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OTORHINOLARYNGOLOGY II

2023

COMENIUS UNIVERSITY IN BRATISLAVA

JESSENIUS FACULTY OF MEDICINE IN MARTIN

Jessenius Faculty of Medicine in Martin, Comenius University in Bratislava, 1st edition
ISBN 978-80-8187-150-4, EAN 9788081871504

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CONTENT

Dear readers,

this book is continuation of Otorhinolaryngology I. It focuses on the function and diseases of esophagus, external, middle and inner ear and includes a short chapter on phoniatrics. Similar to the first part, emphasis is placed on practical information for undergraduate students and healthcare professionals with interest in otorhinolaryngology. Family practitioners and pediatricians may encounter many conditions summarized in the book, among them especially inflammation and tumors of the ears. The aim is for medical and dental students to understand better the etiology, symptoms, diagnosis, including ENT screening methods, differential diagnosis, treatment and prognosis of the diseases of esophagus and ears.

V.Č.

1 ESOPHAGUS

1.1 CLINICAL ANATOMY AND PHYSIOLOGY

The esophagus is a 25 cm long hollow muscle tube with slit lumen. It starts as free continuation of the pharynx and connects with the stomach in the *ostium cardiacum*. It has three parts:

1) the cervical part (*pars cervicalis*); it is about 6 cm long and located at the level of the cervical vertebrae C_6-C_8

2) the thoracic part (*pars thoracica*); it measures 16-20 cm and is the longest one, it is located in the posterior mediastinal space at the level of Th_8-Th_9

3) the abdominal part (*pars abdominalis*); it is only 1-2 cm and is located in the peritoneal cavity

From a clinical point of view, there are three significant physiological narrowings (Fig.1):

1. narrowing (Killian's sphincter) - at the beginning of the esophagus, at the transition of esophagus between the ring cartilage and the spine. In this locality, a foreign body is most often stuck.
2. narrowing - the passage of the esophagus between the thoracic aorta and the left bronchus
3. narrowing - the passage of the esophagus through the diaphragm (*hiatus oesophageus*)

Knowledge on topographic relationships is especially important when introducing a nasogastric tube. The distance from the incisors to the beginning of the esophagus (Kilian's sphincter) is about 15 cm, to the cardia about 40 cm.

The esophagus is lined with a multilayered non-keratinizing squamous epithelium. The esophageal wall is formed by an outer (longitudinal fibers) and an inner (concentrically arranged fibers) muscle layer. In the upper third, the transverse striated muscle fibres predominate, they gradually wane and in the lower part there is only smooth muscle.

The vascular supply is abundant, the cervical portion is supplied through *a. thyroidea inferior*, thoracic part with *rami oesophagei aortae thoracicae* and abdominal part with *a. gastrica* and *a. phrenica inferior*.

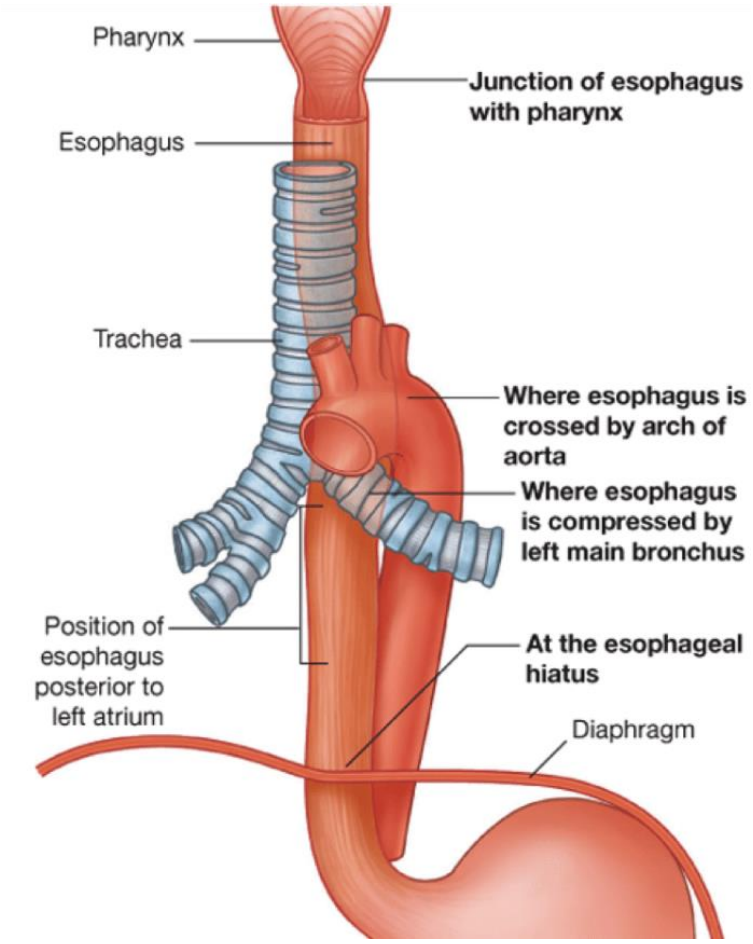

Fig.1 Anatomy of esophagus (from https://medika.life/the-esophagus/ under a Creative Commons License CC BY-NC-ND)

Lymphatic drainage goes through multiple lymph nodes, which are located along the esophagus. The esophagus is innervated by branches *n. vagus* and sympathetic fibers. Sensory fibers are under-represented in the esophagus; more of them is in the neck portion. Swallowing is a reflex. It begins voluntarily by shifting food with the tongue into the pharynx (phase 1). By contraction of the pharyngeal sphincter, the food is transferred to the esophagus through the cricopharyngeal sphincter (phase 2). The third phase, the food is moved into the stomach by peristaltic waves. The cardia reflexively opens and bolus of the food enters the stomach. The mechanism of deglutination is different in infants. The infant is able to breathe and suck, and possibly swallow at the same time. This is made possible by higher position of the larynx and relatively large tongue. The air from the nasopharynx can flow directly into the larynx without the risk of aspiration. Nutritional problems may occur with nasal congestion (for details, see chapter on Acute inflammation of the nasal mucosa).

1.2 EXAMINATION METHODS

It is crutial to correctly take a history. Diseases of the esophagus cause dificulty or painful swallowing and feeling of a foreign body in the throat. Disorders can be caused by the changes in the lumen up to its obturation, or motility disorders. A change of ezofageal lumen can be caused from inside or outside. Clinical examination begins with inspection and palpation and continues with examination of the hypopharynx and larynx (indirect or direct hypopharyngolaryngoscopy). X-ray examination of the esophageal passage is done as native and with the contrast barium mass. At suspected penetration injury to the esophagus, a contrast barium mass has to be replaced by iodine contrast preparations.

Endoscopic examination of the esophagus - *esophagoscopy* - is a frequent direct examination method. Based on esophagoscope type, we distinguish between flexible esophagoscopy, more often performed by a gastroenterologist, and more rarely rigid, which is used in otorhinolaryngology. Indications for endoscopic examination may be for diagnostic or therapeutic reasons, often at the same time together.

Rigid esophagoscopy is performed under general or local anesthesia. An important criterion for choosing the type of anesthesia is the age, general condition and presumed diagnosis of the patient. The dominant indication for rigid esophagoscopy is a wedged foreign body. The instrument has a better gripping ability than the flexible technique.

Flexible esophagoscopy is preferably used in diagnostic procedures. Local anesthesia is preferred.

1.3 DISEASES OF THE ESOPHAGUS

Diseases of the esophagus are divided into acquired and congenital. *Congenital* esophageal *defects* include atresia, possibly with tracheoesophageal fistula, dilatation, and short esophagus (*brachyoesophagus*). The treatment is surgical.

Acquired esophageal *disorders* include inflammation, burns, foreign wedged bodies, tumors and esophageal diverticulum (*diverticulum, pharyngoesophagicum*). Diverticules are further divided into pulse (Zenker's diverticulum), traction and congenital.

1.3.1 Esophageal foreign bodies

The foreign body stuck in the esophagus occurs at any age. The spectrum of foreign bodies is wide (Figs.2,3). In childhood, the most common are pieces of toys, coins, in older age bodies related to dental disorders - pieces of meat, bones, teeth prostheses, etc. In the past, typical and frequent injury arised from self-inflicted trauma in prisoners, when they swallowed, e.g. an open switch pin wrapped in breadcrumbs. Most foreign bodies are stuck in the first physiological narrowing (above Kilian's sphincter). Large foreign body is usually stuck in the hypopharynx, with obturation of larynx and subsequent suffocation.

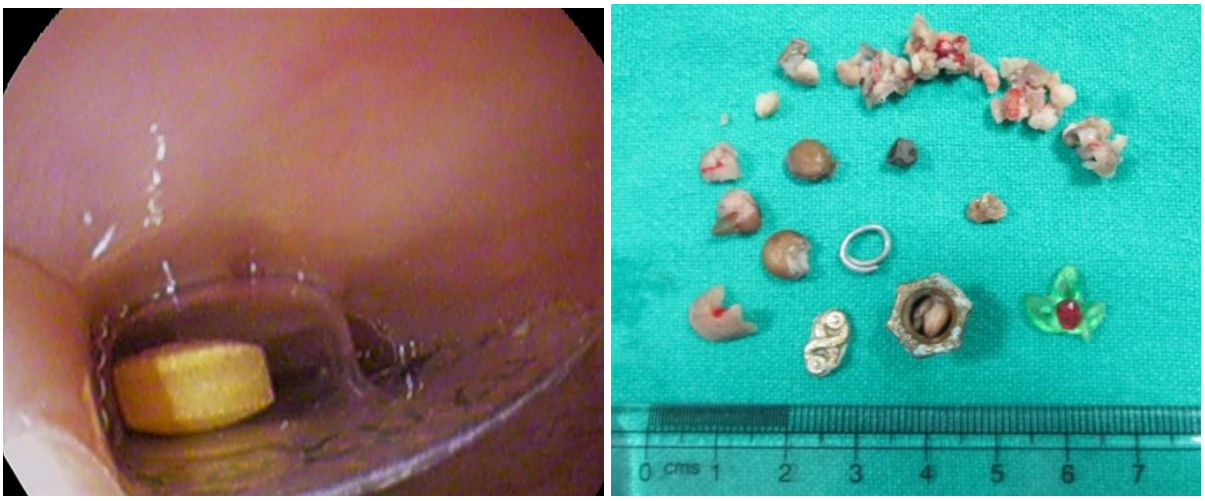

Fig.2 (left) Endoscopic image of foreign body in esophagus, female, 53 y.old by melvil is licensed under CC BY-SA 4.0. To view a copy of this license: https://creativecommons.org/licenses/by-sa/4.0/?ref=openverse, unchanged)

Fig.3 (right) Multiple esophageal foreign bodies in an infant; a rare case of serious parental neglect (from Chakravarti et al., 2016, https://creativecommons.org/licenses/by-nc/3.0/, unchanged)

Symptoms: difficulty and painful swallowing, inability to swallow even saliva, pressure behind the sternum, increased salivation

Diagnosis: anamnestic data, indirect hypopharyngolaryngoscopy, according to the character of the foreign body X-ray native or contrast (no barium mass at risk of esophageal perforation!)

Treatment: esophagoscopy with removal of a foreign body is indicated, to be performed as soon as possible in order to prevent the pressure ulcers and life-threatening complication - mediastinitis. If perforation is suspected, a control X-ray examination of the esophagus is indicated. For perforation, parenteral antibiotic treatment is given, in some cases surgical revision of the injured area and complete parenteral nutrition. Oral food intake is contraindicated. A serious complication of the surgery is iatrogenic perforation of the esophagus. Postoperatively, it is necessary to monitor the patient in the intensive care unit. In rare cases, when it is not possible to endoscopically remove the foreign body, an external surgical approach is indicated.

1.3.2 Esophageal burns *(corrosive oesophagitis)*

Esophageal burns occur by accident or even intentionally by swallowing various substances that have different mechanisms of action. Coagulation necrosis occurs after acid burns. After alkali burn, colliquation necrosis affects the deep structures of the esophageal wall. Changes in pharynx depend on the amount and concentration of substance ingested. The mucosa or even the submucosal tissue and muscle can be damaged. In the most severe forms of burn the esophageal wall is perforated, with consequent life-threatening mediastinitis. An adverse effect of burn repair is stenosis which occurs approximately 1-2 months after injury.

Symptoms: burning sensation, pain in the mouth, behind the sternum and in the epigastrium, vomiting, increased salivation, but inability to swallow the saliva. General symptoms may result from shock, intoxication, renal failure and brain damage.

Diagnosis: history, inspection (burnt lips, oral cavity), esophagoscopy. The extent of changes in the oral cavity may not correlate with the extent of esophageal damage.

Treatment: the administration of neutralizing solutions *per os* as a first aid is highly discussed. Gastric lavage through the probe is only possible if there is no risk of esophageal perforation. Anti-shock therapy, analgesics, antibiotics (prevention of secondary infection), corticosteroids are indicated. Complete parenteral nutrition is essential in extensive burns. As a rule, mucosal changes in the esophagus are of a similar extent and intensity as mucosal changes in the oral cavity and pharynx. After improvement of patient health status, we continue with corticosteroids orally, as they also have an antiproliferative effect (prevention of stenosis). During this period, the oral nutrition of the patient is restored. In the last stage, in about 2-3 months, stenosis can develop in the affected area, which again worsens oral food intake. It is necessary to identify this problem by X-ray of the esophageal passage, followed

by endoscopic dilatation of stenosis. Dilatations are performed by the otorhinolaryngologist with conically enlarging rigid dilatation probes. The gastroenterologist dilates the stenotic areas with balloon probes. If dilation is not possible, stent placement is indicated. The last option is the resection of the stenotic area, or the extreme option is gastrostomy.
Prognosis: it is ambiguous due to possible recurrence of the stenosis.

1.3.3 Tumors of esophagus

Esophageal tumors are divided into benign and malignant. *Benign tumors* are rare. These include myoma, pedunculated fibroma, papilloma, hemangioma and cysts. Rarely, a pediculate polyp may occur. Of the *malignant tumors* the most common is carcinoma, which mostly affects men. The range of symptoms is wide, dominated by dysphagia and odynophagia. At tumors located in the middle part of the esophagus, the symptoms appear later. The diagnosis is made by esophagoscopy (Fig.4), native and contrast X-ray, and histopathology. Treatment is the responsibility of the surgeon and gastroenterologist.

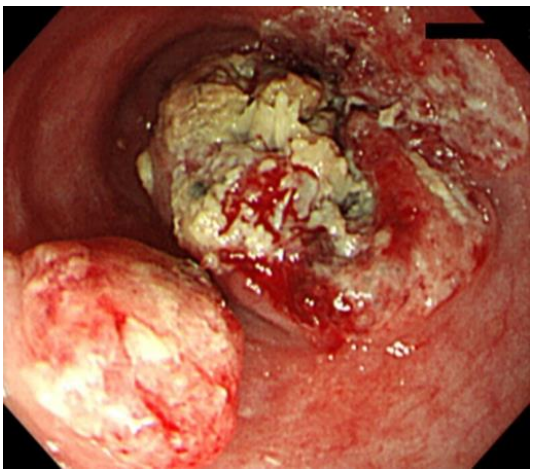

Fig.4 A large neoplasm detected in the lower esophagus at esophagoscopy (from Yamaguchi et al. 2017 under licence https://creativecommons.org/licenses/by/4.0/, unchanged)

Treatment: depends on the overall condition of the patient and the extent of the injury. These include anti-shock measures, respiratory protection (including intubation or tracheotomy), analgesics, antibiotics, corticosteroids, and in some cases surgical revision is indicated.

2 EAR

2.1 CLINICAL ANATOMY

The ear (stato-acoustic organ) consists of a peripheral and a central part. *The peripheral part* consists of the auricle, the outer auditory canal, the middle and inner ear, and *n. vestibulocochlearis*. *The central part* includes the nuclei in the medulla oblongata, auditory nerve pathways, supracortical and cortical centers. The auditory cortical center is located in the first two gyri of the temporal lobe, the terminal vestibular pathways project onto the cerebrum and cerebellum.

2.1.1 External ear *(auris externa)*

The outer ear consists of the auricle and the external auditory canal. The importance of the auricle in humans is questionable. Some authors refer to human ear as rudimentary organ. Auricle mobility in humans is significantly reduced. Its partial significance is in the better reception of sound waves. On the other hand, patients with auricle ablation did not suffer from significantly impaired hearing.

The auricle is made of preformed cartilage, covered with firmly adhering skin (Fig.5). Cartilage has no blood vessels on its own; nutrition is provided be perichondrium. In the lower part there is an earlobe, which does not have a cartilaginous base. The entrance to the external auditory canal is partially overlapped by tragus. The basis of the tragus is cartilage (the cartilage with perichondrium is suitable and often used material in reconstructive middle ear surgery). The external auditory canal is S-shaped, in the adult it is approximately 25 mm long. The S-shape protects the middle ear from direct injury. The ear canal is narrowest at the junction of the cartilaginous and bony part. The outer part is cartilaginous, the inner part is bony. The relationships between the posterior wall of the auditory canal and the pneumatic system of the temporal bone are important, especially for the spread of inflammation (mastoiditis). The anterior and lower walls of the bony portion of the auditory canal forms a part of the well of the temporo-mandibular joint. The ear canal is covered with skin, in the cartilaginous portion there is a subcutaneous connective tissue, hairs (visible mainly in older men) and sebaceous glands producing earwax. In the bony part of the auditory canal, the skin is softer, hairless, subcutaneous structures are reduced. The auditory canal is closed by the tympanic membrane, which has an almost vertical position in adults.

The newborn has only a cartilaginous part. The bone develops during 3th to 4th year of age. The tympanic membrane is almost horizontal and forms a sharp angle with the long axis of the auditory canal.

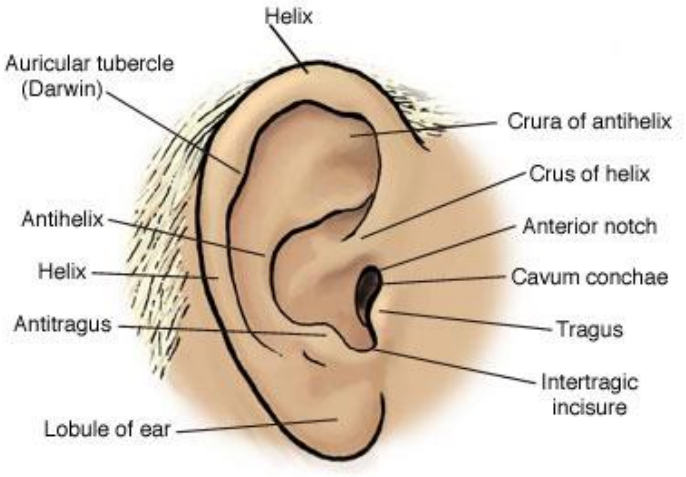

Fig.5 External ear (from https://medika.life/the-esophagus/ under a Creative Commons License CC BY-NC-ND)

Sensitive innervation is provided by branches trigeminal and vagus nerves (responsible for inducing a cough reflex when manipulating the external auditory canal) and branches of facial nerve.

The vascular supply is from *a. temporalis superficialis, a. auricularis posterior*, *a. auricularis profunda*. The lymphatic system of the outer ear is rich, the lymph drains into the pre- and retroauricular and parotic lymph nodes.

2.1.2 Middle ear *(auris media)*

The middle ear includes the tympanic membrane, the middle ear cavity, the auditory bones, the pneumatic system of the temporal bone, and the auditory (Eustachian) tube (Fig.6).

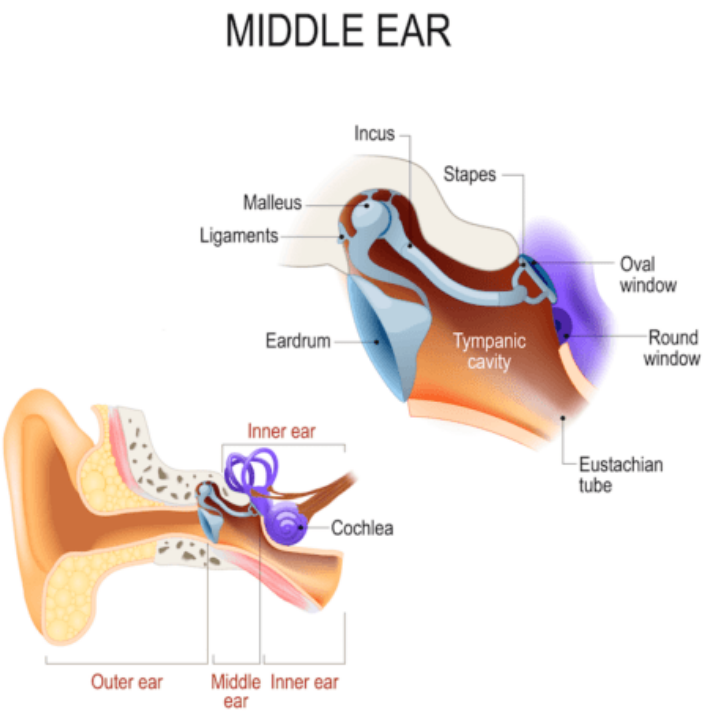

Fig.6 Middle ear (from https://medika.life/the-esophagus/ under a Creative Commons License CC BY-NC-ND)

The tympanic membrane (Fig.7) separates the external auditory canal from the middle ear cavity. It is thin, at the edge, except for the upper part, reinforced by a fibrocartilaginous ring (*anulus fibrocartilagineus*). The ring is inserted into the tympanic sulcus (*sulcus tympanicus*). The membrane of the drum is in the shape of a shallow funnel, open to the external auditory canal.

The central part of tympanic membrane is retracted, this place we call *umbo membranae tympani*. The lateral processus of malleus is fused with tympanic membrane and created smaller elevation on membrane *processus mallearis* .The manubrium of malleus is fused with tympanic membrane. This connection created typical anatomical structure *stria mallearis* . / It is structure between umbo and processus mallearis . Forward and backward from processus mallearis we describe front and back mallear strips /*plica mallearis anterior et posterior*. Between these strips is located *plica chordae tympani*, which one lifting tympanic membrane and created *plica chordae tympani*.

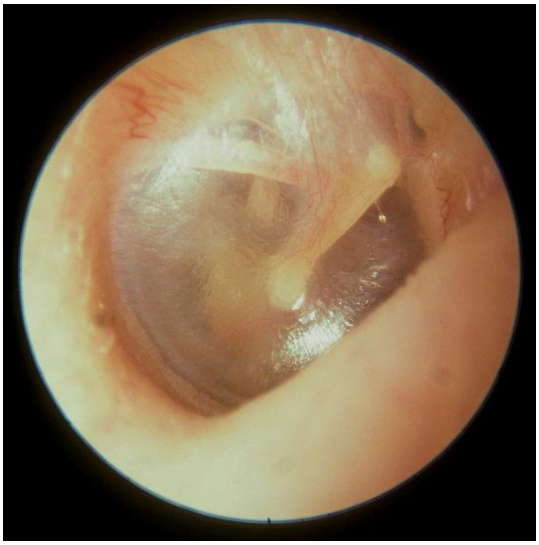

Fig.7 Physiological picture of the right tympanic membrane. The chorda tympani nerve passing lateral to the long process of the incus. (by Michael Hawke MD, CC BY 4.0 <https://creativecommons.org/licenses/by/4.0>, via Wikimedia Commons)

A triangular light reflex is directed from the umbo caudally and forwards. The reflex together with *stria mallearis* and *prominentia mallearis* form so called the Bezold´s triad, which represents the three basic physiological features of the tympanic membrane. As mentioned above, the adult tympanic membrane is set almost perpendicularly to the long axis of the external auditory canal. The part of the tympanic membrane placed above both *plicae malleares* is thin, less stretched, it is called *pars flaccida* (*membrana Schrapnelli*). The lower part of the membrane is thicker, tightly stretched and is called *pars tensa*. From a histological point of view, there is a significant difference between the two structures. *Pars tensa* consists of three layers. The epidermal layer is a continuation of the skin of the outer auditory canal. The middle layer is important for elasticity. It consists of elastic fibers, which are arranged circularly in the inner part and radially in the outer part. The third, inner layer is formed by the epithelial (mucosal) lining, covering the middle ear cavity. *Pars flaccida* is thinner as it does not contain a middle elastic layer.

The outer part of the membrane receives the blood supply through *a. manubria mallei externa et interna* and *a. auricularis profunda*. The medial area is supplied through *a. tympanica*. The outer side of tympanic membrane is innervated from *r. membranae tympani n. auriculotemporalis* and *r. auricularis n. vagi*. The inner surface is innervated by fibers from *plexus tympanicus*.

2.1.2.1 The tympanic cavity (the middle ear cavity)

The tympanic cavity is in the shape of a biconcave lens or the sand-glass and is bounded by 6 walls. From a clinical point of view, it is divided into three parts, which are highlighted by extending the upper and lower lines of the auditory canal wall. The upper part lies behind and above the level of the tympanic membrane (the interface of *pars flaccida* and *pars tensa*), it is called the epitympanum (*recessus epitympanicus*). The middle part is the mesotympanum and the lower part, located behind and below the level of the tympanic membrane, hypotympanum. The lateral wall of the middle ear cavity is made of the tympanic membrane, the medial wall consists of the lateral part of the bone labyrinth capsule of inner ear. The beginning of the basal turn of the cochlea protrudes into the tympanic cavity as a *promontorium*. There are two openings on the laryrinth (medial) wall of the middle ear, separated by a membrane from the inner ear - an oval window (*fenestra cochleae*), into which the base of the stapes is inserted and the free round window (*fenestra vestibuli*) . The anterior wall separates tympanic cavity from the carotid canal. For its intimate relationship with internal carodid artery it is also called as carotid wall. It containes the openning of the auditory canal. On the mastoid - posterior wall (*paries mastoidea*) there is an opening (*aditus ad antrum mastoideum*) through which one the middle ear communicates with the pneumatic system of the temporal bone. The upper wall *(paries tegmentalis)* is thin, sometimes perforated at the site of the *tegmen tympani*. It separates the tympanic cavity from the middle cranial fossa. The spreading middle ear infection to the intracranial space throuh this wall was relatively frequetly in the past. The lower wall *(paries jugulare)* is in contact with the *bulbus v. jugulare*. The middle ear mucosa is covered by a thin cubic epithelium. Vascular supply is provided by *a. stylomastoidea*, *a. meningea media* and *a. pharyngea ascendens*. The innervation is provided by the *plexus tympanicus*. Middle ear cavity is filled by the air and it s conected through *tuba auditiva* / *Eustachian tube*/ with epipharynx. The free connection is the basic condition for good function of middle ear.

2.1.2.2 Auditory ossicles

Three middle ear ossicles include *malleus*, *incus* and *stapes*. Under physiological conditions, they form a moving chain. Together with the tympanic membrane, they belong to the structures that significantly amplify the sound in the middle ear area. Stapes is the smallest ossicle in the human body. The weight of malleus (adult person) is approx. 22 mg, incus

approx. 27 mg and stapes 3,3 mg. The width of stapedial footh plate is approx 2,7 mm. There are two striated intratympanic muscles: *m. stapedius* and *m. tensor tympani*. *M. stapedius* has a protective function - it contracts when exposed to excessive noise, which increases the resistance of the middle ear apparatus and dampens sound transmission. *M. tensor tympani* stretches the eardrum.

2.1.2.3 Auditory (Eustachian) tube (*tuba auditiva)*

The auditory tube connects the tympanic cavity with the nasopharynx (Fig.8). It balances the air pressure in the middle ear cavity with atmospheric pressure and ensures the drainage of fluids from the middle ear. The lateral part is formed by the bone, which ensures a constant size of the lumen. The medial part is cartillaginous, the lumen is slit-shaped and changes its size. Auditory tube is no permanently open. System open and close is changed during day many times. The lumen change is particularly important in deglutination, where the tube opens and the pressures equalize. The mucosa consists of multilayered ciliary epithelium. Cilia oscillate towards the nasopharynx and are involved in the mucociliary transport for passing away secretion from middle ear to nasopharynx. The tube is long approx. 3,5 – 4,0 mm, directed forwards and down. Children has relatively very short and widerly open tube. It is one of the causes of frequently middle ear infection in this group – spreading infection from nasopharynx. Vascular supply is provided by *a. pharyngea ascendens, a. maxillaris, a. meningea media, a. carotis interna*. The innervation is provided by fibers of *n. vagus, n. glossopharyngeus, n. facialis,* and motor fibers of *n. trigeminus*.

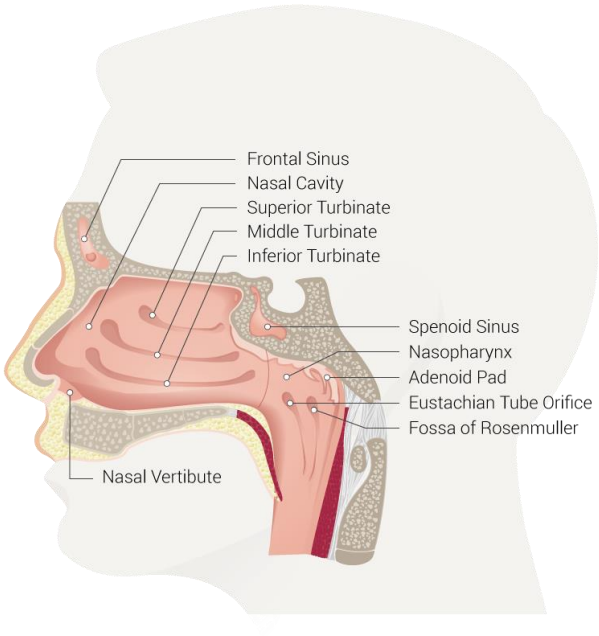

Fig.8 The orifice of Eustachian tube to nasopharynx (from Szymanski A, Agarwal A. Anatomy, Head and Neck, Ear Eustachian Tube. [Updated 2022 Jun 27]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2022 Jan-. Available from: https://www.ncbi.nlm.nih.gov/books/NBK482338/)

2.1.3 Inner ear *(auris interna)*

The inner ear is located in the hardest bone of the human body (petrous part of temporal bone – *os petrosum*). It consists of a bone capsule and a membranous labyrinth (Fig.9). The bony capsule consists of an atrium (*vestibulum*), three semicircular canals (*canales semicirculares ossei anterior, posterior et lateralis*) and a cochlea. The vestibule has an oval window (*fenestra vestibuli*) on the lateral wall, a *recessus utriculi* on the inner and a *recessuss sacculi* caudally. In these structures lie the *utriculus* and *sacculus* of the membranous labyrinth. The semicircular channels are interconnected in the vestibule and organized in mutually perpendicular planes. From a clinical point of view, the lateral canal has a special place. It is placed in a horizontal plane and in chronic inflammation of the middle ear the bone capsule may be destroyed. Afterwards, the toxins enter the inner ear, which is manifested clinically by vestibular symptoms of varying degrees.

The cochlea is formed by a 2.5 turns of the spiral around a bony spindle (*modiolus*). A bony plate (*lamina spiralis ossea*) protrudes from the modiolus into the hollow system of the cochlea and divides it incompletely into two parts (*scala vestibuli* and *scala tympani*). At the top of the kochlea these two compartments are interconnected *via* a *helicotrema*. *Scala*

vestibuli leads to the vestibule, *scala tympani* faces the oval window. The length of the kochlea is about 35 mm. The membranous labyrinth consists of two parts - auditory (*pars auditiva*) and static (*pars statica*). The static part consists of three semicircular canals (*ducti semicirculares*), *utriculus* and *sacculus*. *Utriculus* and *sacculus* are placed in the atrium. They are interconnected by a duct (*ductus utriculosaccularis*) from which the endolymphatic duct (*ductus endolymphaticus*) protrudes. The endolymphatic duct penetrates through the back wall of the pyramid into the endolymphatic sac (*sacculus endolymphaticus*) near the sigmoid sinus. On the medial wall of utriculum and sacculum there are special static hair sensory cells (*macula statica utriculi* and *macula statica sacculi*). The hairs are covered with an elastic layer containing position crystals (*statoconia*). Three semicircular ducts emerge from the sacculus. Each duct/canal has one arm enlarged (*crus ampulare*), the other is simple (*crus simplex*). *Crus membranacea simplex* of the upper and posterior semicircular duct merge into a common arm (*crus membranaceum commune*). In the ampular part, there is a ridge inside (*crista ampullaris*) which is placed perpendicular to the axis of the canal. *Crista ampularis* contains sensory hair cells. The membranous labyrinth is filled with endolymph. The cochlear duct *(ductus cochlearis)* has a triangular shape on its cross section. The upper, atrial wall facing the *scala vestibuli* is formed by the atrial membrane (*membrana vestibularis Reissneri*). The lower wall, facing the *scala tympani*, is formed by the *lamina basilaris*. This wall/lamina is a site of the sensory Corti´s organ, mediating the transformation of mechanical - sound energy into electric potentials. Organ of Corti consists of two obliquely placed Corti's pillars, between which there is free space - Corti's tunnel. There are hair cells on both sides of the tunnel. The outer cells are in three rows and inner in one row. There are also supporting Deiters, Hensen and Claudius (at lowest etage) cells. There are approximately 3.500 inner and 15.000 outer hair cells in the human cochlea. The surface of all cells is covered by a tectorial membrane (*membrana tectoria*), through which the hairs of the sensory cells grow. The hair cell bodies are attached to the dendrites of ganglion cells.

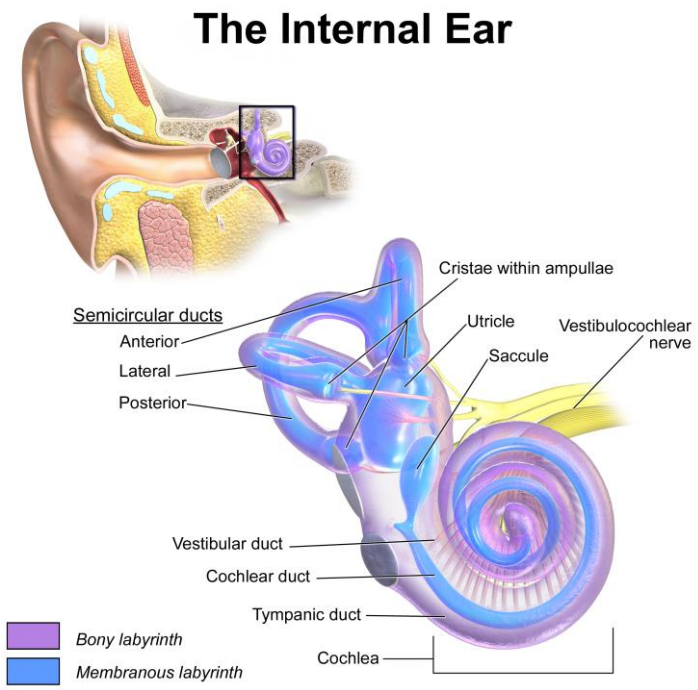

Fig.9 Inner ear anatomy (Szymanski A, Geiger Z. Anatomy, Head and Neck, Ear. [Updated 2022 Jul 25]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2022 Jan. Available from: https://www.ncbi.nlm.nih.gov/books/NBK470359/)

The cavities of the membranous labyrinth are filled with a endolymph. The space between the bony and the membranous labyrinth is filled with a perilymph. The endolymph is characterized by a high potassium and low sodium concentration. The mineral composition of the perilymph is opposite, it has a high Na^+ and low K^+ levels. The different mineral composition is important for transformation of mechanical energy to the electrical potentials in the Corti's organ.

Vascular supply of inner ear is done by *a. labyrinthi*, terminal branch of *a. cerebelli inferior* or *a. basilaris*. The inner ear has no lymphatic vessels.

2.1.4 Otoscopy

It is a basic examination of the external auditory canal and the tympanic membrane with a sight (inspection). Based on changes of the tympanic membrane, we can evaluate pathological process in the middle ear (e.g., inflammation). A headlight and a funnel-shaped ear mirror with different diameters are used, depending on the patient's age. We grasp the ear of the patient with one hand and pull it dorsolaterally, slightly upwards. This maneuver compensates

for the sigmoidal shape of the auditory canal. Subsequently, we introduce an ear mirror and examine the structures of the auditory canal and the tympanic membrane. If the patient is examined outside of a specialized workplace, we use an otoscope with a built-in light source and a magnifying glass (Figs.10,11).

For a detailed examination of changes of the tympanic membrane, perfoming microprocedures on tympanic membrane, it is recommended to use a microscope. The method is called otomicroscopy.

Fig.10 Otoscope (by One Candle Photos is licensed under CC BY-ND 2.0. To view a copy of this license, visit https://creativecommons.org/licenses/by-nd/2.0/?ref=openverse.)

Fig.11 Otoscopic exam (by eliaspunch is licensed under CC BY-NC-ND 2.0. To view a copy of this license, visit https://creativecommons.org/licenses/by-nd-nc/2.0/jp/?ref=openverse)

2.2 PHYSIOLOGY OF THE ACOUSTIC ANALYZER

The sound wave is picked up by the ear and then propagated through the external auditory canal. The sound waves come into the contact with the eardrum and vibrates it. The vibrations are then transmitted through the middle ear ossicular system to the cochlear part of the inner ear. The transmission system of the middle ear together with the tympanic membrane works on the principle of an "amplifier", which is given by the different size of area the tympanic membrane (approximately 55 mm^2) and the base of the stapes (3.2 mm^2). The basic conditions for the sound transmission are continuous external auditory canal, an intact tympanic membrane, the mobility of the ossicles and the situation in the tympanic (middle ear) cavity as pressure and absence of the content. The equal pressures in the external auditory canal and in the middle ear are considered optimal, at altered pressure situation can disrupt sound transmission. It is so-called resistance (impedance) of the gearing middle ear system. Mechanical energy is further transmitted through the base of the stapes to the inner ear. In the cochlea, the mechanical energy is changed to bioelectrical one. Subsequently, it is transmitted through the auditory pathway to the hearing center in the Heschl´s gyrus of the temporal lobe of the cerebral cortex. The mechanism of hearing is not entirely clear at this moment.

In general, Bekesy theory of hearing is accepted. The movement of the stapes through the membrane of the oval window causes the perilymph to move. A “wandering” wave that travels through the cochlear structures is originated. As the fluid is incompressible, the membrane in a round window finally flares outward. The moving pressure wave gradually oscillates the tone-specific ends of the hair cells of the Corti´s organ. After reaching a certain stimulation value in the hair cells, an action potential is created. Action potencial is subsequently summed from the individual hair cells and is removed by the afferent cochlear part of cranial nerve VIII *(n. vestibulocochlearis*). The hair cells of the Corti´s organ are sound-frequency specific. The distribution is typical - on the basal turn the cells are sensitive to higher tones, on top to lower tones.

2.3 PHYSIOLOGY OF THE VESTIBULAR ANALYZER

The perception and maintenance of the body position is ensured by the coordinated activity of the vestibular part of the inner ear, cerebrum, cerebellum, peripheral nervous system, visual analyzer and the activity of the skeletal muscles.

The vestibular system with sensory cells in the *macula utriculi* and *macula sacculi* serves to perceive the position of the head in space and responds to accelerated linear motion (the so-called otolith system). The system of membranous semicircular canals responds to angular acceleration and rotational motion. Similar to the auditory analyzer, by stimulating specific hair cells with an endolymph, a bioelectrical action potential is generated, which is then propagated centrally by the vestibular part of cranial nerve VIII to the CNS.

If the rotational movement of the head is uniform and steady, the movement of the endolymph in the semicircular canals also stabilizes over time, so that the hair cells stop generating action potentials. If the body movement slows or stops, the movement of the fluid by continues inertia. Physiological stimulation of the vestibular system is not perceived by humans. With strong stimulation of the vestibular apparatus (above the threshold level), uncoordinated skeletal muscle activity and disorders of the autonomic nervous system occurs.

2.4 HEARING DISORDERS AND HEARING EXAMINANTION

The quality of human hearing is relatively low compared to some animals. In general, we the tones are divided according to the spectrum into pure (with the same frequency) and mixed. Sound is the part of the spectrum of mechanical waves of air that man is able to perceive. The unit of frequency is Hertz (Hz). Another important characteristic is intensity; the unit is decibel (dB). One is able to perceive a relatively wide frequency band between approximately 20-20.000 Hz. Lower or higher values are not perceived by human hearing. Sound with a frequency lower than 20 Hz is called infrasound (perceived by elephants, for example), with a frequency higher than 20,000 Hz is called ultrasound (perceived by bats up to 150,000 Hz). The lower limit is relatively stable, the upper limit varies significantly and decreases very rapidly with age. The band in the range of 250 - 4000 Hz is important for verbal communication. Hair cells sensitive to sounds in this band are relatively resistant to damage and remain fully functional for a long time. If the patient is able to hear sound up to 20 dB,

hearing at this frequency is normal. The intensity of ordinary speech is in the range of 40 - 45 dB. Sounds with an intensity of 90 dB and more can cause hearing loss, tones with an intensity above 130 dB cause a painful sensation. The rate of onset and extent of hearing loss is very individual, depending on the nature of the sound, the time of exposure, etc.

2.4.1 Hearing disorders

Hearing disorders are divided into conductive (*hypacusis conductiva*), sensorineural, also called perception (*hypacusis sensorineuralis*) and mixed (*hypacusis mixta*).
The type of hearing disorder depends on the location of the problem that leads to the impairment. At pathologies in the external auditory canal and middle ear, there is conductive hearing disorder characterized by limited perception of sound with low frequencies. When the inner ear and higher structures are damaged, we speak of a sensorineural disorder with limited perception of sounds at high frequencies. In the case of an isolated cochlear dysfunction, we talk about *cochlear type* of disorder, at damaged of cranial nerve VIII and higher structures (and intact cochlea), it is *supracochlear type* of sensorineural hearing disorder. The combination of a conductive and sensorineural disorder results in a *mixed disorder*, with pathology at the level of the external auditory canal or the middle ear, and the inner ear or higher structures.
Complete deafness is called *anacusis*. Good hearing is important for speech development. In people with hearing impairment, the speech development is incorrect or delayed.
Practical deafness (*surditas practica*) is a condition where the remnants of hearing are not sufficient for the everyday life of the patient, even with the use of compensatory tools (hearing instruments). As a limit value of the average hearing threshold at tone audiometry without a hearing device the level of 90 dB and more is considered. According to its origin, the deafness is divided into *prelingual* - it occurs before the development of speech (approximately until the 2 year of age), *perilingual* - it occurs during speech development, before its fixation (3 - 6 year of age) and *postlingual* - after speech development.
In the case of cochlear disorder, it is possible to restore hearing with cochlear implants. Even if cochlear implants are relatively expensive, they arepart of standard therapy.

Under standard conditions, sound information is transferred through all auditory pathways to central nervous system. It is so-called *air conduction*, which uses external auditory canal,

middle ear and inner ear. This type of sound conduction is used for normal verbal communication. When sound propagates through the skull directly to the auditory receptors (out of exernal ear and middle ear), the cochlea is directly stimulated, it is called *bone conduction*. By comparing these two types of sound conduction, we can determine hearing loss. In general, air conduction is more efficient due to the amplifying role of the middle ear.

2.4.2 Hearing examination

The examination methods are divided into subjective (cooperation of the patients is required) and objective (without the patient´s cooperation).

2.4.2.1 Qualitative subjective hearing examination

The simplest qualitative hearing test is performed by a loud and whispered speech. These methods are used in basic screening. The principle of the examination is to determine the greatets distance between the patient and the examiner, which allows the patient repeat the words correctly after the examiner. Both sides (ears) are examined separately. The patient stands sideways to the examiner in a quiet room and covers his eye on the given side to exclude reading from the lips. Hearing through the other ear is avoided by Barany box (inserted in the ear and creatings a loud buzzing sound), or simply by pushing the finger on the tragus, when external ear canal is blocked . Simple words, used in everyday life, are used for testing. To examine higher sound frequencies, we use words containing sibilants (e.g. essence, sleep, glass, scent), to evaluate low frequencies the words with containing deep tones are used (oak, lamp, one ...). The physiological limit is a distance of 6 meters. Whisper is investigated by residual air that remains in the lungs after normal expiration. The examiner starts from a distance of 3 m and depending on the patient's reasponse, this distance increases or decreases. The loud speech is investigated by similar way.

Another option is to test hearing with tuning forks. The set contains tuning forks with a frequency from 32 to 4096 Hz. The first, we perform an orientation examination with a whole set of tuning forks. We gradually place the sounding tuner in front of the auricle. The patient indicates whether he/she hears the sound and whether the intensity of the sound is the same. An impaired perception of tuning forks with lower frequencies indicates a conductive disorder, problems to hear higher frequences is indicative for sensorineural hypacusis. To better determine the type of hearing disorder, we use a special tuner with a frequency of 512

Hz and compare the quality of air and bone conduction of sound. The air conduction is examined by placing the sounding tuner next to the auricle, the bone conduction by placing it on *planum mastoideum*.

Rinne (R) test compares bone and air conduction in one side. Vibrating fork is placed close to the auricle (air conduction). When the patient stops to hear it, the fork is placed on the *planum mastoideum* (bone conduction). If the patient hears longer by air conduction, we assume that his/her hearing is normal or there is perception hearing impairment. The Rinne test is positive (R+). The opposite result is called Rinne negative (R–); we assume the conductive, or mixed hearing disorder.

Weber (W) test compares simultaneously the bone conductions in both ears. We place the vibrating fork on the front part of the skull or on its top in the midline. If the patient hears the sound equally in both ears, he/she "does not lateralize" (←W→). If the hearing quality in the ears is different, we call it "lateralization" (referred to as ←W or W→). The arrow points to the side of better hearing. In the case of conductive disorder, the sound is lateralized to the more impaired ear (bone conduction is better), in the case of a perception (sensorineural) disorder it is lateralized to the healthy, or better hearing ear.

Schwabach (Sch) test compares the patient's bone conduction unilaterally with the examiner, provided that the examiner's hearing is physiological. The vibrating fork is placed on the patient's mastoid process. When patient stops hearing, the examiner places it to his/her own planum mastoideum without further vibrating (using residual vibrations). Then the examination is performed in the reverse order. If the time interval is the same, bone conduction (function of the patient´s cochlea and higher structures) is normal. If the patient hears by bone conduction for longer, the Schwabach is prolonged meaning the conductive hearing impairment at that side. If the bone conduction is shorter, Schwabach is shortened meaning the patient has a sensorineural disorder.

Gelle (G) test is a very useful, simple test used at suspected fixation of the middle ear transmission system. The principle is the air pressure change in the external auditory canal during direct stimulation of the inner ear and higher structures with a tuning fork (bone conduction). If the middle ear system is working well, the pressure change is transmitted

directly to the inner ear. Under physiological circumstances, when the pressure increases, the perception of the sound produced by tuning force placed to the planum mastoideum decreases, when the pressure equalizes, the perception increases (G+). If there are no changes in the perception of sound when the pressure in the external auditory canal changes, we may assume the disorder of the middle ear system at the intact tympanic membrane (G–), i.e. conductive hearing loss. However, it should be emphasized that a definitive diagnosis cannot be based on the single test. The patient must also undergo an examination by loud speech and whispering, as well as a complete examination with tuning forks, imaginazing methods – high resolution CT (HRCT).

2.4.2.2 Quantitative subjective hearing examination – audiometry

Audiometric examination is more detailed and it allows more precise specification of hearing disorders. It is devided to pure-tone threshold audiometry and speech (verbal) audiometry. The undergraduate students should become familiar with the principles of pure-tone threshold audiometry. The principle of the examination is the exact specification of hearing loss in terms of determining certain frequency disorder and the rise of the hearing threshold. The hearing threshold is the value of the minimum acoustic energy (volume) at a given frequency that the patient is able to hear. Thus, if we say that the threshold of hearing increases, it is necessary to stimulate the patient with a tone of higher intensity (louder tone).

We usually investigate the frequency band in the range of 125 - 8000 Hz. The aim is to determine the minimum intensity of acoustic energy at a given frequency in air and subsequently in bone conduction. The results are tabulated and the final recording is called an *audiogram*. Air conduction is marked with a solid line, bone conduction with dashed line. Hearing on the right is marked in red, on the left side in blue. If the patient hears at the air conduction tones up to 15 - 20 dB in all frequencies, hearing is normal. If it is necessary to use a higher intensity, while the bone and air conduction are identical, it is a *sensorineural hearing disorder* (disorder at the level of the cochlea and higher structures). A typical example is the *presbyacusis*. This term refers to an increase in the hearing threshold for high tones in relation to aging.

If the bone conduction is normal (up to 20 dB) and the air conduction is disturbed (more than 20 dB), the patient has a hearing loss (e.g., disconnection of the middle ear system, perforation of the tympanic membrane). In a situation where the bone conduction is weaker

(loss of more than 20 dB), but the air conduction is even weaker, the patient has a *mixed hearing loss* (e.g., chronic epitympanic otitis media).

One should be aware that by air conduction of sound, we examine the complete auditory pathway and by bone conduction we investigate the inner ear and higher structures, i.e. we "omit" the external auditory canal and middle ear. It means that in an audiometric test, air conduction can never be better than bone conduction (the benefit of the middle ear "amplifier" does not apply here – only during audiometry test).
By detailed audiometric examination, special tests, it is possible to specify in more detail the sensorineural hearing disorder (cochlear vs. supracochlear type).

2.4.2.3 Objective examination methods

Patient cooperation is not necessary in objective examination methods; it means they can be used in infants as well as in patients with expected simulation or aggravation. The methods are different: we can examine hearing, the resistance of the middle ear apparatus or to do a simple screening of hearing in newborns - otoacoustic emissions.

2.4.2.3.1 Tympanometry

It is a simple, non-invasive examination of the resistance of the middle ear apparatus. Under physiological conditions, the air pressure in the outside environment and the middle ear is the same. As the pressure changes, the tone and position of the tympanic membrane varies as well, and its flexibility is being changed. Membrane compliance and middle ear content are most important factors determining the resistance of the middle ear system, and also affect the sound transmission. Increased, but also reduced resistance leads to conductive hearing disorder.

2.4.2.3.2 Objective audiometry

It is an examination of hearing by evoked potentials. There are several techniques based on the place of the registration of potential: electrocochleography (cochlear recording), brainstem audiometry (BERA - brain stem electric response audiometry) and cortex audiometry (CERA – cortex electric response audiometry).

The principle is the stimulation with the equal, short, repetitive sound stimuli in the same intervals and registration of the evoked potentials. It is similar to the classical encephalography (EEG). The change in electrical potentials in relation with stimulation is evaluated. Based on the magnitude or latency of potentials we can determine hearing loss. As this is a borderline issue in medicine, in some medical facilities these examinations are performed by neurologist.

2.5 DISEASES OF THE EAR

2.5.1 Congenital anomalies of the ear

There are a large number of congenital anomalies of the ear. In the past, the prevailing opinion was that most of them are genetically determined. This idea is not fully accepted, only about 5-20% of all congenital anomalies are inherited and the rest are acquired during intrauterine development. The most important are the factors that affect embryonic development in the first 3 months of intrauterine life. These include viral diseases of the mother (such as rubella) in the 8^{th} and 9^{th} week of pregnancy, which can impair the embryonal development of hearing. Some drugs used during pregnancy have a similar effect, which can be manifested in newborns by deformities and disorders of the development of the outer, middle and inner ear, facial nerve palsy and the like.

Congenital disorders of the external ear (Fig.12) can be associated with disorders of the middle and inner ear. The development of the facial part and the lower jaw may also be impaired. The auricle can be developed excessively (*macrotia*), insufficiently (*microtia*) or may be absent (*anotia*).

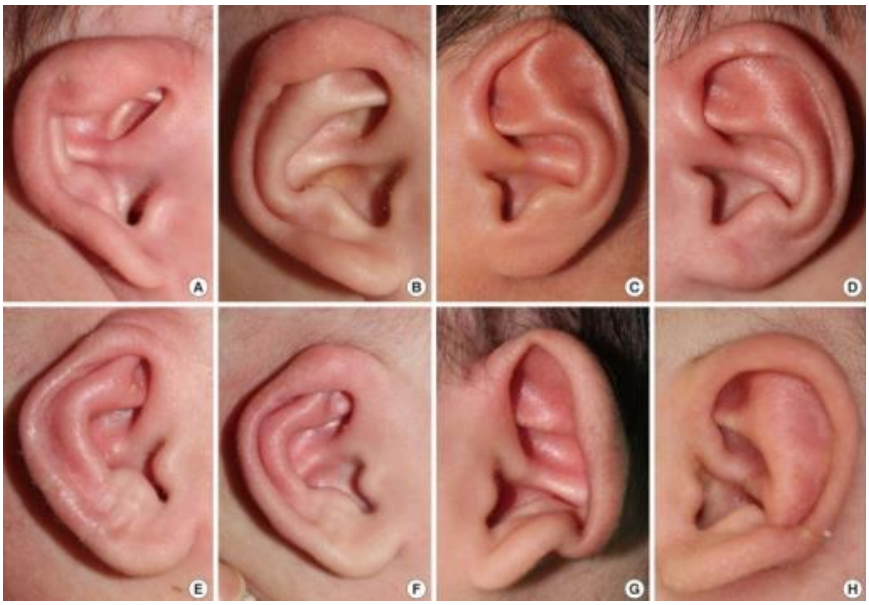

Fig.12 Examples of preprocedure and postprocedure photographs of patients with favorable outcomes. (A) Constricted ear, preprocedure; 23 days old. (B) Constricted ear, postprocedure; after 26 days, at the end of treatment. (C) Stahl ear, pre-procedure; 29 days old. (D) Stahl ear, postprocedure; after 32 days, at the end of treatment. (E) Cryptotia, preprocedure; 9 days old. (F) Cryptotia, postprocedure; after 26 days, at the end of treatment. (G) Prominent ear, preprocedure; 14 days old. (H) Prominent ear, postprocedure; after 25 days, at the end of treatment. (from Woo et al., 2016 under licence https://creativecommons.org/licenses/by/4.0/, unchanged)

The ears are sometimes attached to the skull (*cryptotia*) or, conversely, they stand out very noticeably (*apotasis auriculae*). Deviations in the relief of the ear are also common. Some anomalies are inconspicuous, such as Darwin's hump, others may resemble the ears of animals (cat's ear, monkey's ear). There are also congenital fistulas, several millimeters long channels that end blindly. They have a fibrous wall covered with squamous epithelium. They most often occur in the preauricular region before the *crus helicis*. If they get infected, they tend to be painful. Then they appear as an inflammatory infiltrate that can be seen in front of the auricle.

Atresia of the external auditory canal occurs in connection with an underdeveloped auricle, or congenital disorders of the middle ear, such as an underdeveloped tympanic cavity, ossicles, stapes fixation, and the like. Congenital inner ear disorders affect both the bony and the membranous labyrinth. Several pathological changes may occur: reduced number of cochlear turns, insufficiently developed Corti's organ, deformities or defects of the utriculus, sacculus, semicircular canals, nerve degeneration and the like.

The most common are two congenital dysplasias of the inner ear:

1) **Scheibe dysplasia**

It affects phylogenetically younger parts of the inner ear (sacculum and cochlea). In the area of *stria vascularis*, the areas of aplasia and hyperplasia alternate. Reissner's membrane tends to be weak and is located on the stria and on the rudimentary Corti´s organ. The supporting components of the organ are disrupted and the hair cells are less or completely missing.

Cochlear neurons are not damaged and therefore it is possible to use electrical stimulation with a cochlear implant to establish hearing. This type of disorder accounts for 70% of the causes of congenital deafness.

2) **Mondini dysplasia**

It is a dysplasia of bony and membranous structures, which leads to a hereditary-degenerative type of deafness. The number of turns is reduced to 1.5. The Corti´s organ may be missing or reduced to a group of undifferentiated cells. This type of dysplasia is well identified by CT scan. In very rare cases, the entire labyrinth is missing. These disorders may rarely be associated with congenital malformations of the outer and middle ear.

Measures:

1) **prophylaxis** – exclusion of predisposing factors, examination of Rh-compatibility of parents (or mother and fetus).
2) **therapy** – functionality is assessed, but also an esthetic aspect. Reconstructive surgery is used, in case of auricle defects it is possible to implant an artificial one (Silastic). There is a wide range of reconstruction procedures for the middle ear. In some cases, hearing aids are used to rehabilitate hearing, such as cochlear implants ("artificial inner ear").
3) **education**
    - Auditory training
    - Lip reading
    - Sign language
    - Speech training
    - Role of the family

Currently, the number of children with congenital deafness or partial hearing impairment who can be educated in an integrated way in classical schools is increasing. Children who cannot

be integrated receive education in special schools. It is important for these children to be in the company of normally hearing people as much as possible, or to return to this environment as soon as possible.

2.5.2 Excessive production of earwax

Earwax (cerumen) is a mixture of excretions of the ceruminous glands, pilosebaceous glands and exfoliated keratin. It is formed in the cartilagous part of the auditory canal. The consistency of the wax depends on the proportion of its components. Under normal circumstances, it is excreted from the channel in the form of scales, while its movement is aided by the movement of the jaw. In some people, earwax plug (*accumulatio ceruminis*) forms, which is a result of excessive production and retention by hair in the auditory canal, desquamation, exostosis, and some other processes.

Symptoms: with complete obstruction of the auditory canal, conductive hearing loss, sometimes tinnitus ("ringing" in the ear), pain and dizziness.

Treatment: rinsing the ear canal with a syringe filled with water heated to body temperature, removal with a probe, a hook.

2.5.3 Inflammation of the ear

2.5.3.1 Inflammation of the external ear (*otitis externa*)

The external auditory canal is to some extent protected by secrets of the seborrheic glands. Inflammation of the skin of the auditory canal can occur under the influence of noxious factors. Inflammation of the external auditory canal is most often found in people with diabetes or in those who often bathe in chlorine and thermal water, or work in a humid environment. Erosion of the epidermis, which can occur, for example, when cleaning the ear or the external auditory canal, also plays an important role. The clinical picture of inflammation of the skin of the external auditory canal is the same as in other parts of the body.

2.5.3.1.1 Eczema *(eczema)*

In 90% of cases of dermatoses of the auricle or external auditory canal, there are various forms of eczema.

Etiology: in children it is usually constitutive. It is often caused by a skin allergy to various chemicals, bacteria, fungal toxins, or food allergens. The outer ear is sometimes the only location where skin eczema manifests.

Symptoms: in *acute* moist *form*, the skin is congested, macerated and covered with crusts. Sometimes a yellowish exudate leaks from the auditory canal. In *chronic eczema*, the skin is dry and peels off. Sometimes hyperplastic inflammation develops in the corium, leading to stenosis of the auditory canal. Skin affected by eczema is often infected with pyogenic microorganisms, especially when the patient is injured, due to intense itching and scratching. In this case, increased exudation and pain occur.

Treatment: the treatment is complicated and long-lasting. In moist eczema, substances with a disinfectant and astringent effect (1% resorcinol solution or "solutio Castelani") are used, in the case of secondary infection antibiotics combined with corticosteroids. In dry eczema corticosteroid ointments are usefull. It is important to exclude allergens. If the cause of eczema is chronic purulent otitis of the middle ear, it should be treated as a cause.

2.5.3.1.2 Furuncle of the external auditory canal *(otitis externa circumscripta, furunculus)*

Etiology: often caused by a staphylococcal infection /Staphylococcus aureus/of the sebaceous glands or hair follicle in the cartilaginous part of the external auditory canal, which contains skin adnexa. Sometimes when the posterior wall of the canal is inflamed, the tissues around the retroauricular groove become swollen and the auricle stands out. The condition resembles a subperiosteal abscess in mastoiditis. A special patient´s group are diabetics who are at risk for the uncontrolled spread of infection.

Symptoms: pain of varying intensity, which is exacerbated by pressure on the tragus, pulling of the pinna or chewing. The pain is usually sharp when the inflammation spreads to the perichondrium. Sometimes we detect auditory canal obstruction with a hearing disorder. Fever may not be present.

Diagnostics: inspection. In the case of differential diagnosis, it can sometimes be difficult to distinguish between furuncle and mastoiditis, especially because these conditions can occur simultaneously. Classical X-ray is not more used, CT examination of the temporal bone is indicated.

Treatment: local application of antibiotics and ointments, cleaning of the external auditory canal. At large swelling and temperatures above 38 °C, antibiotics are applied systematically. Incision and drainage are rarely indicated, only in the case of an abscess growth.

2.5.3.1.3 **Diffuse external otitis** (*otitis externa diffusa*)

Acute diffuse otitis externa (inflammation of external ear canal) is an infectious dermatitis that begins in the external auditory canal or in surroundings. Sometimes it can affect the whole ear. The predisposing factor is maceration, injury, or harsh handling. It is most often caused by streptococci, but also staphylococci, *Pseudomonas aeruginosa, Bacillus proteus* and *Escherichia coli*. The treatment is similar to furuncle. Camphor-ichthyol ointment, 3% boric acid solution, or another local desinfections are suitable.

2.5.3.1.4 **Auricular perichondritis** (*perichondritis auriculae*)

This is a rare inflammation of the outer ear. Cartilage become infected as a result of trauma or auricle surgery. The most common cause of perichondritis is the furuncle of the external auditory canal. Inflammation can spread to the entire auricle. In case of inadequate or late treatment, purulent exudate accumulates in the space between the cartilage and the perichondrium, with subsequent cartilage nutrition disorder leading to auricular necrosis.

Symptoms: enlarged and intensively hyperemic pinna, pain, fever.

Treatment: symptomatic antibiotic treatment is often required, in case of abscess - incision and drainage. In the case of cartilage sequestration, the auricle retracts, deforms and shrinks.

2.5.3.1.5 **Granular myringitis** (*myringitis granularis*)

At granular myringitis (inflammation of the tympanic membrane) we detect small granulations on the surface of the tympanic membrane. Inflammation occurs as a result of:

1. localized form of external otitis
2. contact of the ear drum with a hard earwax plug or foreign bodies (e.g a ventilation tube in the middle ear)
3. intense influenza

Aluminum acetate at 8% is suitable for its astringent effect, local desinfection solutions. If the granulations do not subside, the cauterization with silver nitrate solution may be helpful. In case of persistence of granulations, myringoplasty is required.

2.5.3.1.6 **Bullous hemorrhagic myringitis** (*myringitis bullosa hemoragica*)

The disease often occurs as a complication of influenza.

Symptoms: presence of hemorrhagic blisters on the tympanic membrane (Fig.13) and adjacent parts of the auditory canal that rupture rapidly. The predominant symptom is pain. We detect a conductive hearing disorder caused by obstruction by blisters or concomitant inflammation of the middle ear. In some cases, sensorineural hearing loss may also be present due to viral damage to the inner ear.

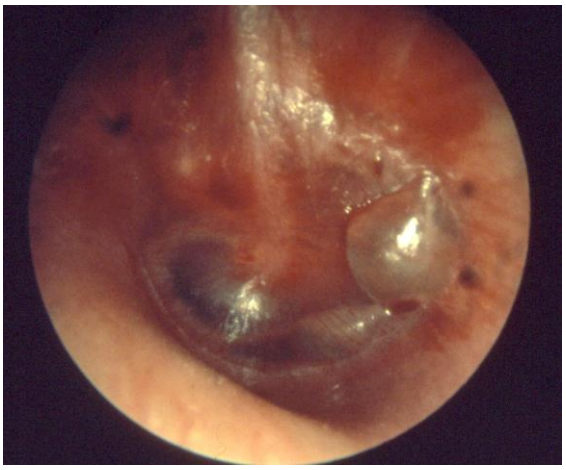

Fig.13 Otitis media bulosa **(**by B. Welleschik is licensed under CC BY-SA 3.0. To view a copy of this license, visit https://creativecommons.org/licenses/by-sa/3.0/?ref=openverse)

Treatment: analgesics, prevention of secondary infection, treatment of otitis media, vasoactive treatment in sensorineural hearing loss.

2.5.3.1.7 Malignant external otitis (*otitis externa maligna*)

Etiology: Inflammation is most often caused by a pseudomonas infection. Quite often, it can be fatal in elderly, diabetic or immunocompromised patients. The infection that initially develops in the ear canal can spread at the area junction of the bone and cartilage to the adjacent bone. Ostitis and osteomyelitis may occur. There is a high risk of spreading the infection to the sigmoid sinus and meninges.

Symptoms: pain predominates, ear discharge, granulation. Paresis of cranial nerves (VII, IX, X, XI, XII) may also occur.

Treatment: wide-spectrum antibiotics in some cases combined with surgical treatment

2.5.3.2 **Otitis media** (*otitis media*)

.

Acute and chronic inflammation of the middle ear (*otitis media*) (Figs.14,15) are the most common diseases affecting the ear. These are various forms of inflammation, from acute tubotympanic catarrh which heals without sequelae within a few days, to chronic otitis media with cholesteatoma which lasts for several years and can have serious complications. Several factors contribute to inflammation. These include the most common inflammations of the upper respiratory organs, the condition and function of the Eustachian tube, the condition of the mucosa of the middle ear, the extent of pneumatization of mastoid procces, the virulence of pathogens, the general condition of the patient and others.

In newborns, aseptic inflammation can develop during the first days of life when amniotic fluid enters the middle ear cavity through a short and widly open Eustachian tube. This type is called *hyperplastic otitis media of newborns*. It plays a role in the further development of the middle ear, as it leads to changes in the mucous membrane of the middle ear and influences the development of the pneumatic system of the mastoid process. Later inflammation of the altered hyperplastic mucosa of the middle ear is more resistant to treatment.

Inflammation of the middle ear is classified based on the etiology (Fig.16), pathology, duration and age of the patient.

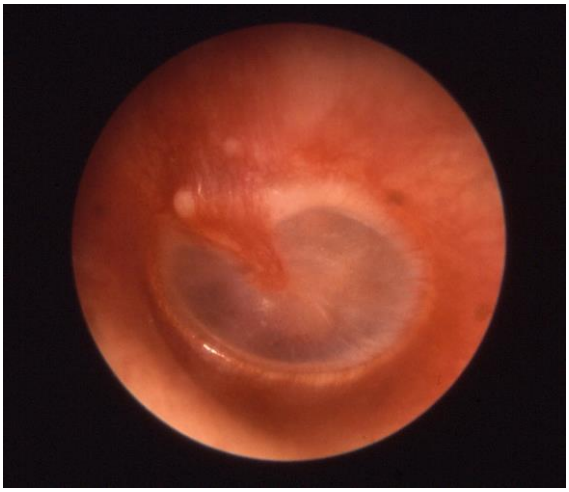

Fig.14 Otitis media incipient (by B. Welleschik is licensed under CC BY-SA 3.0. To view a copy of this license, visit https://creativecommons.org/licenses/by-sa/3.0/?ref=openverse)

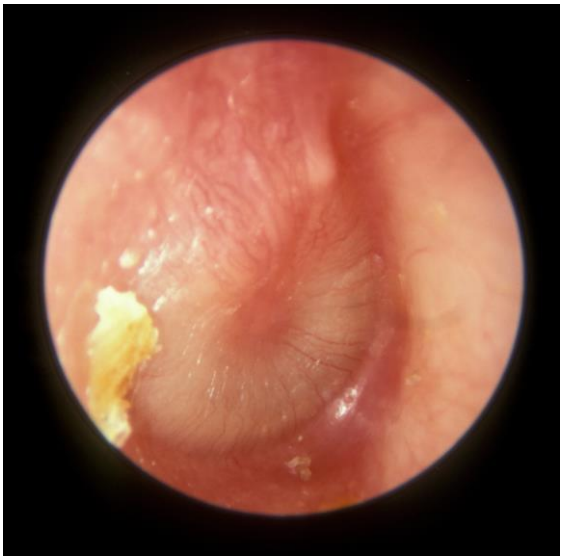

Fig.15 Otitis media at the stage of resolution. The middle ear is filled with a creamy white mucopurulent exudate which is causing the tympanic membrane to bulge laterally. Note the dilatation of the radial blood vessels of the tympanic membrane which appear like the spokes of a wheel. (by Michael Hawke MD is licensed under CC BY 4.0. To view a copy of this license, visit https://creativecommons.org/licenses/by/4.0/?ref=openverse).

2.5.3.2.1 Tubotympanic catarrh

Acute tubotympanic catarrh

The disease is often accompanied by catarrh of the upper respiratory organs of viral origin. It is a serous inflammation of the mucous membrane of the middle ear. If the inflammation also affects the mucous membrane of the Eustachian tube, its function may be partially or completely impaired. The air in the middle ear cavity and the pneumatic system of the mastoid process is resorbed relatively quickly. A negative pressure is created, which causes the retraction of the tympanic membrane and sometimes transudation into the tympanic cavity - *hydrops e vacuo*.

Symptoms: feeling of fullness in the ear, tinnitus and hearing loss of various degrees; during otoscopic examination, the membrane of the eardrum is gray in color, retracted. The lateral process of the malleus is raised. The tympanic reflex is shortened or not present at all. Sometimes you see a yellow serous fluid behind the tympanic membrane.

Treatment: concomitantly with treatment of upper respiratory tract infection. We apply substances with astringent and disinfectant effect to the nasal cavity, sometimes drops with antimicrobial effect and corticosteroids. After remission of the disease or alleviation of the

symptoms of rhinitis, it is recommended to the patient to perform a Valsalva maneuver to support the aeration of the middle ear cavity. Sometimes it is necessary to performe an air shower. If the Eustachian tube malfunction persists after a treated upper respiratory tract infection, it is necessary to perform Eustachian tube catheterization in order to improve hearing. The last solution is paracentesis (creation of a small hole in the lower part of the tympanic membrane) with suction of the contents of the middle ear cavity. In case of repeated problems, prolonged ventilation and middle air drainage are indicated by inserting a ventilation tube into the opening after paracentesis.

Chronic tubotympanic catarrh

Chronic tubotympanic catarrh develops as a result of repeated episodes of acute inflammation. Several factors contribute to the development of the disease, such as adenoid vegetation in children, nasal septal deformities, chronic hypertrophic rhinitis, allergy and others. Pathological changes in the middle ear have a larger extent. The mucosa is congested and swollen, with secretion, mucus and sometimes erythrocytes in the middle ear. Degradation of erythrocytes promotes the formation of granulations. A few years after healing, we can detect adhesions or tympanosclerosis in the middle ear. The patient has a conductive hearing disorder. Untreated chronic granulomatous otitis media can lead to cholesteatoma.
The principle of treatment is the restoration of physiological conditions in the middle ear. Long-term drainage of the middle ear and its aeration (air showers, ventilation tube) are indicated. Antibiotics, corticosteroids can be applied topically through the ventilation tube.

2.5.3.2.2 Acute purulent otitis media (*otitis media acuta suppurativa*)

The most common causes of purulent otitis media are pyogenic bacteria *Streptococcus pyogenes, Diplococcus pneumoniae* (in children type I, II; in adults more often type III) and *Staphylococcus pyogenes*. If it is primarily of viral etiology, the exudate in the middle ear is serous or hemorrhagic. Secondary infection with streptococci or staphylococci occurs rapidly and the exudate turns purulent. Pathogens most often enter the middle ear through the Eustachian tube, less often through the perforated tympanic membrane, or through the bloodstream. In the initial phase of inflammation, the tympanic membrane, the mucous membrane of the middle ear and the pneumatic system of the mastoid process are swollen and

congested. Leukocytes penetrate through the vessel wall into the middle ear cavity and become a part of the exudate.

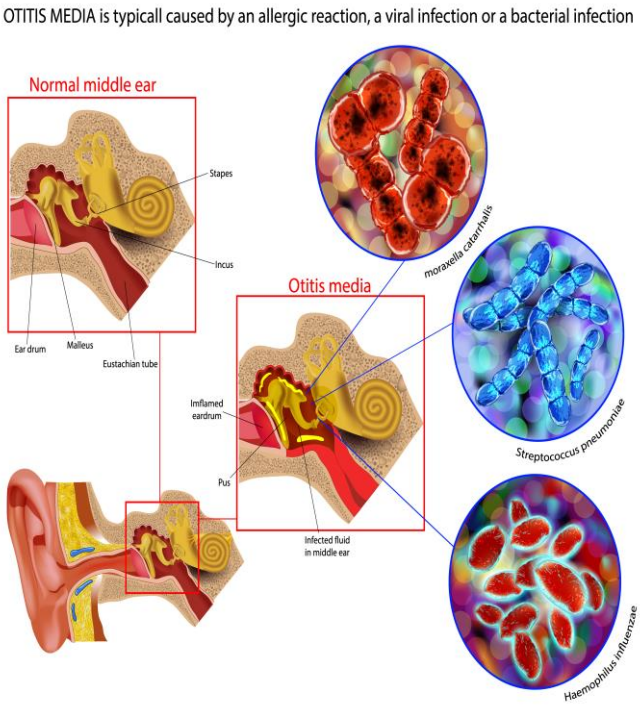

Fig.16 Most common etiology of otitis media. (from Danishyar A, Ashurst JV. Acute Otitis Media. [Updated 2022 Jan 21]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2022 Jan-. Available from: https://www.ncbi.nlm.nih.gov/books/NBK470332/)

Symptoms: intense ear pain that may radiate to the occipital area and to the teeth. Pushing or tapping the mastoid process can be painful - a symptom called *mastoidism*. This means that the inflammation also affected the mastoid process. The body temperature is above 38 °C.

Diagnosis: during otoscopy we can see dilated vessels on the tympanic membrane (radial injection). Later, the entire tympanic membrane is red, diffusely congested and thicker. The back half of the eardrun bulges due to exudate, it can also rupture spontaneously. The rupture of the tympanic membrane is usually small, but it is sufficient for the purulent exudate to be drained from the middle ear cavity into the external auditory canal. Conductive hearing loss is present.

Treatment: rinsing the nasal cavity with a low concentration saline solution, nasal drops with an astringent, disinfectant effect, pain killers. In case of fever and general alteration, antibiotic treatment is also indicated. If the problem persists, and if exudate is present and the tympanic

membrane is bulged, it is necessary to puncture it (myringotomy, paracentesis). Myringotomy is performed under local anesthesia, in the posterior lower quadrant of the tympanic membrane. Its aim is to drain the exudate. The tympanic membrane defect usually closes spontaneously and quickly. The complete cure of purulent otitis media takes 3 to 4 weeks.

Acute otitis media in neonates and infants

Otitis media in children is a common cause of pyrexia and meningeal irritation, while the symptoms of ear damage may be minimal. The predisposition to infection is a wide and short Eustachian tube. In children feeded from bottle contaminated milk is also expected to enter the Eustachian tube. A common cause is rhinitis associated with the first growth of teeth. In some children, otitis media is associated with gastroenteritis. If convulsions are also present, it is a neurotoxic syndrome. The tympanic membrane can be also gray, dull, often without bulging, although there is a purulent content in the middle ear.

2.5.4 Complications of otitis media

2.5.4.1 Mastoiditis *(mastoiditis acuta)*

Mastoiditis is a complication of acute purulent otitis media. It occurs when the inflammation of the mucosa of the mastoid air cells spread to the bony structures (Fig.17). It is necessary to distinguish mastoidism, percussive pain on the *mastoid process*, as one of the symptoms of acute otitis media. In this case, the bone is not affected and after antibiotic treatment the condition improves quickly.

Mastoiditis develops relatively quickly post otitis media, sometimes more weeks later. The main condition for its development is the retention of exudate in the cavities of the pneumatic system. Peripherally placed cells may be sometimes occluded with edematous mucosa, mucus or granulations. The granulations can also close the *aditus* or *antrum* directly, thus separating the pneumatic system from the tympanic cavity. Inflammation in the middle ear cavity heals, but in the pneumatic system it may persist or progress, respectively. The mucosa of the antrum and periantral spaces is hyperemic and swollen. The bony septa between the spaces are decalcified and destroyed. In some cases, large cavities are filled with granulations, bone sequestration and pus formation occur. Inflammation also spreads to the cortical bone and periosteum leading to periostitis.

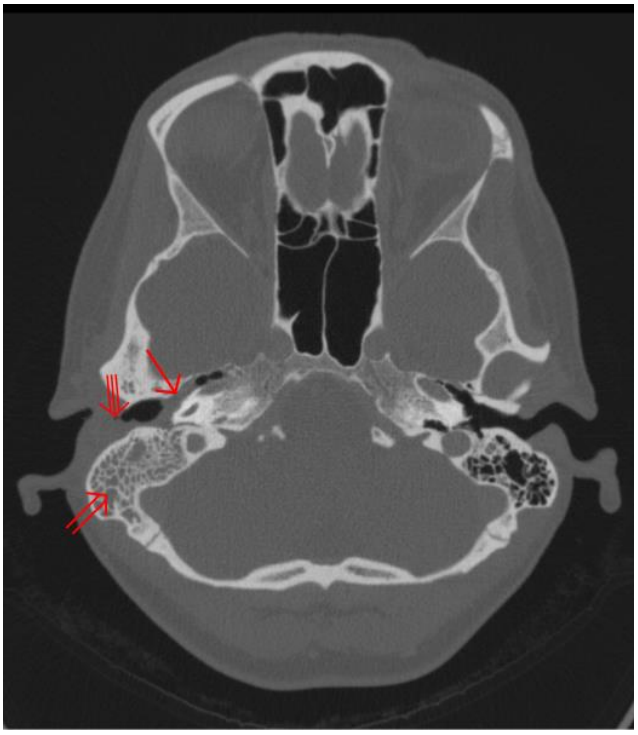

Fig.17 Otitis media (simple arrow) and mastoiditis (double arrow) of the right side (left side in image). The external auditory canal is partially occupied by suppuration (triple arrow). 44-year-old woman. (by Jmarchn is licensed under CC BY-SA 3.0. To view a copy of this license, visit https://creativecommons.org/licenses/by-sa/3.0/?ref=openverse.)

Symptoms: are quite typical; the 3rd or 4th week of antibiotic treatment in acute purulent otitis media, the condition improves only minimally, or redness and swelling develop in the retroauricular area (a sign of periostitis). The retroauricular groove is smoothed, the pinna protrudes. Ear discharge may be intensified. The pain reappears, most intensely in the area of the mastoid process, body temperature rises above 38 °C. The patient may have a forced head position - tilted to the affected side, as the inflammation may spread to the upper part of the *m. sternocleidomastoideus*. The patient often reports a hearing disorder, it is of conductive type.

The situation is different with latent mastoiditis. It develops gradually, asymptomatically, and may be complicated by sigmoid sinus thrombophlebitis. These atypical forms of mastoiditis occur in children, elderly patients, or when symptoms are relieved by antibiotics. The pressure applied on the mastoid process is painful. Fluctuation is present in the subperiosteal abscess in the postauricular region. At periostitis of the anterior part of the mastoid process, the posterior wall of the auditory canal is destroyed. A drop in the upper posterior wall of the external

auditory canal is a poor prognostic sign. Eardrum is dull, hyperemic. The purulent exudate accumulates behind the intact bulged tympanic membrane, or flows out of the middle ear through spontaneous perforation. CT examination of the temporal bone is necessary for the diagnosis (destruction of septal system of temporal bone, granular tissue).

Treatment: surgical, parenteral antibiotic treatment. In the past, mastoiditis was a very serious and feared complication, today it is less common.

2.5.4.2 Petrositis *(petrosititis)*

Petrositis is a purulent inflammation of the pneumatic system of the pyramid. It occurs in acute otitis media or at the same time as mastoiditis. Sometimes a more severe infection can cause a petrous apex abscess. The purulent process in the petrous bone is usually associated with serous meningitis in the middle cranial fossa. Later, an extradural abscess or septic meningitis may develop. Rarely, the infection may spread along *a. carotis interna* and form parapharyngeal abscess.

Symptoms: petrositis is usually associated with mastoiditis, persistent otorrhoea. Pain may be present in the temporal or retroorbital area due to irritation of cranial nerve V. Sometimes paralysis of cranial nerve VI is also present. The complex of symptoms is called Gradening's syndrome.

Treatment: radical surgical and parenteral antibiotic

2.5.5 Chronic otitis media *(otitis media chronica)*

Chronic otitis media is characterized by permanent or intermittent serous-purulent discharge from the external auditory canal and permanent perforation of the tympanic membrane. It occurs in about 1-2% of the population. It was more common in the pre-antibiotic era and in less developed countries is more frequent . Chronic otitis media includes mesotympanic and epitympanic forms.

2.5.5.1 Mesotympanic chronic otitis media *(otitis media chronica mesotympanica, simplex)*

It usually occurs concomitantly with inflammation of the upper respiratory system. The infection spreads to the tympanic cavity through the Eustachian tube from the epipharynx, for example in chronic epipharyngitis, sinusitis, adenoid vegetations. The mucosa of the Eustachian tube and tympanic cavity is inflamed and thickened. There may also be polyps and

granulomatous altered tissue on the mucosa, which sometimes grows into the ear canal. Rarely, ostitis of middle air ossicles or of the wall of the tympanic cavity may appear. The tympanic membrane is perforated, the perforation being of various shapes and sizes and located centrally in the *pars tensa*, not exceeding the *anulus fibrocartilagineus*, which is intact. It is more common in patients with recurrent otitis media in childhood, with reduced immunity and poorly developed temporal bone pneumatization.

Diagnosis: history, odorless discharge from the external auditory canal (there is no ostitis), otoscopy reveals a central perforation of the tympanic membrane (Fig.18). Temporal bone CT reveals poor pneumatization of the mastoid process, but without destruction of the bone septa. There is a conductive, sometimes mixed hearing dysfunction.

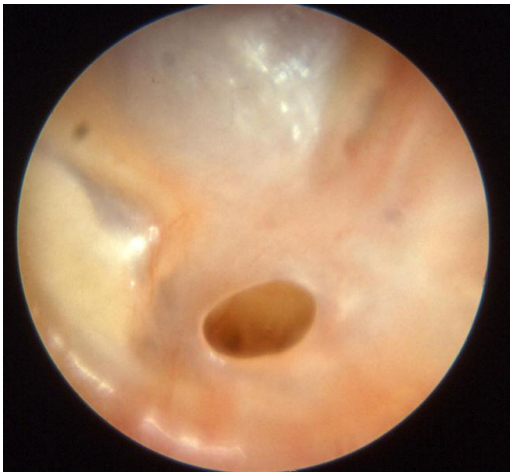

Fig.18 Otitis media mesotympanica (by Welleschik, licensed under CC BY-SA 3.0. To view a copy of this license: https://creativecommons.org/licenses/by-sa/3.0/?ref=openverse).

Treatment: Chronic mesotympanic otitis should be treated together with upper respiratory system. Adenotomy is performed in children. If necessary, septoplasty or endoscopic surgery of the nasal and paranasal cavities (FESS) is performed. Climatotherapy is effective in some patients. After healing of upper airway infection, when the ear is "dry", without secretion, surgical treatment is indicated - reconstruction of the tympanic membrane, or even auditory ossicles. The patient´s hearing post treatment is obviously better, too. Generally, prognosis is very good in most cases.

2.5.5.2. Epitympanic chronic inflammation with cholesteatoma, with ostitis *(otitis chronica epitympanica cum cholesteatomate, cum ostitide)*

This form of inflammation differs from mesotympanic in that there is often no direct association with inflammation of the epipharynx, nasal cavity, or paranasal sinuses. Chronic epitympanic otitis media is more severe form of inflammation. It most often arises from repeated, prolonged, or insufficiently treated acute otitis media. It develops gradually and may be asymptomatic for a long time. Ostitis of temporal bone always develops and 90% of patients develop cholesteatoma. **Cholesteatoma** is simply explained as the presence and growth of the epidermis at an inappropriate site in the body, in this case in the middle ear (Fig.19). There are several theories that explain the origin of cholesteatoma. As to the first theory, cholesteaotoma is caused by the migration of epidermal cells through the perforated membrane of the tympanic membrane. The second theory is based on the fact that cholesteatoma is formed from the retraction pockets of the tympanic membrane, especially in the *pars flaccida*. Retraction pockets are invaginations of the weakened part of the eardrum into the eardrum cavity and adjacent spaces of the middle ear. The third theory presupposes the development of cholesteatoma from the epidermal metaplasia of the middle ear mucosa. The fourth theory assumes the creation of the so-called primary or congenital cholesteatoma arising from an embryonic ectodermal graft. It can be located in the tympanic cavity, mastoid process or *capsula otica*. The dead cholesteatoma cells accumulate on the surface, the central vital part - the matrix - grows. The formation enlarges and presses on the surrounding tissues. It gradually replaces the mucosa of the middle ear. Bone is destroyed mechanically and enzymatically (by collagenase), and ostitis occurs. This leads to formation of a pathological cavity. The changes can spread to dura mater and venous sinuses. With its growth, the cholesteatoma can damage the canal of the facial nerve and cause its paralysis, or lead to the formation of a fistula to the inner ear, most often to the semicircular lateral canal. The auditory ossicles are also damaged.

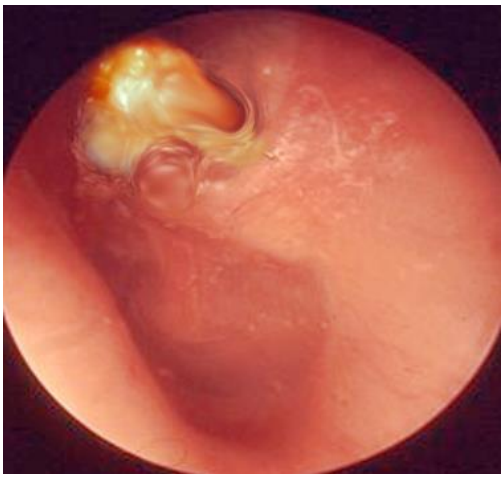

Fig.19 Cholesteatoma of the right ear (from Kennedy KL, Singh AK. Middle Ear Cholesteatoma. [Updated 2022 Jul 4]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2022 Jan-. from: https://www.ncbi.nlm.nih.gov/books/NBK448108/)

Symptoms: foul-smelling secretion (because of ostitis), conductive or mixed hearing loss. Vertigo, nausea and neuralgic pain are very serious symptoms. Peripheral paresis of cranial nerve VII can be detected (rare).

Diagnosis: history, marginal defect of tympanic membrane, extending beyond the *anulus fibrocartilagineus* in the posterior upper quadrant or *pars flaccida*. Sometimes we can see whitish masses protruding through the perforated membrane. These masses mimic squamous cell carcinoma, which must be considered in the differential diagnosis. Examination of the hearing, vestibular system, and CT of the temporal bone are required to uncover destruction of the pneumatic system.

Treatment: Surgical treatment is 1) remedial in order to remove the inflammatory-altered pneumatic system and 2) reconstructive, which includes a wide range of procedures. Their goal is to improve hearing by covering the tympanic membrane defect, restoration, or creation a "new" transmission middle ear system. There are several types of surgical procedures in regard to the extent of the disease. In some cases, remediation and reconstruction are performed at the same time. Another time, patient's treatment runs in two phases: remediation and subsequent reconstruction. Postoperative care, in some cases lasting for years, is important (cleaning).

Conservative treatment - local, oral application of antibiotics, corticosteroids etc. is *non lege artis*. The symptoms alleviate or disappear, but it is just temporary effect. Primary goal of treatment is the remediation of inflammation, sometimes such treatment does not lead to the

satisfactory functional effect (hearing quality) for the patient. Improperly chosen treatment can still have fatal consequences, in terms of life-threatening complications.

2.5.6 Intracranial otogenic complications

Intracranial complications have been common in the past and had high mortality. At present, they are rare, but their prognosis is uncertain and sometimes fatal. Effective treatment of acute otitis media and early surgical treatment of chronic epitympanic otitis media with cholesteatoma are good prevention of intracranial complications. Infection and inflammation can spread from the middle ear to the intracranial space in several ways. In the case of ostitis, it spreads directly - *per continuitatem*. Spread is also possible through fissures and channels in the temporal bone, which connect the temporal bone to the intracranium (*meatus acusticus internus, canalis nervi facialis, ductus cochlearis, tegmen tympani, etc*.). When inflammation spreads to the dura mater, its further spread depends on the virulence of pathogens, the resistance of the dura mater and the overall condition of the patient. Dura mater is very resistant and can resist the spread of infection to the intracranium for a relatively long time. Dural inflammation, *pachymeningitis externa,* can often be asymptomatic and is sometimes a surprising finding in the surgical treatment of chronic otitis media. Highly virulent pathogens cross the natural barrier of the dura mater and inflammation spreads to leptomenings (arachnoid and pia mater) and leptomeningitis develops. The infection further spreads to brain tissue. Following encephalitis can lead to a brain or cerebral abscess.

2.5.6.1 Pachymeningitis *(pachymeningitis externa)*

Pachymeningitis externa is the most common otogenic intracranial complication. Inflammation affects only the temporal side of the dura mater. It occurs in mastoiditis or chronic epitympanic otitis media with cholesteatoma. Dura mater changes color, becomes thicker and granulating tissue forms on its surface. When pus accumulates between the lamina propria of mastoid process and the dura mater, an extradural abscess is formed.

Symptoms: The symptoms of an extradural abscess may not be severe, dominated by headache on the affected side, especially at night. In abscess near sigmoid sinus, shiver attacks accompanied by high fever may occur, indicating involvement of the sigmoidal sinus wall and the onset of thrombophlebitis. Local symptoms occur in very large extradural

abscesses. Large extradural abscesses in the middle cranial fossa can lead to impaired motor activity, aphasia, or can mimic a temporal lobe abscess.

Treatment: combine surgical treatment of the primary inflammatory lesion, the neurosurgical uncovering the dura mater and the intravenous application of antibiotics penetrating the blood-brain barrier.

2.5.6.2 Inflammation of pia mater, leptomeningitis *(leptomeningitis)*

Leptomeningitis occurs in chronic epitympanic otitis media, but can also be a complication of mastoiditis. Approximately half of leptomeningitis cases develops on from an extradural abscess. Much less common is leptomemningitis based on labyrinthitis.

Symptoms: fever to sepsis (at pyemia), intense headache radiating to the back neck, often to the entire spine. The gradually developing stiff neck is due to the spasm of the extensors. Kernig and Brudzinski signs are positive. Consciousness change is frequent and progressive. It starts with drowsiness, followed by confusion. The next stage is stupor, when the patient responds only to simple commands, followed by semicoma, when responds only to painful stimuli. In a deep coma, the patient does not respond to painful stimuli. Vomiting is common. Cerebrospinal fluid is cloudy, leukocyte count and protein content increase, glucose levels decrease; it must be examined microbiologically and for pathogen resistance to antibiotics. Common pathogens are pneumococci, *Streptococcus haemolyticus*, *Meningococcus*, *Staphylococcus, H. influenzae*. Sometimes lesions of cranial nerve III, VI, VIII, IX, X or XI are present. The patient with meningeal irritation should always be examined by an ENT specialist to rule out an otogenic cause.

Treatment: surgical treatment of the primary inflammatory lesion. Intravenous administration of antibiotics penetrating the blood-brain barrier is essential. Repeated relieving lumbar puncture with culturing of cerebrospinal fluid and possible adjustment of antibiotic treatment is indicated.

2.5.6.3 Trombophlebitis of sigmoid sinus *(trombophlebitis sinus sigmoideus)*

Sigmoid sinus is highly exposed to contact infection, especially in mastoiditis, due to its size and anatomical location. In mastoiditis, the infection spreads rapidly to the brain and cerebral fossa. In chronic epitympanic otitis media with cholesteatoma, sigmoidal sinus

thrombophlebitis begins almost regularly in the exacerbation phase of inflammation. The infection can spread from the mastoid process to the sinus through small veins that create a rich bed in the wall of the sinus and terminate in it. Pus can accumulate in the surroundings of the sinus forming an abscess. Later the sinus wall is thickened and the endothelium is damaged due to periflebitis. In the final phase, thrombophlebitis of the sinus may develop, followed by thrombolytic spread into the bloodstream and sepsis. In extreme cases, a large thrombus may extend to the *sinum confluens* or to the jugular vein. The inflamed wall of the sinus is reddish- brown, covered with granulations and may be necrotized.

Symptoms: thrombophlebitis of sigmoid sinus leads to otogenic sepsis; symptoms of sepsis dominate. Typical are fluctuating fever, shiver and poor general patient´s condition caused by bacteremia and toxemia. Shivering begins 20-30 minutes after the massive entry of bacteria into the bloodstream, and it is accompanied by fever above 40 ° C. In children, shivering is rare and the temperature curve is continuous. With prolonged antibiotic treatment and pneumococcal infection, the clinical picture can be atypical without shivering and fever. The pressure on the area behind the posterior edge of the mastoid process is painfull (Greisinger symptom). In descending thrombophlebitis, the jugular vein can be palpated as a hard band. Rarely, severe headache caused by haemostasis during the occlusion of one of the paired sinuses, occurs.

Diagnosis: history, CT of the temporal bone, blood culture.

Treatment: priority is surgery of the primary lesion (ostitis; chronic otitis media). Resection of the diseased sinus is performed after a previous ligation of *v. jugularis*, which is a preventive measure against the spread of infection and air embolism. Parenteral treatment with wide-spectrum antibiotics is also indicated.

Prognosis: prognosis of a patient with otogenic sepsis and sigmoid sinus thrombophlebitis is unfavorable. Despite intensive treatment, 10% of patients die. With a metastatic abscess to the brain and lungs, the prognosis is even more unfavorable.

2.5.6.4 Cerebral and cerebellar abscess *(abscessus cerebri et cerebelli)*

Cerebral and cereberal abscesses of otogenic origin are less common. They most often occur in chronic epitympanic otitis media with cholesteatoma and its exacerbation. The abscess cavity, bounded by the pyogenic membrane, is filled with pus and necrotic brain tissue (Fig.20). It is located in the temporal lobe or in the cerebellum. Sometimes there is a tendency

for purulent encephalitis to occur, or the abscess may perforate into the ventricles or subarachnoid space.

Symptoms: depend on virulence of pathogens, abscess location and value of intracranial pressure. In initial stage severe headaches, nausea, vomiting and fever are typical. Inflammatory parameters in plasma and cerebrospinal liquor increase, pressure of cerebrospinal fluid raises. Later, epileptiform attacks may occur, the patient is tired, the neurological symptomatology enhances: *aphasia* (inability to speak), *alexia* (inability to read), *agraphia* (inability to write). In a cerebellar abscess, balance disorders and a reduced muscle tone dominate. In the terminal stage, we detect disorders of heart rhythm and respiration. Mental symptoms are manifested by depression, drowsiness and restlessness. Diagnosing a brain abscess is not easy. It requires interdisciplinary cooperation. The definitive diagnosis is confirmed by imaging methods (CT, MRI).

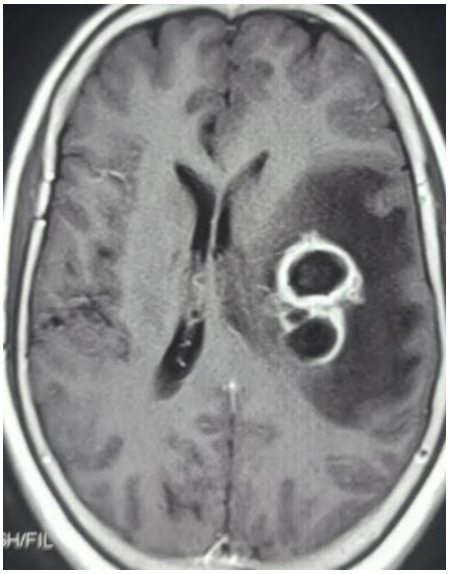

Fig.20 Cerebral abscess (from Bokhari MR, Mesfin FB. Brain Abscess. [Updated 2022 May 11]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2022 Jan-. Available from: https://www.ncbi.nlm.nih.gov/books/NBK441841/

Treatment: combined - radical surgical and pharmacological (widewide-spectrum antibiotics are given parenterally and corticosteroids are often indicated). It is necessary to surgically treat the primary lesion - most often chronic epitympanic otitis media (otorhinolaryngologist), as well as complications (neurosurgeon). The treatment is based on the relevance of the patient's condition. It is possible to solve both problems immediately after each other or with a certain time interval. Clearly, both issues need to be resolved. Mortality from serious

intracranial complications has decreased to 10% in recent years. Accompanying encephalitis and brain edema are serious problems, which are the immediate cause of death in fatal cases.

2.5.7 Inner ear diseases

Several factors are involved in inner ear diseases. The most important are infections, endogenous and exogenous intoxications, vascular disorders and injuries. Inner ear diseases are accompanied by hearing and balance disorders, which are difficult to treat. About 1-2% of the world's population has a hearing impairment, and this percentage increases significantly in people over 60 years of age.

2.5.7.1 Labyrinthitis *(labyrinthitis)*

Inflammation of the inner ear is rare. Labyrinthitis can occur as a complication of acute or chronic otitis media. In acute otitis media, the infection spreads to the inner ear through a round or oval window. In chronic epitympanic otitis media, the infection usually penetrates the inner ear through a fistula in the lateral semicircular canal. We distinguish two forms of labyrinthitis, which have different symptomatology: diffuse and circumferential.

2.5.7.1.1 Serous diffuse labyrinthitis *(labyrinthitis serosa diffusa)*

The disease is caused by toxins that penetrate the labyrinth from the middle ear. It leads to high fluid pressure in the inner ear. The patient has nausea, vertigo and vomits. Irritational nystagmus with the rapid component directed towards the affected ear, is present. Serous labyrinthitis usually heals without functional consequences. In some cases, hearing impairment occurs and vestibular symptoms of varying degrees may persist.

2.5.7.1.2 Purulent diffuse labyrinthitis *(labyrinthitis purulenta diffusa)*

This form of labyrinthitis occurs when bacteria enter the inner ear. Purulent inflammation of the membranous labyrinth will damage the structures of the inner ear and may spread further through the inner auditory canal into the intracranium. Otogenic meningitis and cerebellar abscess may occur. The clinical picture is dominated by severe vertigo, nausea, vomiting, tinnitus. Hearing loss develops relatively rapidly. Irritational nystagmus soon changes direction to a healthy ear (destructive nystagmus). The change in the direction of the

nystagmus indicates complete destruction and loss of inner ear function. The patient may have fever and symptoms of meningeal irritation.

2.5.7.1.3 Circumscribed labyrinthitis *(labyrinthitis circumscripta)*

The disease occurs in chronic epitympanic otitis media with cholesteatoma and ostitis, due to disruption of the bony wall of the lateral semicircular canal. There is a fistula between the inner and middle ear. The patient has vertigo of varying intensity depending on the size of the fistula and the condition of its occlusion. Vertigo can be caused by an increase in air pressure in the external auditory canal, irritational nystagmus towards the affected ear (fistula sign).

The **treatment of labyrinthitis** runs together with the treatment of otitis media. In chronic epitympanic otitis, we surgically treat the primary lesion and the fistula. Parenteral treatment with wide-spectrum antibiotics is necessary. In some cases, treatment with corticosteroids is necessary. A more conservative approach is sufficient in the treatment of serous labyrinthitis. In more serious situation with purulent labyrinthitis, it might be necessary to remove the labyrinth surgically - labyrinthectomy. Hearing loss in the inner ear damage is irreversible, but vestibular dysfunction may not be definitive. Vertigo of varying intensity may persist. At prolonged vertigo, which disables the patient, a labyrinthectomy or transection of the vestibular nerve is performed, which allows the patient a normal life.

2.5.7.2 Neuritis and degeneration of the statoacustic nerve

The auditory analyzer is phylogenetically younger than the vestibular analyzer, and therefore it is more sensitive to various noxious agents such as infections, exogenous intoxications and metabolic disorders. Relatively common causes of hearing loss are childhood infectious diseases - measles, scarlet fever and epidemic parotiditis; at a later age influenza and *herpes zoster oticus*. In the past, typhus, malaria and syphilis often led to hearing loss.

Some exogenous industrial toxins such as mercury, lead, aniline dyes, methyl alcohol or CO also cause hearing or vestibular disorders. In this context, it is also necessary to mention drugs, especially antibiotics from the group of aminoglycosides (STM, GEN, NEO, KAN and others), whose ototoxic effect may be potentiated by insufficient excretion. Of the metabolic

disorders, diabetes mellitus is particularly important, which has an adverse effect on the function of the inner ear.

Symptoms: sensorineural hearing disorder of various degree up to complete hearing loss, tinnitus and vertigo.

Treatment: to exclude the adverse factors. Corticosteroids are indicated. The issue of detoxification is controversial, even if an "antidote" is available. Vasoactive, nootropic substances, vitamins A and B and oxygen therapy are important. Sedatives are given for tinnitus.

Prognosis: vestibular symptomatology usually resolves and vertigo disappears. Tinnitus and hearing loss generally do not tend to improve.

2.5.7.3 Meniere´s disease *(Morbus Meniére)*

Meniere's disease is characterized by a feeling of fullness or pressure in the ear, attacks of rotational vertigo, tinnitus, nausea to vomiting and sensorineural hearing loss. Vertigo can last for minutes, hours or even days. Hearing loss is mild in the initial stages, gradually worsens in the entire frequency band, and in the extreme case it terminates with deafness. The symptoms are usually one-sided. The length of seizures varies, the period between seizures is relatively calm.

The etiology is unknown. The disease often affects relatively healthy people with no potential noxious factors in the history or disease with possible interference with inner ear function (metabolic and cardiovascular diseases, allergies, infections). Psychosomatic factors may trigger Meniere's disease, as patients themselves report the psychological tension preceding a seizure. There are also some signs of dysfunction of the autonomic nervous system.

An increased endolymphatic pressure, i.e. endolymphatic hydrops of the labyrinth is the chief pathophysiological mechanism of an attack in Meniere's disease. Hydrops may be induced by excessive production of endolymph by *stria vascularis* or its delayed resorption. The cochlear duct enlarges and the Reissner´s membrane bulges. The utriculus and sacculus are also enlarged, the semicircular canals do not change shape.

Due to the increased pressure of the endolymph, the Reissner's membrane or the wall of sacculum may rupture. Endolymph, which contains more K^+ ions and less Na^+, and perilymph, which has the opposite mineral ratio, are mixed. This results in severe inner ear dysfunction characterized by vertigo, tinnitus and hearing loss. After the membrane defect

heals, the concentration of electrolytes in the perilymph and endolymph normalizes and the symptoms disappear. The situation may repeat itself.

In **differential diagnosis** the diseases with cochlear-vestibular symptomatology should be distinguished. The *neurinoma of the auditory nerve* differs from Meniere's disease in that the auditory loss is supracochlear ("behind" the cochlea - more centrally; in may be differentiated by stapedial reflex, audiometric examination and brainstem evoked potentials).
CT or MRI shows the enlargement of the internal auditory canal, caused by expansion tumor. In Barré-Liéou syndrome caused by irritation of the posterior cervical sympathetic system, the symptomatology may be similar to that of Meniere's disease. X-ray examination confirms degenerative changes of the cervical vertebrae. Cervicocranial syndrome differs from other symptoms that are not present in Meniere's disease such as headache, otalgia, dysphagia, ophthalmic symptoms, and the like.
Treatment: pharmacological. Bed rest is indicated during an attack with the infusion of mannitol to reduce hydrops, antiemetics, corticosteroids, sedatives, treatment of the internal environment. After stabilization, vasodilators and nootropics that improve the metabolism of the inner ear are appropriate. In remission, a good professional and family background of the patient, a healthy lifestyle with the exclusion of alcohol, nicotine and caffeine are important. A diet with a reduced fluid and salt intake is recommended. In treatment-resistant vertigo, which does not allow the patient to live normally, destruction of the labyrinth (medicamentous - gentamicin destruction or surgical transection of the vestibular nerve) is indicated, which leads to a relative improvement in health status.

2.5.7.4 Sudden sensorineural hearing loss

Sudden sensorineural hearing loss, partial or complete, occurs within minutes or hours, especially in young people between 30 and 40 years of age. The etiology is unknown. Vascular disorders in the inner ear such as spasm, embolism, thrombosis and bleeding, viral infection, allergy or mental "stress" are anticipated.
The sudden onset of hearing loss indicates an embolism of *a. labyrinthi*. In viral infections, such as parotiditis or influenza, the virus directly destroys the sensorineural epithelium, neuritis of cranial nerve VIII, or causes edema of the capillary endothelium. Sudden

sensorineural hearing loss is relatively common in cerebelopontine lesions, e.g. in acoustic nerve neurinoma. With hearing loss, a tinnitus may or may not be present. In all patients (especially in unilateral disorder), mainly MRI is indicated to rule out an organic origin.

Treatment: early treatment is important to prevent complete destruction of hair cells. Corticosteroids are applied, hyperbaric oxygen therapy is suitable. The time factor of initiating treatment from the onset of the disorder is important (the sooner the better). Delayed treatment reduces the chance for complete recovery. In some patients hearing may return spontaneously.

2.5.7.5 Otosclerosis

Otosclerosis is a localized disease of the labyrinth capsule (*capsula otica*). From the pathological-anatomical point of view, it is a dystrophy of the enchondral layer of the bony capsule of the labyrinth. The capsule is resorbed first at a certain point by the activity of osteoclasts. The spongiosis is newly formed in the affected area, which later sclerotizes. Changes can occur anywhere in the labyrinth. The location of the lesion delineates the symptomatology. Clinically significant is the so-called non-labyrinthized form, which predilectionly affects the anterior edge of the oval window, with fixation of the base of stapes. There is genetic predisposition; the disease can occur in collagenoses (*osteogenesis imperfecta* and others) and it is more common in women. The clinical manifestation begins between 20 - 30 years of age. Pregnancy as well as the use of contraception and hormone supplementation therapy can accelerate the course of the disease. In the past, suspicion of otosclerosis was a possible indication for abortion after woman´s consent.

Symptoms: conductive hearing loss, in 80% bilateral. It can start unilaterally with damage to one ear. Its development is gradual, but may be also sudden. The perception of high frequencies is disturbed in later stages when the cochlea is affected. The disease can rarely manifest as sensorineural hearing loss when the new bone affects the cochlea and not the base. This is the labyrinthized form which is sometimes difficult to distinguish in differential diagnosis. *Paracusis Willisi* may be present meaning that patient hears better in a noisy environment. There is often tinnitus of varying intensity.

Diagnosis: otoscopy is physiological, sometimes increased vascularization of the promontory can be seen through the tympanic membrane; it is referred to as Schwartze sign (reddish brown color leasion, behind intact eardrum). The Eustachian tube is intact. In non-

labyrinthized form conductive, sometimes mixed hearing impairment is characteristic. By classical hearing examination with tuning forks we diagnose a hearing loss at lower frequencies (conductive disorder); the Gelle test is also important. Tympanometry reveals an increased resistance of the middle ear. HRCT (high-resolution CT) is indicated, which can display pathological foci too.

Treatment: in the past, conservative treatment was preferred. In the early days of the disease, in an effort to stabilize the otosclerotic process, fluoridation (NaF) was performed, especially in cochlear or combined otoclerosis. The effectiveness was questionable. Microsurgical treatment is currently preferred. Its principle is to create a hole in the base of stapes, into which the prosthesis is inserted and the other end is fixed to the incus. The prosthesis replaces a non-functional stapes. It transfers sound energy from the incus to the vestibule, instead of the fixed stapes removed. In most patients, hearing improves, and the tinnitus often subsides.

2.5.7.6 Hearing loss and the noise

2.5.7.6.1 Sudden hearing loss (short-term exposure to excessive noise)

Sudden hearing loss is due to excessive noise. Depending on the time factor of exposure to acoustic energy, either the inner ear may be damaged (very short interval, e.g. shot; leads to sensorineural type of hearing loss) or the middle and inner ear at the same time (longer time interval, e.g. explosion, given 1.5 ms and more). It leads to conductive or mixed type of hearing loss. At otoscopy perforation of the tympanic membrane can be identified. The patient often complains of concomitant tinnitus of varying intensity and duration. In acoustic trauma prevention is the most important, especially in some workplaces the use of hearing protectors is necessary.

2.5.7.6.2 Longterm exposure to the noise

Noise-induced hearing loss is a damage to the inner ear caused by prolonged exposure to loud noise, especially in certain industries. Continuous exposure to sound with an intensity higher than 80 dB is dangerous. The degree of hearing loss is proportional to the length of exposure, although there are interindividual differences in susceptibility.

Pathophysiology: the changes begin in the basal turn of cochlea, which is responsible for the perception of higher frequencies. This region of the basilar membrane is more rigid and is therefore exposed to larger tension; in this area the bone capsule is weakened. The hair cells of the basal turn gradually die and their number decreases.

Symptoms: when staying in a noisy environment, the hearing deteriorates - the hearing threshold increases, later can returning to normal. This is a *temporary shift* of the hearing threshold induced by noise. After a certain time, a *permanent shift* of the threshold establishes. Hearing loss is characteristic in the range of 4 kHz and higher.

Treatment: Rest and rehabilitation help with early symptoms. The most important thing is prevention. Employees in endangered fields must wear hearing protection and must be inspected regularly. The most suitable solution is to use technologies with reduced noise.

2.5.7.7 Presbyacusis

After reaching the age of 60, in some cases sooner, a hearing disorder of a certain degree develops, which is obviously bilateral and symmetrical. Age and noise exposure are basic causal factors, gender differences are not known. Degenerative changes result from vascular disorders in atherosclerosis and thrombosis. Atrophy of epithelial cells in the basal turn of cochlea, reduced number of hair cells, and damage to afferent and efferent nerve fibers lead to loss of high-frequency tone perception (Fig.21). Atrophy also affects nerve tissue at other locations - in ganglionic cells and *stria vascularis*. The elasticity of the basilar membrane is lost. All these changes lead to a sensorineural hearing loss, with a gradual loss of tone perception around from 2 kHz and above. The patient hears low- and mid-frequency tones relatively well. Typically, the patient does not understand speech, especially if it is fast, loud, and when he/she is in a noisy environment. It is necessary to speak clearly, slowly and not too loudly to a person affected in this way. Hearing loss is progressive and can result in severe social disability. Hearing aids are used at an advanced stage.

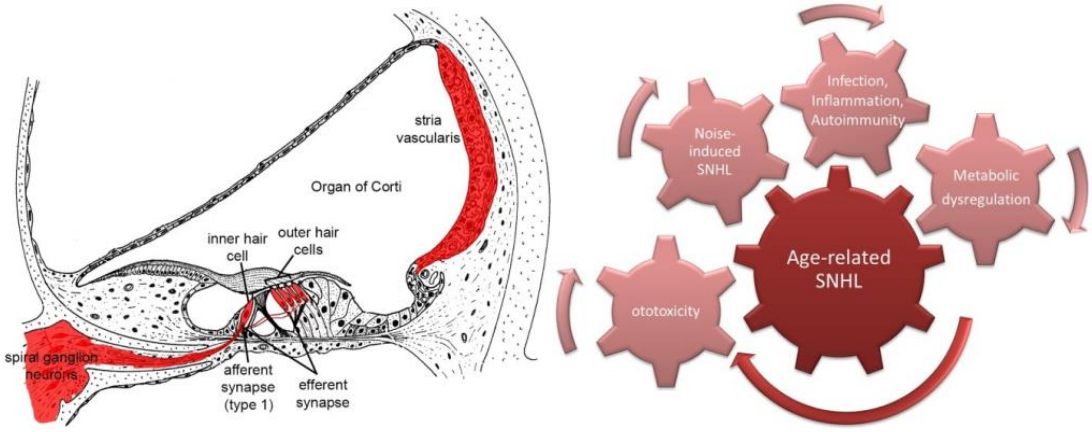

Fig. 21 Left: Illustration highlighting cochlear tissues that are prone to irreversible damage and cell death in sensori-neural hearing loss (SNHL) in red. Damage to any of these tissues reduces hearing; right: Cumulative/synergistic damage hypothesis of age-related hearing loss (ARHL). Causes of SNHL that accumulate over a lifetime combine with endogenous susceptibility to exacerbate age-related SNHL (from Wong and Ryan, 2015; https://creativecommons.org/licenses/by/4.0/, unchanged)

2.5.7.8 Tinnitus *(tinitus)*

Tinnitus is the perception of the noise without or with a hearing loss (so-called subjective tinnitus). It must be distinguished from hallucinations, which are of psychogenic origin. Melodic or rhythmic sounds may occur in epilepsy caused by the pathologies in temporal lobe or in other locationss. Rarely, tinnitus may be heard by the examiner as a click or buzzing (so-called objective tinnitus); in that case the vascular tinnitus must be ruled out.

2.5.7.8.1 Tinnitus with hearing loss

In most cases, tinnitus is associated with hearing loss, even when the patient is unaware of the hearing disorder. Tinnitus is often the first sign of incipient hearing loss, ototoxic damage or neurinoma of cranial nerve VIII, especially when it is unilateral. Unilateral pulsating tinnitus may indicate tumor of *glomus caroticum* or *jugulare*.

2.5.7.8.2 Tinnitus without hearing loss

Tinnitus without hearing loss can occur with certain lesions out of the statoacoustic analyzer, such as focus, carious "wisdom tooth," migraine, anemia, atherosclerotic plaques in the carotid artery, and intracranial vascular tumor, hypertension. During auscultation of the skull,

a tinnitus of vascular origin may be heard. Rarely, objective tinnitus is caused by clonic twitching of intratympanic or palate muscles. Degenerative changes in the cervical spine are also a common cause of tinnitus. Patients complain of persistent tinnitus especially in a quiet environment and they have a problem with sleep.

Treatment: when the origin of tinnitus is unknown, its treatment is very problematic. Intense tinnitus can lead to suicidal tendencies. Causal treatment is not always possible and therefore symptomatic treatment is applied. From a short-term perspective, sedative treatment is appropriate. It is clearly necessary to rule out an organic cause.

2.5.8 Tumors of the ear

2.5.8.1 Benign tumors

Benign ear tumors are relatively rare. Hemangiomas, fibromas or lipomas can develop on the pinna (Fig.22). Exostosis or osteoma, possibly adenoma occur in the external auditory canal and they can lead to its partial or complete obstruction. Complete obstruction of the ear canal can cause conductive hearing loss.

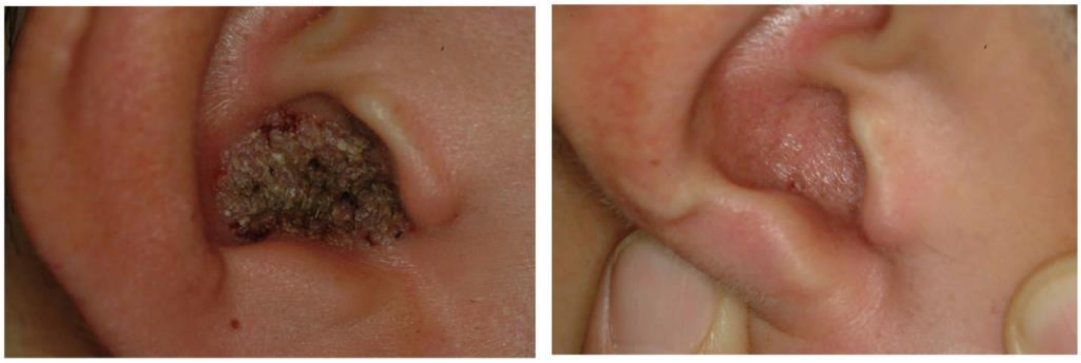

Fig.22 Left: a well-demarcated 1×1 cm, dark-brown to black papillomatous plaque near the external auditory canal of right ear; right: clearance of the skin lesion (by laser abrasion). Kim and Park, 2013; https://creativecommons.org/licenses/by-nc/3.0/; unchanged)

In the *middle ear*, the most common tumor is **paraganglioma**, a tumor growing from the *glomus jugurare*. It arises from achromaffin cells in the adventitia of *bulbus v. jugularis* and grows into the middle ear, labyrinth and mastoid process. It destroys the temporal bone and grows into the intracranium. Early symptoms include pulsating tinnitus and conductive hearing loss. At otoscopy we detect the bulged lower part of the tympanic membrane, or the

tumor growing into the external auditory canal. Because it is well vascularized tumor, it tends to bleed. This is why biopsy verification is inappropriate. Diagnosis is confirmed by contrast CT or MRI examination.

Treatment of benign tumors is surgical. It is problematic in paraganglioma if the tumor affects a large part of the temporal bone or grows into the intracranium. Then extensive surgery with lateral access to the skull base and otoneurosurgical removal of the tumor is required.

2.5.8.2 Malignant tumors

Malignant tumors mainly affect the pinna, where they represent 85% of all ear tumors. They rarely affect the external auditory canal. In the middle ear the carcinoma is most common. From the histological point of view, it is mainly basal cell and squamous cell carcinoma. The basis of diagnostics is a biopsy examination.

Basal cell carcinoma of the pinna grows slowly. After a few years, it destroys the auricle and infiltrates the mastoid process. It does not metastasize. The treatment is surgical. *Spinocellular carcinoma* has a much worse prognosis. It grows faster, penetrates surrounding tissues and metastasizes to regional lymph nodes.

Middle ear carcinoma has the worst prognosis. It is rare, often grows asymptomatically. The symptomatology in the early stages is similar to that of epitympanic otitis media. A foul-smelling, sanguinolent discharge from the external ear canal is present, followed by hearing loss of conductive, later of mixed or perception type. Deafness or paresis cranial nerve VII may develop when tumor grows into the inner ear. The problem is that this type of tumor is not considered in diagnosis because it is so rare. It it forms metastases in the cervical lymph nodes, invades to the surrounding structures (temporomandibular joint, parotid salivary gland, intracranium). Diagnosis is relatively simple - history, otoscopic and biopsy examination. CT is necessary. Despite radical, often mutilating surgical treatment, in combination with radiotherapy and chemotherapy, its effectiveness and 5-year survival is very low. Together with melanoma, it has one of the worst prognoses of malignancies in the ENT area.

Malignant *melanoma of the auricle* is characterized by rapid growth, metastases, not only to the regional lymph nodes, but also to the lungs and other organs. Biopsy verification is strictly contraindicated due to the enormous risk of spreading. *Sarcomas* are extremely rare.

2.5.8.3 Neurinoma of statoacoustic nerve

Neurinoma of the statoacoustic nerve (VIII) is a relatively rare benign tumor growing from Schwann cell sheat. It grows non-invasively and appears as a solid nodular formation of yellowish color. This tumor usually occurs in the upper part of the vestibular nerve and affects both men and women, usually between 30 to 60 years. The growing tumor compresses the brain and brainstem, and destroys the adjacent cranial nerves (Fig.22). Histologically, it consists of bundles of connective tissue cells, the nuclei of which are arranged in palisades.

Symptoms: unilateral sensorineural hearing loss with tinnitus is a common first symptom. Can occur also as bilateral disorder which is often associated with neurofibromatosis - Recklinghausen's disease.

The disease can be easily overlooked. Hearing loss may occur suddenly, but gradual hearing disorder is more typical. Vertigo may occur, but mild vestibular symptoms as stability disorders are more common. Trigeminal symptomatology appears in medium and large tumors. Corneal sensitivity and corneal reflex tend to be weakened. The headache is dull, located behind the ear. It occurs before the increase in intracranial pressure. The injury to cerebellum is accompanied by cerebellar symptomatology (ataxia, adiadochokinesis, titubation). Nystagmus is usually rapid, of first degree, away from the lesion, with a slow component towards the tumor. In large tumors, facial nerve palsy may be present. Tumor growth can lead to dilatation of the ventricles - hydrocephalus, intracranial hypertension, and persistent generalized headache, nausea, vomiting and papillary edema. In the terminal stages, the patient loses sight and falls into a coma.

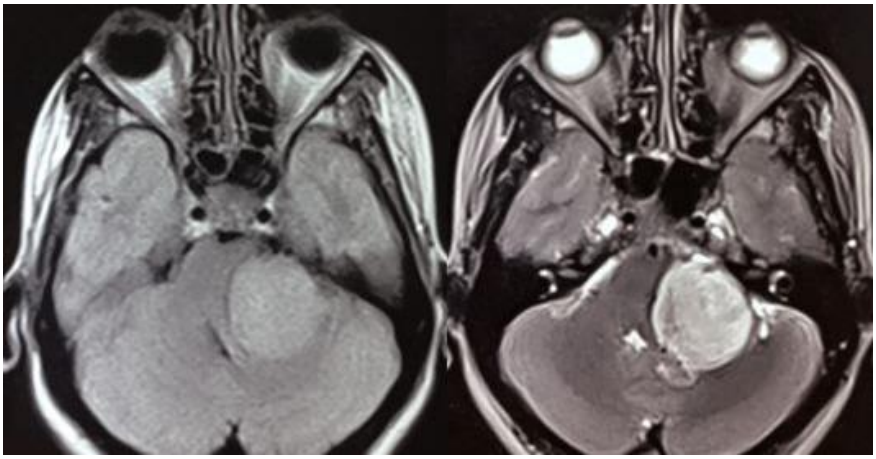

Fig.23 Acoustic neurinoma (Greene J, Al-Dhahir MA. Acoustic Neuroma. [Updated 2022 Jun 4]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2022 January; Available from: https://www.ncbi.nlm.nih.gov/books/NBK470177/)

The tumor of cranial nerve VIII is most common in the cerebropontine angle. The similar clinical picture can be seen in meningioma of the posterior cranial fossa, neurinoma of trigeminal nerve, cerebellar tumors, primary cholesteatoma, aneurysms and arachnoiditis, however they are much rarer.

Diagnosis: audiology with evoked potentials reveals perception supracochlear hearing loss. Vestibular examination reveals an ipsilateral vestibular lesion. In case of any suspicion, an MRI is clearly indicated.

Treatment: the aim is complete surgical removal of the tumor. Otoneurosurgical approaches are preferred, especially translabyrinth, combined with microsurgical techniques. Interdisciplinary cooperation is important, especially between otorhinolaryngologist and neurosurgeon.

2.5.9 Ear injuries

2.5.9.1 External ear injuries

Lacerated wounds. They are of varying degrees (Fig.24). The treatment consists of surgical revision of the wound. In general, even large-scale injuries have a good prognosis with early treatment. The complete detachment of the pinna can be corrected by the prosthesis.

Othematoma. It is formed by separation of perichondrium from the cartilage and consecutive bleeding into this space. Therapeutically, the incision and aspiration of the hematoma is done. Sometimes the "glove drain" is inserted, which prevents the incision from closing, and allows long-lasting drainage. Compression of the affected area promotes hemostasis and prevents refilling of the hematoma. Thorough compression and timely treatment are important also for nutrition of cartilage. The cartilage has no vessels and its nutrition is done by diffusion through the vascular system of the perichondrium. Incorrect or late treatment carries a risk of thrombus fibrotization with consequent deformation of the auricle.

Ear frostbite. They start with red or blue coloration in certain areas of the helix, which later turn white. The ear swells, is red, painful, blisters and later gangrene may form. In the early stages, the ear can be gently warmed. The gangrene is treated later, in some cases more radically.

Foreign bodies. These are most often the objects in the external auditory canal. At complete obturation of the canal they cause a conductive hearing loss. Insects can also enter the ear canal, causing irritation and are a source of "noise, buzz". Foreign bodies are removed with tweezers, rinsing or suction.

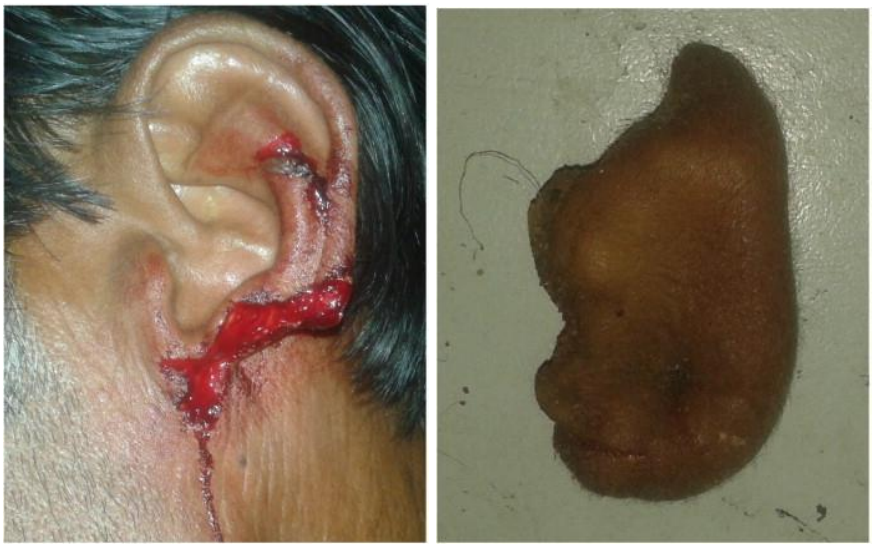

Fig.24 Auricle injury due to human bite. (from Sinwar 2015; https://creativecommons.org/licenses/by-nc-nd/3.0/; unchanged)

2.5.9.2 Middle ear injuries

Foreign bodies, high pressure in the external auditory canal or in middle ear, or a fracture of the temporal bone can cause a rupture of the tympanic membrane, and sometimes even injury to the structures of the middle ear. The tympanic membrane usually heals spontaneously, in some cases a reconstructive surgery - myringoplasty, is necessary.

At the moment of rupture, the patient may feel severe pain, which later weakens or disappears. Hearing loss depends on the size and location of the perforation. Tinnitus and vertigo may occur temporarily. During otoscopy, we usually see the blood in the external auditory canal. If there is a foreign body, it must be removed. Flushing of the external auditory canal is strictly contraindicated, mainly to avoid the irritation of the inner ear. In some cases, it is possible to perform membrane reposition. It is recommended to cover the defect with cigarette paper, which lead to spontaneous and quick healing.

As a result of the penetration of keratinized epithelium (skin layer) through the site of perforation into the middle ear covered by the mucosa, cholesteatoma may develop.

Prophylactically the antibiotics are applied. The patient is instructed about the care which includes the prevention of water flow into the ear canal, prevention of a sudden air pressure increase in the middle ear during sneezing and the like. In head injuries (temporal bone), the middle ear osscicles may separate. The most common lesion is the separation of the incus and stapes, on the other hand, the connection between the malleus and the incus is very rarely disrupted. Fractures of both crus of stapes can also occur. Separation of the base of the stapes from the oval window leads to a progressive conductive hearing loss. We think of this diagnosis when the conductive hearing idisorder persists even after the tympanic membrane has healed. Tympanometry reveals high compliance (low resistance) and a missing stapedius reflex. The treatment is surgical.

2.5.10 Fractures of temporal bone (laterobasal injuries)

2.5.10.1 Longitudinal fractures

The longitudinal fracture of the temporal bone is the most common; it represents up to 80% of all fractures. The fracture line is in the longitudinal axis of the petrous bone and passes through the tympanic cavity, the tympanic membrane and the bony part of the external auditory canal.

Usually, conductive hearing disorder occurs that can resolve spontaneously. Damaged skin of the ear canal can be the source of bleeding, or blood flows through the ruptured membrane of the eardrum. When the tympanic membrane is intact, accumulation of blood in the tympanic cavity forms *hemotympanum* (Fig.25). It is manifested by the blue-black coloration of the drum, which is an important diagnostic feature. During otoscopy, we can also see the deformity of the bony part of the ear canal. Facial nerve paresis may occur. The outflow of cerebrospinal fluid is not a standard finding. Diagnosis is based on the finding of hemotympanum and CT examination (Fig.26).

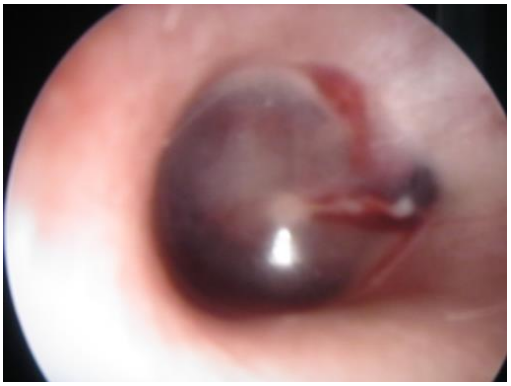

Fig.25 Hemotympanum. Endoscopic view of left tympanic membrane as a result of spontaneous epistaxis. (from Fidan et al., 2011, https://creativecommons.org/licenses/by/2.0/; unchanged)

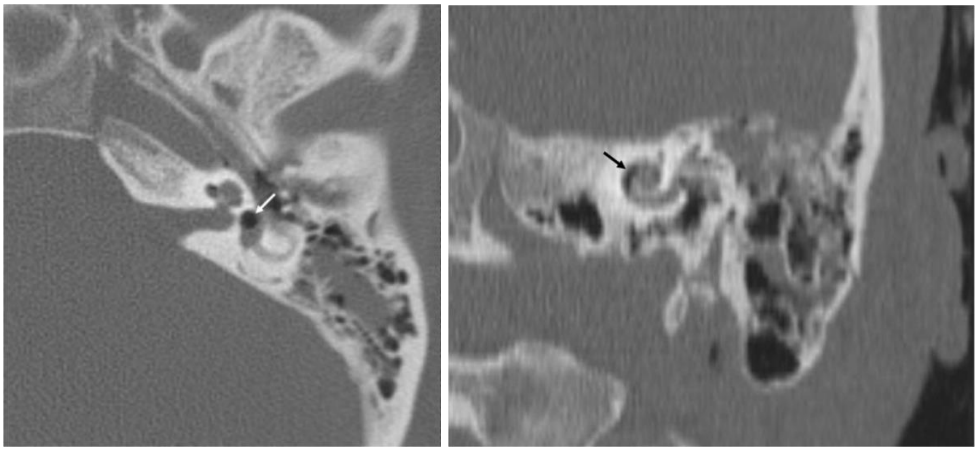

Fig.26 Left: Axial computed tomography scan of the temporal bone showing air within the left vestibule (arrow); right: Coronal computed tomography scan of the temporal bone showing air within the basal turn of the cochlea (arrow). (from Bacciu et al., 2014, https://creativecommons.org/licenses/by-nc/3.0/; unchanged),

2.5.10.2 Transversal fractures

This is a less common type of fractures. The fracture line passes perpendicular to the long axis of the petrous bone and affects the labyrinth, *meatus acusticus internus*, rarely the middle ear. The clinical picture depends on the location of the fracture line. Hearing loss is common. Depending on the location of the injury it is conductive or perception. The fracture line most often runs through the vestibule of the inner ear, i.e. the patient suffers from perception hearing disorder. Vertigo and nystagmus are also common and last for different lengths of time until loss of vestibular function is compensated. Hemotympanum may be present as well as facial nerve paresis. Depending on the degree of damage, nerve palsy can resolve spontaneously. Diagnostically, the intact external auditory canal and the entire tympanic

membrane are important. Sometimes we find hemotympanum and hearing loss. Definitive diagnosis is based on CT.

Treatment: initially conservative - prophylactically we apply broad-spectrum antibiotics penetrating the blood-brain barrier. We secure vital functions - breathing, or circulating volume during bleeding. Temporal bone fractures are associated with a risk of meningitis and an increase in intracranial pressure. Surgical treatment is indicated if meningitis is suspected at mastoiditis, and bleeding from the sigmoid venous sinus. Over time, we can surgically repair impressive fractures of the skull, solve disorders of the conductive system, etc.

3 PHONIATRICS

Phoniatrics is a special postgraduate field of otorhinolaryngology, dealing with the problems of the communication system. Phoniatrist uses otorhinolaryngological examination methods, works closely with a neurologist, psychiatrist, pediatrician, dental surgeon and other specialists.

3.1 VOICE DISORDERS

Voice disorders are temporary or permanent. They can be congenital, and more often acquired. They are manifested by a change of voice - dysphonia to aphonia. The background is mainly the unevenness of the medial edge of the vocal cord, and the disorder of the closure or tone of the vocal cords. They are classified to organic and functional. There might by no precise boundary between these groups.

3.1.1 Organic voice disorders

The most common causes of organic voice disorders are inflammation, tumors and paralysis of the nerves innervating the vocal cords. Important are also endocrine changes, thyroid dysfunction, menopause and puberty. In boys during puberty, there is a significant acceleration of growth, including the larynx. The larynx enlarges and the vocal cords lengthen, leading to phonation discoordination. The voice "jumps", decreases in frequency, it is rougher. The change is relatively short-term; prolonged mutation (*mutatio prolongata*) may

occur. It is necessary to rule out a hormonal disorder. After it is excluded, phoniatric exercises may be necessary to adjust the voice.

3.1.2 Functional voice disorders

They can occur without an organic cause. They are often the result of dysfunction of the voice apparatus, or more severe inflammation of the vocal cords. Secondary to the functional disorder, an organic change can also occur.

Functional disorders include *hysterical aphonia*. This disorder occurs suddenly, the vocal cords gently touch each other when trying to phonate and immediately separate. *Spasmodic dysphonia* is characterized by spasm of the laryngeal muscles. This causes the voice to break and have a tight, strained or strangled sound. Its creation requires a lot of effort. The laryngoscopic image is normal. Spasms only arise during speech; singing, coughing and laughing are physiological. The treatment of both problems is demanding, psychotherapy is suitable. Popular treatment is application of bottox.

Another disorder is the *ventricular voice*; it is rough, deep, and exhaled air column vibrates also thickened ventricular plicae. Typical example for this problem is chronic hyperplastic laryngitis, which one is precancerosis.

3.1.3 Speech disorders

Speech is a conditioned reflex. The basic precondition for the correct speech development is good hearing, intellect, stimulation environment, correct development of speech organs. The child begins to pronounce the first words before the first year.

Dyslalia is one of the most frequent speech disorders. It is a malfunction of some vowels, certain vowel groups are omitted, or may be replaced by others. It is a disorder of the pronunciation of the sound "s" - *sigmatism* (rustling), "r" - *rhotacism*, "l" - *labdacism*. The causes of dyslalia can be functional or organic. Organic causes include abnormalities of lips, tongue, teeth, hard and soft palate, and others.

Rhinophonia is a disorder of nasal resonance in speech. It can be closed (*rhinophonia clausa*) in impaired passage of nasal cavity (rhinitis, tumor), or open (*rhinophonia aperta*) in developmental defects such as cleft palate or lips.

Stuttering (balbuties) is a spastic coordination speech neurosis. It is manifested by spasms of the respiratory, phonation or articulatory muscles.
Tumultus sermonis is a speech disorder that is also classified as a neurosis. It is characterized by fast, poorly articulated speech.

3.1.4 Deaf-mutism *(surdomutitas)*

It is a condition that arises from uncompensated deafness from an early age. The speech apparatus can be developed normally. Deaf-mutism may be inherited, or acquired during embryonic development, perinatally, or early in the childhood.

3.1.5 Deafness and rehabilitation options

At present, with irreversible damage to the cochlear neuroepithelium, this problem can be solved by using the cochlear implant (Fig.27). It is a microdevice, which changes the acoustic stimuli into electrical impulses and conducts them by microelectrodes to *scala tympani*.

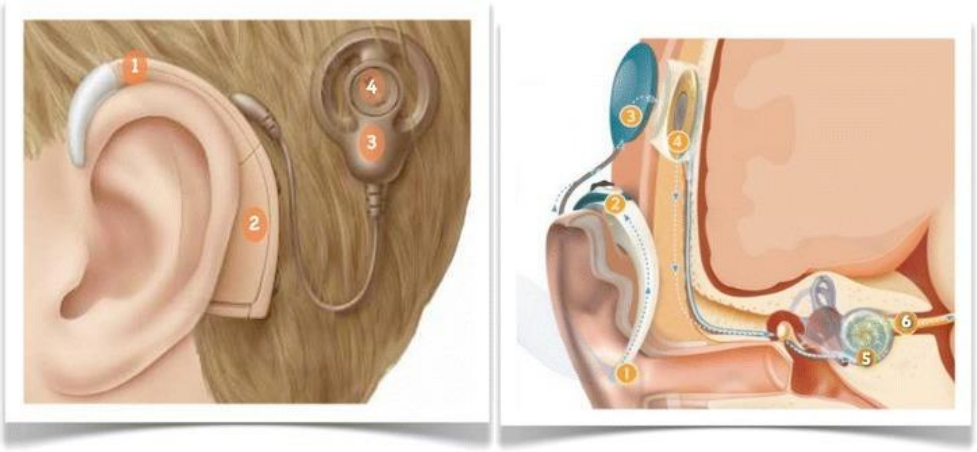

Fig.27 Left: cchlear implant (external part). 1: microphone; 2: speech processor; 3: external antenna; 4: magnet; right: how a cochlear implant works. The microphone (1) picks up sound; the speech processor (2) analyzes and transforms sound into digital information; the magnetic headpiece (3) transmits the coded signal to the surgically implanted part (4); the intracochlear electrodes (5) stimulate the cochlear nerve fibers (6). (from Vincenti et al., 2014; https://creativecommons.org/publicdomain/zero/1.0/ and https://creativecommons.org/licenses/by/4.0/, unchanged)

The perilymph is conductive environment thus the electrical signal may directly reach the auditory nerve and pass to the auditory center. This process mimics physiological conditions. This treatment is in many cases very effective but relatively costly, therefore strict indication criteria apply. It should be emphasized that this type of treatment requires a multidisciplinary approach, as in addition to diagnosis and surgical treatment, subsequent rehabilitation is necessary.

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