, difficulty of swallowing, enlarged lymph nodes
E. sharp hyperemia, infiltration, one-sided protrusion of tonsil with the arches

9. A patient of 17 years old complains of a bad sore throat, which is intensified in swallowing, high body temperature, general malaise, headache, absence of appetite. He fell ill 3 days ago after overcooling. Objectively: The pulse is 88 beats per min, rhythmic. Bright hyperemia of the mucous membrane of the palatine arches, tonsils are revealed. There are white coatings on the surface of the tonsils, which are easily removed. Deep neck lymph nodes are enlarged, painful to palpation. What is diagnosis?

A. catarrhal angina
B. follicular angina
+C. lacunar angina
D. ulceromembranous angina
E. necrotic angina

10. A patient complains of a bad pain in swallowing, irradiating into both ears, pain in the joints, high temperature. On pharyngoscopy the tongue is covered with a coating, the palatine tonsils are enlarged, hyperemic, there are yellowish-white points in the submucous layer. Regional lymph nodes are enlarged, painful to palpation. What is the diagnosis?

A. acute pharyngitis
B. catarrhal angina
+C. follicular angina
D. lacunar angina
E. both side peritonsillar abscesses

11. A patient complains of a sore throat at rest and in swallowing, general malaise, the body temperature is up to 37.8°C. He fell ill three days ago after overcooling. He was treated by gargling of the throat, aspirin. In examination: insignificant hyperemia of the mucous membrane of the nasal cavity.The mucous membrane of the uvula is hyperemic, infiltrated. Light hyperemia of the mucous membrane of the arches and tonsils. Neck lymph nodes are enlarged, painful to palpation. On laryngoscopy, which is sharply difficult to perform because of the tenderness in putting out of the tongue, hyperemia, infiltration of the lingual tonsils, coatings in the form of white islets on its surface are determined. The mucous membrane of the larynx is not changed, the vocal chords are white. How is to treat the patient?

A. antibacterial treatment
B. desintoxication treatment
C. diuretics
D. inhalation of antibiotics, corticosteroids
+E. all enumerated

12. A patient complains of a headache, an increase in the temperature to 40°C. On examination hyperemia of the pharynx mucous membrane is determined, tonsils are enlarged, friable. The cervical, axillary, inguinal lymph nodes are enlarged, painful to palpation. The liver and spleen is moderately enlarged, painful to deep palpation. In blood analysis - leukocytosis with the prevalence of mononuclear leukocytes and virocyts. What is the diagnosis?

A. catarrhal angina, cervical lymphadenitis
+B. mononucleosis
C. agranulocytic angina
D. ARVI
E. tonsillogenic sepsis

13. A patient complains of difficult opening of the mouth, impossibility to swallow the food because of the intensive pain in left half of the throat, general malaise, high body temperature. He is ill the fourth day. He was unsuccessfully treated at home (Aspirin and Analgin per os, gargling of the throat by the extract of sage). On examination enlarged and painful lymph nodes are determined in the left retromandibular region. The trismus of masticatory muscles presents. The mucous membrane of the oropharynx is hyperemic, the palatine tonsilla is displaced to the centre, infiltration is determined, which involves the anterior arch and part of the soft palate. What is the diagnosis?

+A. peritonsillar abscess
B. retropharyngeal abscess

C. parapharyngeal abscess
D. intratonsillar abscess
E. tumor of the palatine tonsilla

14. A patient complains of a mild sore throat on the left. Objectively: the general state is not disturbed, the body temperature is 36.6°C. There are no changes in the internal organs. A greyish coating is determined at the upper pole of the left tonsil, after removal of which sufficiently deep ulcer with the uneven bottom is found. Other ENT organs are normal. What is a probable diagnosis?

+A. ulceromembranous angina of Simanovsky-Vensan
B. lacunar angina
C. syphilitic angina
D. cancer of the palatine tonsil
E. tuberculosis angina

15. A mother complains of uneasiness of her eight-month baby, cry, sleep disorder, refusal of food, difficult nasal breathing, high body temperature during four days. 10 days ago it had acute respiratory infection. The difficulty of nasal breathing developed against the background of high temperature. The state of the child is of moderate severity, the body temperature is 38.0°C, the skin is pale. Respiration is with a snoring sound. Submaxillar lymph nodes are enlarged, painful to palpation on the right. The mucous membrane of the posterior wall of the pahrynx is hyperemic, protruded on right half. What is the diagnosis?

A. peritonsillar abscess
+B. retropharyngeal abscess
C. parapharyngeal abscess
D. phlegmon of the neck
E. mononucleasis

16. A patient complains of a several pain in swallowing, headache, general weakness, pain in the knee joints, increase in the body temperature up to 38°C. He is ill the second day, he associates the disease with overcooling. The general state is satisfactory. The appetite is reduced. The pulse is 102 beats per minute, of satisfactory filling. Pharyngoscopy: the mucous membrane of the pharynx is hyperemic with white coatings on the surface of both tonsils. Neck lymph nodes are enlarged, painless to palpation. What is the diagnosis?

+A. lacunar angina
B. follicular angina
C. catarrhal angina
D. ulceromembranous angina
E. acute pharyngitis

17. What day from the onset of the disease in peritonsillar abscess is its opening performed?

A. on the first day
+B. on the third- fifth day
C. on the second day
D. on the seventh day
E. in 14 days

18. A patient presents complaints on intensive pain in the throat on the right, profuse salivation, impossibility to swallow even liquid. His voice is nasal. He has been ill for seven days, was treated in polyclinic for lacunary angina; substantial improvement began, but the day before the body temperature increased to 38°C and gradually developed the phenomena described above. On examination the mouth is opened with difficulty; the fauces is narrowed due to the significant bulging of the right half of the palatine arch and tonsil and displacement of the edematic uvula to the left. Infiltration is extended along lateral wall to the right pear-shaped sinus. In the right retromandibular region the painful infiltration and lymph nodes is palpated. What is the diagnosis?

A. right-sided peritonsillar abscess
B. phlegmon of the neck

+C. right-sided peritonsillar abscess, parapharygeal abscess
D. lacunary angina
E. parapharygeal abscess

19. A patient complains of severe one-sided sore throat, intensified in swallowing, increase in the body temperature up to 39.0°C, general malaise. What symptom does not correspond to the clinical picture of paratonsillar abscess?

A. sharp hyperemia, infiltration, one-sided protrusion of the tonsil with the arches, limitation of opening of the mouth
+B. protrusion and edema of the tonsils, covered with a white-grey coating, coming beyond the arches, absence of trismus
C. unpleasant smell from the mouth
D. enlargement of the lymph nodes of the neck
E. high temperature, nasal voice

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 favourable 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 convenient 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, he adache, decreased working capacity, the temperature is often subfebrile. For many patients, sore throat in the anamnesis is the only complaint.

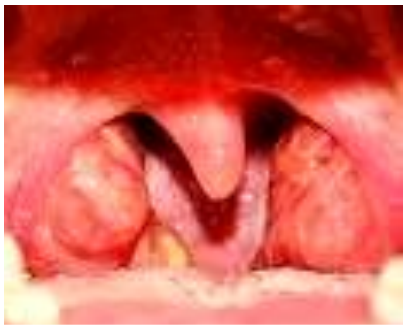

Fig.96. Chronic tonsillitis

Inspection of the tonsils (fig.96) 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.

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 tonsillar function in form of residual tonsillitis, paratonsillitis, peritonsillar abscess, different pathological reactions, diseases of other organs and systems. In the formulation of diagnosis in decompensation condition precise form of decompensation are indicated. Examples of formulating diagnosis: chronic tonsillitis, compensatory type; chronic tonsillitis, decompensatory type (residual tonsillitis, rheumatism).

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

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

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

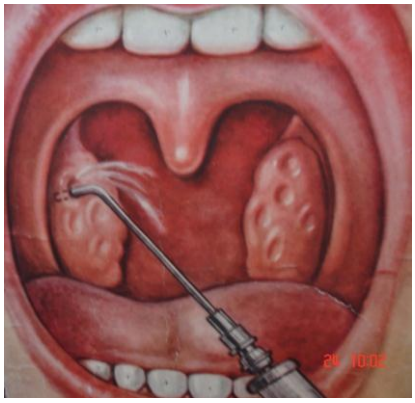

Fig.97. Irrigation of the lacunas

The toxico-allergic form should be treated surgically, but the first degree of this disease can also be treated conservatively (1-2 courses). If treatment is not sufficiently effective, tonsillectomy is indicated. Toxico-allergic symptoms of the second degree are direct indications for tonsillectomy. If this operation is contraindicated (e.g. in the presence of haemophilia), cryotherapy with liquid nitrogen should be recommended. In 1972 in ENT department professor V.D.Dragomiretsky practically introduced cryosurgical method of treatment of chronic tonsillitis by using autonomic cryoapparatus. Clinical and immunological investigation shoved that extreme cold not only leads to remove pathological changes of parts of palatine tonsils but have stimulating effect on organism of type tissue therapy of Academic V.P.Filatov. It has hyposensibilising action and possesses immune-regulator property. Cryoaction doesn't accompany general and local reaction of organism and these gives us to using cryosurgical method ambulatory to the patient to whom surgical method are contraindicated with high degree of risk. Cryosurgical method has the following advantages: cryodestruction is less painful and in most cases is performed without anaesthesia; there is no blood loss and method is useful 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 (fig. 97) with various antiseptic solutions (furacin, boric acid, ethacridine lactate, potassium permanganate) and also mineral alkaline water, peloidin and interferon is effective. A special syringe with a long curved cannula is used for the purpose.

Among physiotherapeutic methods are UV rays, electromagnetic UHF and SHF oscillations, and ultrasound.

Indications for tonsillectomy are the following:

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

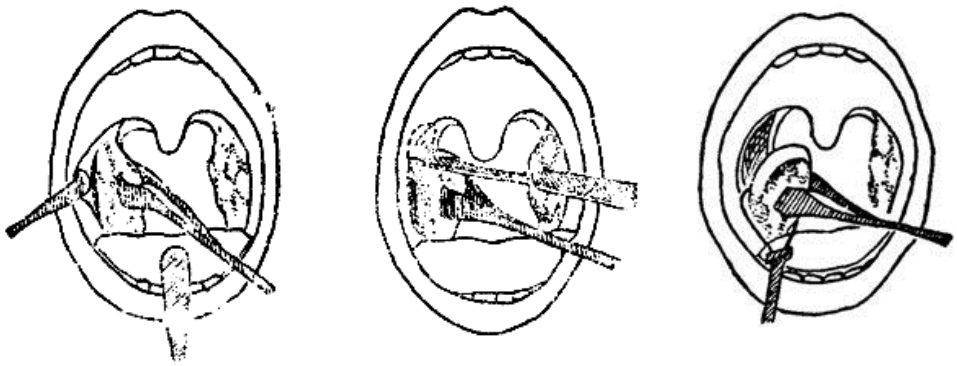

Fig.98. Tonsillectomy

Tonsillectomy (fig. 98) 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 syn drome) 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 Ketgut 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 unfavourable environmental conditions. Acute tonsillitis is often preceded by local or general chilling. Hence the importance of general and local hardening of the body: regular exercises and sports, air baths, and sponging with water (with gradually lowering temperature). But all these measures should be taken gradually and regularly.

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

HYPERTROPHY OF LYMPHOID TISSUE OF THE PHARYNX

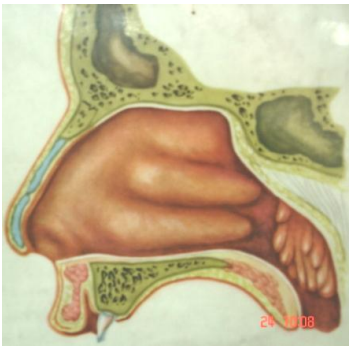

Fig.99. Pharyngeal tonsil hypertrophy

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

Hypertrophy of the pharyngeal tonsil (adenoids) (fig. 99)**.** Adenoids usually grow at the age from 3 to 15, but they also occur in younger patients and in adults.

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

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

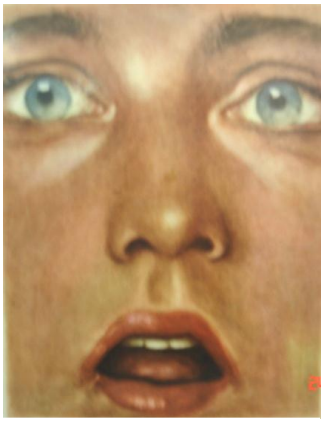

Fig.100. Adenoid face

Three degrees of adenoid growth are distinguished: degree I-adenoids cover to one third of the vomer; degree II-about half of the vomer is covered; degree III-the vomer is covered to two thirds or almost completely. Degree I adenoids do not impair significantly respiration through the nose in child. If a child is ill for a long time, the face bones become distorted: the dropping jaw becomes narrow and long, while the hard palate undergoes malformations: it becomes high and narrow; incorrectly growing teeth cause malocclusion. These changes give a specific dull expression to the face of children with adenoid growths (fig. 100).

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

Treatment of adenoids is commonly surgical. Conser vative treatment is helpful only if hypertrophy is insignificant or there are contraindications for the operation. Anti histaminics and Calcium Gluconate help in some cases.

The surgical removal of the adenoids (adenoidectomy) is performed in cases where the enlarged tonsil impedes respiration through the nose. The operation is usually performed at the age from 5 to 7, but infants and adults can also be operated on if nasal breathing is pathologically impeded, the hearing function is impaired, or other diseases concur. Children can be operated under out-patient conditions, while adults only in hospital. Children should not take breakfast on the day of the operation. The operation can be performed either without anaesthesia, or after instilling 5 drops of a 10 per cent Lidocaine solution into each side of the nose. Contraindications for adenoidectomy are diseases of the blood, severe diseases of the cardiovascular system, and infectious diseases (the patient may be operated on only in 1-2 months after the disease). An important pre-operative measure is immobilization of the child. The nurse sits on a stool or in a surgical chair facing the surgeon and holds the child in her laps so that his legs are fixed between the nurse's knees; the right arm is used to hold the child's arms and the trunk, while the left arm holds the child's head. A sterile cloth should cover both the nurse and the child The operated child is placed on his side in bed on a low pillow for 25-30 minutes. Hot or spicy foods should not be given, since bleeding can resume for 4-5 days. Physical strain, active movements and chilling should be avoided.

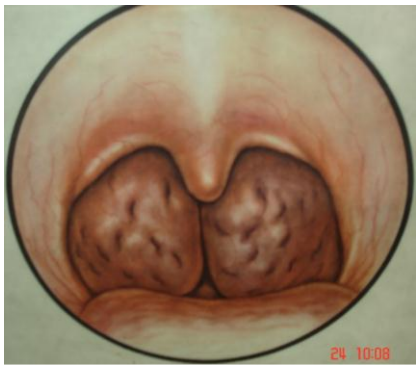

Fig.101. Hypertrophy of the palatine tonsils

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 (fig.101) 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 tonsillitis, 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.

Acute and Chronic Pharyngitis

Acute Inflammation of the Nasopharynx. Acute rhinopharyngitis is in most cases secondary to inflammation of the inferior portion of the pharynx and the nasal cavity which is associated with acute or exacerbated chronic pharyngitis, rhinitis or sinusitis.

The etiological factors of acute rhinopharyngitis are the same as of acute rhinitis. The disease is common in infants and children, especially in the presence of adenoid growths in asthenic children with various forms of diathesis. Children's acute infections (measles, scarlet fever, etc.) often promote the onset of acute nasopharyngitis. Various cocci, e.g. streptococci, staphybcocci, diplococci, pneu-mococci, and other microflora found in the nasopharynx in a non-pathogenic form, are the causative factors of the disease.

Symptoms include discomfort in the nasopharynx (burning, dryness, tickling), accumulation of mucous secretion which is sometimes stained with blood and drains with difficulty from the nasopharynx. Headache and pain in the occipital region are frequent. Respiration through the nose is impeded; the voice becomes nasal, especially in children. If the inflammation extends to the mucosa of the auditory tubes, the patient complains of clicking and pain in the ears, and impaired hearing (defective conduction).

Nasopharyngitis in adults is usually apyretic, while in children the body temperature can significantly rise, especially if the inflammation extends to the larynx and the trachea, as in acute respiratory infection.

Acute non-specific rhinopharyngitis differs from diphtheria by the absence of fibrinous exudate. In dubious cases, the discharge should be tested for Corynehactenum diphtheritic. The disease lasts from a few days to two weeks.

Treatment is the same as in acute rhinitis. Instillation of a 2 per cent (5 per cent for adults) solution of protein silver or colloid silver, 5 drops 3 times a day into each nostril, is effective. If inflammation is marked, Polidexa should be instilled into the nose for a few days; this should be followed by instillation of vasoconstrictors. Acetylsalicylic acid and antibactcrial preparations are recommended for elevated temperature. Vitamins and physiotherapy (UV rays on the soles and also UHF on the nose) are indicated.

Acute Inflammation of the Oropharynx. Acute inflammation of the middle portion of the pharynx alone occurs in rare cases. More frequently it occurs as a descending acute inflammation from the nasal cavity and the nasopharynx, or in combination with inflammation of the mouth cavity and the tonsils.

Symptoms of the disease are dry and sore throat, and hyperaemic mucosa. Pain intensifies and can radiate to the ear when the swallowing act is imitated. A mucopurulent discharge can be seen on the

posterior wall of the pharynx. Hyperaemia and swelling extend from the posterior wall of the pharynx onto the posterior palatine arches and the uvula. Marked forms of acute pharyngitis are associated with regional lymphadenitis and elevated temperature (in children). Complications of acute pharyngitis are the same as in rhinopharyngitis. The disease lasts up to two weeks.

Treatment. Coarse or irritating food should be excluded. Inhalations and spraying of antiseptics and antibacterial solutions are prescribed. In the presence of elevated temperature, antibacterial preparations and acetylsalicylic acid are indicated per os. The disease has the tendency to convert into its chronic form. The acute inflammation should therefore be thoroughly treated.

Chronic Pharyngitis. The disease is classified as chronic catarrbal, hypertrophic (granular and lateral) and chronic atrophic pharyngitis.

Chronic pharyngitis is a common disease of the pharynx. It occurs in children mostly in its simple and hypertrophic forms. Middle-aged and aged persons often develop chronic pharyngitis. The incidence is higher in men (the hypertrophic form), while in women it occurs mostly in the atrophic form.

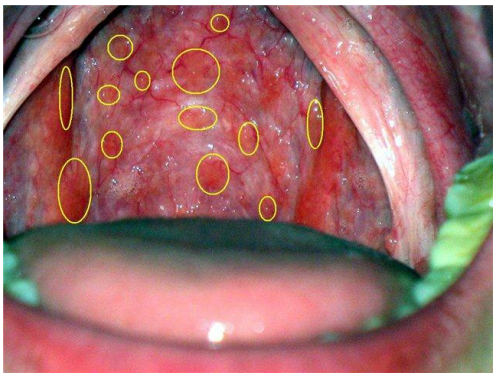

Fig.102. Chronic hypertrophic pharyngitis

The etiology of chronic pharyngitis is determined in most cases by local irritation of the pharyngeal mucosa, which is usually persistent. The hypertrophic form (fig.102) of pharyngitis is characterized by thickening and edema loosening of the connective-tissue stroma of the mucosa proper and of the submucous layer. Serous exudate which fills the mucosa is gradually organized and replaced by cell elements. The mucosa becomes thicker and firmer. The lymphoid granules that are hardly seen on normal mucosa thicken and broaden significantly and often fuse into larger formations. Hypertrophy affects mostly the mucosa of the posterior wall of the pharynx (granular pharyngitis) or the lateral parts of the pharynx (lateral hypertrophic pharyngitis).

In atrophic chronic pharyngitis the mucosa is thin; its lymphoid tissue and the submucous layer are partly replaced by connective tissue. The number of mucous glands and their size decrease. The number of vessels decreases too. The walls of the vessels are often thick while their lumen narrow; the vessels are sometimes obliterated. The mucosa is covered with epithelium which is mostly keratinized and desquamated at places.

The symptoms of the simple and hypertrophic forms of catarrhal pharyngitis are sore throat and tickling in the throat, which provokes slight hacking coughing with expectoration and swallowing of the accumulated discharge, especially in the morning. These symptoms are more pronounced in hypertrophic pharyngitis. The patient sometimes complains of stuffed ears (the symptom subsides after several swallowing movements). The main complaint in atrophic pharyngitis is dry throat, especially during long conversation, difficult swallowing, especially during imitation of the swallowing act, and unpleasant breath. It should be noted that the complaints often do not agree with the objective pharyngoscopic picture: no complaints while the changes in the pharyngeal mucosa are significant, and vice versa, complaints may be numerous in the presence of only slight objective changes in the throat.

Simple chronic catarrhal pharyngitis is characterized pharyngoscopically by hyperemia, mild edema and thickening of the pharyngeal mucosa, the presence of clear or cloudy mucus on the posterior wall of the pharynx.

Treatment should first of all be directed at elimination of the general and local causes of the disease (chronic purulent foci in the nose and the paranasal sinuses, in the tonsils, etc.). Possible irritating factors such as smoking, exposure to dust or harmful gases, inhalation of polluted air, spicy foods and others should be eliminated. Systemic chronic diseases that promote the development of pharyngitis should be treated properly.

Local treatment includes removal of mucus and crusts by irrigating the pharyngeal mucosa. Inhalation of medicinal aerosols, surface application and instillation of drops into the nose are also prescribed.

Hypertrophic forms of pharyngitis should be treated by gargling the throat with a 0.5-2 per cent warm solution of sodium hydrocarbonate and a 1 per cent sodium chloride solution. These solutions are also helpful when inhaled in the atomized form. Application of a 5-10 per cent tannin emulsion in glycerol, 1-2 per cent silver nitrate solution, 3-5 per cent (sometimes 10 per cent) solution of protein silver or colloid silver lessens edema of the pharyngeal mucosa. Gargling with tea or sage decoction is also recommended. Large granules should be belter removed by using extreme cold; cauterization of these lymphoid formations with a concentrated (30-40 per cent) silver nitrate solution (or with the solid substance) is also recommended. Ultrasound (phonophoresis) also produces a good therapeutic effect on the pharyngeal mucosa.

Treatment of atrophic pharyngitis includes daily irrigation of the pharynx to remove the mucopurulent discharge and crusts from its surface. An isotonic or a 1 per cent sodium chloride solution containing 4-5 drops of a 10 per cent iodine tincture per 200 ml of the liquid should be used for the purpose. Regular and prolonged irrigation of the pharynx with this solution removes irritation of the mucosa, and alleviates the unpleasant symptoms of pharyngitis. The posterior wall of the pharynx should be treated with Lugol's solution (with glycerol). Ten-day courses should be repeated at intervals. The composition of the solution is as follows: iodine tincture 0.1 g; potassium iodide, 0.2 g; glycerol 10.0 ml. The posterior pharyngeal wall should be treated once a day. A 30 per cent potassium iodide solution is prescribed: 8 drops 3 times a day per os. The patient should first be tested for sensitivity to iodine. Concentrated vitamin A, E should be given.

Pronounced symptoms of chronic pharyngitis such as paraesthesia, burning, dryness, etc, can be removed with Novocain block into the lateral parts of the posterior wall of the pharynx; Novocain block is often combined with aloe injections.

Control tests

1. A patient suffers from recurrent anginas, which are accompanied by the tonsillogenic intoxication, exacerbation of rheumatism. On pharyngoscopy the palatine tonsils are of I degree, there are purulent plugs in the lacunas, the edges of the palatine arches are infiltrated, cicatricial adhered to the tonsils. The regional lymph nodes are enlarged on the neck on both sides. What is diagnosis?

+A. chronic decompensated tonsillitis
B. chronic compensated tonsillitis
C. lacunary angina
D. leptotrichosis
E. hypertrophy of palatine tonsils

2. A patient complains of frequent anginas with a high temperature in the last 8 years, sometimes 2-3 times per year. After last angina he had pains in the heart, subfebrile temperature persisted approximately one month. In pharyngoscopy: hyperemia, thickening of the edges of the palatine arches, the palatine tonsils are of I degree, friable, adhered to the arches, there is purulent plugs in the lacunas. The regional cervical lymph nodes are enlarged. What is the diagnosis?

A. chronic compensated tonsillitis
B. chronic subcompensated tonsillitis
C. catarrhal angina
D. lacunar angina, cervical lymphadenitis
+E. chronic decompensated tonsillitis

3. A patient complains of unpleasant smell in the mouth, periodic insignificant sore throat, general fatigue. On pharyngoscopy: the palatine tonsils are of II degree, friable, there is liquid pus in the lacunas, symptoms of chronic tonsillitis of Zak, Gieze, Preobrazhenskiy are positive. What is a therapeutic tactics?

+A. conservative therapy, cryotonsillotomy
B. dissection of the lacunas of the tonsils
C. tonsillotomy
D. tonsillectomy
E. only physiotherapy

4. A patient suffers from frequent anginas, the last one was complicated by peritonsillar abscess. In the last year he complains of periodic pain in the heart. The objective signs of chronic tonsillitis are expressed on pharyngoscopy. How can you treat the patient?

A. conservative therapy
B. cryotonsillotomy
C. tonsillotomy
+D. tonsillectomy
E. dissection of the lacunas of the tonsils

5. A patient complains of a constant sore throat, subfebrile temperature, general weakness, poor sleep, pains in the joints. Conservative treatment is ineffective. On examination: the tongue is covered with a whitish coating, the tonsils are atrophic, adhered to the arches, there is pus in the lacunas, deep cervical lymph nodes are enlarged. What is the diagnosis?

A. chronic tonsillitis, simple form
B. chronic tonsillitis, toxicoallergic form, first stage
+C. chronic tonsillitis, toxicoallergic form, second stage
D. lacunar angina
E. hypotrophy of palatine tonsils

6. A patient was admitted to hospital with the diagnosis of tonsillogenic sepsis. On examination the diagnosis was confirmed. What form of treatment is indicated?

A. only conservative therapy
B. tonsillectomy
+C. tonsillectomy and conservative therapy
D. tonsillotomy and conservative therapy
E. cryotonsillotomy and conservative therapy

7. A child of 4 years old is frequently ill with respiratory diseases. A notable enlargement of the palatine tonsils is determined in the throat, which are practically closed across the central line. There are no hyperemia of the mucosa, coatings and purulent plugs in the tonsils. What is the diagnosis?

A. chronic compensated tonsillitis
+B. hypertrophy of the palatine tonsils
C. chronic decompensated tonsillitis
D. double-sided peritonsillitis
E. chronic tonsillitis, simple form

8. A child of 5 years old breathes through the nose, he is frequently ill with respiratory diseases, he sleeps badly, yells in the sleep, night enuresis is noted. Objectively: the skin is pale, half-open mouth, smoothness of the nasolabial folds, high Gothic sky. What is the assumed diagnosis?

+A. hypertrophy of the adenoid tissue
B. chronic adenoiditis
C. acute rhinitis
D. chronic allergic rhinitis
E. hypertrophy of palatine tonsils

9. A patient complains of frequent angina. He does not present complaints between anginas. On pharyngoscopy there is determined injection of the vessels along the edges of the palatine arches, tonsils are friable, with purulent plugs in the lacunas. The cervical lymph nodes are enlarged. Internal organs are without any peculiarities. What is diagnosis?

A. chronic tonsillitis, simple form
+B. chronic tonsillitis, toxicoallergic form, first stage
C. chronic tonsillitis, toxicoallergic form, second stage
D. lacunar angina
E. hypertrophy of palatine tonsils

10. What classification of chronic tonsillitis is proposed by the academician I. B. Soldatov?

+A. compensated, decompensated
B. primary, secondary
C. compensated, subcompensated, decompensated
D. mild, moderate severity, severe
E. simple form, toxicoallergic form, first stage, second stage

11. What symptoms are characteristic of the compensated form chronic tonsillitis?
+A. there are local signs of inflammation, neck lymphadenitis
B. enlargement of the tonsils
C. there are no signs of local inflammation
D. posterior wall of the pharynx is hyperemic
E. tonsillogenic intoxication

12. A patient aged 17 suffers from chronic decompensated tonsillitis. Decompensation is expressed by frequent anginas, infectious polyarthritis, pyelonephritis. What is rational method of treatment of this form of chronic tonsillitis?
A. cryotonsillotomy
+B. tonsillectomy
C. ultrasonic disintegration
D. conservative treatment
E. electro-caustics of the tonsils

13. Name the ear diseases, caused by adenoidal vegetations, except one:
A. acute middle otitis
B. chronic middle otitis
C. conductive hearing disorder
+D. sensoneural hearing disorder
E. salpingootitis

14. A patient complains of frequent anginas with a high temperature in the last 8 years, sometimes 2-3 times per year. After last angina 5 months ago he had pains in the heart, subfebrile temperature was kept for approximately one month. Objectively: there are no pathologic changes in the internal organs. Examination of the ENT organs determined hyperemia of the mucous membrane along the edge of the palatine arches. The palatine tonsils somewhat protrude over the edge of arches, they are friable, adhered to the arches, the tonsil lacunas contain purulent detrite. The upper cervical lymph nodes are consolidated. What is the diagnosis?
A. chronic compensated tonsillitis
+B. chronic decompensated tonsillitis
C. chronic pharyngitis
D. follicular angina
E. hypertrophy of palatine tonsils

15. A patient complains of frequent anginas, from which he has been suffering since childhood. At the age of 24 angina was complicated by peritonsillar abscess, in the last year the patient complains of pain in the region of the heart. Objectively: the state of the patient is satisfactory. Pulse is rhythmic, 76 beats per minute. The functional heart sound is revealed on auscultation. Other changes in the internal organs are not revealed. There is mild hyperemia of the mucous membrane of the anterior palatine arches. The tonsils are dense, cicatricial, adhered to the arches. There is liquid purulent detrite in the tonsil lacunas. The deep neck lymph nodes are consolidated, enlarged to palpation. The mucous membrane of the posterior wall of the pharynx is pink, with single enlarged granules. What symptoms of chronic decompensated tonsillitis present in patient, except one:
+A. enlarged granules of the pharynx
B. peritonsillar abscess
C. frequent anginas
D. hyperemia of the mucous membrane of the anterior palatine arches
E. enlargement of deep neck lymph nodes

16. A patient referred to the otolaryngologist with complaints on frequent anginas. On oropharyngoscopy: the tonsils are of small size, friable, two yellowish rounded formations of 1-2 mm in size are seen through the epithelial cover of the tonsils. The methods of treatment are:

A. irrigation of the lacunas of the tonsils
B. paint of the surface of the tonsils with Lugol solution
C. physical therapeutic methods
D. lacunotomy
+E. all enumerated

17. A 5-year-old child sleeps badly at night, according to his parents, breathing is noisy, mouth is always opened. Voice is nasal, he swallows solid food with difficulty, eats slowly and for a long time. Objectively: the tonsils are smooth, of large size, converge across the central line. The palatine arches are unchanged, lacunas are clean, regional lymph nodes are not enlarged. Prescribe treatment.

+A. adenotomy, tonsillotomy
B. physiotherapy
C. vitamin therapy
D. tonsillectomy
E. tonsillotomy

18. Conservative therapy of chronic adenoiditis includes everything, except:

A. irrigation out of the nasal cavity with antiseptic solutions
B. use of preparations of the local antibacterial therapy
C. use of the topical bacterial immunocorrectors
D. physiotherapy
+E. systemic antibacterial therapy

19. A patient of 46 years old complains of constant dryness in the throat, periodically discomfort becomes worse, pain develops in swallowing. Objectively: the mucous membrane of the nose and posterior wall of the pharynx is dry, its surface is smooth, shines, it is covered with viscous phlegm. What is the diagnosis?

A. acute rhinopharyngitis
+B. chronic atrophic rhinopharyngitis
C. chronic vasomotor rhinitis
D. ozena
E. chronic hypertrophic rhinopharyngitis

20. The chronic adenoiditis differs from adenoidal vegetations by:

A. duration of the disease
B. presence of the ear symptoms
+C. bacterial inflammation
D. addition of disorders of general nature
E. belonging to children with allergic reactions

21. A 8-year-old patient complains of discomfort in the throat: tickling, burning, which appeared after consumption of cold food. On examination by the ENT doctor acute pharyngitis is diagnosed. What are its clinical symptoms?

A. hyperemia and moderate infiltration of the mucous membrane of the posterior wall of the throat
B. hyperemia of the mucous membrane of the pharynx, the lymphoid granules are visible on its posterior wall
C. posterior wall of the pharynx is covered with viscous mucous secretion
D. protrusion, hyperemia of the posterior wall of the pharynx
E. positive symptoms of Gieze and Zak

22. Which is not related to complications of acute tonsillitis:

A. tonsillogenic sepsis
B. retropharyngeal abscess
+C. anaphylactic shock
D. purulent lymphadenitis
E. tonsillogenic mediastinitis

23. A patient referred with complaints on tickling, a feeling of a foreign body in the throat, pain in "empty" swallow. A similar state has been disturbing periodically for 10-12 years, the temperature is normal, the general state of the organism is satisfactory. Hyperemia of the mucous membrane of the pharynx is noted on pharyngoscopy, the lymphoid granules are visible on its posterior wall, the palatine tonsils are of II degree, pink, without pathologic contents in the lacunas. What is the diagnosis?

A. chronic catarrhal pharyngitis
B. acute pharyngitis
+C. chronic hypertrophic pharyngitis
D. chronic compensated tonsillitis
E. chronic sub-atrophic pharyngitis

24. A 5-year-old child was made a diagnosis: the adenoidal vegetations of III degree. There are no signs of chronic adenoiditis. Name the leading symptom in this form of pathology, except:

A. difficulty of nasal breathing
+B. muco-purulent discharged from the nose
C. recurrent acute middle otitis
D. night enuresis
E. closed nasality

25. A child of 3 years old is frequently ill with respiratory diseases. According to his mother, during sleep in the lying position his nasal and oral breathing is disturbed, there is snoring, agitated sleep. The otolaryngologist made a diagnosis of hypertrophy of the tonsils. What is a clinical picture of the disease?

A. enlarged tonsils with purulent contents in the lacunas on pharyngoscopy
+B. enlarged tonsils, which are practically closed in the central line, there is no hyperemia of the mucosa, there are no coatings and purulent plugs in the tonsils
C. enlarged tonsils, positive symptom of Zak on pharyngoscopy
D. enlarged tonsils, positive symptom of Gieze on pharyngoscopy
E. enlarged tonsils, which are covered with purulent or fibrinous coatings

26. A 4-year-old child is made a diagnosis: adenoidal vegetations of III degree. There are no signs of chronic adenoiditis. Name the consequences, which may be associated with this pathology, except:

A. allergic rhinitis
+B. atrophic rhinopharyngitis
C. chronic hypertrophic pharyngitis
D. recurrent middle otitis
E. open nasality

27. The causes for development of chronic tonsillitis include the following, except:

A. infectious agent (more frequent β- hemolytic streptococcus)
B. allergization and autosensitization to the bacterial and tissue antigens
C. reduction in the reactivity of the organism
D. virulence of the microflora
+E. realization of the factors of specific and nonspecific protection of the mucous membranes of the upper respiratory tract

28. What data are characteristic of the compensated stage of chronic tonsillitis:

A. recurrent acute tonsillitis in the anamnesis; the tonsils are of the middle size, liquid pus in the lacunas; the band-shaped thickening of the anterior and posterior palatine arches;
B. 3 degree of hypertrophy of the palatine tonsils; the mucous membrane of the tonsils is of the pink colour;
C. frequent angina in the anamnesis, periodic pain in the region of the heart, joints, rapid fatigue. On pharyngoscopy: the tonsils are of 2 degrees of hypertrophy, the symptoms of Zak, Gieze, Preobrazhenskiy are positive;
+D. tonsils of 1 degree of hypertrophy, cicatricial adhesions between the tonsils and arches are noted;
E. on pharyngoscopy: tonsils of the middle size, liquid pus in the lacunas, Preobrazhenskiy's symptom is positive, the bands of Koritskiy are palpated. In the anamnesis: angina with a frequency of 1-2 times per year, with sharply pronounced intoxication. He is ill with rheumatism.

29. In what cases the conservative treatment of chronic tonsillitis is not indicated?

A. recurrent acute tonsillitis (angina) in the anamnesis; on pharyngoscopy: the tonsils are of the middle size, liquid pus in the lacunas; the band-shaped thickening of the front and posterior palatine arches is noted;

B. on pharyngoscopy: 3 degree of hypertrophy of the palatine tonsils; the mucous membrane of the tonsils is of the pink colour;

C. frequent angina in the anamnesis, periodic pain in the region of the heart, joints, rapid fatigue. On pharyngoscopy: the tonsils are of 2 degrees of hypertrophy, the symptoms of Zak, Gieze, Preobrazhenskiy are positive;

D. tonsils of 1 degree of hypertrophy, cicatricial adhesions between the tonsils and arches (in the past he had tonsillotomy) are noted;

+E. on pharyngoscopy: tonsils of the middle size, liquid pus in the lacunas, Preobrazhenskiy's symptom is positive, the bands of Koritskiy are palpated. In the anamnesis: angina with a frequency of 1-2 times per year, with sharply pronounced intoxication. He is ill with rheumatism.

30. The compensated form of chronic tonsillitis (classification by I.B.Soldatov) is diagnosed in presence of:

+A. objective local signs of chronic tonsillitis;

B. recurrent acute tonsillitis;

C. tonsillogenic intoxication of different degree of manifestation;

D. metatonsillar diseases of the distant organs and systems;

E. local complications of chronic tonsillitis - paratonsillitis, paratonsillar abscess, cervical lymphadenitis with abscess formation.

DISEASES 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 may is 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.

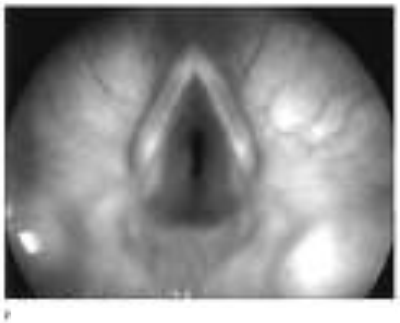

Fig.103. Subglottic laryngitis

Subglottic Laryngitis (False Croup) is a variety of acute catarrhal laryngitis which develops in the subglottic space (fig. 103). It occurs in children ageing from 2 to 5 and is associated with the anatomy of their larynx (narrow lumen and loose connective tissue in the subglottic space). The onset of the disease is as a rule connected with acute inflammation of the mucosa of the nose or the pharynx. False croup occurs mostly in children who tend to develop laryngospasm and suffer from diathesis. The onset of the disease is sudden: an attack of barking cough occurs during night sleep. The child wakes up and tosses in his bed. Breathing becomes very difficult and whistling; inspiratory dyspnoea is pronounced. The nails

and the visible mucosa become cyanotic. The child is frightened and this intensifies coughing. Inspection of the child reveals retraction of the soft tissues of the jugular fossa, supra- and subclavicular spaces, and the epigastric region. This condition lasts from a few minutes to half an hour; the child then sweats excessively and his respiration becomes almost normal. The laryngoscopic picture in subglottic laryngitis is characterized by ridge-like swelling of hyperaemic mucosa in the subglottic part.

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

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

Treatment. A 10 per cent Calcium Chloride solution, vitamin K, and an expectorant are quite effective to arrest small haemorrhage. The subsequent treatment is the same as for acute laryngitis.

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

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

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

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

Chronic Inflammatory Diseases of the Larynx

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).

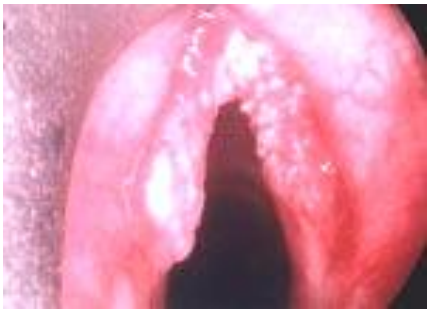

Fig.104. Chronic hypertrophic laryngitis

Chronic Hyperplastic Laryngitis (fig. 104) is characterized by hyperplasia of the laryngeal mucosa. Local and diffuse forms of the disease are distinguished by the extent of involvement. The main complaint of the patients is hoarseness and even aphonia, which are usually due to uneven thickening of the vocal folds and paresis of the vocal muscles. Direct and indirect laryngoscopy reveal hypertrophy of the mucosa which is usually symmetrical on both sides of the larynx and in the 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 histological and cytological 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.

Laryngeal Pachydermia is characterised by heaping up of epithelium in the interarytenoid region and vocal processes of arytenoids. 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.

Polypous Degeneration of Vocal Cords (Reinke’s Oedema) (fig.105)**.** 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.

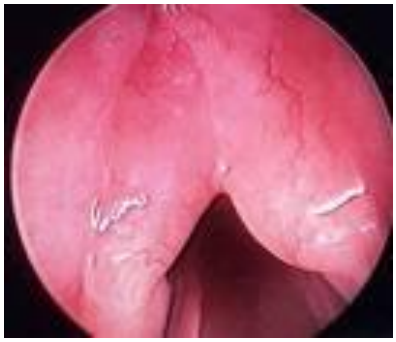

Fig.105. Reinke’s oedema

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 infusion therapy, big doses of antibiotics which eliminate inflammation. The patient with chondroperichondritis should be given pasty non-irritating food. Tube feeding is not recommended, because the gastric tube can irritate the laryngeal tissues. The general reactivity of the body can be increased by biological stimulants (aloe, vitreous body, etc.). Surgical intervention is indicated for an abscess which should be emptied to remove the necrotized tissues. The presence of fistulae is also an indication for surgery, by which the fistula is opened and necrotized tissue removed.

DISORDERS OF LARYNGEAL NERVES.

Sensory and motor disorders of the laryngeal nervous apparatus are distinguished.

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

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

Hyperaesthesia can be of various intensity. In some cases it can take the course of neuralgia. If sensitivity increases, perverted sensations may appear (paraesthesia). Hyperaesthesia is usually caused by the systemic nervous diseases (neurasthenia, hysteria) or changes in the peripheral nerves of the mucosa. The disorder is characterized by the tingling sensation when breathing and talking; sometimes the patient feels an urge to cough-up mucus.

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

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

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

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

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

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

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

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

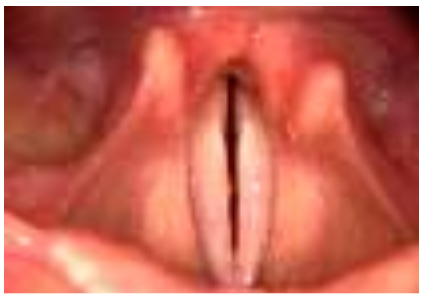

Fig.106. Paresis of both vocal cords

Paresis of both vocal cords (fig. 106) 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

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, hemodynamic, 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 hemodynamic 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.

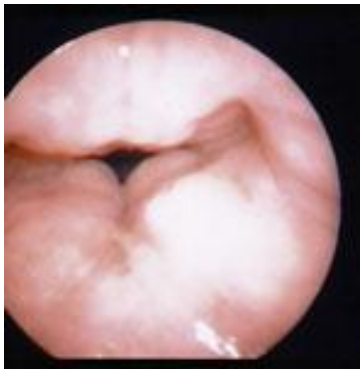

Fig.107. Laryngeal oedema

Acute stenosis of the larynx can be caused by local inflammatory diseases such as the laryngeal oedema (fig. 107), 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 ves sels, of the lungs, the kidneys, etc. Depending on the degree of stenosis, stridor develops. Examination reveals re traction 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 decentralisation 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 perspires; his lips, the nose tip and the terminal phalanges are cyanotic; the pulse is fast. The glottis is 2-3 mm wide.

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

Treatment depends on the cause and stage of acute stenosis. Emergency care in stenosis caused by oedema and inflammation of larynx: antiinflammatory therapy ; use of corticosteroids (3-5 mg. per kg. mass). Gglucocorticoids give antiinflammatory, as well as antiallergic affect; use of 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 Euphillin, 10% solution of Calcii 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 anaesthe sia in intensive therapy departments. Asphyxia (stage IV) requires urgent conicotomy and then tracheotomy.

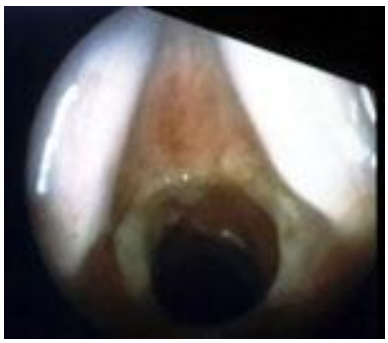

Fig.108. Chronic stenosis of the larynx

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: chondroperichondritis (traumatic (fig. 108), infectious, radiation); disturbed mobility of the cricoarytenoid joint; dysfunction of the inferior laryngeal nerves due to toxic neuritis, following strumectomy, compression by a tumour, and the like; tumour, tuberculosis, syphilis, or scleroma.

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

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

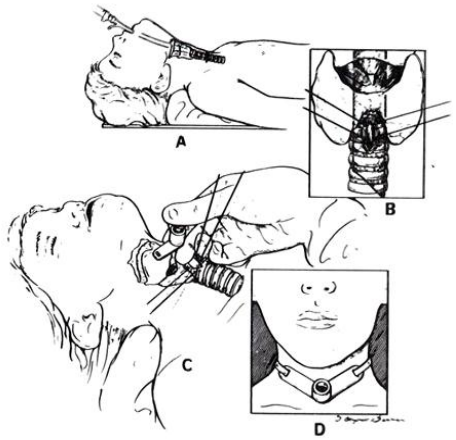

Fig.109. Intubation of the larynx (a) and tracheotomy (b, c, d)

Tracheotomy (fig.109) 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 anaesthesia but in the event of asphyxia where time is a factor of overriding importance no anaesthesia 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.

Control tests

1. A 2- year-old child developed hoarseness 3 months ago. Labored breathing was growing gradually which has recently become noisy. Data of direct laryngoscopy: the glottis is partially closed by fine tuberous formations of gray color. Make a preliminary diagnosis.

A. chronic stenosis of the larynx
B. diphtheria of the throat
+C. papillomatosis of the larynx
D. edema of the larynx
E. chronic hypertrophic laryngitis

2. Symptoms of chondroperichondritis of the larynx are next, except:
A. increase in the body temperature
B. intensification of pain on swallowing
+C. unchanged voice
D. presence of inflammatory edema in laryngoscopy
E. hoarse voice

3. Characteristic of acute subglottic laryngitis:
+A. barking cough
B. bronchospasm
C. expiratory dyspnea
D. latent onset of the disease
E. ringing voice

4. On indirect laryngoscopy a patient is found to have "putrid" position of both vocal folds, he had operation on the thyroid gland two weeks ago. What function of the larynx suffers in bilateral paralysis of the infralaryngeal nerve?
A. resonator
B. protective
+C. respiratory
D. voice-forming
E. olfactory

5. A patient complains of a bad sore throat, which intensifies in swallowing, labored breathing. He has been ill for three days, after drinking cold water. Objectively: the body temperature is increased to 38.8°C, there is inspiratory dyspnea. The cervical lymph nodes are enlarged and painful to palpation. Data of laryngoscopy: edema of the epiglottis, sites of abscess formation on it, the glottis is not seen. Make a preliminary diagnosis.
A. hematoma of the larynx
B. acute laryngitis
C. phlegmonous laryngitis
D. lingual angina
+E. abscess of the epiglottis

6. A patient complains of a bad sore throat, which intensifies in swallowing, labored breathing, hoarseness, cough. He has been ill for three days, after drinking cold water. The diagnosis is phlegmonous laryngitis. Describe the objective picture.
A. hyperthermy
B. inspiratory shortness of breath
C. painful enlarged lymph nodes
D. hyperemia and infiltration of the mucous membrane of the larynx
+E. all of the listed

7. A patient complains of the sensation of tickling in the throat, dry cough, hoarseness and rapid fatigue of voice. He has been ill for 2 days, overcooling preceded the disease. Objectively: the patient's general state is satisfactory. The diagnosis is acute laryngitis. Which of the therapeutic measures is uncorrect?
A. antibiotic therapy
B. vocal regimen
C. local corticosteroids
+D. sedatives
E. antiedematous therapy

8. Complications of phlegmonous laryngitis are next, except:
A. sepsis
B. asphyxia
+C. phlegmon of the oral cavity
D. retropharyngeal abscess
E. pneumonia
9. Formation of "vocal" nodules is associated with:
A. anomaly of the larynx
+B. overloading of vocal folds during singing
C. hereditary tendency
D. abuse of alcohol
E. smoking
10. Principles of the treatment of acute phonasthenia are next, except:
A. sedative therapy
B. strict vocal regimen for one week
C. multivitamins, general strengthening therapy
D. "switching off" of the auditory control
+E. respiratory gymnastics
11. The form of chronic laryngitis includes:
A. allergic
+B. atrophic
C. serous
D. cystic
E. papillomatous
12. A patient complains of severe pain in turnings of the head, swallowing, chill, increase of the body temperature to 38°C. The disease began sharply 3 days ago. The mucous membrane of the throat and larynx is hyperemic, moderately edematous on indirect laryngoscopy. There is a rounded protrusion, with a yellow spot on the top on the lingual surface of the epiglottis. The lower parts of the larynx are not seen. What is the diagnosis?
A. acute catarrhal laryngitis
B. diphtheria of the larynx
+C. abscess of the epiglottis
D. tumour of the larynx
E. subglottic laryngitis
13. A 3- year-old child was admitted to the ENT department with complaints on labored breathing, barking cough and slight hoarseness. He has been ill for 4 days, when rhinitis, cough, increased body temperature developed. The respiration sharply deteriorated during sleep. The child is excited, the skin is pale, auxiliary musculature participates in the act of respiration. During direct laryngoscopy the mucous membrane of the larynx is reddened, elevations of red color are visible in the subglottic space, the glottis is narrowed. Name the disease.
A. diphtheria
B. retropharyngeal abscess
C. foreign body of the larynx
D. phlegmonous laryngitis
+E. subglottic laryngitis
14. A patient took a new antibiotic. She has complaints on a feeling of foreign body in the throat, significant difficulty of respiration, hoarseness, edema of the face and neck. The diagnosis is established: allergic edema of the larynx. Indicate laryngoscopic data which confirm the diagnosis.
A. hyperemia and immobility of the vocal folds;
B. contraction of the glottis;
C. coatings on the lingual surface of the epiglottis;

+D. edema of aryteno-epiglottic folds, posterior wall of the entrance in the larynx;

E. tumor-like formation of yellow- transparent color on the posterior wall of the pharynx.

15. A patient complains of sharp general weakness, increase of the body temperature to 38.5℃, sore throat, difficult swallowing, choking during consumption of food, loss of voice, unbearable voiceless cough with periodic discharge of a dark gray patches. Data of indirect laryngoscopy: the mucous membrane of the larynx is hyperemic, covered with a gray patches, which narrows glottis. Make a preliminary diagnosis.

A. phlegmonous laryngitis

B. acute laryngitis

C. diphtheria of the larynx

D. chondroperichondritis of the larynx

E. edema of the larynx

16. A patient complains of hoarseness, discomfort in the throat, periodic dry cough. Work is connected with frequent overcooling and dustiness of the air, the patient smokes. Data of indirect laryngoscopy: the mucous membrane of the vocal folds is pink, unevenly thickened, there is stratification of white color in the posterior part of the vocal folds, both halves of the larynx are symmetrically mobile.
Make the diagnosis.

+A. chronic hypertrophic laryngitis

B. leukoplakia of the vocal folds

C. diphtheria of the larynx

D. chondroperichondritis of the larynx

E. chronic hypertrophic laryngitis, leukoplakia of the vocal folds

17. The form of chronic laryngitis includes:

A. allergic

+B. hypertrophic

C. serous

D. cystic

E. papillomatous

18. In 3 hours after tracheostomy a patient's respiration deteriorated, crepitation of the subcutaneous cellular tissue was noted in the region of the anterior part of the neck. Determine therapeutic tactics.

A. make a revision of the tracheostoma

B. carry out the intubation

C. replace the tracheocanuula for the smaller size

+D. introduce tracheocannula in the trachea opening after revision

E. hormonotherapy

19. What form of tracheotomy is expedient in laryngeal cancer in case of stenosis?

A. upper

+B. lower

C. median

D. lateral

E. any of the enumerated

20. Name the disease which may be the cause of chronic stenosis of the larynx:

+A. paresis of the recurrent nerves after strumectomy

B. subglottic laryngitis

C. paratonsillar abscess

D. pachidermia of the larynx

E. disease of the kidneys

21. What symptom is characteristic of 2 stage of stenosis, except?

A. fear

+B. expiratory dispnoe

C. tachycardia

D. paleness

E. increasing of arterial pressure

22. Name the complications of tracheotomy:

A. hemorrhage

B. emphysema of the mediastinum

C. hypodermic emphysema

D. pneumothorax

+E. all enumerated above

23. A child of 2 years suddenly developed barking cough, hoarseness against the background of ARVI at night. There are emotional and motor restlessness, difficulty of respiration on physical exertion. What is the diagnosis?

A. acute nasopharyngitis

B. acute tracheobronchitis

+C. acute subglottic laryngitis, stenosis of the larynx of the 1 degree

D. allergic edema of the larynx

E. acute subglottic laryngitis

24. Causes of acute stenosis of larynx are:

A. phlegmon of the larynx

B. parapharyngitis

C. chondroperichondritis of the larynx

D. retropharyngeal abscess

+E. all enumerated above

25. A child with stenosing laryngotracheitis developed IY stage of stenosis of the larynx. Determine therapeutic measures.

A. drug destenosis

B. prolonged nasotracheal intubation

C. upper tracheotomy

+D. lower tracheotomy

E. none of the enumerated

26. What symptom is characteristic of 2 stage of stenosis:

A. bradypnoe

B. respiration of Cheyne- Stokes

C. dilated pupils

D. bradycardia

+E. pale nasolabial triangle

27. A patient has been troubled by a bad sore throat for 5 days. He referred to the doctor because of the increasing difficulty of breathing. On examination there are expressed inspiratory dyspnea at rest, acrocyanosis, tachycardia. The patient's position is forced. What inflammatory diseases can be the cause for development of acute stenosis of the larynx?

A. angina of the palatine tonsils

B. acute rhinitis

+C. phlegmon of the larynx, chondroperichondritis

D. acute pharyngitis

E. acute otitis

28. A patient complains that he received a blow on the anterior surface of the neck during fight. Shortness of breath and hoarseness developed in several hours. The patient is excited; tachycardia, inspiratory dyspnea are revealed. Data of laryngoscopy: the tumorous formation of the cyanotic color of the right vestibular region, it overlaps the glottis. Make a preliminary diagnosis.

A. edema of the larynx

B. hematoma of the larynx

C. chondroperichondritis of the larynx

+D. hematoma of the larynx, stenosis of the larynx

E. stenosis of the larynx

29. A patient complains of hoarseness, pain in the region of the larynx, which is enhanced on swallowing, insignificant difficult breathing. He has been ill for 2 days, the disease was preceded by ARD. The body temperature is 38.0°C, on palpation the lymph nodes of the neck are painful and enlarged. Data of laryngoscopy: hyperemia and infiltration of the mucous membrane of the aryepiglottic and vestibullar folds of the larynx, the glottis is narrowed. Indicate the stage of stenosis.

+A. compensation
B. incomplete compensation
C. sub-compensation
D. decompensation
E. asphyxia

30. What measure is most appropriate in 1 stage of laryngeal stenosis of the inflammatory origin:

A. moistened oxygen
B. expectation
C. conicotomy
D. tracheotomy
+E. distracting therapy

31. What symptom is characteristic of 1 stage of stenosis?

A. cyanosis of the lips
B. difficult breathing
+C. hoarseness
D. intermittent respiration
E. normal respiration

32. Stenosis of the larynx is characterized by dyspnea:

+A. inspiratory
B. expiratory
C. mixed
D. paroxysmal
E. intermittent

SPECIFIC GRANULOMAS OF THE ENT-ORGANS

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). Patients need local and general specific antituberculous treatment.

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 pathomorphological 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 usual ly runs a chronic course.

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

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 vacu ole. The infiltrate also contains hyaline globules (Russel's bodies).

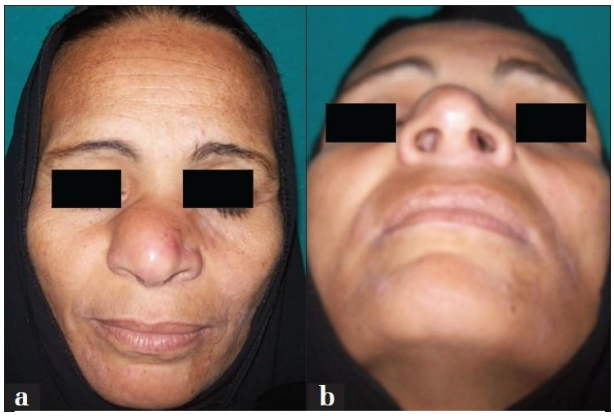

Fig.110. Scleroma

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 (fig. 110) 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 vesti bule 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 antibiotics, corticosteroids. Radiation therapy is sometimes effective. Surgical treatment includes excision of the infiltrates and scars, their elimination by cryosurgery (liquid ni trogen).

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).

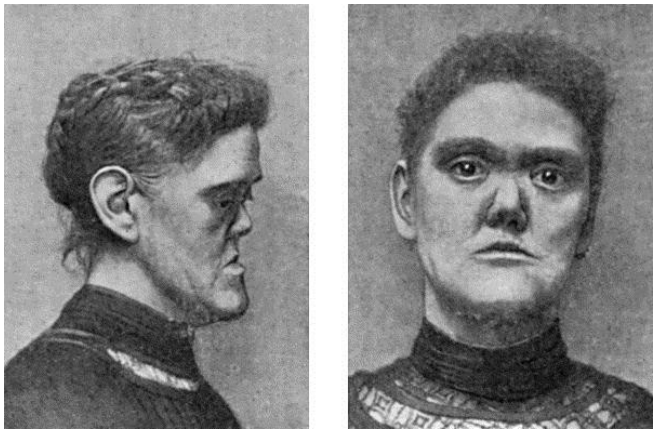

Fig.111. Inherited Syphilis

Syphilis of the Nose (fig. 111) occurs as a primary sclerosis of the secondary and terti ary 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 subglottic 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.

TUMORS OF THE ENT ORGANS

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.

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

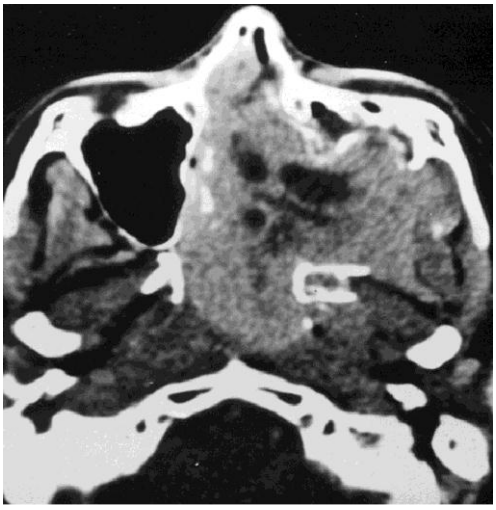

Fig.112. CT: Nasopharyngeal fibroma

The site of origin of the tumour is still a matter of dispute. Earlier it was thought to arise from the roof of nasopharynx or the anterior wall of sphenoid bone but now it is believed to arise from the posterior part of nasal cavity close to the margin of sphenopalatine foramen. From here the tumour grows into the nasal cavity, nasopharynx and behind the posterior wall of maxillary sinus which is pushed forward as the tumour grows. Laterally it extends into pterygomaxillar fossa and thence to infratemporal fossa and cheek (fig. 112).

The essential element of a nasopharyngeal fibroma is dense connective tissue containing a great number of elastic fibres and blood capillaries. The tumour has benign histology, but for its clinical course marked as it is by irresistible growth and destruction of the surrounding tissue, postoperative relapses and frequent copious haemorrhages endangering the patient's life, it may sooner be classified as a malignant neoplasm. It may extend into :

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

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

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

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

In neglected cases where the tumour invades the zygomatic area and paranasal sinuses the removal is performed only after a preliminary operation has been undertaken to provide access to the tumour proper. This access may be obtained through the sinus maxillaris with complete removal of the lateral nasal wall. There may be about 2 litres of blood loss during surgery. Therefore attempts are made, pre-operatively, to reduce the vascularisation of tumour. A course of oestrogen, preoperative radiation therapy may reduce vascularisation of tumour.

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 (fig. 113), and may occur at any age. Children are effected by papilloma between a year and half and five years old. Boys are affected twice more often than girls, men are affected a four times more often than women. Multiple papillomas are found on vocal cords, extend down to subglottic space and trachea, restrict gap of larynx and cause difficult breathing. During first five years of child's life papilloma grow fast, often relapse in spite of therapy, but almost are never malignant. During puberty papillomas may disappear spontaneous. At adult papilloma of larynx is solitary formation on vocal cord with slowly growth. Hard papilloma with proliferous crawling growth is found in every fourth case. Such growth causes transformation papilloma to flat (squamous) cell carcinoma. It is found in 15-20% and gives grounds to regard papilloma of larynx at adult as obligatory precarcinoma.

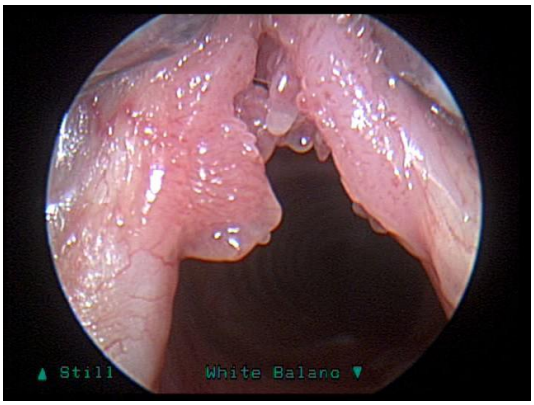

Fig.113. Papillomas of vocal cords

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.

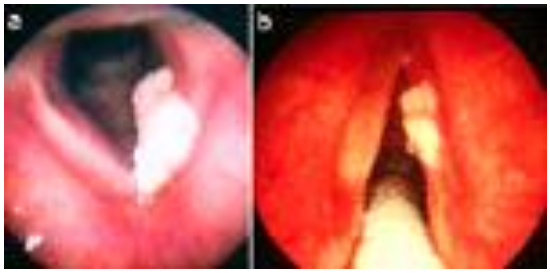

Fig.114. Cancer of larynx

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 (fig. 114) 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. Pat ient'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 dyspnoea, hoarseness and increasing difficult breathing. The tumor which grows exophytic may be discovered with the help of the indirect laryngoscopy.

The symptoms which appear with the further growth of tumor very little depend upon the region of the initial localization. They become common for cancer of larynx (hoarseness or aphonia, cough,

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

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

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

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

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

This operation is kind of saving of organ.

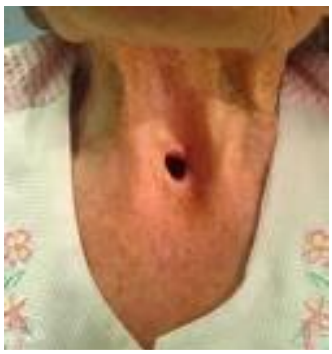

Fig.115. Tracheostoma after laryngectomy

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

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

Combined 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

mettastasing 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: 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 often, 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 (often accompanied with irradiation into ear. Then the signs apply which are caused with tumor germination and involving of the chews, root of the tongue and by collateral oedema 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 piriformis, 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. Tumor germination is accompanied with symptoms of laryngeal affection – hoarseness and difficult breathing. Also, there are a narrowing of the recesses performs and accumulation of saliva inside it, but in case of the postcricoidcarcinoma – moreover there is an oedema arytenoid cartilages and, often, 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 often 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, have a supplementary characterize clinic symptoms. The 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 dense elastic consistantion during palpation. Then, tumor tends to enlargement and involves a surrounding pharyngeal tissues to the tonsil so, that it often ulcerates and is accompanied with pain. The primary tumor may be enlarged over the pharynx and involves the gingival, root of the tongue, surrounding bones, but in case of affection of the rhinopharynx – into nasal cavity. In that case a chewing and swallowing are difficult. Tumor decay is accompanied with very harsh nasty odour out of oral cavity.

Often, the first patient's complains of is a metastatic enlargement of the lymphnodes. If the primary neoplasm is in the palate tonsils, then the regional metastases develop inside the retromaxillar 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 tonsillar tumors. Neoplasm of the lingual tonsil manifests with regional metastases in the upper lymphnodes of the profound jugular chain of neck which is on a place of the common carotid artery bifurcation. They are discovered as a dense elastic nodes which tend to quickly enlargement, compressing neural and vascular trunks so, that causing an acute pain and collateral oedemas.

At present, the general therapeutic method for the radiosensitive tonsillar tumors is a radiotherapy during an adequate chemotherapy. Relapses of the tonsillar tumors, often, are not on a place of the primary focus, but in region of the regional and other lymphnodes (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 favourable prognosis after treatment is provided by any therapy for fifth-years survival rate no more than in 35% of patients.

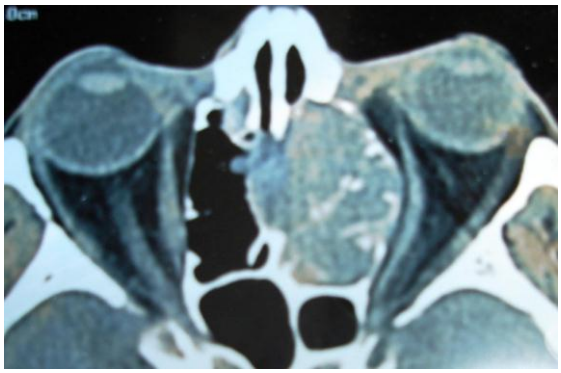

Fig.116. Melanoblastoma of the nasal cavity and PNS

More often, 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 tonsillar tumors in nose – they are: reticulosarcoma, lymphoepithelioma, also as a rarer tumors as – melanoblastoma (fig. 116) 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, often, 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), eodematic 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 tumor 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 bone destructions and focus higher concentration of the tumorotrophic radiopharmopreparation on the gamma-scintigramm that reports about tumor 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. Often, 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%.

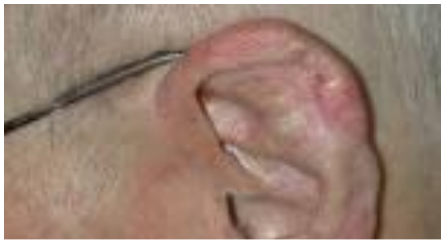

Fig.117. Cancer of auricula

If the all ear's malignant tumors compose 100%, then 85% - are tumors of the auricle (fig. 117), 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 often 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 unfavourable and poor, even if we use a combined therapy. At first, there is used a radiotherapy, then an extended surgical operation. In spite 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 tumor 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.

Control tests

1. A patient aged 63 was admitted to the clinic with complaints on hoarseness of voice. He has been ill for a year, he noted difficult breathing in the last 5 days. Laryngoscopy: the right half of the larynx is occupied by the tuberous tumor, it is immovable, the glottis is narrowed. A dense mobile lymph node is palpated on the neck on the right, of 5 cm in size. No pathology is revealed on the X-ray examination of the chest organs. What is your preliminary diagnosis?

+A. cancer of the larynx of III st
B. tuberculosis of the larynx
C. cancer of the larynx of II st.
D. disease of Wegener
E. scleroma of the larynx

2. What diseases compose to complex “infectious granuloma”?

+A. tuberculosis, scleroma, syphillis of the ENT organs
B. cancer of the larynx, pharynx, ethmoid labyrinth
C. angina of Vensan-Simanovskiy, syphillis of the larynx
D. tuberculosis of the larynx,
E. disease of Wegener

3. On pharyngoscopy a patient is determined to have an irregular whitish tumor with disintegration. The tumor involves the lower pole of the right palatine tonsil with passage to the lateral side of the tongue and cellular tissue of the floor of the oral cavity. The mobility of the larynx is preserved. Planocellular cancer without keratinization is histologically verified. What is your therapeutic tactics?

A. radiation therapy
+B. selective chemotherapy, radiation therapy
C. operation
D. antibiotic therapy
E. cryo-destruction of the tumour

4. A patient aged 52 years has been under clinical observation of the otolaryngologist for 1.5 years for cancer of the larynx of 2 st. He refused the proposed treatment. Objectively: stridor, pale skin, cyanosis of the lips, moderate participation of the accessory musculature in the act of respiration, the patient is conscious, active. On indirect laryngoscopy: extended tumor of the larynx, which shuts its opening, the width of the glottis is 3 mm. What are your therapeutic measures?

+A. operation for a tumour of the larynx
B. tracheotomy
C. drug destenosis
D. intubation
E. none of the enumerated

5. A patient A. aged 67 was admitted to the ENT clinic with complaints on pain, itching and bleeding from the left auricle. According to the words of the patient he has been suffering for a month. He referred for aid after the sequential renewal of bleeding. The ulcerated surface of 1.5 to 2. 5 cm is determined in the region of the helix of dark brown color, the skin around the formation is macerated. Otoscopy is without any peculiarities. Hearing corresponds to the age changes. The histologic type of the tumour: highly differentiated cancer. What is the therapeutic tactics?

A. surgical removal of the tumor within the healthy tissues
B. chemoradiation therapy
+C. segmental resection of the auricle
D. complete resection of the auricle
E. radiation therapy

6. What diseases should cancer of the subglottic part of the larynx be differentiated with?

A. scleroma
B. syphillis

C. Wegener's granulomatosis
D. cicatricial stenosis of the larynx
+E. all of the enumerated

7. Which of the enumerated methods you will use in a patient in 3 st. of stenosis of the tumor genesis:
A. drug destenosis
B. intubation
C. distracting therapy
+D. tracheotomy
E. antipyretic therapy

8. A patient aged 60 referred to the clinic with complaints on nasality of her voice, difficult painless swallowing. She has been ill for 3 months. Pharyngoscopy: the right palatine tonsil, paratonsilar cellular tissue are of pink color, infiltrated, the fauces is asymmetric. The chains of small and large, dense mobile lymph nodes are palpated on the neck on both sides, of 5 cm in size. Enlarged lymph nodes of the mediastinum are determined on X-ray examination of the chest organs. Preliminary diagnosis is lymphogranulomatosis. What methods of diagnostics are necessary for making the diagnosis?
A. culture from the fauces on the right
B. CT of the neck
+C. biopsy of the tonsil, ultrasound examination of the neck, organs of the abdominal cavity, CT of the lungs and mediastinum
D. biochemistry of the blood
E. cytology examination

9. A patient developed difficult nasal breathing on the right against the background of chronic runny nose. According to the words of the patient he has been ill for about three months. He was treated by the ENT doctor of the polyclinic. He took antibiotics, UHF to the nose. The state of the patient deteriorated: apathy, adynamia, weakness and periordic bleeding from the right half of the nose developed. He noted worsening in vision in the right eye and discomfort in the eye. On rhinoscopy: the mucous membrane of the nose is of pink color on the right, tumerous formation with the bleeding areas is determined in the upper and middle nasal passages. The lower nasal passage is free. The paranasal sinuses are painless to palpation. There is no revealed lymphadenopathy on the neck. The assumed diagnosis is cancer of the ethmoid labyrinth. What methods of examination should you make?
A. cytological study
+B. computer study of the paranasal sinus, biopsy of the tumor
C. blood count, biopsy of the tumor
D. posterior rhinoscopy
E. bacteriological examination

10. A patient was admitted to the clinic with complaints on hoarseness of voice, shortness of breath. The patient smokes and drinks a lot. He was treated 1-2 times a year by the therapeutist for chronic tracheobronchitis. He has been ill for 2 years, he noted difficult breathing in the past 5 days. Laryngoscopy: the right half of the larynx is protruded in the opening of the larynx, the surface of the protrusion is smooth, the larynx is immobile, the glottis is narrowed. There is no revealed lymphadenopathy on the neck. On X-ray examination of the chest organs no pathology is revealed. What physician should to do?
A. cytological study
B. computer tomography of the larynx, biopsy of the tumor
+C. tracheotomy, biopsy
D. bronchoscopy
E. antibiotic therapy, diuretics.

11. A patient was admitted to the clinic with difficult nasal breathing, periodic nasal bleedings. He has been suffering for 3 months. He was not treated, he referred for aid only after an increase in frequency of nasal bleedings. Rhinoscopy is without any peculiarities, because of the deviation of the nasal septum the posterior parts could not be examined. A tumorous formation with ulceration is determined on fibroscopy,

the formation shuts the upper and middle part of the choanna. The histologic type of the formation: low-differentiated cancer of the nasopharynx. There is no revealed lymphadenopathy on the neck. What is your therapeutic tactics?

A. surgical removal of the tumor by the external access

+B. chemoradiation therapy

C. surgical removal of the tumor under the control of sight

D. antibiotic therapy

E. chemotherapy.

12. A patient of 72 years old is admitted to the clinic with complaints on hoarseness of voice and tumorous formation on the neck on both sides. In the recent two weeks he developed pains on the right with irradiation to the right ear and shortness of breath on physical exertion. The patient smokes and drinks a lot. Objectively: the right half of the larynx is immobile, the glottis is clean, it is sufficient for respiration, the right pear-shaped sinus is not opened, there is a lake of saliva at its entrance. Preliminary diagnosis: cancer of the pear-shaped sinus. What additional methods of study should you make for exception or affirmation of cancer of the pear-shaped sinus?

A. diagnostic lymphadenectomy

+B. biopsy of tumor of the pear-shaped sinus

C. US investigation of the larynx and neck

D. CT examination

E. cytology, bacteriology examination

13. A patient (hypersthenic) was urgently admitted to the oncologic ENT department with complaints on shortness of breath and hoarseness of voice. Harmful habits are absent. The patient was treated at the therapeutic department for bronchial asthma, she was discharged three days ago. She was given a hormonal therapy. Her state insignificantly improved. She was brought to the ENT hospital by ambulance with stenosis of the larynx of II degree. On indirect laryngoscopy hypertrophy of the tissue is determined in the region of the right vocal fold. It is impossible to examine the larynx in detail because of the anatomical peculiarities. The ENT doctor made the preliminary diagnosis of cancer of the middle part of the larynx. What is his therapeutic tactics should be?

+A. tracheostomy, biopsy

B. chemoradiation therapy

C. surgical removal of the tumor under the control of sight

D. antibiotic therapy

E. puncture of the tumor

14. A patient complains of expressed headache, pain in the left ear, bleeding from the ear. He has been ill for eight months. Otoscopy revealed a tuberous infiltration obturating the auditory meatus with presence of blood-containing necrotic masses with unpleasant smell. Moderate bleeding started in an attempt to make toilet and revision. A preliminary diagnosis is cancer of the middle ear. What studies should be carried out first of all?

A. computer tomography of the temporal region

B. biopsy

C. bacteriologic study

+D. biopsy, computer tomography of the temporal bone

E. blood count, coagulability, duration of hemorrhage

15. The patient of 17 years old has been treated for chronic hyperplastic rhinitis, frequent nasal bleedings (3-4 times per week), hemoptysis for a long time; posterior rhinoscopy visualized the tuberose tumour occupying the fornix of the nasopharynx and lateral walls of the nasopharynx, the choanae are closed in the upper and middle part. What is your preliminary diagnosis?

+A. juvenile fibroma

B. cancer of the nasopharynx

C. foreign body of the nasopharynx

D. choanal polyp

E. abscess of the nasopharynx

16. What endoscopic methods of study help to make early diagnostics of cancer of the larynx?

A. fibroscopy

B. electronic stroboscopy

+C. direct micro-laryngoscopy

D. indirect micro-laryngoscopy

E. none of the enumerated

17. After examination at the clinic a patient is made a preliminary diagnosis - cancer of the larynx. What studies must be carried out first of all?

A. computer tomography of the larynx

B. direct laryngoscopy

+C. biopsy of the tumor

D. ultrasonic study of the larynx

E. bacteriological study

FOREIGN BODIES OF THE ENT ORGANS

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

Fig.118. Removing of foreign body with syringing

Most of the seed grains and smooth objects can be removed with syringing (fig. 118). Smooth and hard objects like steel ball-bearing should not be grasped with a forceps as they tend to move inwards and may injure the tympanic membrane. In all impacted foreign bodies or those where earlier attempts have been made, it is preferable to use general anaesthetic and an operating microscope. Occasionally postaural approach is used to remove foreign bodies impacted in deep meatus or those which have been pushed into the middle ear. Unskilled attempts at removal of foreign bodies may lacerate the meatal lining, damage the tympanic membrane or middle ear ossicles.

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

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

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

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

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

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

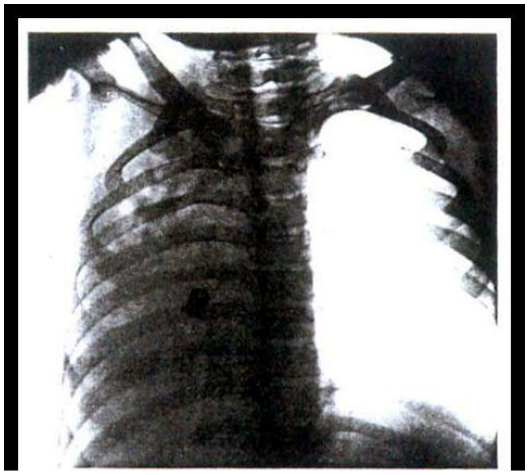

Fig.119. X-ray: Foreign body of the right bronchus, total obstruction, right lung atelectasis.

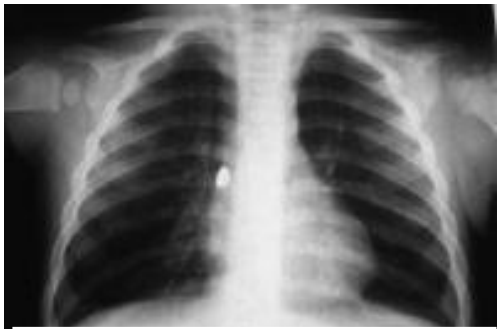

Fig.120. Retained foreign body in the right bronchus

Bronchial foreign body. Most foreign bodies enter the right bronchus because it is wider and more in line with the tracheal lumen. A foreign body may ***totally obstruct*** a lobar or segmental bronchus causing atelectasis (fig. 119) or it may produce a ***check valve obstruction*** - allowing only ingress of air but, not egress, leading to obstructive emphysema. Emphysematous bulla may rupture causing spontaneous pneumothorax. A foreign body may also shift from one side to the other causing change in physical signs. A ***retained*** foreign body in the lung (fig. 120) may later give rise to pneumonitis, bronchiectasis or lung abscess.

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

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

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

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

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

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

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

Oesophagus. Usual foreign bodies lodged in the oesophagus are a coin, piece of meat, chicken bone, denture, safety pin, or a marble. Children are more often affected. They have a tendency to put

anything in the mouth. Playing while eating is another factor. Use of upper denture prevents tactile sensation and a foreign body is swallowed undetected. Loss of consciousness, epileptic seizures, deep sleep or alcoholic intoxication are other factors. Pieces of food may be held up in cases of oesophageal stricture or carcinoma. The first symptom of carcinoma oesophagus may be sudden obstruction from a foreign body such as a piece of meat, fruit or vegetable. Foreign body may be swallowed with an attempt to commit suicide.

Esophageal Foreign Body

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

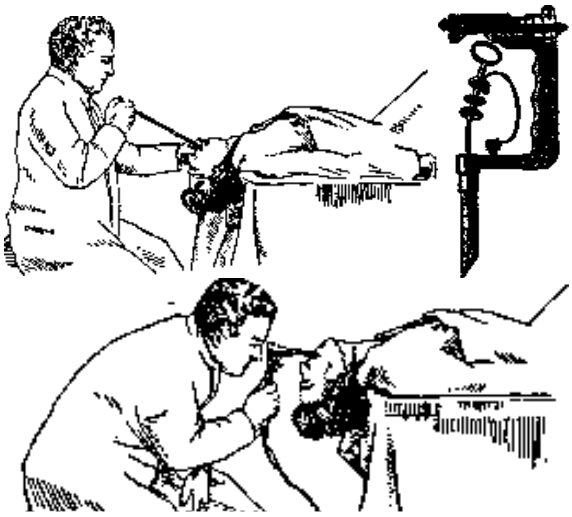

Fig.121. Rigid esophagoscopy

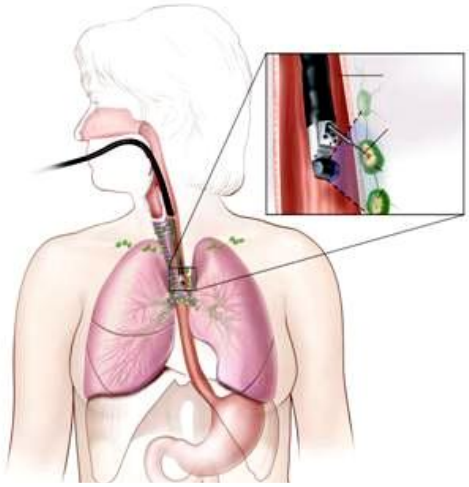

Fig.122. Fibroesophagoscopy

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

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

Management. *Oesophagoscopic* removal (fig. 121, 122). Most of the foreign bodies in oesophagus can be removed by esophagoscopy under general anaesthesia. *Cervical oesophagotomy*. Impacted foreign bodies or those with sharp hooks such as partial dentures located above thoracic inlet may require removal through an incision in the neck and opening of cervical oesophagus. *Transthoracic oesophagotomy.* For impacted foreign bodies of thoracic oesophagus, chest is opened at the appropriate level. A foreign body which has reached the stomach may pass through the rest of gastro-intestinal tract without difficulty; stools should be examined everyday.

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

Control tests

1. The basic methods of removal of the foreign bodies of the nose:
   A. rhinoscopy, removal with the aid of the clamp
   B. removal with the aid of the fibroscope
   C. with the aid of the nose pincers
   D. rhinoscopy (anaemization of the mucous membrane, removal by the hook)
   +E. by the hook; in large foreign bodies - splitting with bone forceps and further removal by the hook.
2. Symptoms of the nasal foreign bodies and rhinoliths, except:
   A. unilateral stuffiness of the nose, purulent runny nose, hyperemia of the mucous membrane of the nose, granulation.
   +B. atrophy of the mucous membrane of the nose, wide nasal passages.
   C. unpleasant smell from the nose.
   D. nasal bleeding
   E. lacrimation, worsening in the sense of smell, headache.
3. Methods of diagnostics of foreign bodies of the upper respiratory tract are:
   A. anamnestic information (presence of short-term asphyxia at the moment of passage of the foreign body through the glottis)
   B. physical methods
   C. X-ray of the larynx and lungs.
   D. endoscopic methods.
   +E. all enumerated.
4. The characteristic of organic foreign bodies of the respiratory tract, except:
   +A. roentgenocontrast
   B. get swollen, cause the processes of decay in the tracheobronchial tree.
   C. crumble in removal (second plurality of foreign bodies).
   D. get inflammation in lung
   E. X-ray no contrast
5. The method of removal of the foreign bodies of the larynx in an adult:
   A. bronchoscopy.
   +B. indirect laryngoscopy.
   C. digital removal.
   D. through laringofissura
   E. direct laryngoscopy
6. The method of the removal of the foreign body of the larynx in a child:
   A. bronchoscopy.
   B. indirect laryngoscopy.
   C. digital removal.
   D. through laringofissura
   +E. direct laryngoscopy.
7. During consumption of the food a patient choked with a fish bone. He made an attempt to push it by consumption of rough food. He developed piercing pain, which intensified on swallowing. The ENT doctor of the polyclinic did not reveal a foreign body. Next day the symptoms increased and patient referred to the ENT clinic. Indirect laryngoscopy showed edema and hyperemia of the arytenoid mucous membrane and entrance in the esophagus. What is a preliminary diagnosis, diagnostic tactics?
   A. consultation of the phthisiologist
   B. revision of this zone by the finger of the doctor
   C. antibiotics, hypopharyngoscopy
   D. spasmalytics, antibiotics
   +E. diagnostic esophagoscopy

8. A patient of 75 years old is admitted to the clinic with complaints on pain in the substernal area, between the scapulae and nonpassage of food through the esophagus. He has been ill for 1 day. According to the words of the patient he choked with a part of the maxilla denture during dinner. He was administered the antiinflammatory therapy and went home. On the second day the pain intensified in the substernal area and between the scapulae, the temperature was 38°C in the evening. The diagnosis: a foreign body of the esophagus (dental prosthesis). What is a therapeutic tactics?

A. consultation of the therapeutist

B. fibroscopy with removal of the foreign body

C. mediastenotomy with removal of the foreign body

+D. esophagoscopy with removal of the foreign body, antibiotics

E. diagnostic esophagoscopy

9. A child of 7 years old drank the concentrated solution of the caustic soda when he was three year old. No treatment was given at that time and up to now he felt well. 4 days ago the boy ate a small piece of the fried lard; he eats or drinks nothing after this. The child is exhausted, hypodermic fatty layer is expressed weakly, the tongue is dry. He walks with difficulty, suffers from unquenchable thirst and requires water all the time, which he immediately ejects back by emetic motions. The pharynx and larynx are without visible changes. What diseases should we think of?

A. cicatricial stenosis of the esophagus

B. food obstruction.

+C. cicatricial stenosis of the esophagus, food obstruction.

D. acute esophagitis

E. nervous disorders of the esophagus

10. A patient who choked with a meat bone, was made a control X-ray examination of the neck in the side projection after esophagoscopy. The narrow strips of the air in the prevertebral soft tissues were determined on the X-ray . What is your conclusion?

+A. perforation of the esophagus

B. cicatricial contraction of the esophagus

C. burn of the esophagus

D. acute esophagitis

E. food obstruction

11. A girl of 7 years old, while playing with beads, pushed one of them in the auditory passage. On-duty nurse, to whom they referred for aid, tried to remove a foreign body by pincers, but the attempt failed – the bead fell in the depth of the auditory passage. The girl is brought to the ENT department. Objectively: on examination mild infiltration of the tissues of the left auditory passage is noted, there are single bruises on its the skin. A foreign body is visualized in the depth of the auditory passage, behind the isthmus. The eardrum is not visible. The attempt to remove the foreign body from the auditory passage by washing failed. What is tactics?

A. removal of the foreign body by surgery

B. removal of the foreign body by pincers

+C. removal of the foreign body by the hook

D. removal of the foreign body by the clamp

E. irrigation of the external auditory canal

12. A mother of the 3- year-old child notes runny nose from the left half of the nose, absence of nasal breathing for a month. He was treated by the pediatrician for a week with deterioration. He developed edema of the lower eyelid on the left, sleepless nights, t 38-39°C. He was examined by the ENT doctor. Rhinoscopy on the left is impossible because of edema of the mucous membrane of the nose, abundant quantity of mucopurulent discharge with unpleasant smell. What examination should be made?

A. computer tomography of the nose and PNS

+B. anemization of the nasal mucosa, rhinoscopy

C. blood count

D. consultation of the oculist

E. bacteriological study

13. A 3-year-old girl is brought to the clinic. Her parents noted that about 2 hours ago she played with a button and pushed it in the right half of the nose. An attempt to take out the button failed, the button passed into the depth of the nasal passage. Rhinoscopy: the mucous membrane of the nasal cavity is hyperemic on the right. The foreign body is determined in the depth of the general nasal passage, nasal breathing is difficult on the right. How can the foreign body be removed?

A. general narcosis, removal with the aid of the pincers

+B. anemization of the nose mucosa, removal with the aid of the hook

C. irrigation of the nasal cavity

D. removal of the foreign body by the clamp

E. removal of the foreign body by surgery

14. A 6-year-old girl was brought by her parents with complaints on cough, shortness of breath, which developed after choking with a button. Objectively: the skin is pale, moist. Difficult breathing develops during cough, in this case the skin becomes cyanotic. On examination the ENT organs are without any peculiarities. The glottis is wide, the mucous membrane of the larynx is moderately hyperemic. There is mucus in the subglottic part. What is diagnostic tactics of the doctor?

A. blood count

B. bacteriological examination

+C. R-scopy of the tracheobronchial tree, tracheobronchoscopy

D. consultation of the pediatrician

E. immunological examination

15. A patient referred to the ENT doctor with complaints on a sore throat and a feeling of tingling. She associates the disease with eating fish two days ago. The patient made an attempted to help herself by taking a great amount of bread crusts. Objectively: moderate edema of the mucosa of the fauces is noted on the left and small wound areas of the front palatine arch, palatine tonsil. The end of the small fish bone is determined between the arch and capsule of the palatine tonsil. What is tactics of the doctor?

A. general narcosis, removal with the aid of the pincers

+B. anesthesia of the fauces mucosa, removal with the aid of the clamp

C. R-tomography of the pharynx, removal with the aid of the fibroscope

D. removal with the aid of Kocher's clamp

E. bacteriological study of the pharynx

16. A patient was admitted to the clinic with complaints on pain in the substernal area, between the scapulae and nonpassage of food through the esophagus. She has been ill for 3 days. According to the words of the patient she choked with a hen bone during supper. She did not refer for help. On the second day the pain intensified in the substernal area and between the scapulae, the temperature was 38°C in the evening. On the day of reference the temperature was 39°C. What is diagnostic tactics?

A. R-graphy of the esophagus

+B. R- scopy of the esophagus with barium

C. R-computer tomography

D. bacteriological study

E. consultation of the thoracic surgeon

17. A patient was admitted to the clinic with complaints on pain in the substernal area, between the scapulae and nonpassage of food through the gullet. According to the words of the patient she choked with a hen bone during supper. She has been ill for 3 days. She did not refer for help. On the second day the pain intensified in the substernal area and between the scapulae, the temperature was 38°C in the evening. On the day of reference the temperature was 39°C. A preliminary diagnosis is a foreign body of the esophagus (hen bone).What is a therapeutic tactics?

A. general narcosis, esophagoscopy with removal of the bone

B. intramuscular spasmolitics

C. mediastinotomy with removal of the bone

+D. local anesthesia with 10% Lidocaine, esophagoscopy with removal of the bone, antibioticotherapy

E. antibioticotherapy

BURNS OF THE ESOPHAGUS

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

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

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

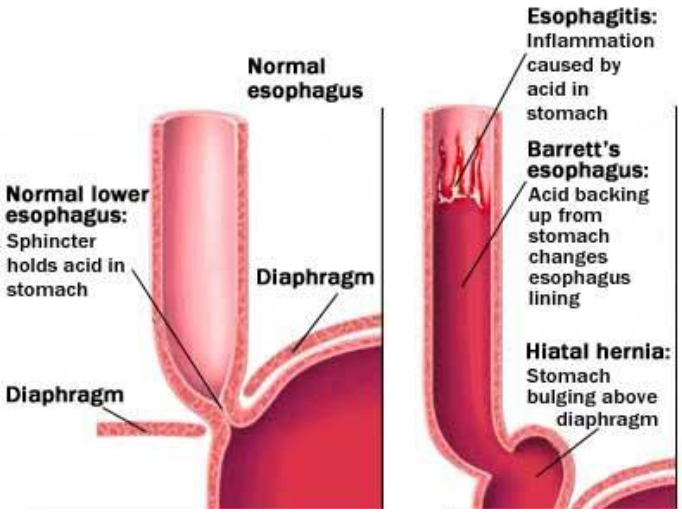

Fig.123. Fourth stage of burns of the esophagus.

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

It lasts several weeks The fourth stage (*scarring)* (fig. 123) is that of formation of stenosis.

There are three clinical stages of the pathology under discussion.

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

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

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

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

Treatment should begin as soon as possible. In chemical bums, the poisonous substance should be neutralized during the first 6 hours. If the poison is an alkali, the patient should be given a weak solution of acetic, tartaric, or citric acid. If it is an acid, magnesia or chalk should be given. If the antidotes are not available, warm boiled water should be given, adding half of the volume of milk or egg white. If a gastric tube cannot be passed into the stomach, the patient should drink 5-6 glasses of washing liquid and then the tongue root should be pressed down with the fingers or a spatula to induce vomiting. The stomach should be lavaged several times with 10 litres of liquid.

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

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

Follow the patient with oesophagoscopy every two weeks till healing is complete, for the development of any stricture. If stricture develops it can be treated by: 1. Oesophagoscopy and prograde dilatations, if permeable, 2. Gastrostomy and retrograde dilatation, if impermeable, 3. Esophageal reconstruction or by-pass, if dilatations are impossible.

Patients of corrosive injuries of oesophagus may require life-long follow up.

Control tests

1. Morphological changes in burn of the esophagus of III degree:
   - A. damage of the superficial epithelial layer.
   - B. damage of the whole thickness of the mucous membrane.
   - C. necrosis of the mucous and muscular layers of the esophagus.
   - D. inflammatory changes penetrate the deep-lying formations (periesophageal cellular tissue of the mediastinum).
   - +E. all enumerated
2. Therapeutic measures (most important) in the first twenty-four hours after burn of the esophagus:
   - A. accelerated diuresis with application of 4% solution of soda.
   - +B. fight with shock, intoxication, dehydration, stenosis of the larynx (in its development), hunger, corticosteroid preparations, antibiotics.
   - C. only antipyretic treatment.
   - D. corticosteroid preparations, antibiotics, rehydration, spasmolytics, pain-killers, parenteral nourishment or the diet № 1.
   - E. bougienage of the gullet + measures, indicated in D.
3. Therapeutic measures (most important) in the first seven days after burn of the esophagus:
   - A. accelerated diuresis with application of 4% solution of soda.
   - B. fight with shock, intoxication, dehydration, stenosis of the larynx (in its development), hunger, corticosteroid preparations, antibiotics.
   - C. only antipyretic treatment.

+D. corticosteroid preparations, antibiotics, rehydration, spasmolytics, pain-killers, parenteral nourishment or the diet № 1.

E. bougienage of the gullet + measures, indicated in D.

4. What chemical substances cause more profound alterations in the walls of the esophagus in burns?

A. Acids

+B. Alkali

C. Alcohol

D. Phenols

E. Petrol

5. What chemical substances cause coagulation necrosis of the tissues in burns of the esophagus?

+A. Acids

B. Alkali

C. Alcohol

D. Phenols

E. Petrol

6. At what period after burn of the esophagus is it expedient to carry out esophagoscopy for the diagnostic purpose?

A. on the first 24 hours

B. on the 5th day.

+C. on the 10th day.

D. on the 20th day.

E. on the 30th day.

7. What chemical substances cause coagulation necrosis of the tissues in burns of the esophagus?

A. Acids

+B. Alkali

C. Alcohol

D. Phenols

E. Petrol

8. What of the factors indicated depend on the degree of burn of the esophagus?

A. concentration of poison.

B. amount of poison.

C. duration of the influence on the tissue.

D. character of damage (acid, alkali).

+E. all enumerated

9. Pathologoanatomic stage of burn of the esophagus, in which maximum development of the connective tissue occurs:

A. stage of necrosis (I stage)

B. II stage (ulcerations)

C. III stage (granulations)

D. IV stage (cicatrizations)

+E. III -IV stage

10. How many degrees of burn of the esophagus are distinguished?

+A. three degrees.

B. four degree.

C. five degrees

D. one degree.

E. two degree

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The following list comprises some of the most widely read journals in otolaryngology. They are available in most medical libraries.

Acta Oto-Larangologica (Stockholm)
American Journal of Otolaryngology
American Journal of Otology
American Journal of Rhinology
Annals of Otology, Rhinology and Laryngology
Archives of Otolaryngology-Head and Neck Surgery
Clinical Otolaryngology
Ear, Nose and Throat Journal
Head and Neck Surgery
International Journal of Pediatric Otorhinolaryngology
Journal of Laryngology and Otology (London)
Journal of Otolaryngology
Laryngoscope
Otolaryngologic Clinics of North America
Otolaryngology-Head and Neck Surgery
Journal of Ear, Nose and Throat Diseases (Kyiv)