



Zaporozhye State Medical University

**OTORHINOLARYNGOLOGY**

**THE SELF-STUDY TUTORIAL**

*for english medical students*

**НАВЧАЛЬНО-МЕТОДИЧНИЙ ПОСІБНИК**

*з оториноларингології для самостійної роботи*

*студентів вищих медичних навчальних закладів з англійською формою  
навчання*

*Department of Otorhinolaryngology*

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## **Preface**

The speciality of Ear, Nose and Throat, now better known as Otolaryngology: Head and Neck Surgery, stands at cross-roads with several other specialities and subspecialities such as neurology, neurosurgery, oncologic surgery, ophthalmology, paediatrics, plastic and reconstructive surgery, respiratory medicine and critical care, gastroenterology, allergy and immunology. The boundaries between these specialities and ours are indistinct and frequently transgressed. This has also led to the development of several subspecialities (better called superspecialities) within Otolaryngology thus giving birth to Otology, Otoneurology, Paediatric Otolaryngology, Skull Base Surgery, Head and Neck Oncology, Endoscopic and Minimal Access Surgery, Laryngology and Phonosurgery. With advances in technology such as advanced computer imaging techniques with 3D reconstructions, endoscopes, powered instrumentation, navigational surgery, lasers, intensity modulated radiotherapy, stereotactic radiosurgery, etc. patient care has greatly improved. This has also put a greater demand on new generations of medical students who should be introduced to these advancements.

## I. STUDY PLAN

STRUCTURE	H o u r s				Course	Cinds control
	Altogether / credits	Audience		USW		
		Lecture	Practical lessons			
Modulus 1 Modulus 3	90/3 Credit ECTS	10	40	40	4	Initial, boundary
Altogether	90/3 Credit ECTS	10	40	40	4	Initial, boundary

## II. THEMES OF DISCIPLINE

### 2.1. Lectures themes.

№	THEMES AND ITS CONTENTS	Lectons
		Hours
1	Development of the modern otorhinolaryngology, achievements of modern otorhinolaryngology. Clinical anatomy, physiology and methods of examination of the middle ear. The contemporary methods of examination of the cochlear apparatus. Acute middle otitis. The peculiarities of acute otitis in children. Kinds of mastoiditis, clinical symptoms, diagnosis, treatment.	2
2	Chronic middle suppurative otitis. The contemporary methods of surgical treatment of chronic suppurative middle otitis (sanative and hearing restore operations). Nonsuppurative pathology of the ear: sensoneural deafness, otosclerosis, Meniere's disease.	2

3	Clinical anatomy, physiology and methods of examination of the nose and paranasal sinuses. Acute and chronic rhinitis. Acute and chronic sinusitis. Rhinogenic intraorbital complications.	2
4	Clinical anatomy, physiology and methods of examination of the throat. Acute and chronic tonsillitis and their complications. Acute secondary tonsillitis at infectious diseases and at blood system diseases. Hypertrophy of the lymph tissue of the throat.	2
5	Acute and chronic laryngitis. Versions of the local forms of the chronic laryngitis (hyperkeratosis, pachydermia, leukoplakia). Infection granulomas of the upper respiratory tract. Tumores of the larynx.	2
<b>Altogether hours</b>		<b>10</b>

## 2.2. The practical lessons.

<b>№</b>	<b>Themes</b>	<b>Hours</b>
1	Clinical anatomy, physiology and examination of the nose, paranasal sinuses.	4
2	Clinical anatomy, physiology and examination of the pharynx, larynx, trachea, bronchi.	4
3	Anatomy, physiology, methods of examination auditory analyzer and vestibular analyzer, external and middle ear, structure of the cochlea.	4
4	Diseases of the nose (acute catarrhal rhinitis, lesion of the nasal mucosa in measles, scarlet fever and diphtheria, chronic rhinitis).	4
5	Diseases of the paranasal sinuses (acute and chronic form)	4

	sinusitis).	
6	Diseases of the pharynx (acute tonsillitis, peritonsillar abscess (quinsy, tonsillitis in patients with blood diseases, parapharyngeal and retropharyngeal abscesses, acute pharyngitis, chronic tonsillitis, chronic pharyngitis, pharyngomycosis, adenoids)	4
7	Diseases of the larynx (acute laryngitis, subglottic laryngitis (false croup), submucous laryngitis (angina laryngea), phlegmonous laryngitis, chondroperichondritis of larynx, acute and chronic stenosis of the larynx, tumors of the larynx)	4
8	Diseases of the external and middle ear (inflammation of the external acoustic meatus, otomycosis, earwax plug, acute catarrh of the eustachian tube, acute inflammation of the middle ear, acute otitis media in children, acute mastoiditis, antritis, chronic suppurative otitis media)	4
9	Tumors of the larynx. Infection granulomas of the upper respiratory tract.	4
10	The traumas, foreign bodies, bleedings of the ENT organs and first aid. (the traumas of the nose, the nasal bleedings and first aid, foreign bodies of the respiratory tracts and esophagus, burns of esophagus)	4
<b>Altogether hours</b>		<b>40</b>

### 2.3. The unassisted student's work (USW)

№	Topic	Hours
<b>1</b>	<b>Preparation for practical lessons</b>	22
<b>2</b>	<b>USW at topics</b>	
2.1	Variants of chronic hyperplastic laryngitis	2
2.2	The rhinogenic orbital and intracranial complications	2
2.3	Contemporary methods of surgical treatment of chronic purulent middle otitis	2
2.4	Methods of a stopping of nasal bleedings	2
<b>3</b>	<b>Individual USW</b>	
3.1	Libray research paper preparation	8
3.2	Libray review preparation or research work	2
<b>Altogether hours</b>		<b>40</b>
<b>4</b>	<b>USW at topics which are not included to auditorial classes</b>	
№	Topic	Hours
4.1	Secondary tonsillitis at infection diseases and diseases of the blood system	2
4.2	Forms of mastoidites	2
4.3	Hypertrophy of lymphoid tissue of the pharynx	2
4.4	Burns of the esophagus	2

## **Topic 1:**

### **«Variants of chronic hyperplastic laryngitis»**

*The number of hours – 2*

**Reason:** Larynx being a part of the air conducting tracts of the organism, participates in fulfilling the main functions of breathing, phonation and speech. The violation of the normal anatomical and functional relationships in the larynx leads to different pathological processes, firstly discovered by the development of the nose, larynx and voice disfunction. Chronic larynx diseases running with the disturbance of breathing and vocal functions, are often met in the clinical practice. Sick persons with chronic laryngitis are subjected to differential diagnosis from other larynx diseases, benign and malignant tumours.

**The educational purposes:**

*The students should know:*

1. Cause invoking chronic hyperplastic laryngitises;
2. Main clinical signs of this pathology;
3. Principles of treatment.

*The students should know how:*

1. To fulfil an indirect laryngoscope;
2. To put the diagnosis and to carry out the differential diagnosis.
3. To select conforming medical tactics.

**The information block.**

Three forms of the disease are encountered: catarrhal, hyperplastic, and atrophic. Disturbed vocal function is typical for all three forms. Occupational hazards, smoking, and vocal overstrain help maintain chronic inflammation in the larynx.

High-temperature cigarette smoke dries mucosa of the larynx, trachea, and bronchi. In addition, it evokes congestive hyperaemia of the mucosa, thus maintaining inflammation with a large amount of sputum. The latter, in turn,



provokes cough, especially in the morning because smoke suppresses the function of ciliated epithelium of the larynx, trachea, and bronchi, and therefore during the daytime the cough reflex is reduced: transportation of the accumulated sputum to the infraglottic cavity is difficult. At night, ciliated epithelium restores its function to some extent and can transport sputum to the lower larynx to stimulate the cough reflex and facilitate expectoration. Cough may be violent since viscid sputum adheres to the walls and is expectorated with difficulty. Resultant microinjuries to the laryngeal mucosa also maintain inflammation.

**Chronic catarrhal laryngitis.** Persistent, mild, and diffuse hyperaemia of the entire larynx, and moderate oedema are typical.

**Chronic atrophic (dry) laryngitis** resists treatment, and only palliative therapy can be used. Dystrophy developing in the mucosa of the larynx, nose, pharynx, and trachea underlies this form of chronic laryngitis. This is a systemic disease of the mucous membrane. The patient's complaints include severe dryness and a feeling of a foreign body in the throat, voice impairment (up to aphonia), and recurrent crusts. The final diagnosis can be established after indirect laryngoscopy which reveals pale, exsanguineous, and thin mucosa; thin vocal cords fail to join in the midline: viscous yellowish sputum forms crusts in the lumen of the rima glottidis.

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

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

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

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

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

**Polypoid degeneration of vocal cords (Reinke's oedema)**. It is bilateral symmetrical swelling of the whole of membranous part of the vocal cords, most often seen in middle aged men and women. This is due to oedema of the subepithelial space (Reinke 's space) of the vocal cords. Hoarseness is the common symptom. Patient uses false cords for voice production and this gives him low-pitched and rough voice. Vocal cords show pale, translucent fusiform swellings. Ventricular bands may appear hyperaemic and hypertrophic and may hide view of

the true cords. Treatment: Decortication of the vocal cords, i.e. removal of strip of epithelium, is done first on one side and 3-4 weeks later on the other. Voice rest. Speech therapy for proper voice production.

***Stuffs for selfverification.***

**Questions for selfverification:**

1. Name joints of a larynx.
2. Name cartilages of a larynx.
3. Name external muscles of a larynx.
4. Than the vestibule of the larynx is derivated.
5. Name anatomical derivations of a middle department of a larynx.
6. Enumerate main functions of a larynx.
7. Scheme of stages of survey of a larynx at an indirect laryngoscope.

***Theme of educational research work of the students on the given subject:*** To draw laryngoscopy picture of one of the circumscribed forms of a hyperplastic laryngitis.

## **Topic 2:**

### **«Rhinogenic orbital and intracranial complications»**

*The number of hours – 2*

The theme actuality. Despite of successes of modern medicine in diagnostics and treatment of acute and chronic sinuites, still quite often there are cases of rhinogenic orbital and intracranial complications. More often it is connected that the patients sometimes are engaged in a selftreatment and do not access is well-timed behind a medical care. Are be of accidental errors of diagnostics and inadequate treatment. The given pathology can result in loss of vision and even morses. Therefore well-timed treatment and competently assigned therapy are success of liquidation of these complications.

#### **The educational purposes.**

##### ***The students should know:***

1. Clinic of intracranial and orbital complications;
2. The indications to conservative and surgical treatment;
3. Peculiarities of support of the postoperative period.

##### ***The students should know how:***

1. To determine a degree of the inflammatory process in paranasal sinuses, orbit, head cavity;
2. To assign adequate conservative treatment or surgical intervention on paranasal sinuses, orbit, head cavity;
3. To organize postoperative support of the given patients.

#### **Theoretical contents of a subject.**

##### ***Orbital complications include:***

(a) *Inflammatory oedema of lids.* This is only reactionary. There is no erythema or tenderness of the lids which characterises lid abscess. Eyeball

movements and vision are normal. Generally, upper lid is swollen in frontal, lower lid in maxillary, and both upper and lower lids in ethmoid sinusitis.

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

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

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

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

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

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

**Intracranial complications.** Rhinogenic intracranial complications are very dangerous. In 75 per cent of the cases, rhinogenic intracranial complications arise

due to chronic inflammation in the sinuses, and in 25 per cent of the cases, they are secondary to acute sinusitis. The infection can spread by the contact, haematogenic and lymphogenic pathways. Frontal, ethmoid and sphenoid sinuses are closely related to anterior cranial fossa and infection from these can cause: (a) Meningitis and encephalitis, (b) Extradural abscess, (c) Subdural abscess, (d) Brain abscess,

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

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

**Stuffs for selfverification.**

***Questions for selfverification.***

1. Name a paranasal sinus, which inflammation in early childhood yields orbital and intracranial complications.
2. What pathes of spreading of an infection contamination to a head cavity and orbits?
3. Name orbital and intracranial complications.
4. Change of vision, position of an eyeglobe at orbital complications.
5. Clinic retrobulbar absces.
6. Clinic of a phlegmon of an orbit.
7. Clottage of cavernous sine.
8. Feature of conservative and surgical treatment of orbital complications.
9. Name rhinogenic intracranial complications.
10. The methods of diagnostics of intracranial complications.
11. The data of spinal liquor at different intracranial complications.
12. What main purpose of the operation at intracranial complications?
13. What essence of prophylaxis of orbital and intracranial complications?

***Subject of educational research work of the students:*** To make the classification table of orbital and intracranial rhinogenic complications.

### **Topic 3:**

#### **«Contemporary methods of surgical treatment of chronic purulent middle otitis»**

*The number of hours – 2*

**Theme:** Contemporary methods of surgical treatment of chronic purulent middle otitis.

**The actuality of the theme.** Chronic purulent middle otitis is the most frequent disease of the ear and you can meet it in 20-25 per cents of cases among the all pathology of ENT organs. But unsymptomatically taking chronic otitis, especially epitympanitis, can suddenly causes the hard intracranial complications (meningitis, sepsis, brains abscesses, etc.) The frequency of chronic suppurative inflammation of the middle ear, its aggravation leading to a temporary and sometimes permanent loss of working ability, to the development of diminished hearing and other dangerous complications define social significance of the disease. Any physician must know the symptoms of chronic suppurative middle otitis and its complications. He must to be able to prevent its development, and if necessary he must send the patient to the hospital for urgent treatment. That's why the knowing of contemporary methods of surgical treatment of chronic purulent middle otites is necessary for doctors of different types.

#### **The educational purposes.**

##### ***The students should know:***

- The indications and contraindications to various forms of an operative intervention at chronic purulent middle otites;
- The indications to various forms of sanative operations at chronic purulent epitympanitis;
- The indications and contraindications to hearing restoring operations;



- Forms of sanative operations;
- Stages and types of tympanoplasties;
- Peculiarities of current and support of the postoperative period.

***The students should know how:***

- To determine a degree of the destructive process in the middle ear at chronic purulent otitis;
- To assign an adequate method of a surgical intervention in dependence on the form of a chronic purulent middle otitis and given acumen;
- Correctly to assign medicament therapy in the postoperative period.

**Theoretical contents of a subject.**

Treatment of chronic suppurative epitympanitis is more difficult than of chronic suppurative mesotympanitis. Conservative treatment is effective in cases with anterior epitympanitis. Local treatment includes daily irrigation of the attic by attic needle with the following warm solutions: 40% alcohol, 3% alcohol solution of boric acid, 0,25% solution of formaldehyde. Conservative treatment is usually ineffective in cases with the medial and posterior location of the marginal perforation in the superior parts of the tympanic membrane. A surgical intervention is necessary in such cases. If chronic inflammation is confined to the attic, atticotomy is performed. Attico-antrotomy is necessary if the process extends to the antrum. Both operations are sparing; the hearing function is preserved. If caries extends to the cavities of the middle ear, the diagnosis is confirmed otoscopically, roentgenographically, and by other instrumental examinations, a radical surgery is necessary. The presence of signs of intracranial complications (sinus thrombosis, meningitis, cerebral abscess and abscess of the cerebellum) is the absolute (vital) indication for a radical operation on the ear in suppurative otitis media. The operation should be performed immediately. Surgical intervention is also required in the presence of signs of mastoiditis, paresis of the facial nerve, and

labyrinthitis. In the other cases the extent of operative intervention should be determined with consideration of the auditory and vestibular functions of the ear.

***The radical operation.*** The radical operation essentially consists in the tympanic cavity, the epitympanic recess, the antrum with the remaining mastoid cells and the external auditory meatus being thrown into one wide cavity. Therefore, this operation is also known as radical mastoidectomy. A thorough removal of carious bone and the cholesteatoma will ensure free pus drainage through the auditory canal and prevent possible intracranial complications.

The operation begins with opening the antrum, as in mastoidectomy; next follows the removal of the upper section of the posterior bony wall of the external auditory meatus and the external wall of the attic. Here, in the depth of the operative cavity, great care must be taken to avoid injury to the facial nerve, as the descending knee of the facial nerve canal is located in the depth of the posterior bony wall of the auditory meatus. The concluding stage of the operation is removal of all necrotic auditory ossicles apart from the stapes. Polyps, granulations and carious bone are carefully removed with a curette. The operation is rounded off with a plastic repair in order that the walls of the operative cavity may later be overgrown with epidermis. For this purpose one or two flaps are cut out of the skin of the posterior wall and roof of the external auditory meatus and are transplanted on to the lower or upper parts of the wound. The flaps serve as a source of epidermis for the whole trephination field. The skin wound behind the ear is either sutured or left open if the meninges are exposed. The operation area is packed with a tampon soaked in iodoform or antibiotic solution. Dry dressing is first applied on the sixth to eighth day following the operation, provided there is no fever or pain in the wound. The postoperative treatment is rather complicated and normally continues for at least six to eight weeks. In some cases, tympanoplasty is performed if there is no affection of the middle ear together with signs of an intracranial complication. The aim of this operation is not only to remove

pathological tissue from the middle ear but to repair the drum. Not infrequently the hearing also improves as a result of this operation.

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

Contraindications for tympanoplasty are the following: (a) exacerbation of chronic otitis and especially the presence of labyrinthine, intracranial or septicopyemic complications; (b) considerable affection of the sound perception apparatus; (c) obstruction of the auditory tube. Reconstructive surgery of the ear has been greatly facilitated by development of operating microscope, microsurgical instruments and biocompatible implant materials.

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

in the middle ear. *Functioning eustachian tube* to provide aeration to the middle ear. *A functioning sensorineural apparatus*, i.e. the cochlea and VIIIth nerve.

**Types of tympanoplasty.** Wullstein classified tympanoplasty into five types:

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

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

**Type III** Malleus and incus are absent. Graft is placed directly on the stapes head. It is also called myringostapediopexy or columella tympanoplasty.

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

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

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

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

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

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

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

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

### **Stuffs for selfverification.**

#### ***Questions for selfverification:***

1. Name the absolute indications to the radical operation.
2. What relative indications for execution of the sanative operation on a middle ear?
3. What main purpose of sanative operations?
4. Name principles of the radical operation.
5. Main surgical stages of the radical operation.
6. What complications of the radical operation are possible?

7. Peculiarities of a postoperative period current of trepan cavum.
8. What final aim of postoperative period current of trepan cavum?
9. What clinical signs and the diagnostic aspects determine a choice of a method of sanative the operation?
10. The indications and contraindications to an atticotomy.
11. Name principles of the atticotomy.
12. The indications to tympanoplasties.
13. Name contraindications to hearing restoring operations.
14. What main stages of hearing restoring operations?
15. Name main types of a tympanoplasty on Wulshtein, indication to performing of this or that type.
16. What peculiarities of medicamental therapy and dressings after the operation of a tympanoplasty?

***Subject of educational research work of the students:*** To make a diagrammatic representation of the radical operation of a middle ear, atticotomy, 5 forms of a tympanoplasty.

## **Topic 4:**

### **«Methods of a stopping of nasal bleedings»**

*The number of hours – 2*

**Theme actuality.** The nasal bleedings in medical practice meet frequently. They are be so strong, that there is a threat for life. Therefore knowledge of methods of a stopping of nasal bleedings is necessary for the doctor of any profile the same as at bleedings from mild, stomach, uterus, trunk pots.

**The educational purposes.**

*The students should to know:*

- Peculiarities of anatomy of a mucosa of a nasal cavity and of paranasal sinuses;
- Peculiarities of a circulation of a mucosa and external tissues of a nose;
- Etiology and pathogenesis of nasal bleedings;
- Methods of chemical caustic of a mucosa of a nose, cryolysis, anterior and posterior tamponade of a nasal cavity;
- The indications to a dressing of an external carotid artery;
- The indications to ethmoidotomy;
- Technique of the operation of a dressing of an external carotid artery.

*The students should know how:*

- To stop a bleeding from Kisselbach's plexus;
- To make a anterior and posterior tamponade of a nasal cavity;
- To assign medicamental therapy for lowering arterial pressure, improve of the coagulating system of a blood.

## **Contents of a subject.**

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

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

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

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

***Clinical picture.*** It should be remembered that blood can get into the nose from other parts of the upper airways, e.g. from the pharynx, larynx, trachea, oesophagus, the lung and sometimes even from the middle ear through the auditory



tube. The diagnosis is established by rhinoscopy, pharyngoscopy, and inspection of the other related organs.

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

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

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

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

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

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

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

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

*Posterior tamponade* is done with special sterile tampons. Gauze is folded several times into 3 x 2.5 x 2 cm tampon which is then tied up crosswise with two 20-cm long silk threads. One end is cut off, while the other three ends remain. The

posterior tamponade is begun with passing a thin rubber catheter into the bleeding side of the nose until its end enters the nasopharynx to appear in the middle of the pharynx. The end of the catheter is taken with a forceps and pulled outside through the mouth. Two threads of the tampon are tied up to this end of the rubber catheter and pulled back through the nose. The second finger of the right hand should be used to help to seat the tampon behind the soft palate in the nasopharynx and press it tightly to the corresponding choana. The next manoeuvre is to pull the two threads through the nose. The threads should be held strained while the nose is packed with the turunda and the thread ends are then tied tightly over a gauze pad at the nasal vestibule. The thread in the mouth will be used to withdraw the tampon. Its free end is fixed on the cheek with an adhesive tape.

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

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

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

### **Stuffs for selfverification.**

#### ***Questions for selfverification.***

1. Name the endogenic etiological factors of nasal bleedings.
2. Name the exogenous etiological factors of nasal bleedings.
3. What anatomical features of a structure of a mucosa of a nasal cavity?
4. Why the bleedings from ethmoidal arterias are dangerous?
5. Local conservative methods of a stopping of nasal bleedings.
6. Surgical methods of a stopping of nasal bleedings:
  - a. operation of a dressing of an external carotid artery;
  - b. ethmoidotomy with endonasal and external access;
7. Medicamental therapy of a stopping of nasal bleedings.

***Subject of educational research work of the students:*** To make the plan of a sequence of operations for a stopping of nasal bleedings.

## **Topic:5**

### **«Secondary tonsillitis at infection diseases and diseases of the blood system»**

*The number of hours – 2*

**The theme actuality.** The angina as a sign of other disease meets at children's illnesses (scarlet fever, diphtheria, mononucleosis), at virus lesions of upper respiratory tracts, at blood diseases. In this connection in each concrete case there is a necessity legibly to designate character of disease, its clinical signs showing in upper respiratory routes. Knowledge of the given subject are necessary for the doctors of many specialties.

#### **The educational purposes:**

##### ***The student should know:***

1. Cause of acute secondary anginas, their clinical exhibiting.
2. To differentiate features of inflammatory changes in a pharynx in dependence on the etiological factor.
3. Characteristic changes of morphological composition of a blood at an agranulocytosis, contagious mononucleosis, ulcerative anginas.

##### ***The student should know how:***

1. To collect the anamnestic data and to estimate pharyngoscopy picture.
2. To carry out the differential diagnosis at various lesions of a blood.
3. To decrypt the data of laboratory researches.

#### **Contents of a subject.**

### **AFFECTIONS OF THE PHARYNX IN SYSTEMIC DISEASES**

**Infectious mononucleosis.** This infectious disease is probably caused by a special lymphotropic virus which occurs together with *Listerella* genus. It is

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

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

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

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

*Symptoms.* The prodromal period is characterized by indisposition; it lasts 1-2 days. Fulminant, acute, and subacute forms of agranulocytosis are distinguished. In the former two cases the disease begins with high temperature (to 40° C), chills,

and bad general condition. Necrotic and ulcerative changes in the pharynx, mainly in the region of the palatine tonsils, occur simultaneously. Necrosis often spreads onto the mucous of the pharynx, gums, and the larynx. In rare cases, the destructive changes occur in the intestine and the urinary bladder. Necrosis can extend onto deep underlying soft tissues and bones.

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

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

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

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

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

Ingestion of this grain, in particular millet, will cause a bitter taste and a burning sensation in the mouth, pharynx, esophagus and stomach, as well as numbness in the tongue. These symptoms are often accompanied by nausea, vomiting, and headache. Yet in other cases, the absorption of this food for only two or three weeks is followed by headache, prostration and weakness. Punctate hemorrhage looking like flea bites appears on the skin. Already at this early period of septic angina, blood analysis will reveal a progressive reduction in the leukocyte count, viz., onset of the period of leukopenia. The whitish or yellowish-brown membrane which appears on the tonsils marks the onset of necrosis which soon, in

fact in 24 hours, causes deep ulcers . This ulceration commonly affects not only the tonsils which soon collapse completely but other aggregations of lymphadenoid tissue as well, and may extend to the palatine, pharyngeal and esophageal mucosa and, sometimes, to that of the oral cavity.

Withdrawal of toxic products from food at the initial period of the disease, prior to the onset of anginal symptoms, may often bring recovery, especially if the total amount of toxic food eaten has been moderate. Advanced septic angina is frequently fatal.

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

Intramuscular antibiotics injections have been used with success.

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

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



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

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

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

**Table of Distinctive Symptoms of Diphtheria and Lacunar Tonsillitis**

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

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

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

### **Stuffs for selfverification.**

#### ***Questions for selfverification.***

1. What is an angina?
2. Secondary anginas, their classification, cause.
3. Feature of clinical current of a ulcerative angina.
4. Treatment of an angina Symanovsky-Vensan.
5. Forms of a diphtheria of a pharynx.
6. Differential diagnosis of a lacunar angina and a diphtheria of a pharynx.
7. Clinical changes at a infection mononucleosis.
8. Characteristic changes of the formula of a blood at a infection mononucleosis, agranulocytosis, alimentar-toxic aleukia.
9. What medicinal preparations render toxic effect on an bone brain?
10. The principles of treatment of an agranulocytosis.

***Subject of educational research work of the students:*** To make graphological frame of a subject «Acute secondary tonsillitis».

**Topic:6**  
**«Forms of mastoidites»**

*The number of hours – 2*

**Actuality of topic:** Acute purulent mastoidites is called inflammatory infectious disease of mastoid processes. Today mastoidites occurs quite frequently within the population of different age groups and particularly frequent in child age due to anatomic peculiarities of structure of middle ear in this age, as well as tendency towards infectious diseases, which are complicated by diseases of ear. Suffered mastoidites may be the reason of stable hardhearing, of development of chronic inflammation of middle ear, threatening intracranial complications. Probability of the latter is related with no diagnosis at right time, as well as with mistakes in treatment tactics of mastoidites. Therefore doctor of many different specialities come across with contingent of such patients.

**The educational purposes.**

***The students should know:***

- Possible terms of a beginning of an inflammation in a mastoid;
- Clinical signs of mastoidites;
- Methods of diagnostics of mastoidites;
- The clinical forms of typical and atypical mastoidites;
- Methods of treatment of mastoidites;
- Tactics of treatment in the postoperative period.

***The students should know how:***

- On a basis of knowledge of a clinical signs to put the diagnosis a mastoiditis;
- To estimate of otoscopy picture of the patient with a mastoiditis;

- To estimate a roentgenogram on Shuller;
- To determine a stage of an acute mastoiditis;
- To assign adequate treatment in dependence on a stage of a mastoiditis;
- Correctly to carry out conservative treatment of the first stage of a mastoiditis.

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

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

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

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

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

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

The mastoid process, especially the apex, and sometimes its posterior margin, are very tender to palpation. Inflammation in the mastoid process can be

activated causing subperiosteal abscess due to passage of pus from the mastoid cells to the periosteum. The differential blood count shifts to the left; the leukocyte count is moderately high; the ESR gradually increases.

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

**Zygomatoc** abscess. It is due to infection of zygomatic air cells situated at the posterior root of zygoma. Swelling appears in front of and above the pinna. There is associated oedema of upper eyelid. Pus in these cases collects superficial or deep to temporalis muscle.

The apex- cervical forms of mastoiditis:

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

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

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

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

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

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

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

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

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

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

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

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

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

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

each particular case, especially in children.

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

**Basic differential diagnostic symptoms of AMO and mastoiditis.**

Symptoms	AMO	Mastoiditis
General (overall) condition	Improves	Inspite of treatment deteriorates
Pain in ear	After perforation decreases	Inspite of perforation does not decrease
Noise in ear	Gradually decreases	Inspite of treatment does not decrease
Hearing	Improves	Does not improve
Excretion from ear	Stands less, after then disappears. From serous - blood and mucoid-purulent stands mucoid	Purulent; purulent-blood in very big quantities
Palpation of mastoid process	Painless, may be painful during the first days of disease (mastoidal reaction)	Sharply painful
Skin of postauricular region	Unchanged	Infiltrated, swollen mastoid process, smoothness of postauricular fold



Change in tympanic membrane and external acoustic meatus	Correlative to stages	Infiltrated, thickened (mastoidal type); hanging of postero-superior wall of acoustic meatus
Percussion of mastoid process	Painless	Painful

**Differentiative symptoms of mastoiditis and furuncul of external acoustic meatus.**

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

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

Indication:

1. Acute coalescent mastoiditis.
2. Incompletely resolved acute otitis media with reservoir sign.
3. Masked mastoiditis.
4. As an initial step to perform:
  - a. endolymphatic sac surgery
  - b. decompression of facial nerve
  - c. translabyrinthine or retrolabyrinthine procedures for acoustic neuroma.

Patient lies supine with face turned to one side and the ear to be operated uppermost. A curved incision is made behind and following the attachment of the auricle. The incision extends from a point on a level with the upper margin of the pinna to the mastoid tip. In infants and children up to 2 years, the incision is short and more horizontal. This is to avoid cutting facial nerve which is superficial in the lower part of mastoid. Incision cuts through soft tissues up to the periosteum. Temporalis muscle is not cut in the incision. Periosteum is scraped from surface of mastoid and posterosuperior margin of osseous meatus. Tendinous fibres of sternomastoid are sharply cut and scraped down. The lips of the wound are drawn apart with retractors to keep the mastoid surface open for examination. Should a fistula be darkened and soft portions of bone be discovered, the operation must be started at this place. Should a fistula be absent, the operation must be started in a typical place determined by landmarks. The upper border of the operative area is the temporal line; the anterior border is the spine above the external auditory meatus and the latter's posterior wall. Trephination is begun by attacking the bone right behind the spine on the *planum mastoideum* to the antrum. In an adult antrum lies 12-15 mm from the surface. Horizontal semicircular canal is identified.

All the carious and soft bones should be removed carefully until the antrum has been exposed. The antrum is then widened somewhat with a small curette, and the granulations are thoroughly scraped out with utmost care. Care must be taken

in opening the mastoid process to avoid injury to the sigmoid venous sinus, the dura mater, the middle cranial fossa, the facial nerve and the external semicircular canal. Lateral wall of the mastoid tip is removed exposing muscle fibres of posterior belly of digastric. Zygomatic cells situated in the root of zygoma, retrosinus cells lying between sinus plate and cortex behind the sinus are removed.

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

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

*Complications:*

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

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

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

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

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

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

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

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

### **Stuffs for selfverification.**

#### ***Questions for selfverification:***

1. Etiological factors of development of a mastoiditis.
2. Role of virulence of a microflora, reactivity of an organism in development of a mastoiditis.
3. Role of the local factors (structure of a mastoid, nasal respiration, presence of the locuses of an infection contamination of upper respiratory ways).
4. Stage of current of a mastoiditis.
5. Early and late clinical signs of a mastoiditis.
6. Otoscopy picture of an acute mastoiditis.

7. X-ray diagnostics of purulent mastoidites.
8. Type of mastoidites on localization.
9. Atypical current of mastoidites.
10. The differential diagnosis of a mastoiditis with a furuncle of external acoustical passage.
11. The differential diagnosis of a mastoiditis with a neuralgia of an occipital nerve.
12. Conservative treatment of mastoidites (local and general), indication, criterion of an assessment of efficacy.
13. Surgical treatment of mastoidites, indication.
14. Technique and possible complications of a mastoidotomy.
15. Peculiarities of postoperative period, assignment of adequate conservative therapy.

**The tests for selfverification.**

***Subject of educational research work of the students:*** To make the table of the typical and atypical forms of acute purulent mastoidites in dependence on primary localization of a suppurative focus of an osteomyelitis of a temporal bone (in figure of temporal bone to designate places of localization of the process).

## **Topic: 7**

### **«HYPERTROPHY OF LYMPHOID TISSUE OF THE PHARYNX»**

*The number of hours – 2*

**The theme actuality:** In dependence on a level of function activity the adenoid tissue of a pharynx undergoes appreciable changes in volume. The dimensions of lymphatic derivations depend on influence of many factors, such as, state of protein metabolism, endocrinologic function, transferred inflammatory diseases, feeding habits, conditions of surrounding. In some cases the increasing of volume of an adenoid tissue wears nonperishable character, that produces distress of functions of a nose, ear, voices and unfavorably has an effect for a common state of the patients. Differential diagnostics of a hypertrophy pharynx and palatine tonsils with other volumetric processes for a choice of rational tactics of treatment is important.

#### **The educational purposes.**

#### ***The students should know:***

1. Cause of appearance of a hypertrophy of an adenoid tissue of a pharynx.
2. Clinical sings of adenoid growths and hypertrophy of palatine tonsils.
3. Methods of diagnostics of the given pathology.
4. Indication to operating treatment.
5. Possible complications of operating treatment.

#### ***The students should know how:***

1. To fulfil front and back rhinoscopy.
2. To fulfil digital examination of a nasopharynx.

3. To carry out the differential diagnosis between a hypertrophy of an adenoid tissue of a nasopharynx with tumoral lesions.
4. To fulfil a pharyngoscope.
5. To assign the indications to a tonsilectomy.
6. To assign conservative therapy in the postoperative term.

### **The information block.**

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

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

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

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

Three degrees of adenoid growth are distinguished: degree I-adenoids cover to one third of the vomer; degree II-about half of the vomer is covered; degree III-the vomer is covered to two thirds or almost completely. Degree I adenoids do not

impair significantly respiration through the nose in child. If a child is ill for a long time, the face bones become distorted: the dropping jaw becomes narrow and long, while the hard palate undergoes malformations: it becomes high and narrow; incorrectly growing teeth cause malocclusion. These changes give a specific dull expression to the face of children with adenoid growths (adenoid facies).

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

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

The surgical removal of the adenoids (adenoidectomy) is performed in cases where the enlarged tonsil impedes respiration through the nose. The operation is usually performed at the age from 5 to 7, but infants and adults can also be operated on if nasal breathing is pathologically impeded, the hearing function is impaired, or other diseases concur. Children can be operated under out-patient conditions, while adults only in hospital. Children should not take breakfast on the day of the operation. The operation can be performed either without anaesthesia, or after instilling 5 drops of a 10 per cent lidocaine solution into each side of the nose. Contraindications for adenoidectomy are diseases of the blood, severe diseases of the cardiovascular system, and infectious diseases (the patient may be operated on only in 1-2 months after the disease). An important pre-operative measure is immobilization of the child. The nurse sits on a stool or in a surgical chair facing the surgeon and holds the child in her laps so that his legs are fixed between the nurse's knees; the right arm is used to hold the child's arms and the trunk, while the left arm holds the child's head. A sterile cloth should cover both the nurse and the child. The operated child is placed on his side in bed on a low pillow for 25-30 minutes. Hot or spicy foods should not be given, since bleeding can resume for 4-5



days. Physical strain, active movements and chilling should be avoided.

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

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

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

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

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

complications are catarrhal or purulent otitis media, retropharyngeal abscess, suppuration of the regional lymph nodes, and also general infectious complications.

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

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

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

The essential element of a nasopharyngeal fibroma is dense connective tissue containing a great number of elastic fibres and blood capillaries. The tumour is histologically benign, but for its clinical course marked as it is by irresistible growth and destruction of the surrounding tissue, postoperative relapses and frequent copious hemorrhages endangering the patient's life, it may sooner be classified as a malignant neoplasm.

It may extend into :

1. Nasal cavity causing nasal obstruction, epistaxis and nasal discharge.
2. Paranasal sinuses. Maxillary, sphenoid and ethmoid sinuses can all be invaded.
3. Pterygomaxillary fossa, infratemporal fossa and cheek.

4. Orbits giving rise to proptosis and "frog-face deformity". It enters through the inferior orbital fissure and also destroys apex of the orbit. It can also enter the orbit through superior orbital fissure.
5. Cranial cavity. Middle cranial fossa is the more common.

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

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

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

In neglected cases where the tumour invades the zygomatic area and paranasal sinuses the removal is performed only after a preliminary operation has been undertaken to provide access to the tumour proper. This access may be obtained through the sinus maxillaris with complete removal of the lateral nasal

wall. There may be about 2 litres of blood loss during surgery. Therefore attempts are made, pre-operatively, to reduce the vascularity of tumour. A course of oestrogen therapy may reduce vascularity of tumour. Preoperative radiation also helps to reduce vascularity but is not generally.

**Stuffs for selfverification.**

***Questions for selfverification:***

1. Name the causes of a profound hypertrophy of an adenoid tissue of a pharynx.
2. Classification of a hypertrophy of a pharyngeal tonsil.
3. Main clinical signs of a hypertrophy of a pharyngeal tonsil.
4. Indication and contraindication to an adenotomy.
5. Possible complications of an adenotomy.
6. Features of support of the postoperative term after an adenotomy.
7. Main clinical signs of a hypertrophy of palatine tonsils.
8. Classification of a hypertrophy of palatine tonsils.
9. Indication and contraindication to a tonsilectomy.
10. Possible complications after a tonsilectomy.
11. Differential diagnostics of a hypertrophy of a pharyngeal tonsil and youthful fibroma of a nasopharynx.

***Subject of educational research work of the students:*** To make a diagrammatic representation of degrees of a hypertrophy pharyngeal and palatine tonsils.

## Topic:8

### «Burns of the esophagus»

*The number of hours – 2*

**Reason.** Laryngeal and esophageal burns with different chemical substances are met rather often. It is a severe pathology with a great percentage of fatality because of different complications. Unfavorable outcome of the disease may occur both on the first days after burn and during its treatment.

All mentioned above proves that patients with chemical burns need urgent medical aid which should be given by any practitioner.

As example we give the following observation: patient L., 60 y.o. had been admitted to the hospital because of acute chemical esophageal burn with vinegar essence. The patient complained of the pain in the mouth, pharynx, along esophagus and epigastric area, bloody vomiting, acute general weakness. Case history cleared that a day ago he drank 100 ml of vinegar essence. At the local hospital he got the first medical aid: his stomach had been drained with the solution of  $\text{Na}_2\text{CO}_3$ , cardiac remedies had been injected. Because of worsening of his general condition and anuria (30 ml of urine per a day) the patient had been transported to the central hospital.

**OBJECTIVELY:** patient's general condition is very severe, he is slow, adynamic, skin and visible mucosal membranes are icteric, covered with cold sweat. Pulse is 100 beats per minute, arrhythmic, blood pressure is 90/50 mm Hg, cardiac sounds are dull. There is a bright hyperemia as stripes to the chin. Mouth mucosal membrane, pharynx is covered with rough white patch and starting ulcerations in the area of the tongue and posterior wall of the pharynx. In spite of resuscitation performed, the patient died in 12 hours after admission under aggravating cardiac weakness.

**CONCLUSION** of the forensic medical examination: poisoning with vinegar essence, vast necrosis of the esophageal mucosal membrane. Acute nephronnecrosis. Intoxication.

The example given above shows that promoted shifts took place in the homeostasis due to the intoxication and burn with vinegar acid. Thus, during this classes the students have to learn complex of therapeutical measures of the first medical aid for this group of patients. At the same time one should realize that variety of symptoms and clinical course of esophageal burns, possibility of complications development, complication of therapeutical process need admission of a patient to the in-patient department immediately after the incident (reanimation, surgical, E.N.T.).

Duration of the classes - 90 min.

The aim of the classes.

*A student should know:*

1. causes of esophageal burns;
2. factors influencing on the degree of damage under chemical burns;
3. pathologoanatomical stages of esophageal burns;
4. clinical symptoms of esophageal burn (3 stages).

*A student should be able:*

1. to give urgent medical aid during first hours after esophageal burn;
2. to determine range of therapeutic measures for the whole period of treatment.

**Tasks for independent students' work.**

For the achievement of the aim of the classes a student should have basic knowledge from the previous themes on otorhinolaryngology:

1. Peculiarities of esophageal structure, its physiological and anatomical narrowing;
2. Structure of esophageal walls.

**Tasks for self-control of the basic level of knowledge.**

1. On what level does the esophagus begin in the adults?
  - a) on the level of the VI th cervical vertebra;
  - b) on the level of the II nd cervical vertebra.
  - c) on the level of the IV th cervical vertebra;
  - d) on the level of the II nd thoracic vertebra
2. What kinds of esophageal narrowing do you know?
  - a) physiological
  - b) mechanical
  - c) anatomical
3. Name the esophageal walls: a), b), c)
4. Name the methods of examination of esophagus: a), b), c), d)

**Patterns of the answers to the task.**

1. a
2. a, b
3.
  - a. mucosal membrane with supramucosal base
  - b. muscle layer
  - c. adventitial layer

4. a) X - ray examination; b) roentgenography, c) fibroscopy, e) esophagoscopy

### **Tasks for independent preparation of the students to the classes**

#### **Approximate chart for the students independent preparation**

1. Types of esophageal damages with chemical substances:

- a. notion of colliquacionic necrosis of esophageal walls;
- b. notion of coagulative necrosis.

2. Factors influencing on the degree of esophageal burns:

- a. type of the chemical substances admitted
- b. amount of the poison admitted;
- c. concentration and duration of the solution action

3. Pathologoanatomical changes under chemical burns of the esophagus:

- a. stage of necrosis
- b. stage of ulcers and granulations
- c. stage of scarring
- d. stage of stenosis

4. Degrees of esophageal burns:

- a. local changes under I stage
- b. local changes under II changes
- c. local changes of the mucosal membrane under the 3rd stage

5. Urgent aid under esophageal burns at the first 2 - 3 days:

- a. neutralization of the burning substance
- b. irrigation of the stomach with thick gastric probe



- c. antishock measures
  - d. desintoxicative therapy, measures against dysphagia
  - e. parenteral feeding, 2 days
  - f. antibacterial therapy(anti-inflammatory)
  - g. hormonal therapy
  - h. drugs improving water-electrolitical balance
  - i. cardiac and other remedies
6. Prophylaxis and treatment of esophageal stenosis.
- a. bougieurage with elastic boogies
  - b. use of peritoneal tubes
  - c. retrograde bougieurage
  - d. gastrostomy
  - e. plastic operations on the esophagus

### **CONTENT OF THE LEARNT MATERIAL**

The first stage of esophageal burn is necrosis. It lasts 1 week. After the rejection of the necrotic masses the second stage that of ulcers begins. It lasts near 1 week. Ulcers are covered with succulent granulations. They become dense, newly formed connective tissue wrinkles, becomes scarring, tightens the esophageal walls and does its lumens less. So gradually develops the III stage that of granulations. . It lasts several weeks. The fourth stage (scarring) is that of formation of stenosis. Strictures may be transversal and tubular. The first of them are not more than 2 - 3 cm in the length, and the transversal are longer. The duration of the III and IV stages is from two months to several years.

There are three clinical stages:

1. An acute period;
2. Latent period or a period of sham prosperity;
3. Period of esophagus stenosis.

They diagnose the burn of the *first degree* if under esophagoscopy they determine hyperemia and edema of the gastric mucosal membrane, the patient's self-feeling is satisfactory. Under the burn of the *second degree* they observe ulceration or small, non - combined, necrotic patches on the mucosal membrane of the esophagus. The patient's self - feeling is severity, symptoms of intoxication are not promoted distinctly. For the *third degree* of a burn the presence of combined patches on the esophageal mucosal membrane is typical. Intoxication is promoted, the patient's condition is hard. Sometimes intoxication is so very significant and local damages so vast that the patients die at the nearest hours or days after poisoning.

One of the measures of the first medical aid under the esophageal burns is removing and neutralization of a caustic substance (it should be done at the first four hours after the burn). They should do the following: irrigation of the esophagus and stomach with thick gastric probe with a great amount of water (till 10 liters). If the poisoning had happened because of vinegar acid the irrigation should be done to the lack of smell. Under poisoning with alkalines (caustic soda) the irrigation should be done with 0,5% solution of vinegar or 1% solution of lemon acid. Under the poisoning of acids (sulfuric, nitric) the irrigation of the stomach should be done only with water, as under the use alkaline solution an elimination of a great amount of the CO<sub>2</sub> is possible. If any hesitations are present they should irrigate the stomach with buffer solution or milk.

**Check yourself with the tests given below:**

1. What of the factors mentioned below influences less on the degree of the esophageal burn:
  - a. concentration of the poison
  - b. amount of the poison
  - c. duration of the influence upon tissues
  - d. patient's psychic condition at the moment of burn
  - e. character of damage
2. Name pathologoanatomical stage of esophageal burn: a); b); c); d)
3. Name clinical stages of the esophageal burn: a); b); c); d)
4. Local changes of the mucosal membrane (hyperemia, edema, limited areas of necrosis) are typical for:
  - a: I stage ;
  - b: II stage ;
  - c: III stage
5. Where do the strictures of the esophagus form more often after a burn:
  - a. at the aperture of the esophagus;
  - b. at the bronchial stricture;
  - c. in the supra diaphragmal area of the esophagus;
  - d. at the area of physiological strictures
6. What are the most important therapeutical measures in the first days after esophageal burn:
  - a. forced diuresis with 4% solution of  $\text{Na}_2\text{CO}_3$
  - b. fight against shock, lack of water, laryngeal stenosis (if it develops);
  - c. stomach irritation and neutralization of the caustic substances;

- d. corticosteroids, antibiotics, rehydration, spasmolytics, analgetics, parenteral nutrition;
- e. bougieurage of the esophagus

7. To what department should the patient with an acute esophageal burn be admitted:

- a. reanimation and intensive care department;
- b. therapeutical department
- c. department of otorhinolaryngology
- d. department of surgery

### **CONTROL QUESTIONS.**

1. What is the character of the acids damage action upon the esophageal wall?
2. What is the character of the alkalines damage action upon the esophageal wall?
3. Describe the local changes of the mucosal membrane in the first degree of esophageal burn.
4. What is typical for the second degree of the esophageal burn?
5. Characterize the third degree of the esophageal burn.
6. What caustic substances have a deeper damage effect upon the esophageal wall?
7. Describe morphological changes under the esophageal burns.
8. What medical mistake may be done under the stage of a sham prosperity of esophageal burns?

9. What neutralizing solutions will you use under the poisoning with alkalines?

10. What neutralizing solutions will you use under the poisoning with acids?

11. What are the urgent measures under esophageal burns?

12. What are the therapeutic tactics under esophageal stenosis?

13. What are the complications of the chemical esophageal burns?

14. What is the prophylaxis of esophageal chemical burns?

### **RESEARCH WORK OF STUDENTS.**

Complete a scheme of urgent measures under acute stage of esophageal burn.

Do the following tasks :

**TASK N1.** A patient of 7 y.o. at the age of 3 y.o drank a strong solution of the caustic soda. No treatment had been conducted at that period, self-feeling was nice, the patient followed mechanically mild diet. Four days ago (from mother's words) a child ate a small piece of a roasted fat and after that ate and drank nothing. The child is broken down, subcutaneous layer is weakly expressed, the tongue is dry. Walks with difficulty, has an unquenchable thirst and asks for a water every time. The water given is removed immediately because of vomiting. Both pharynx and larynx are without changes.

What disease can you suspect? What additional methods of investigation will you offer? What should be the therapeutic tactics?

**TASK N 2.** A patient of 23 y.o. had been admitted to E.N.T.- unit in 1/2 of hour after she drank nearly 100 ml of the vinegar essence. The condition is severe, a promoted inspiratory dyspnea, 36 respiration per min., skin is pale, blood

pressure 100/60 mm Hg, pulse 96 beats per min. Under auscultation they determine dry and wet rales in the lungs.

What should be the therapeutical tactics?

**PATTENS OF THE ANSWERS FOR SELF-CONTROL.**

1. e;

2. a) necrosis; b) ulcers and granulations; c) scarring; d) stenosis

3. a) acute; b) a sham prosperity; c) esophageal stenosis

4. b;

5. a,b,c;

6. b, d;

7. a

## Topic:9

### «Acute and chronic stenosis of the larynx. Stages of stenosis and emergency aid»

**Reason.** Pathological conditions leading to the narrowing of the lumen of rima glottidis present a special category of laryngeal diseases.

#### ACUTE STENOSIS OF THE LARYNX

Acute stricture of the glottis may be caused not only by inflammatory diseases of the laryngeal mucosa but also occurs as a result of trauma or allergic oedema induced by intolerance of some drugs used in dentistry (antibiotics, iodine tincture, and others). An abrupt stenosis sometimes occurs—spasm of muscles dilating the rima glottidis develops due to the ingress of a foreign object (food, fluid, fragments of removable dentures, etc.). Obstruction of the glottis by food is infrequent in drunken persons: foreign bodies may be inhaled due to the reduced control of reflexogenic zones of the oral cavity, pharynx, and larynx.

Depending on the underlying cause of acute laryngeal stenosis, the physician takes either conservative or surgical measures. The ability of a physician to estimate the condition of the patient is of great importance. Therefore, the physician must be well familiar with the clinical picture of the disease, since the width of the rima glottidis is the sign that determines the clinical picture of laryngeal stenosis. An adult at rest normally inhales about 7 litres of air per minute; this volume contains  $225 \text{ cm}^3$  of pure oxygen. In patients with stenosis, the act of respiration (consisting of inspiration, physiological pause, and expiration) must undergo some transformation in order to inhale the adequate amount of air. Respiration is regulated automatically and depends upon the amount of carbon dioxide diluted in blood, which stimulates the respiratory centre. Increased amount of carbon dioxide in patients with stenosis changes their respiration—the necessary amount of air that must pass through the constricted rima glottidis makes the inspiration phase longer and the pause and expiration phase shorter. Thus, inspiratory dyspnoea (difficult inspiration) develops.

Clinically, four stages of stenosis are distinguished and guide the physician in choosing proper measures to save the patient.

### **STAGES OF LARYNGEAL STENOSIS**

*Stage I (compensation).* The patient tries to compensate for deficiency of air inhaled through the narrowed glottis. This stage is marked by slow (bradypnoea) and noisy respiration, prolonged inspiration, short physiological pause, short and abrupt expiration followed by another short pause and another long inspiration. A person at rest does not feel dyspnoea (exertional dyspnoea). This is a mild form of stenosis.

*Stage II (relative compensation).* The patient still manages to compensate for deficiency of air by activating accessory muscles, but this compensation is difficult. This stage is marked by pronounced inspiratory dyspnoea: difficult long inspiration, complete absence of the pause between inspiration and short violent expiration. Bradypnoea progresses. Retraction of the yielding intercostal spaces, supraclavicular and suprasternal fossae becomes vivid. The patient is forced to occupy sitting position with the head tilted back and hands leaning against the bed. The patient is excited and restless; acrocyanosis develops.

*Stage III (decompensation).* The patient's anxiety is extreme. He may run about the room, tear his collar in an effort to facilitate the inhalation of air. The face is covered with sticky cold sweat, the eyes seem to be getting out from the orbits and express fright. First the face and then the neck and chest become cyanotic. Tachypnoea develops, but respiration becomes shallow and respiratory movements are scarcely distinguishable because the chest is practically immobile and only the laryngeal prominence (Adam's apple) rapidly moves up and down. Voice and cough are silent, pulse is rapid, the patient is conscious.

*Stage IV (asphyxia)* is the terminal stage of laryngeal stenosis. Senses are lost or consciousness is confused, respiratory excursions are unnoticeable, or Cheyne-Stokes respiration develops. The skin is cyanotic and pallid. The heart continues contracting, the pupils are sharply dilated.



Choice of emergency care in acute laryngeal stenosis depends on the stage of stenosis, its cause (foreign object, oedema, trauma, false or diphtheritic croup), and the patient's condition.

*Emergency care in acute laryngeal stenosis* may be conservative or surgical. Conservative measures are given to patients with the first stage stenosis due to the inflammatory or allergic oedema of mucosa of the infraglottic space, the area of the arytenoid cartilages, interarytenoid space, and ventral surface of the epiglottis. It should be remembered that oedema of any part of the larynx may quickly extend to the vocal cords and the infraglottic space; the patient with oedema of the larynx must therefore be urgently hospitalized.

Treatment of laryngeal stenosis caused by oedema comprises the following procedures:

(1) hot foot baths (the patient's legs are dipped into hot water to the knee level);

(2) mustard plasters to the calves (plasters must not be wetted in hot water since they become ineffective);

(3) diuretics (furosemide, lasix) lessening tissue oedema and drugs reducing permeability of the vascular wall (dimedrol, 10 per cent solution of calcium chloride intra venously, pipolphen, suprastin, hydrocortisone, and 40 per cent glucose solution);

(4) vascular stimulants (1 ml of a 1 per cent lobeline);

(5) antibiotic aerosols (200 000 U);

(6) inhalations with humidified oxygen;

(7) careful nasotracheal intubation in children. Preparations of morphine hydrochloride that inhibit the respiratory centre must not be used.

### **EMERGENCY SURGERY**

Prompt and correct identification of the patient's condition (in the presence of extreme restlessness and progressing asphyxia) plus assessment of the possibilities at hand comprise the main difficulties encountered by the physician who renders first aid to the suffocating patient. Plethoric cervical veins and

sometimes specific anatomy of the neck (excess subcutaneous fat, short neck) interfere with surgery.

The physician must choose the technique of opening the airways.

In the absence of conditions for a classic tracheotomy, a dentist must be able to perform surgery that saves the patient's life and allows transport to a hospital where tracheotomy is performed. The operation that can be performed in most inappropriate conditions (in traffic, in the street, in public places) is coniotomy.

*Coniotomy* is making entry to the trachea through the cricovocal membrane (conus elasticus) which joins the arch of the cricoid cartilage and inferior margin of the thyroid cartilage. The conus elasticus covers a small depression between the margins of the thyroid and cricoid cartilages, which is well palpable in a patient with his head tilted back. Large blood vessels are absent in this area. The skin covers conus elasticus beneath which lies the infraglottic cavity. After incising the overlying skin and cricovocal membrane, one can easily penetrate into the trachea in the absence of bleeding using any cutting instrument at hand (a kitchen-knife, blade, scissors, etc.). Non-collapsible (even of a small diameter) tube should then be inserted into airways through the created incision. This will allow the patient to breathe until tracheotomy (with the insertion of a cannula) is done.

The danger of coniotomy lies in possible damage to the cricoid and thyroid cartilages and the mucosa of the infraglottic cavity, which can later provoke granulation, perichondritis and, consequently, narrowing of the infraglottic cavity due to fibrous tissue. This, in turn, causes narrowing of the trachea at late postoperative period. Therefore, this operation is regarded as an emergency one because the physician cannot do anything else under the given circumstances. Fixing the tube in place by any suitable means (even holding it in hand), the physician has a chance to transport the patient to a hospital where tracheotomy is carried out.

To perform the procedure, the head of a sitting patient is tilted back, which helps to identify the site of a cricovocal membrane. Its determination is easier by a fingertip that slides along the anterior margin of the thyroid cartilage until gets into

the depression. After the point is found, a stab horizontal incision through the skin and cricovocal membrane is done without local anaesthesia to save time, and the physician penetrates into the infraglottic cavity. Posterior structures of the cavity should not be damaged. The inserted tube must not be wide, since otherwise it exerts excess pressure on the surrounding tissues and entails reactive inflammatory reaction. Figure 1 shows levels for tracheotomy.

*Tracheotomy* has been known since antiquity as a method of saving patient's life in various diseases and injuries to the larynx. This operation still remains highly important nowadays: timely tracheotomy saves the patient's life and promotes a complete cure.

Indications for tracheotomy include acute or subacute stenosis (acute respiratory-viral and other infections) and severe cranial injuries. It is also used for inferior bronchoscopy, i.e., insertion of a bronchoscope through a previously created tracheostoma.

Tracheostoma is placed according to the following rules.

1. The most common patient's position is supine, though in some cases tracheotomy is performed in a sitting patient with his head tilted back. First, for conveniency of manipulations, a bolster is placed under the patient's neck. The bolster is then moved under the patient's back to make easier manipulations on the trachea. At the onset of operation the bolster should not be placed under the patient's back since it impedes breathing even more. Only when the trachea is exposed and few seconds are left before its opening, the bolster may be placed under the patient's back.

2. Anaesthesia. Emergency tracheotomy is usually done under local anaesthesia. If asphyxia develops and the operation should be performed immediately, no anaesthesia is applied. For the planned operation intubation anaesthesia is rational, since artificial lung ventilation provides conditions for the most sparing surgical intervention.

Novocaine or trimecaine solutions (1-2 per cent) are used for local anaesthesia. Figure 2 shows schematically the administration of topical anaesthetics.

3. There are clear land marks on the anterior surface of the neck (the laryngeal prominence, arch of the cricoid cartilage, and suprasternal notch) above and below the incision site on the tracheal wall. The skin must be incised accurately by the midline, otherwise, the surgeon risks to miss the trachea.

4. The surgeon stands on the right of the patient during superior and median tracheotomy, and on the left in inferior tracheotomy. In these positions it is convenient to make incisions at various levels.

5. A 5 to 6 cm incision is made on the skin from the middle of the laryngeal prominence downward to the suprasternal notch.

6. The skin and subcutaneous fat are incised with a bellied scalpel (using a sharp-pointed scalpel is dangerous because its tip can sink deep and damage not only the muscular tissue, but also the isthmus of the thyroid gland). Linea alba cervicalis (where the sternohyoid muscles join) is then found. If this line is seen in the wound after separation of the tissues, the direction is correct.

7. Linea alba should be dissected all along its course. To prevent damage to the underlying isthmus of the thyroid gland, this line should be dissected with scissors (not with a scalpel). Before that, approximately at the middle of the linea alba, the tissue is lifted by two forceps, and a transverse incision is made with scissors. A grooved probe is inserted into this incision upward and down ward, and the linea alba is dissected along this probe to the length of incision on the skin.

8. After the muscles are drawn apart, the plethoric isthmus of the thyroid gland covering the trachea is exposed. For a superior tracheotomy, the isthmus must be preliminarily mobilized by dissecting the fascia that holds it to the tracheal wall. Once the isthmus is pulled down by a blunt hook, the first tracheal rings are exposed. For an inferior tracheotomy, the isthmus is displaced superiorly, thus exposing the third, fourth, and fifth tracheal rings.

9. Absolute haemostasis is advisable before opening the trachea. To

suppress the cough reflex, 1 ml of a 5 per cent cocaine or a 1 per cent dicaine solution should be instilled into the tracheal mucosa by piercing the band between the rings. During superior tracheotomy, the second and third rings are cut; in median tracheotomy, the third and the fourth; and in inferior tracheotomy, the fourth and fifth rings are dissected. Care is taken not to damage mucosa of the infraglottic cavity while dissecting the second ring during superior tracheotomy. Injury to the tissues of this area and prolonged presence of a tracheotomy tube provoke granulations with subsequent stable cicatrization that narrows the laryngeal lumen, as a result of which a patient may become a tube carrier for a long time.

In laryngeal stenosis the trachea and larynx rapidly move up and down. This necessitates larynx fixation before dissecting the tracheal wall. To that end, the assistant puts a sharp single-toothed hook into the arch of the cricoid cartilage, pulls the larynx up and keeps in this position until the trachea is opened.

10. The tracheal rings are dissected with a sharp-pointed scalpel. Care should be taken not to damage the posterior tracheal wall. To that end, the belly of the scalpel should be wrapped in wet cotton-wool with its tip left open to cut the tracheal wall to the depth of 0.5 cm. After dissecting the two rings or band between them, a dilator is inserted into the wound. After dilatation of the wound, a tracheotomy tube, metal or plastic, of a corresponding diameter (Nos 1-5) is inserted. It consists of the inner and outer tubes which are necessary for postoperative care. When the inner tube is plugged with mucus, sputum, or crusts, it must be removed, while the outer tube remains in the trachea. After cleaning the inner tube is repositioned. The tracheotomy tube should be inserted as follows: grasping the flange of the tube between the index finger and thumb, it is first put across the patient's neck moving its tip to the stoma. When the tip of the tube enters the trachea, the tube should be turned to align with the neck axis and inserted into the trachea. The tube should be tightly held in place, the strings must obligatorily be knotted to prevent their spontaneous undoing as a result of which the tube may slip out. The strings should be tied on the neck side so as not to bore

the supine patient and to be easily controlled by the medical personnel. After a cannula has been inserted, the incision on the skin is sutured with one or two stitches above and below the tube.

11. The patient with a tracheotomy tube should be under constant medical surveillance. During the first postoperative hours patient's respiratory movements must be watched, since it is difficult for the patient to adapt to unusual breathing through the cannula. In some cases the respiratory centre is stimulated by the administration of lobeline.

*Possible complications of tracheotomy.*

1. Loss of orientation after incision of soft tissues. Severe bleeding due to injury to the isthmus of the thyroid gland.

2. Blood aspiration during hasty and unprepared dissection of the tracheal wall. A damage to the posterior tracheal wall and penetration into the oesophagus as a result of unreasonably deep incision in cases with in adequate fixation of the larynx and trachea.

3. The insertion of a tracheotomy cannula between the separated, but not dissected tracheal mucosa and the tracheal wall leads to tracheal obturation.

4. Subcutaneous emphysema in complete closure of the incised skin and disagreement between the size of wound in the tracheal wall and the diameter of cannula. During breathing and especially coughing the air penetrates into subcutaneous fat to cause emphysema. The latter first extends to the neck and face and then may spread over the chest.

After emergency tracheotomy all efforts must be directed at eliminating the causes of acute stenosis.

### **CHRONIC LARYNGEAL STENOSIS**

This condition is controversial in otorhinolaryngology, since in the majority of cases chronic laryngeal stenosis may be caused by neoplasms, disordered innervation, and cicatrization. Stenoses may also be the result of traumas and burns of the larynx, which leads to inflammation of the laryngeal soft tissues and cartilages. The diagnosis of chronic laryngeal stenosis does not present difficulty:

the severity of stenosis depends on the size of rima glottidis. Cicatricial tissue is removed surgically (cryodestruction, ultrasound, laser beam), and the laryngeal channel is then dilated by a T-tube.

Treatment of patients with chronically stenosed larynx is aimed at restoration of spontaneous respiration through the mouth and nose.

## **THE STUDY-METHODICAL LITERATURE OF DISCIPLINE**

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