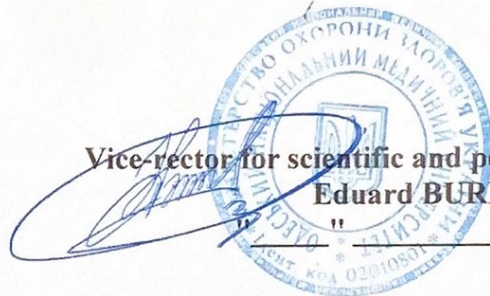


MINISTRY OF HEALTH OF UKRAINE
ODESA NATIONAL MEDICAL UNIVERSITY

Faculty: International
Department: Ophthalmology



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Vice-rector for scientific and pedagogical work
Eduard BURIACHKIVSKIY
" _____ " 2023.

METHODOLOGICAL DEVELOPMENT

TO THE LECTURES ON THE EDUCATIONAL DISCIPLINE

Faculty: Medical, IV year

Educational discipline: OPHTHALMOLOGY

Approved
at the meeting of the Department of Ophthalmology

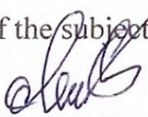
Protocol № 1 dated "29" august 2023

Head of Department  Liudmyla VENGER

Approved by the subject-cycle methodological commission for surgical disciplines of
ONMedU

Protocol № 1 dated "30" august 2023

Head of the subject-cycle methodological commission for surgical disciplines


_____ Vasyl MISHCHENKO

Developer: Head of the Department of Ophthalmology, Doctor of Medicine,
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Doctor of Medicine, Associate Professor Svitlana YEPISHEVA

Lecture №1

Topic: Introductory lecture. the history of the Odessa Ophthalmological School. physiology of the visual analyzer. refraction and accommodation of the eye.

Physiology of the visual analyzer, visual acuity, color perception, visual field, light perception, binocular vision, medical and social examination, refraction and accommodation of the eye, optical system of the eye, diseases of the lens.

Relevance of the topic: With the help of the organ of vision, a person receives 95% of information about the surrounding world. Knowledge of the anatomy of the organ of vision is necessary for further study of ophthalmology, for all stages of establishing a diagnosis of an ophthalmological patient. Knowledge of the anatomy of the visual analyzer is important for any clinician, because it facilitates the diagnosis of diseases, helps to predict the ways of the spread of the pathological process, its dynamics and prognosis.

A necessary condition for establishing the correct diagnosis is the ability to perform a full examination of the patient. In ophthalmological practice, it includes a detailed examination of the anterior and posterior parts of the eye. There are a significant number of research methods that can be used to obtain objective information about the state of the organ of vision. It is essential that the doctor not only has the necessary knowledge of the symptoms of eye diseases, but also possesses the practical skills of examining patients with pathology of the organ of vision.

The doctrine of refraction and accommodation of the eye cannot be overemphasized. The formation of a clear image on the retina of surrounding objects is one of the main conditions for normal vision.

Cataract is one of the most common eye diseases in adults and children, and one of the main causes of curable blindness.

Thanks to the intensive development of surgical means of its treatment, in recent decades, significant progress has been made in the development of surgical methods of intraocular and other types of correction, achieved by the domestic ophthalmological centers of Kyiv and Odesa, which also remain the most accessible to patients.

A doctor of any specialty should be able to diagnose cataracts, prescribe the necessary help and treatment, and resolve the question of surgery in a timely manner.

Diseases of the retina and optic nerve can be a manifestation of a number of general diseases of the body, such as hypertension, diabetes, inflammatory diseases (rheumatism, influenza, syphilis, etc.), a consequence of the toxic effects of alcohol and nicotine, and the result of complications of a high

degree of myopia. Therefore, knowledge of the most common diseases of the retina and optic nerve, the ability to provide timely emergency care should be known to every doctor.

Goal:

Students should know:

- the structure of the orbit, vessels and nerves passing through the holes and slits of the orbit, oculomotor muscles, their mechanism of action and innervation features;
- structure of eyeball shells;
- departments of the visual pathway, localization of subcortical and cortical centers of the visual analyzer;
- the order of eye examination, research methods: the front part of the eye, the internal environments of the eyeball, the fundus;
- features of eye examination in children of different ages;
- development of visual functions in the process of ontogenesis.
- the concept of central vision acuity and what elements of the retina provide it.
- the principle of building tables for determining visual acuity.
- concept of color perception; anatomical and physiological aspects of color perception.
- the principle of diagnostic methods for hereditary and acquired color perception disorders.
- what is the visual field, the method of visual field research.
- characteristic changes in the field of vision in the case of damage to the peripheral part, the chiasm of the optic tract and the central part.
- types of clinical refraction, astigmatism, anisometropia, methods of their establishment, clinical characteristics and principles of correction with glasses.
- modern methods of correction and treatment of the main types of clinical refraction and their combinations.
- clinic, complications, diagnosis, treatment and prevention of progressive myopia.
- features of establishing and treating strabismus.
- anatomical and physiological features of the lens and their age-related changes.
- lens research methods.
 - classification of congenital, acquired and secondary cataracts, clinical features, diagnosis, treatment and prevention of lens diseases.
 - modern methods of aphakia correction.
 - features of ophthalmic surgery.
 - a picture of a healthy fundus.
 - etiopathogenesis, clinic and treatment measures for retinal detachment, optic nerve atrophy.

Students should be able to:

- to analyze the peculiarities of the structure and functions of the eyeball membranes, the structure and function of the refractive media of the eye, the additional apparatus;
- to analyze the anatomical prerequisites of complications of inflammatory and traumatic lesions of the orbits;
- carry out an examination of the front part of the eye using a binocular magnifier;
- examine the front part of the eye and the lens by biomicroscopy;
- perform direct and reverse ophthalmoscopy;
- interpret the picture of the fundus;
- determine the acuity of the central vision according to the distance tables.
- examine the field of vision of each other in a control manner.
- determine the type and angle of strabismus according to the Hirshberg method.

At the lecture, issues of duty, conscience, dignity, medical secrecy and responsibility are resolved.

Educational goals. Professional training will be combined with ethical education. It is explained to the students that there is a kind of relationship between the doctor and the patient, which is regulated both by moral and ethical norms and by law. The norms of behavior of doctors in their relations with patients and colleagues were developed over many generations and became the basis for the formation of medical ethics and deontology.

Basic concepts: Anatomical and functional features of the organ of vision of an adult and a child, methods of research of the front and back parts of the eye, methods of studying the central and peripheral field of vision and color perception. Types of clinical refraction, astigmatism, anisometropia, methods of their establishment, clinical characteristics and principles of correction with glasses.

Plan and organizational structure of the lecture:

№	The main stages of the lecture and their content	Time allocation (min.)
1	2	3
	Preparatory stage: 1 Determination of educational goals. 2 Providing positive motivation The main stage: 3 Presentation of lecture material. 1. Physiology of the visual analyzer 2. Visual acuity 3. Color perception 4. Field of vision 5. Light perception 6. binocular vision 7. refraction and accommodation of the eye 8. diseases of the lens 9. optical system of the eye The final stage: 4 Summary of the lecture, general conclusions. 5 The lecturer's answers to possible questions 6 Tasks for student self-training	3 2 10 10 10 10 5 5 10 5 5 10

Content of the lecture material:

Today, many questions are defined that all students should study, regardless of the profile of the faculty and the future specialty. Therefore, the purpose of teaching ophthalmology at a medical university is to form in students the basics of clinical thinking, medical deontology, examination skills and abilities, providing emergency and first aid for eye diseases and injuries, as well as the ability to carry out preventive measures that prevent the occurrence of epidemiological outbreaks and traumatic injuries organ of vision and the development of a severe form of disability - blindness.

PHYSIOLOGY OF THE VISUAL ANALYZER

The eye is the most important instrument of cognition of the surrounding world. The main information from the surrounding world enters the brain precisely through this analyzer.

The energy of light radiation serves as an adequate stimulus of the eye. The human eye perceives light from 380 to 760 nm of wavelength.

The act of vision is a complex neurophysiological process, in which many of the details are not yet clarified. It has been established that this process has 4 main stages:

1. With the aid of the eye optic media an actual, but upside-down image of things of the outer world is being formed on the photoreceptors of the retina.

2. The light energy acts on photoreceptors where a complicated photochemical process is going on leading to dissociation of visual pigments with their subsequent regeneration with the aid of vitamin A. This photochemical process promotes the transformation of the light energy into nervous impulses. The light, dark and colored image details excite the retinal photoreceptors in different ways and make it possible to perceive light, color and form of things from the surrounding world.

3. The impulses that arise in the photoreceptors are transmitted along the nervous fibres to the optic centers of cerebral cortex.

4. In the cortical centers a transformation of the nervous impulse energy into the visual sensation and perception takes place

Photosensitivity of the eye is the basis of all visual functions. The functional ability of the retina is not the same along its full length. It is the highest in the region of the macula lutea, especially in fovea centralis. Here the retina is presented by neuroepithelium — highly differentiated cones. Rods are located at the peripheral part of the retina. In this connection one should differentiate central and peripheral vision. The central vision is realized by the inner apparatus of the retina, it is characterized by visual acuity, color perception and form perception. While the peripheral vision.

A person with normal colour perception is *a normal trichromatic*. Colour perception dysfunction can be congenital and acquired. The abnormalities in trichromasy, such as *protanomaly*, *deuteranomaly* and *tritanomaly*, are insignificant dysfunctions. *Dichromasy* is the absence of perception of one colour: *protanopia* (when red color is not perceived), *deuteranopia* (when green colour is not perceived) and *tritanopia* (when blue color is not perceived). There may be cases of *achromasia* when a person does not discern colours at all.

Congenital dysfunction of colour perception is observed in 8-10% of males and in 0.5% of females, while the acquired ones are observed in diseases of the retina and optic nerve.

VISUAL FIELD

Visual field is an area which is simultaneously perceived by the eye when the eye and head are not moving and immobilized. Visual field ensures orientation in space. The size of the visual field in a normal eye is defined both by the borders of the optically active part of the retina and by the configuration of the parts of the face neighbouring to the eye (such as ridge of the nose, the upper edge of the eye orbit).

Normally, the borders of visual field for the white color are the following: outward — 90° , down — 70° , up — 55° , inward -60° . In the temporal part of the visual field the blind spot (optic disc) is determined -18° from the point of fixation, its size is -9° vertically and -8° horizontally. To study the visual field the following methods are used: the control method, perimetry, spheroperimetry, campimetry.

Changes in the visual field: scotomas, visual field narrowing, hemianopsias.

Scotomas can be physiologic (the blind spot is a projection of the optic nerve disc into the visual field; angioscotomas — spots corresponding to retinal vessels) and pathologic (in retinal and optic nerve disease).

Scotomas can be subdivided into positive (when a patient perceives subjectively a black spot within the field of vision) and negative (when it is not perceived by the patient); absolute (complete loss of vision in some area of the visual field) and relative (when the vision is impaired in some area of the visual field).

According to the localization, the scotomas can be central, paracentral, peripheral; according to the form they can be circular, annular, sectorform, irregular, arced, etc.

The narrowings of the visual field can be concentric (in pigment degeneration of retina), mostly on one side (in glaucoma — on the nasal side, in retinal detachment — in the upper part).

Hemianopsia is loss of a half of the visual field; it can be heteronymous and homonymous.

Heteronymous hemianopsia can be observed in the visual pathway lesion in the area of optic chiasm.

If the lesion is in the center of the chiasm (hypophyseal tumors, sella turcica walls), bitemporal hemianopsia occurs. If the visual pathway has lesions in the subcortical and cortical centers of the optic analyzer, the homonymous (right- or leftsided) hemianopsias occur. Besides, the central part of the visual field is preserved if the lesion occurs in the center. Thus, the character of the changes that take place in the visual field help the topical diagnosis in the diseases of the central nervous system.

LIGHT PERCEPTION

The most ancient function of the optic analyser is **light perception**, i.e. the ability of the eye to perceive light of different brightness. This ensures the twilight and night vision, and is characterized by the light perception threshold at different levels of illumination (adaptation). To study adaptation different control and test apparatuses called adaptometers are used.

BINOCULAR VISION

Binocular vision is a very complicated physiological function and presents the highest stage of evolutionary development of the optic analyzer. Binocular vision is sight in both eyes when the two images merge into one, an absolutely new image of stereoscopic perception. The first signs of such vision occur in a child of 2-3 months of age. It becomes steady by 6-7 years, sometimes by 12 years of age. There are many ways of checking binocular vision, the most available one is the test using two pencils, or an apparatus with 4 points called colortest.

The vision organ functions are very important for job.

Medical and social examination

Persons with colour perception dysfunctions cannot work in transport or textile industry, they cannot be doctors. The jobs connected with small and moving objects demand high visual acuity and binocular vision.

Disability groups due to vision dysfunction are fixed only in case of a lowered function of vision if the disease is incurable.

The 1st disability group is determined for the patients who need constant help of the other person(s) due to severe visual dysfunction. Such disabled persons are those who have their visual acuity on the better eye not over 0.03 with correction, or those who have bilateral concentric narrowing of the visual field down to -10° .

The 2nd disability group is determined for the patients who have their visual acuity lowered down to 0.04-0.08 with correction on the better eye. This disability group should also be given to those who have a combination of severe anatomical defects (having no limbus, blindness of one of the eyes or the visual acuity of the eye is not over 0.02).

The 3rd disability group is determined for the patients who have a necessity to change their job because of the eye condition. The 3rd group is also given to those who are completely blind or have very low visual acuity on one eye in connection with a military or occupational trauma, this procedure is independent of the kind of work performed by the patient.

REFRACTION AND ACCOMMODATION OF THE EYE

The anomalies of eye refraction are extremely widespread. Especially important problem of ophthalmology is nearsightedness (myopia). In different areas of our country the frequency of this disease among the schoolboys composes 3.5-33%. A high nearsightedness — the most often reason

of physical inability owing to eye diseases. All this puts the struggle with myopia on a level of the state problem and indicates necessity of realization of active and broad measures of prevention of this disease and its complications, that should be realized not only by oculists, but also by doctors of a general profile.

The refraction is a refractive ability of any optic system (re-fractivity). A refraction of the eye is its refractivity in the condition of accommodation, i.e. refractive force of the eye owing to its anatomic constitution.

OPTIC SYSTEM OF THE EYE

The refractive apparatus of the eye consists of the cornea, aqueous humor (liquid of the anterior chamber), lens and vitreous. The refractive apparatus of the eye consists of a few refractive surfaces and environments and represents a difficult compound optic system.

The refraction force of any optic system is measured in diopters. The unit of the refractive force is the force of the lens with a focal length of 1 m. The less a focal length, the more the force of lens.

For example, the force of lens with a focal length of 50 cm amounts to two diopters; the force of lens with a focal length of 25 cm amounts to four diopters etc.

The optic values consist of: 1) curvature radiuses of the cornea and lens surfaces; 2) factors of environments refraction; 3) distances between refractive surfaces, i.e. between the cornea and anterior surface of the lens between anterior and posterior surfaces of the lens. Knowing these values, it is possible to calculate a position of cardinal points, i.e. two main points, two nodular and two main focuses, and then focal length (these concepts are known for you from a course of physics).

The optic values are determined with the help of very delicate and sometimes very difficult methods of investigation. As these values have personal oscillations in this connection the mean values are obtained as a result of measurements of series of eyes. Such an eye with mean optic indices is named a schematic eye.

The schematic eye of Gullstrand is most widespread, it consists of six refractive surfaces: the anterior and posterior surfaces of the cornea, anterior surface of the lens, anterior and posterior surfaces of the lens nucleus, posterior surface of the lens. They divide seven environments: air, the cornea, fluid, anterior and posterior cortical layers of the lens and its nucleus and vitreous. The refractive force of a schematic Gullstrand's eye is 58.64 D.

The schematic eye is used for the solution of many problems of physiologic optics but in the whole number of cases the calculation of the data which is necessary for the clinical purposes is possible with the help of the most simplified scheme of the optic eye model. It is named as the reduced eye in which there is one average index of refraction, one refractive surface and one main plane. The reduced eye of Verbitski is the perfect model. The constants of this eye include the index

of refraction — 1.4; the radius of refractive surface curvature — 6.8 mm; radius of retina surface — 10.2 mm; length of eye axis — 23.4 mm.

Last years the simplified schemes of optic indices calculation gain large practical value for definition of focal point of the optic eye system at opticoreconstructive operations.

CLINICAL AND PHYSICAL REFRACTION

Using the data of optic values, it is possible to calculate refractive force of optic eye system, which is on the average from 58.0 to 60.0 D, but it may oscillate from 52 up to 80 D. It is a physical refraction of the eye. However, in clinic the position of main focus concerning to retina has significance for refractive force of eye environments. This determines concept of **clinical refraction** — correlation between physical refraction and length of eye axis.

Three versions are possible: the main focus is placed on a retina, before it, or behind it. In this connection, there are three kinds of clinical refraction. The first one when the parallel rays, having refracted, incorporate on a retina; it is a proportional refraction — *emmetropia*. When harmony is not present, *ametropia* takes place (nearsightedness or hyperopia). If the parallel rays, having refracted, incorporate in main focus in front of retina, it is nearsightedness (myopia, strong clinical refraction). When the parallel rays having refracted incorporate, behind a retina, the hyperopia (hypermetropia, weak clinical refraction) is watched.

Thus, the kinds of a clinical refraction are characterized by a position of a main focus concerning to retina.

Ametropia can depend on that the diopter apparatus refracts stronger or weaker comparing with the norm (refractive ametropia), or on that longer or shorter than normal the axis of the eye (axial ametropia).

The kinds of clinical refraction are characterized also by attitude to rays of different direction.

At emmetropia the parallel rays incorporate on the retina, i.e. the rays, which go from infinity. In myopic eye the divergent rays going from distance closer than 5 m are focused on the retina. At hyperopia theoretically only the convergent rays could collect on a retina which do not exist in the nature.

The point, *the most remote from the eye*, which is visible at accommodation rest is named as a further point of clear vision (*punctum remotum*). In emmetropia it is infinity, in myopia it is closer than 5 m, and in hypermetropia it does not exist.

Development of the refraction. The most part of the children are born with a strong physical refraction (80 D). However, owing to that length of eye axis is small (18.5 mm), clinical refraction of such an eye is weak (hypermetropia — about .0 D). To years the physical refraction makes up D, practically without changing in further and the clinical refraction becomes more stronger

averaging +2.0 D to 4 years, + 1.0 D to -8 years and after -12 years there is emmetropia. The length of eye axis in these periods is accordingly .5; 23.7 and mm.

Ways of Definition of Clinical Refraction

There are subjective and objective methods of clinical refraction definition. The method based on selection of corrective lens concerns to a subjective method. By the kind of lenses, with which the eye can see better, it is possible to define the character of refraction (myopia or hypermetropia), and the force of these lenses determines a degree of anomaly. The objective methods consist of direct ophthalmoscopy, shadow test — skiascopy, refractometry (on Hartinger refractometer and dioptrone).

For children and young people the refraction is determined only by objective methods after cycloplegia (atropinization).

Clinic of Refraction Anomalies

So, there are three kinds of clinical refraction: emmetropia, hypermetropia and myopia. The refraction depends on two factors: the force of the eye refractive apparatus and length of the eye axis. The majority of children are born with hypermetropia and short eye axis. In the process of organism growth the eye grows also, the degree of hypermetropia decreases up to 20-25 years, when the refraction is formed the hypermetropia remains in 50-55% of the people, the emmetropia comes to 30% and in the others (20-25%) the nearsightedness and astigmatism develops.

Emmetropia (E) — the proportional refraction. Such an eye connects the parallel rays on the retina. It is established on the indefinitely remote point. The people with emmetropia can see well both at a distance and closely; with the age, after years old when accommodation starts to weaken they begin to use glasses only for near vision.

At **hyperopia (H)** the main focus lies behind the eye, and the circle of light dispersion is formed on the retina. In order that the focal point has got on retina, the rays must gather in the front of the eye and come into the eye already convergent. Such rays are not present in the nature, therefore hypermetropic eye does not see well either far, or near, all the time it must accommodate.

That part of hypermetropia, which is determined by lenses, is named as obvious hypermetropia, and that, which is hidden by accommodation tension is latent hypermetropia. The full hypermetropia is the sum of its obvious and latent parts. Degrees of hypermetropia: weak — up to D, mean — up to D and high — more than D.

In connection with that hypermetropic eye always accommodates, and the fatigue of the eye comes while working without glasses. There is feeling of pressure, nausea, dull ache around the eye and in the forehead. It is the accommodative asthenopia. In small children the development of

convergent strabismus, spasm of accommodation, refractive amblyopia is possible. Frequently chronic blepharoconjunctivitis is watched.

The weak degrees of hypermetropia, at which the vision is normal do not require the correction. When the hypermetropia causes asthenopic phenomena, it requires the correction by biconvex lenses the same as in hypermetropia of mean and high degrees.

The hypermetropia is only mismatch between dioptric force of the eye and length of eye axis because of this any changes on fundus of the eye is not watched. Sometimes in cases of its high degree the boundaries of optic papilla seem unclear. Such picture is like in neuritis, but as the visual functions are not broken, the treatment is not required.

Myopia — is a refraction, at which the parallel rays converge before the retina. The further point of clear vision lies in front of eye. The visual acuity at a distance is reduced considerably, closely — it is good. There are the circles of light dispersion on the retina. To reduce them the people with myopia must screw up their eyes. In such a way the visual acuity increases. There are three degrees of myopia: weak — up to .0 D, mean — 3.0-6.0 D and high — more than .0 D. In etiology of myopia the following factors have a large value:

1. Genetic predisposition (Steiger's theory).
2. Unfavorable conditions of environment (professional and school myopia).
3. The primary weakness of accommodation, that conducts to compensatory stretch (Avetisov's theory).
4. Disorder between accommodation and convergencia that conducts to spasm of accommodation (false myopia) which then turns into true myopia.
5. Changes in the posterior part of the eye - dystrophy processes in a sclera that promotes its stretch and increase of length of eye axis.

In the *pathogenesis* of nearsightedness the lengthening of the eye axis plays a great role. The malignant myopia is accompanied by pathologic processes on the fundus of the eye. There are subjective complains (flying flies), which can be explained by opacities of vitreous, decrease of visual acuity into the distance, the muscle asthenopia, spasm of accommodation, development of divergent strabismus. At ophthalmoscopy near temporal edge of optic papilla it is marked a white sickle myopic conns, ;it high degree ol myopia posterior staphyloma (false). At malignant myopia it may be limited sclera prominence true staphyloma. If myopia degree increases by 0.5 D within a year, it is considered as slowly progressive myopia, if more than D — it is fast progressive myopia. At high degree of nearsightedness, as a rule, the pathologic changes on fundus of the eye develop. It is a so-called complicated nearsightedness. The most severe myopic changes, which lead to loss of central vision are the changes in the field of a macula lutea, i.e. central chorioretinites. These are atrophic processes owing to strong stretch of posterior part of eyeglobe. In the course of

time the white foci of irregular configuration in the field of macula lutea occur. They can occupy a large part of fundus of the eye. Quite often in the macula lutea field the pigmented focus (Fuks spot) develops after a haemorrhage or without it. There can be too heavy complications: detachment of retina, retinal or vitreous haemorrhage. At high myopia the complicated nuclear cataracts and rough opacities of the vitreous frequently develop.

Treatment of Myopia

The myopia can be corrected by divergent contact or combined spheroprismatic lenses. The conservative treatment includes:

1. Removal of accommodation spasm.
2. Special exercises, medicines, electro- and phonophoresis with aloe, vitamins, phosphene electrostimulation, laserstimulation for strengthening of ciliar muscle tonus.
3. Spectacles with positive glasses for reading.
4. Common health therapy: vitamin and tissue therapy, massage of colar zone, acupuncture.

If myopia makes progress the operative treatment is necessary for strengthening of sclera (retrosclerosealing, scleroplasty). The technique, elaborated at our department with strengthening not only posterior, but also anterior part of the sclera is especially effective.

In case of non-progressive myopia it is possible to make the keratomileusis, the extraction of transparent lens. Laser correction is wide-spread.

Prevention of myopia

1. Harmonic physical development of the child.
2. Fulfilment of the sanitary-hygienic conditions of illumination and organization of working place of schoolchildren.
3. Picking out of the risk groups among children with reduced volume of accommodation, who have myopia in their family.

All myopes should be registered in the health center. The patients with weak or mean degree of myopia must be watched not less, than twice a year, with high degree — not less than times a year with carrying out of corresponding courses of treatment.

Anisometropia is the unequal refraction of both eyes. It is accompanied by various sizes of the retinal images — aniseikonia. Usually the image of one eye is suppressed in the cortex of brain, that leads to development of refractive amblyopia. It is possible to correct the anisometropia up to .0 D (for children — maximum .0 D) by the help of eyeglasses with the difference of refraction more than .0 D wearing of isoiconic spectacles, contact correction or surgical treatment (refractive keratoplasty, keratotomia, excimer laser) is recommended.

Astigmatism — is an anomaly of refraction, at which there are different kinds of refraction of different degrees of the same refraction at various meridians of the same eye. Astigmatism more often arises owing to incorrect curvature of mean part of the cornea, more rare due to lens.

There are regular and irregular astigmatism. The regular astigmatism is usually the inherent astigmatism, at which the refracting force within the limits of one meridian is identical, at irregular one — each meridian at various parts refracts light with different force. At irregular astigmatism two meridians are picked out: with the greatest and the least refraction (main meridians). They are disposed perpendicularly to each other. The astigmatism in .5 D is characteristic almost of all eyes; it has not an influence on sharpness of vision, it is considered as physiological and does not require the correction. If the refraction on vertical meridian is more, than on horizontal, it is the direct astigmatism. At indirect astigmatism the refraction is more on horizontal meridian. This form even in case of weak degree considerably reduces the sharpness of vision. The astigmatism at which the main meridian passes between horizontal and vertical is named as astigmatism with oblique axes.

Simple myopic or hypermetropic astigmatism — there is emmetropia in one meridian and myopia or hypermetropia in the opposite one.

Complex myopic astigmatism there is myopia of some degree in one meridian and myopia of the other degree - in the opposite one.

Complex hypermetropia astigmatism - there is hypermetropia in both meridians but of various degree.

Mixed astigmatism — there is myopia in one meridian and hypermetropia in the opposite one.

Correction of astigmatism. Astigmatism considerably reduces sharpness of vision, complicates the work at a close distance and causes the asthenopic phenomena, chronic blepharoconjunctivitis and amblyopia. The correction is possible by cylinder lenses. They are characterized by that rays, which pass in the plane parallel to axis of lens, do not refract, but the rays, which go in the plane, perpendicular axis, refract, i.e. the glass is established so, that the axis of the cylinder is perpendicular to the meridian, which is necessary to correct.

At high degrees of astigmatism which are not corrected by optic glasses, it is necessary the correction by solid contact lenses or refractive operations on the cornea.

ACCOMODATION

The eye has the mechanism which is capable to raise a refraction, to adapt it for seeing at different distances. This dynamic process of intensification of eye refraction and its adaptation to different distances is named **accommodation**.

Mechanism of accommodation is following - The lens in the **eye** is attached with the help of ciliary zonule, the fibers of which go from ciliary body to equator of lens. When the fibers of ciliary muscle are shortened there is a relaxation of ciliary zonule, lens becomes more convex, that increases its refractive force. At relaxation of accommodative muscle there is a return process. It is the classic theory of the accommodation mechanism created by Helm-holtz and acknowledged by the majority of the ophthalmologists. The point which is seen by the eye at maximum tension of accommodation is named **the nearest point of clear vision** (*Punctum prox-imum*). That quantity of diopters, on which the eye can increase its refraction, expresses the force, volume and amplitude of accommodation. The volume of accommodation is calculated by the formula:

$$A = P + R,$$

where A — accommodation, P — force of refraction at the installation of the eye on the nearest point of clear vision, R—refraction of the eye in a state of accommodation rest.

The distance between the nearest and the most remote point of clear vision is named length of accommodation. This linear concept is expressed in measures of length. The accommodations of one eye is absolute. At examining a subject by both eyes the vergence joins to the act of accommodation. That accommodation, which both eyes are capable of at definite convergence, is named a relative accommodation. It always less then absolute and consists of positive and negative parts. The interrelation of these parts has large significance. The less a positive part of accommodation, the faster fatigue arises.

Disorders of Accommodation

The anomalies of refraction are frequently accompanied by different disorders of accommodation. Hypermetropia and astigmatism, as it was already indicated above, may cause the accommo-lative asthenopia, pareses of ciliar muscle, reduction of accommodative volume, appearance of obvious hypermetropia and spasm of accommodation. It is characterized by strengthening of refraction, appearance of false myopia, which disappear at the altitude of cycloplegia (after atropinization).

The spasm of accommodation can arise also in patients with weak degree of myopia. The paresis and paralysis of accommodation are watched at the lesion of parasympathic part of the oculomotor nerve owing to traumas or poisonings.

With age the volume of accommodation decreases, the so-called **presbyopy** develops. The presbyopy arises at the age after 40 years. It is characterized by removal of the nearest point of clear vision from eye, worsening of vision at a short distance. At the age of 65 the accommodation equals 0 and the nearest point of clear vision is removed to infinity.

For correction of presbyopy concave glasses for short distance are prescribed: at 40 years of age — +1.0 D, at — +2.0 D, after and older — +3.0 D. If there are anomalies of refraction, the

degree of refraction is added (at hypermetropia) or deducted (at myopia) to force of glasses for appropriate age.

In the conclusion it is necessary especially to underline once more that it is necessary the early diagnosis of refractive anomalies and carrying out of the special exercises for strengthening the tonus of ciliar muscle, the general treatment and observation of children with ametropia in consulting centers.

DISEASES OF THE LENS

Anatomy of the Lens

The diseases of the lens take about % of eye diseases. The lens (lens crystalline) develops from ectoderm. The lens is one of refractive environments of the eye, its main function is refraction of light rays. The important feature of the lens is its accommodative capability. Without accommodation, when looking at a distance, the refractive power of the lens is approximately 18-20 D, and at a short distance, at a forced accommodation, it can increase up to 35 D in children. Iris and lens together form so called irido-lens diaphragm, which divides anterior and posterior parts of the eye, keeps the vitreous in the posterior part of the eye. Anterior surface of the lens can be seen in the region of a pupil. Its centre is the anterior pole of the lens. The posterior surface is visible by biomicroscopy on slit-lamp, its centre is the posterior pole of the lens. A line of transition of anterior surface to posterior is named as the equator of the lens.

The lens is covered by the unstructured elastic capsule. The part of the capsule, which covers the anterior surface of the lens, is named the anterior capsule, and that one that covers the posterior surface — the posterior capsule. Suspensory Zinn's ligaments attach to the capsule on the equator, and go from the ciliary body. The lens is suspended by these ligaments. Under the anterior capsule there is a layer of cubical epithelium, transparent cells, which reproduce, displace to equator and transform in lens fibres. The old layers of fibres displace to the centre of the lens, condense and form a nucleus of the lens.

Size, form, consistence, colour of the lens change during the life. In newborns the lens form is spherical, colourless, soft, with thickness of 4 mm and diameter of mm. The lens of the child contains % of various proteins, % of water, % of mineral salts (K, Ca, P), vitamins (C, B₂), glutathion, enzymes and lipids. In adults thickness of the lens is about 3.6 mm, diameter — 9-10 mm. In the lens of the adult over -30 years old layers in the centre form a yellowish transparent firm nucleus, and periferal layers form a soft transparent core. The chemical structure of the lens changes with the age: the amount of insoluble protein fractions increases, cholesterol, thirosin collects and the lens becomes yellowish. The amount of Ca increases, the amount of vitamins C and B decreases.

The bigger the nucleus, the firmer the lens and less accommodation ability and the farther from the eye the nearest point of clear vision. But these changes are physiological, the lens in adults, as well as in newborns, remains transparent. The lens has no vessels. Nutrition to the lens is provided by the aqueous humour of anterior and posterior chambers of the eye. The metabolism occurs very slowly. The lens has no nerves, therefore pathological changes of the lens proceed painlessly.

Methods of the Lens Examination

With a wide pupil we may see almost the whole lens, but the edge of the lens is seen only in complete colobome of the iris or in aniridia. Lens examinations are carried out by using of the*following methods:

- lateral illumination;
- biomicroscopy on a slit lamp;
- investigation with passing light;
- ultrasound (in the cornea opacity, for measurement of the lens thickness);
- examination of the eye functions.

The main symptoms of the lens pathology are a loss of transparency, dislocalization, change of the form and size. The diseases of the lens are congenital and acquired.

Diseases of the Lens

The main signs of the lens pathology are loss of transparency, disorder of localisation, changes of its form and size. The diseases of the lens can be congenital and aquired.

Congenital Diseases of the Lens

Congenital dislocations of the lens. Etiology. Hereditary defect of metabolism of the connective tissue. It is often combined with other congenital defects of cardiovascular, musculoskeletal system. *The Marfan's syndrom* is characterised by subluxation of the lens, high growth, arachnodactily, fragility, weakness of the ligamentous apparatus.

The Marchesani's syndrom is characterised by subluxation of the lens, microphakia, myopia, ditachment of retina, low growth, short trunk, neck, extremities, limitation of joints movement.

Lenticonus (anterior and posterior) is a change of the form of the lens surface in which posterior or anterior pole of the lens assumes a conical shape.

Microphakia is a reduction of the sizes of the lens with spherical shape. It is combined with myopia. It can block the pupil aperture, that causes development of the phakotopic secondary glaucoma.

Coloboma of the lens is a defect of equatorial edge of the lens. In large coloboma astigmatism and myopia usually occur.

Congenital cataract may be following. Hereditary in the Dawns disease caused by trisomia of chromosome 21 and antenatal, because of illness of the mother during pregnancy (influenza, rubella, toxoplasmosis) or consequence of influence of the various adverse factors of external and internal environment (intoxication, ionizing radiation, avitaminosis).

The cataracts may be unilateral and bilateral. Due to visual deprivation in congenital cataract complications develop quickly: amblyopia, strabismus, nystagm. Frequently cataract is combined with the anomalies of refraction, microcornea, microph-thalmus, congenital diseases of the retina, choroidia, and optic nerve.

Depending on presence or absence of these complications and accompanying pathology congenital cataracts are divided into:

1. Simple cataract.
2. Complicated congenital cataract.
3. Congenital cataract with associated diseases.

In dependence on localization and form of opacity, congenital cataract may be:

- zonular or lamellar — grey opacity in separate layers of the lens as circle with radial projections resembling the spokes in the wheel, popularly known a "riders";
- complete (diffused);

- polar — anterior and posterior;
- coronary;
- punctate;
- starshaped;
- axial;
- pyramidal;
- spinde;
- atypical (polymorphic);
- membranous;
- semiresolved.

Congenital cataracts of any type may be of three degrees: 1. The visual acuity is 0.3 and more (diameter of opacity is up to 1.5 mm).

2. The visual acuity is 0.2-0.05 (diameter of opacity is 2-3 mm).

3. The visual acuity is less than 0.05 (diameter of opacity more than 3 mm).

Treatment of congenital cataracts is only operative — removal of the lens. At the third degree of congenital cataract it should be operated early. Cataracts of the first and second degree are operated at the age of -4 years. Before this time the pupil is widened, pleoptic treatment is carried out.

Kinds of operative treatment: aspiration-irrigation, phakoemul-sification, laser phacopuncture (laser discision in the membranous cataracts).

Operation and postoperative period have easier current, than in the adult, but the results are worse, because there are complications and associated pathology, which usually are absent in adults.

Acquired Cataracts

It is divided the following kinds of acquired cataracts: senile, traumatic, radiational (thermal, X-rays), toxic, complicated.

Senile cataract. Last thirty years its level has grown 6 times as much (4-6 cases in one thousand of population). 30-40% of operations on the eyes are operations due to cataracts. Usually it arises at the age over 50, but last years cataract is met in younger age. *Etiology.* Cataract is caused by topical and systemic disorders of metabolism. The cataractogenic factors are:

- a) decrease of antioxidative enzymes activity with the age;
- b) defects of microcirculation of the eye caused by diseases of cardiovascular system (hypertention, atherosclerosis);
- c) defects of metabolism in liver and kidneys diseases, diabetes mellitus, a deficiency of vitamins C and B2.

Clinical course of the senile cataract. In the beginning of the disease patients complain to:

- "flies" which move together with the eye. In fixed eye "flies" are immovable (opacity of the vitreous is mobile, in motionless eye it falls down);
- myopia in elderly age, often change of glasses for near distance;
- monocular diplopia, polyopia.

Gradually a decrease of the visual acuity, mist before the eyes appears.

The visual acuity decreases from 1.0 up to a right perception of Due to cataract only one visual function (visual acuity) is impaired. The visual field, light sensitivity remain normal.

It is distinguished cortical and nuclear senile cataract. Cortical cataract is divided into four stages:

- I. The incipient stage. At the periphery of the lens there appear radial opacities ('spokes in a wheel'). At the centre of the lens under the capsule there are the transparent vacuoles, eye fundus can be seen clear. The visual acuity is equal 1.0-0.3. The complaints are "flies", "mist" before eyes.
- II. Immature, intumescent cataract. Considerable opacities often have the form of sectors or "spokes in a wheel" which reach the centre. There is the iris shadow in lateral illumination, there is a reflex from eye fundus on periphery, but its details are not visible. The visual acuity is less than 0.2. The anterior chamber is shallow. There is complication — phacogenic (phacomorphic) glaucoma.
- III. Mature cataract. The entire lens becomes opaque, there is no the iris shadow at lateral illumination, pupil is white or grey, there is no reflex from eye fundus, visual acuity is right perception of light.
- IV. Hypermature cataract. Fibre of the superficial layers of the lens break, cortex of the lens becomes white like milk liquid and partially resorbs. Therefore the anterior chamber becomes deep, tremulousness of the iris arises. The reflex from eye fundus and even low visual acuity may appear. The heavy nucleus falls downwards on the bottom of the anterior chamber. Complications: phacolytic secondary glaucoma, phacolytic iridocyclitis.

In nuclear cataract the opacity begins in the centre of the lens' nucleus. By lateral illumination the gentle opacity like a cloud is visible in the centre. In examination by a passage light the reflex is absent in the centre. It develops slowly, at once the visual acuity considerably decreases. It can be atypical form of the age cataract, i.e. brown or brunescence cataract. The pupil has a dark colour, the reflex from eye fundus is absent. Lens is firm, brown, it has only nucleus, cortex is absent.

Treatment of cataract. The conservative treatment is carried out only in I stage with the purpose to delay the cataract progressing. It is used topically tauphon. vitamin drops (catachrome. sancata-lin. vita-iodurol, vitaphacol etc.).

Beginning with the II stage the surgical treatment is carried out. and the condition of the second eye is taken into consideration. If the visual acuity of the other eye is high, the operative treatment is used in decrease of visual acuity of the ill eye less than 0.3. If the visual acuity of a other eye is low or it is absent, the cataract is operated in visual acuity less than 0.1.

History of cataract surgery. Up to the middle of XVIII century there was carried out reclinatio of cataract. So-called reclinators made it. This operation caused blindness from glaucoma in 40%. The founder of modern methods of lens removal was Jacques Daviel, which has removed cataract through the cornea incision in Marseilles in 1745. At that time about 10% of the patients lost vision due to intraocular infection.

At present there are applied the following methods of removal of cataract (only with microscope):

1. Extracapsular cataract extraction. The posterior capsule is not removed. It prevents loss and hernia of the vitreous, detachment of the retina. Due to microsurgical technique in the most cases it is possible to prevent the main defect of this method — development of a secondary cataract. Secondary cataracts are carried out by laser or knife dissection.
2. Phacoemulsification is the most modern and effective method. It is distinguished with a small incision, not very traumatic operation, fast rehabilitation of the patient. Due to the new achievement in phacoemulsification technique quality of the cataract surgery is very high. The main components of the method is automatic aspiration-irrigation system which supports constant intraocular pressure

during the operation, high quality of coaxial microscope, viskoelastics for prevention of the cornea damage, small tunnel incision, adapted to a small incision of intraocular lens.

3. Intracapsular cataract extraction. It is carried out by means of a cryoprobe (cryoextraction) or with capsule forceps. This method frequently gives complications: hernia of the vitreous, glaucoma, detachment of retina. Now it is carried out only in subluxation of the lens.

Aphakia

Aphakia is absence of the lens. One of aphakia causes is operation of cataract extraction. Clinical signs of aphakia: — the visual acuity is less, than .05; accommodation; high hypermetropia - 10 - 12 D; deep anterior chamber; iridodom — absence of the lens complex at ultrasound examination.

Methods of aphakia correction

1. The intraocular lens (IOL). The first experimental implantation of IOL in rabbits was made by A. Kh. Mikhailov in 30s in Sukhumi. In people the first implantation of IOL was carried out by Ridley (England, 1949).

Now it is impossible to imagine cataract surgery without implantation of the intraocular lens. IOL are implanted almost to all patients after extraction of cataract. Contraindications are only diseases of the cornea, severe cases of diabetes mellitus. The models of the intraocular lens, its material are permanently improved. Now the posterior camera lens with intracapsular fixation are usually applied. They are made from poiyethylmetacrelate. silicon. hydrogel, acril. In our department the new models of intraocular lens with a carbon covering, has been worked out due to which trauma of eye tissue during implantation and excudative reaction have decreased, toxic effect of PMMA and damage effect of ultra-violet rays of light on retina were removed.

Last achievement in development of new models of intraocular lenses is multifocal lenses, which ensure the high vision at long and short distances.

2. Keratophakia — interlammelar refractive keratoplastic.

3. Contact lenses.

4. Iseikonia glasses (in monocular aphakia).

5. The old method. In binocular aphakia it is used glasses from + 10 to +12 D for a long distance and glasses from +13 to +15 D for a short distance (the eye refraction before operation will be taken into account).

12. List of recommended literature:

Basic:

1. Ophthalmology: textbook / O. P. Vitovska, P. A. Bezditzko, I. M. Bezkorovayna et al.; edited by O. P. Vitovska. - Kyiv: AUS Medicine Publishing, 2017. - 648 p. ISBN 978-617-505-598-4
2. Atlas of Glaucoma. Second edition: textbook / Neil T. Choplin, Diane C. Lundy. - Informa healthcare, United Kingdom, 2007. -364 p. ISBN-10: 1841845183.
3. Common Eye Diseases and their Management: textbook / N. R. Galloway, W.M.K. Amoaku, P. H. Galloway and A. C. Browning; -Springer - Verlag London Limited, 2006. – 208 p. ISBN 1-85233-050-32.

Additional:

1. Eye Diseases. Course of lectures: textbook / G. E. Venger, A. M. Soldatova, L. V. Venger; edited by V. M.Zaporozhan. - Odessa: Odessa Medical University, 2005. – 157p.
2. Ophthalmology at a Glance: textbook / JANE OLVER, LORRAINE CASSIDY; - by Blackwell Science Ltd a Blackwell Publishing company, USA, 2005. -113 p. ISBN-10: 0-632-06473-0.
3. Ophthalmology: textbook. / Gerhard K. Lang, edited by J. Amann, O. Gareis, Gabriele E. Lang, Doris Recker, C.W. Spraul, P. Wagner. - Thieme Stuttgart. New York, 2000. - 604 p. ISBN 0-

86577-936-8.

4. EYE Atlas. Online Atlas of Ophthalmology. / All rights Reserved, Oculisti Online. Copyright 2001. -408 p.

5. ABC of Eyes, Fourth Edition: textbook / P. T. Khaw, P. Shah, A. R. Elkington. - by BMJ Publishing Group Ltd, BMA House, Tavistock Square, London, 2005. - 97 p. ISBN 0 7279 1659

13. Electronic information resources

1. <https://info.odmu.edu.ua/chair/ophthalmology/>
2. <https://repo.odmu.edu.ua/xmlui/>
3. <http://library.gov.ua/>
4. <http://www.nbuiv.gov.ua/>
5. https://library.gov.ua/svitovi-e-resursy/dir_category/general/
6. <http://nmuofficial.com/zagalni-vidomosti/biblioteky/>
7. <https://guidelines.moz.gov.ua/documents>
8. www.ama-assn.org – [American Medical Association](http://www.ama-assn.org)
9. www.dec.gov.ua/mtd/home/
10. <http://bma.org.uk>
11. www.gmc-uk.org

Lecture №2

Topic: Red eye

Anatomy of the eyelids, diseases of the eyelids, neoplasms of the eyelids, conjunctiva and its diseases, inflammatory diseases of the conjunctiva (conjunctivitis), diseases of the lacrimal organs, inflammatory diseases of the orbit, diseases of the cornea, diseases of the sclera, diseases of the choroid.

Relevance of the topic: According to statistics, in the general structure of diseases of the organ of vision, diseases of the eyelids make up about 10%. The peculiarity of the morphological features of the structure of the eyelids is also determined by the peculiarity of their pathology. Diseases of the skin, the edges of the eyelids, the meibomian glands, the muscular and vascular-nervous apparatus are especially distinguished.

The pathological process can spread to all layers of the eyelid tissue. Depending on the cause and mechanism of the development of the disease, the eyelids can be inflammatory, dystrophic, tumorous, traumatic, allergic, hereditary anomalies of development. Inflammatory diseases are the most common.

The pathology of the lacrimal organs is mainly inflammatory in nature, but there are also developmental abnormalities, tumor processes, and changes in systemic diseases. These diseases make up 3-6% of the number of ophthalmological patients.

Inflammatory diseases of the eyelids and lacrimal organs are most often found in children, therefore timely diagnosis and adequate treatment are of great importance for the prevention of possible complications.

Inflammatory diseases of the conjunctiva are one of the most common pathologies of the eye. They make up 30-48% of all eye diseases in the structure of patients who sought ophthalmological help in polyclinics. Conjunctivitis can be highly contagious, cause complications (for example, reduced visual functions). That is why a doctor of any specialty needs to be able to make a correct diagnosis. Early diagnosis makes it possible to prescribe treatment in a timely manner, isolate the patient and thereby prevent the spread of the disease.

Diseases of the fibrous membrane of the eye (cornea and sclera), and especially the cornea, which is at the border of the internal and external environment, have always been an urgent problem of ophthalmology due to high morbidity (about 25%) and disability (reduction or loss of vision in almost 50%), which leads to blindness of people of working age.

As in the past, when lesions of the cornea (in the case of injuries and as a complication of smallpox, gonorrhoea, trachoma) were common, so now with the predominance of endogenous infections (as a complication of herpes, tuberculosis, syphilis, etc.) and dystrophies, only a comprehensive approach to diagnosis and treatment with the active participation of doctors of various specialties can reduce the percentage of morbidity and disability.

Goal:

Students should know

- principles of treatment of inflammatory diseases of the eyelids, possible anomalies of the development of the eyelids
- principles of treatment of dacryoadenitis and dacryocystitis
- anatomical and physiological features of the conjunctiva, classification of conjunctival disease;
- features of clinical manifestations of various types of conjunctivitis - epidemic Koch-Wicks, viral, diphtheria, pneumococcal, trachoma.
- anatomical and physiological features of the cornea and its age-related changes, methods of cornea research; the main symptoms of corneal lesions (keratitis, dystrophies, degenerations, opacities, developmental abnormalities); classification, modern methods of diagnosis, treatment and prevention of keratitis and their complications;
- anatomy and diagnostic methods of the vascular tract
- clinic and treatment of infectious diseases (toxoplasmosis, tuberculosis,
- adenovirus infection, etc.), clinic and treatment of venereal diseases (syphilis, chlamydia)
- what laboratory tests should be carried out when examining a patient with iridocyclitis.

Students should be able to:

- to diagnose inflammatory diseases of the eyelids, dacryoadenitis, dacryocystitis of newborns and adults
- examine the conjunctiva of the eyelids, transitional folds of the sclera by the method of lateral (focal) illumination, turn out the upper eyelid for examination.
- distinguish clinical signs of conjunctivitis: hyperemia, swelling of the conjunctiva, increase in follicles, the presence of hemorrhages separated in the conjunctival cavity.
- be able to provide first aid, give professional recommendations
- to explain the peculiarities of etiopathogenesis of acute epidemic, viral conjunctivitis, gonoblenorrhoea, trachoma. Prescribe treatment and provide measures to prevent the further spread of the infection.

- examine the cornea by the method of lateral focal illumination and determine its main properties, examine the sensitivity of the cornea;
- diagnose keratitis, carry out differential diagnosis of infiltrates and opacities, prescribe keratitis treatment;
- to diagnose a purulent ulcer of the cornea and give urgent medical and organizational recommendations;
- formulate the concept of iridocyclite.
- purposefully collect anamnesis from a patient with iridocyclitis, carry out a diff. diagnosis of iridocyclitis and acute onset of glaucoma
- the principles of treating patients with iridocyclite, make the first medical appointment help

At the lecture, issues of duty, conscience, dignity, medical secrecy and responsibility are resolved.

Educational goals. Professional training will be combined with ethical education. It is explained to the students that there is a kind of relationship between the doctor and the patient, which is regulated both by moral and ethical norms and by law. The norms of behavior of doctors in their relations with patients and colleagues were developed over many generations and became the basis for the formation of medical ethics and deontology.

Basic concepts:

On the basis of knowledge of anatomy, physiology of the organ of vision, mastering the basic methods of eye research, modern data on etiopathogenesis, clinic, treatment of blepharitis and lacrimal organs, form the correct diagnosis and appropriate treatment.

To acquaint students with the features of clinical conjunctivitis, the principles and means of their modern medical treatment.

to get acquainted with the current state of the problem of corneal pathology, to learn to identify its main diseases, to provide emergency care, to carry out prevention and treatment of complications.

On the basis of knowledge of anatomy, physiology of the organ of vision, mastering the basic methods of eye research, modern data on etiopathogenesis, clinic, treatment of diseases of the choroid, form the correct diagnosis and appropriate treatment.

Plan and organizational structure of the lecture:

№№	The main stages of the lecture and their content	Time allocation (min.)
1	2	3
	Preparatory stage:	
1	Determination of educational goals.	3
2	Providing positive motivation	2
	The main stage:	
3	Presentation of lecture material.	
	1. Eyelids and their diseases	10
	2. Conjunctiva and its diseases	10
	3. Lacrimal organs and their diseases	10
	4. Orbit and its diseases	5
	5. Corneal disease	10
	6. Diseases of the sclera	5
	7. Pathology of the choroid	10
	The final stage:	5
4	Summary of the lecture, general conclusions.	5
5	The lecturer's answers to possible questions	5
6	Tasks for student self-training	10

Content of the lecture material:**1. THE EYELIDS, CONJUNCTIVA AND LACRIMAL APPARATUS DISEASES****Anatomy of the Eyelids**

The eyelids are the organ of passive and active protection of the eye. Passive protection consists in that the eyelids always cover a part of the eye, and the whole eye in complete closing. Active protection is accomplished by the eyelids in two directions: first, due to reflex apparatus the eyelids close the eye automatically and instantly in danger; second, the eyelids promote removal of foreign bodies, which have gotten into the eye with the help of their movement.

The eyelids are two skin folds, which edges are connected from the external side forming an acute angle, and from the internal side they form an arched curve before connection.

The space between the upper and lower eyelid is called palpebral fissure; its length is 30 mm on an average in adults, and height is from 10 to 14 mm.

There are four layers in the eyelids: 1) cutaneous; 2) muscular; 3) connective tissue (ciliary cartilage) and 4) mucous.

The eyelid skin is thin. There is no subcutaneous fat. Subcutaneous fat is porous that makes the eyelid skin very mobile. Oedema of the eyelids appearing in various local and some general diseases is explained by porosity of the subcutaneous fat.

The circular muscle of the eye (*m. orbicularis oculi*), which is innervated by the facial nerve (*n. facialis*), is located immediately under the skin. The muscle is divided into two parts: a palpebral part, which is nearer to the free eyelid edge (*pars palpebralis*) and an orbital part, which is nearer to the orbital edges (*orbitalis*).

A contraction of the palpebral part provokes eyelid closing, which is observed, for example, during sleep, and also ensures the blinking movements. A contraction of the orbital part takes place in closing of the eye. When closing the eyelids, the eyeballs are deviated upwards (Bell's phenomenon).

The connective layer is localized under the muscle. The semilunar plate is a cartilage, which, however, doesn't correspond to its name, as it consists of not the cartilaginous cells but of compact fibrous connective tissue and occupies its main part. The semilunar plates of the upper and lower eyelid are joined with the help of their medial ligament (*lig. palpebrale mediale*) near the medial angle of the eyelids and near the external angle of the eyelids by the lateral ligament (*lig. palpebrale laterale*).

The fascia, which stretches to the orbital edges (*fascia tarso-orbitalis*), is fastened to the convex upper edge of the cartilage. Thus, the cartilage and fascia form a connective septum (*septum orbitale*), which closes the orbit in front. The muscle (*m. levator palpebrae superioris*) is fastened to its upper edge. Beginning near the orbital apex from the fibrous ring around the visual foramen, the muscle stretches along the upper orbital wall not reaching its edge; it is fan-like, dilated and divided into three parts. The first (tendinous) part is plaited into the skin and eyelid circular muscle. The second part is fastened to the upper cartilaginous edge. The muscle, which consists of the smooth muscular fibers, is fastened here too; it is called Muller's muscle. The third part is fastened to the conjunctiva of the transitional fold. Such division of the muscle fibers lifting the upper eyelid has a great physiological importance, essence of which consists in simultaneous lifting of all eyelid parts: skin, cartilage and mucosa. The muscle lifting the upper eyelid is innervated by the oculomotor nerve except the Muller's muscle, which is innervated by the sympathetic nerve.

The mucous membrane (*conjunctiva*) covers the whole posterior palpebral surface and anterior surface of the eyeball to the corneal edge.

Palpebral edge. There are anterior rounded rib, posterior (right-angle) one, and also intercostal space localized between them on the free palpebral edge. There are 2-3 rows of 100 150 eyelashes on the upper eyelid near the anterior rib and 50 70 eyelashes on the lower one. They are a protective net against foreign bodies and sweat. There are some sebaceous glands (Zeiss' glands) near each eyelash; there are changed sudoral glands between them with secretory ducts opening into the hair follicles of the eyelashes. An even row of the punctate foramina is visible somewhat in front of the posterior rib; they are excretory ducts of the meibomian glands. Their secretion greasing the palpebral edges does not permit pouring of tears over the edge; it protects the palpebral skin from maceration and promotes tight closing of the eyelids and hermetic closing of the conjunctival sac.

Palpebral vessels. Each eyelid is supplied by palpebral arteries: a. palpebralis lateralis (branches of the anterior ethmoidal artery) a. ethmoidalis anterior. A. palpebrales lateralis et medialis are united, and they form vessels of the arterial marginal arch (arcus tarseus marginalis): arcus tarseus superior (on the upper eyelid) and arcus tarseus inferior (on the lower eyelid). They are localized in deeply in the eyelids on the posterior surface of the circular muscle, in front of cartilage at a distance of 3 mm from the free palpebral edge. The shallow sulcus (sulcus subtarsalis) where the small foreign bodies are most often detained corresponds to them on the posterior cartilaginous surface from the conjunctiva side. Branches of these arches pass through the cartilage on the conjunctiva, and supply blood to them.

The eyelids have a well developed system of lymphatic vessels. They consist of two nets joined with each other, one of which is localized on the anterior cartilaginous surface, another one — on the posterior one. The lymphatic vessels of the upper eyelid fail into the parotid, and of the lower eyelid — into submaxillary lymphatic glands.

Diseases of the Eyelids

Inflammatory Diseases

Diseases of the palpebral skin. Diseases of the palpebral skin practically don't differ from the diseases of the face skin. Palpebral oedema that is frequently observed is the result of their anatomical structure — friable junction of the skin with a muscle. Eczema, lupus, vaccine pustules are observed on the palpebral skin. Treatment is given by a dermatologist.

Palpebral abscess. It develops more often after trauma complicated by infection. The eyelid swells up, skin becomes tense, painless, and then fluctuation develops. After a cut or arbitrary opening of abscess these signs calm down quickly.

Blepharochalasis. In this process the drooping skin becomes so thin that it forms numerous small folds; as a result of dilation of the superficial veins the skin becomes red. Blepharochalasis develops after frequent palpebral oedema, for example, after recurrent angioneurotic oedema (Quincke's disease). Treatment is surgical.

Angioneurotic palpebral edema (Quincke's oedema) manifests itself by limited edema in the palpebral tissue. Edema develops and disappears very quickly. The disease depends on disturbance of the vessel innervation, autoimmune disturbances. Treatment should be directed at the improvement of activity of the central and vegetative nervous system, decrease of permeability of vessels and organism's desensitisation.

Blefaritis is the inflammatory disease of the palpebral edge.

Etiology. The uncorrected anomalies of refraction especially hyperopia and astigmatism are the main reasons of this disease. Avitaminoses, diseases of teeth, nasopharynx, digestive tract (gastritis, colitis, gastric ulcer) also can entail inflammation of the palpebral edge. Unhygienic conditions, working in the dusty apartment, in the wind can favour the development of blefaritis. There are following blefaritis forms: simple, ulcerative, squamous, meibomian, angular and demodicous. Simple and squamous blepharitis. Palpebral edges are slightly hyperemic, thickened. The tender greyish squamae are visible near the base of eyelashes. The disease has a chronic course and quite often leads to shedding of cilia. Slight pruritus, increased sensitivity of the eyes, which water easily, especially during evening work, are registered. The eyes are sensitive to light, dust, smoke and get tired quickly. Blepharitis is usually combined with chronic inflammation of the mucosa manifesting as slight hyperemia and sensation of "sand" in the eyes.

Ulcerative blepharitis. The eyelids are oedematous, their edges are very thickened and covered with yellow crusts, under which there is pus. After removal of the crusts weeping, hemorrhagic ulcers remain on their places. In a prolonged course the process diffuses on the hair follicles of the eyelashes. If treatment is insufficient or is not carried out ulcerative blepharitis can lead to incomplete closing of the eyelids, their shortening up to their loss (madarosis). There may be wrong growth of the eyelashes (trichiasis). The course is chronic. When treatment is insufficient, ulcerative blefaritis can result in shortening of the eyelids, smoothing and ectropion.

Angular blefaritis is characterized by considerable hyperemia of the palpebral skin, mainly in the corners of palpebral fissure, appearance of fissures, ulcers, wetting, great amount of mucous secretion and itch.

Meibomian blepharitis. Palpebral inflammation is quite often supported by the disease of the meibomian glands, in which great amount of liquid is accumulated. In meibomian blepharitis the palpebral edges are red, thickened; there is a foamy secretion in the eye angle (secretion of the meibomian glands is beaten up into foam by intensified winking). When pressing the palpebral cartilage, the yellow liquid secretion is excreted from the meibomian glands.

In demodectic blepharitis the patients' complaints are the same as in other forms of blepharitis. Objective examination reveals the same picture as in squamous blepharitis. Thinning, shortening and intense falling out of eyelashes are frequently observed. To detect the tick Demodex

microscopic investigation is made under the low (7x10) and then by high (7x40) magnification of the eyelid epithelium, the secretion of the meibomian glands.

Treatment of blepharitis is etiological, general and local. Local treatment is made as follows. Refraction anomalies are corrected (if they are). The eyelid edges are treated with antiseptic solutions (furacillin solution 1:5000, etc), massage of the eyelids with a glass stick, treatment of the eyelid edges with mixture of alcohol and ether or 1% solution of brilliant green. 30% sodium sulfacyl is instilled or 0.25% zinc sulfate in angular blepharitis 5-6 times a day. The eyelid edges are smeared with antibiotic ointment (1% tetracycline or erythromycin ointment).

In ulcerative blepharitis the scales are removed together with the eyelashes (depilation), and in torpid course of the disease autohemovitamin therapy is given.

Surgical intervention includes separation of the eyelids by the eyelash edge, treatment with 1% solution of brilliant green, diathermocoagulation of the meibomian glands. The course of treatment has to be repeated periodically.

In demodectic blepharitis it is used zinc-ichtiol or butadion ointment, solution of amitrazin on the eyelid edges.

Hordeolum (sty). The hordeolum is an acute purulent inflammation of the hair follicle or sebaceous gland near the root of the eyelash. A painful swelling is formed on the limited space of the palpebral edge; it is frequently accompanied by palpebral oedema, and sometimes by edema of the eyeball mucosa chemosis 3-4 days later the yellowish point (a pustule, which bursts out soon) appears on the external palpebral rib, near the root of the eyelash.

Sometimes the hordeola appear one after another. It is caused by a decrease of stability of the organism. In these cases the hordeola are combined with the general furunculosis. Disturbance of the physiological activity of the gastrointestinal tract is very important.

Treatment: application of desensitizing drops (decametoxin, nor-max, sulfacetamide), treatment of the hordeolum head by 1% solution of brilliant green, 70% alcohol, use of 1% tetracycline ointment, dry warmth, UHF. Bandage, compress are contraindicated. Squeezing out is contraindicated due to a possibility of diffusion of the infection into the orbit and cranial cavity (orbital phlegmon, empyema of the cerebral sinuses, thrombosis of the cavernous sinus).

Meibomitis is an acute purulent inflammation of the meibomian gland, it gives the same clinical picture as the hordeolum. The difference consists in more distant localization of the inflammatory process from the anterior palpebral edge; when the focus of suppuration is opened not near the external palpebral rib, but on the internal palpebral surface, from the conjunctiva side. Treatment is the same as in the hordeolum.

Chalazion. Chalazion is a limited tumor formed in the palpebral cartilage. It usually develops without any visible inflammatory signs and painless; it often develops after meibomitis. Fast

increasing chalazion becomes visible from the skin side. The palpebral skin is mobile over the tumor. Chalazion is translucent with greyish colour from the conjunctiva side. Sometimes chalazion is opened on the conjunctival surface and the granular tissue develops around it. Treatment. Small chalazion resolves after the injection of dexamethasone, sometimes UHF. If the tumour is growing in size, it is necessary to extract chalazion in capsule through the incision from the conjunctival side.

The Diseases of the Palpebral Muscle

Lagophthalmos, a "hare's" eye, is incomplete closing of the eye, provoked by paralysis of the facial nerve (n. facialis), which innervates the circular palpebral muscle. In this disease the eyelids remain open, and the eye waters; when attempting to close the eyelid the palpebral remains open. Keratitis, ulceration of the cornea due to it. Ectropion of the lower eyelid leads to conjunctivitis and conjunctive. Besides the paralysis of the facial nerve lagophthalmos can be caused by congenital shortening of the eyelids, cicatricial ectropion of them, considerable diverticulum of the eye. For example, in the orbital tumors, the Basedow's disease. Loss of consciousness in patients in a severe condition may be also accompanied by lagophthalmos. Treatment. Besides treatment of paralysis of the facial nerve, it is necessary to protect the eye from outward influences; application of ointments with antibiotics and vitamins is recommended for this. The eyelids should be sutured together in stable lagophthalmos to avoid affection of the cornea.

Ptosis is lowering of the upper eyelid can be congenital and acquired, partial (1/3 is covered), incomplete (1/2 is covered) and complete (the whole eyeball is covered).

Congenital ptosis is usually bilateral and depends on insufficient development of the muscle raising the upper eyelid.

Acquired ptosis depends mainly on paralysis of the muscle raising the upper eyelid, and frequently it is combined with paralysis of another muscles innervated by the oculomotor nerve. The acquired ptosis can be isolated that indicates its central origin (nuclear paralysis).

As the smooth Müller's muscle innervated by the sympathetic nerve takes part in raising of the eyelid ptosis may develop in paralysis of the sympathetic nerve (ptosis sympathica).

In these cases retraction of the eyeball into the orbital depth (enophthalmos) and contraction of the pupil (miosis) are observed simultaneously with ptosis. This triad is named the Homer's syndrome (ptosis, miosis, enophthalmos); it is evidence of paralysis of the cervical sympathetic nerve.

Treatment. In congenital ptosis and when the acquired ptosis is not treated conservatively, surgical operation is indicated. The most popular operation is shortening of the levator.

Congenital Anomalies of the Eyelids.

Congenital ptosis has been already said above. Coloboma. This disease often affects the upper eyelid, and it is a triangular defect in the middle part. Quite often a "hare's" lip and palatal fissure

are observed in it. Treatment. Restoration of coloboma edges and their suture. The "plastic operation is indicated in great defects.

Epicanthus. Epicanthus is a skin fold on ridge of the nose, due to it the internal angle of the eye and internal part of the palpebral fissure are closed by a semilunar skin fold.

Treatment. Plastic operation gives good results.

Ankyloblepharon is adhesion of the palpebral edges along their length or in separate places.

Complication is amblyopia of obscure origin.

Treatment is surgical.

Palpebral Tumours

There are various benign and malignant tumours of different histogenesis on the eyelids: epithelial (papillomas, skin horn, adenomas, dermoid cysts), mesodermal (fibromas, lipomas, hemangiomas), neurogenic and pigmental (melanomas, nevus, neurofibromas). Treatment is surgical, cryoablation, surgical diathermy, laser coagulation, close-focal roentgenotherapy by indications.

Carcinomas (epitheliomas) usually arise near the palpebral edge, mainly of the lower eyelid, near the internal angle as a firm nodule. The nodule is ulcerated some time later. Usually new nodules arise nearby. It results in formation of big ulcer with torus-shaped firm edges. The disease lasts for a long time; it can destroy the eyelids, pass onto the eyeball and diffuse on the orbit. Carcinoma may start from the meibomian gland (adenocarcinoma); first it may be erroneously diagnosed as chalazion. Treatment. Cryodestruction and close-focal roentgenotherapy give positive results.

Sarcoma occurs very seldom. Treatment. Chemotherapy, X-ray therapy.

DISEASES OF THE CONJUNCTIVA

Anatomy of the Conjunctiva

The conjunctiva covers all posterior surface of the eyelids, passes to the anterior surface of the eyeball and does not end near the edge of the cornea, and covers it in a little changed kind. The fissure cavity between the posterior surface of lids and anterior segment of the eyeball is called a conjunctival sac. Outside lacrimal caruncle is located.

The conjunctiva of cartilage and fornix is covered by a stratified cylinder epithelium, the conjunctiva of eyeball — stratified flat epithelium.

Glands of the conjunctiva. There are glass-shaped cells of cylinder epithelium in a tarsal part of the conjunctiva, and also tubular deepenings of epithelium (Moll's glands), which product liquid.

Additional lacrimal glands, the ramified tubular glands, which are similar to lacrimal glands by their structure, take place, mainly, near the upper edge of cartilage (Waldeyer's glands), as well as fornix of the conjunctiva (Krause's glands).

Vessels of the conjunctiva. The conjunctiva of eyelids, transitional folds and, partly, the eyeball are supplied by the branches of aa. palpebras mediales et laterales and arcus tarseus — posterior

conjunctival arteries. The conjunctiva, that adjoins the limbus of the cornea, is supplied by anterior conjunctival vessels from the u of anterior ciliar arteries. Anterior and posterior conjunctival vessels anastomose with each other.

While examining the conjunctiva it is needed to pay attention at its color, transparency, smoothness, presence of excretions and scars. A normal conjunctiva is transparent, moistened, has a smooth surface. In a tarsal part meibomian glands are visible through it.

Diseases of the Conjunctiva

The diseases of the conjunctiva can be divided into the following groups:

1. Inflammatory diseases of the conjunctiva:

- inflammatory diseases of the conjunctiva of exogenous origin: infectious conjunctivitis; conjunctivitis caused by physical and chemical factors: allergic conjunctivitis;
- inflammatory diseases of endogenous origin: conjunctivitis in general diseases, autoallergic conjunctivitis.

2. Degeneration of the conjunctiva.

3. Tumours.

Inflammatory Diseases of the Conjunctiva (Conjunctivitis)

Conjunctivitis can have both acute and chronic clinical course. Acute conjunctivitis is characterised by sharp hyperemia of conjunctiva. In case of inflammation mucous membrane swells up, loses transparency and gets red because of vasodilatation. We should distinguish a superficial, or the conjunctival injection, from a deep one, or pericorneal injection, that has an important practical value. The first one is caused by the disease of the conjunctiva, the second one — by the disease of the cornea, iris or ciliar body, that is those parts of the eyeball, which are supplied by branches of anterior ciliar vessels. At inflammation of the conjunctiva the superficial vessels are dilatated. All the vessels net is easily removed together with the conjunctiva while replacing by a glass stick. The conjunctiva of the eyeball has a bright red colour. The injection is mostly revealed in the parts which lie farther from the cornea, in the area of the fornix and eyelids, and as far as it approaches to the limbus it diminishes.

A transitional fold is swollen, in drawing of the lower eyelid or ectropion of the upper one it comes forward as a roller. The surface of conjunctiva can remain smooth, but sometimes it becomes rough, follicles appear. At inflammation of mucous membrane there are always mucoserous, mucopurulent or purulent discharge, which dries up on eyelids and glues together their edges.

Subjectively a patient feels the presence of a foreign body, pressure and causalgia in the eye. A certain eyes sensitiveness to light is marked.

Acute epidemic conjunctivitis. This form of conjunctivitis is caused by the Koch — Weeks' bacillus. The disease has an epidemic character and quite often involves the whole families.

organized groups of people. The disease, as a rule, affects both eyes, thus the second eye falls ill in 2 days after the first one (a period of incubation). Multiple small haemorrhages in a mucous membrane and hemorrhoids of transitional folds especially of the inferior one are characteristic for this form of conjunctivitis. Acute epidemic conjunctivitis is often accompanied by the temperature reaction, headaches, insomnia.

Pneumococcal conjunctivitis. Acute conjunctivitis, more often is met in children, the causative agent is Weikselbaum's pneumococcus. Pellicles which are easily taken off by moist cotton wool appear on the mucous membrane of eyelids. In pellicle conjunctivitis (conjunctivitis membranacea). Pneumococcal conjunctivitis can be mixed up with gonoblenorrhoea or diph- of the eye. therefore bacteriological analysis is necessary. Treatment of sharp conjunctivitis. First of all secretion should be carefully deleted, washing eyes once or twice a day by 3% solution of boric acid, by furacillinum solution or kalii permanganas :5000. Sulfonamides give good results, especially in conjunctivitis, caused by Koch — Wicks' bacillus: sulfacylum solubile is used each hour as powder or 3% solution. In severe forms of the disease sulfonamides act quickly and effectively at internal use. At the acute conjunctivitis, caused by the Koch — Wicks' bacillus, pneumococcus, staphylococcus. there are widely used application of 1% gentamycin solution as drops (each 3 hours), .25% levomycetin solution 8 times a day, normax. tobrex — 4-5 times a day. Overnight in a conjunctival sac it is put 1% tetracyclini or erythromycini ointment. Medical films with sulfonamides are applied.

Angular conjunctivitis is caused by Morax — Axenfeld's diplo-bacillus, always bilateral, has an acute, even chronic clinical course. The characteristic reddening of lids edges of near external and internal angles joins the catarrhal phenomena. Due to this sign the conjunctivitis got its name. Discharge are mostly insignificant, mucous and foamy. The strong itch is subjectively felt. Quite often there is the lesion of the cornea (regional keratitis). Treatment. Zincum oxydum which as 0.5-1% solution is brought into a conjunctival sac 4-5 times per a day is a specific mean. The edges of eyelids are oiled by 1% zinc ointment. Treatment needs to be continued a few weeks more after all sickly phenomena disappeared, otherwise the relapse comes easily.

Gonoblenorrhoea is inflammation of the conjunctiva, caused by the Neisser's gonococci, belongs to very serious diseases of eyes. As well as at gonorrhoeal urethritis, gonococci can spread outside a conjunctiva and cause generalisation of infection with all its own distinctive phenomena and complications. Therefore in gonoblenorrhoea gonitis, myositis, endocarditis, general indispositions and increase of temperature are possible. The incubation period lasts for 1-2 days, first signs of the disease: eyelids swell up strongly, become firm so that it is impossible to open them. Discharge are serous bloody, like "meat" - (the first stage is infiltration). On the 4th-5th day eyelids become softer, the amount of discharge increases, they become purulent. Yellow-green pus flows out from the eye in great amounts (the 2nd stage — suppuration). The conjunctiva is hyperemic, friable,

swollen, has a rough surface. On the 7th—10th day the edema of conjunctiva diminishes gradually, wrinkles and pappilae appear on it (the third stage is pappilar hypertrophy). In -6 weeks conjunctiva goes back to the normal condition.

The danger of gonoblennorrhoea is predetermined by the damage of the cornea. The oedema of the conjunctiva of eyeball, squeezing the regional vascular net of the cornea, violates its supply. Epitheli-um of the cornea is covered by erosions, an ulcer can develop easily. Limited infiltrate of grey color which soon becomes yellow appears in the cornea, disintegrating, grows into an ulcer. Clinical course of ulcer is different. It can clear up and, replaced by connecting tissue, heals over, leaving after itself dimness of the cornea (macula or leukoma). Spreading of ulcer into the deep can lead to perforation of the cornea, that results in leukoma, adherent with an iris (leucoma cornea adhaerens). An ulcer, that takes all the cornea, ends with a complete leukoma (leucoma totale), and sometimes sta-phylocoma formation (a stretched leukoma, that protrudes ahead). At distribution of infection inward the eye panophthalmia (pyoinflam-mation of all membranes of eyeball) can develop, which results in shrinkage of the eyeball — subatrophy of the eye. Treatment of gonoblennorrhoeal conjunctivitis. At considerable purulent discharge frequent eyes washing is needed, solution of po-tassium is preferable. Sulfonamides (30% solution of silfacylum-natrium), penicillin, gentamicin each 2-3 hours are administered. A bandage on a sick eye is contraindicated. General treatment is conducted by a specialist in skin and venereal diseases. Prophylaxis of gonoblennorrhoeal conjunctivitis of newborns: the child after birth is wiped eyelids by the cotton wool moistened with 1% solution of boric acid, then instill '2 drops of 30% solution of silfacylum-natrium in each eye three times an hour.

Diphtherial inflammation of the conjunctiva is caused by con non bacteria of Lefler's diphtheria. It occurs mainly in children.

The disease begins with acute edema of the eyelids, their skin is tense, red; eyelids are firm, as a board; discharge are serous bloody. In intercostal space, as well as on the conjunctiva dirty-gray films are visible, often with numerous hemorrhages. After the removal of films conjunctiva bleeds very much. Afterwards a scar appears nulation tissue. Commissures may form between palpebral mucus and sclera. Since the first days the process involves the cornea which in the case of severe disease i quite often fully destroyed. Diphtheria of the conjunctiva is rarely taking as an isolated disease. More often it is combined with diphtheria of nose, fauces, larynx. Prognosis at diphtheria is always serious both for the eve and life of the patient. Treatment. When suspecting diphtheria, without waiting for the results of bacteriological analysis, a patient should be isolated and injected with antidiphtheric serum (6,000-10,000 AO). Locally there are prescribed sulfacil-natrium, penicillin, norfloxacin, gentamicin in drops, 1% tetracyclini ointment.

Pterygium, a wing-like hymen, is three-cornered fold of conjunctiva, accreted with the surface of the cornea. As a rule, pterygium appears near the internal edge of the cornea, rarely near the external one. It occurs more often in people who all the time yield to the air. that contains many admixtures irritating the conjunctiva and the cornea. Pterygium, making progress, can reach the pupil's area, reducing the visual acuity.

Tumours of the Conjunctiva

Lipodermoids are benign, inborn tumours of the conjunctiva, which locate near the edge of the cornea or in the external part of the conjunctival sac. The former have an appearance of dense small tumours, the others — thick fold of rather yellow-white color, the basis of which penetrates deep into the orbit, where passes to adipose tissue.

Angiomas of the conjunctiva are met often enough. They consist of thick interlacements of the dilatated veins or united vein cavities. They are inclined to subsequent diffuse spread, therefore should be removed.

Birthmarks (naevi) of the eyeball conjunctiva are non-pigmented as white spots and pigmented — as brown-black spots. Birthmarks can grow, but remain benign, however, melanoma can develop from them.

Carcinomas belong to malignant tumours which pass to the conjunctiva from the skin of eyelids. At first they have an appearance of a flat wart. Overgrowing, carcinomas grow into epithelial tissue. Spreading by the epibulbar way, they can cover the cornea. Sometimes carcinomas invade into the eye. Treatment. Close-focus roentgenotherapy, laser cryoablation or operative treatment, up to the excision of the eyeball (enucleation, orbital exenteration)

DISEASES OF THE LACRIMAL ORGANS

Anatomy of the Lacrimal Organs

The lacrimal organs consist of the lacrimal gland, which forms a tear, and organs which conduct it, — lacrimal passage.

The lacrimal gland (glandula lacrimalis) lies in the frontal part of the outer-upper segment of the orbit, into the pit of the same name (fossa glandulae lacrimalis). The tendon of muscle, that lifts an upper eyelid, divides the lacrimal gland into the orbital and palpebral parts. The excreting channels of the orbital and palpebral parts of the lacrimal gland are opened in the conjunctival sac. In conjunctival mucose there are accessory lacrimal glands -Krause's and Waldeyer's glands.

The lacrimal gland is innervated by the lacrimal nerve (n. lac-rimalis) — a branch of the trigeminal nerve which has secretory fibres in the composition. The lacrimal gland has sympathetic innervation. Likable fibres get to the gland with vessels from inter-lacemnt near an internal

carotid. The secret of the lacrimal gland is a tear (lacrimae) — is transparent, a little opalescent liquid. Besides of insignificant quantity of albumen and mineral salts, it contains lysozym which has great bactericidal action.

Under normal conditions the amount of tears, secreted by the accessory Krause's glands is sufficient to moisten the eyeball. The basic lacrimal gland begins to function when it gets irritated re-flexly as a result of eye disease under the influence of psychical moments (weeping).

The lacrimal passage consists of lacrimal puncta, lacrimal canaliculi, lacrimal sac and nasolacrimal duct, that is opened in the cavity of nose in inferior nasal meatus, under inferior nasal concha.

The lacrimal puncta are two openings lying near the posterior margin of each lid, near the inner canthus. The lacrimal canaliculus joins the punctum to the lacrimal sac. It first passes vertically, then horizontally and falls into the lacrimal sac.

The lacrimal sac lies in the lacrimal fossa, formed by the lacrimal bone and the frontal process of the maxilla. The upper portion of the sac, fundus extends above the medial palpebral ligament.

The lower end continues as nasolacrimal duct.

Diseases of the Lacrimal Organs

Inflammation of the lacrimal gland — dacryoadenitis is, as a rule, bilateral. It begins with sharp pains and swelling of the external part of the upper eyelid, in the area of the lacrimal gland, and also lacrimation. The disease is sometimes accompanied by displacement of the eye to bottom and inward, diplopia occurs. At drawing of the upper eyelid, a palpebral part of the lacrimal gland, which comes forward in a transitional fold, is well visible. The clinical course of the disease is benign the infiltrate resolves during 10-15 days, but in weakened patients it can result in formation of abscess and even phlegmon of the orbit. As a rule, the disease is accompanied by fever.

Etiology. Inflammation of the lacrimal gland is complication of infectious diseases, such as flu, angina, pneumonia. More frequent bilateral acute dacryoadenitis is observed in parotitis. Quite often orchitis joins the disease. Treatment. Treatment of general disease is conducted (antibiotics, sulfonamides, analgetics, antihistamins). Physiotherapy is used locally (dry heat, UHF-therapy), washing of conjunctival sac with warm antiseptics and anesthetics, application of ointments with antibiotics and sulfonamides. At abscess dissection is performed.

Inflammation of the lacrimal sac — dacryocystitis. When there are inflammatory processes in the mucous of nasolacrimal duct, and also different processes in the nose, for example, hyperplasia of mucous tunic, atrophic processes in it and nasolacrimal duct, nasal septum deviation, formation of polyps, nasolacrimal duct is narrowed or is fully closed. Microflora which gets into the lacrimal sac with a tear propagates and irritates its walls, mucous of which begins to produce a secret, a sac is full of mucous, mucous-festering or only festering discharge. Chronic inflammation of the lacrimal

sac develops (dacryocystitis chronica). At pressure on the the lacrimal sac area festering contents is discharged from the lacrimal punctum. An eye is always moistened. Margins of eyelids, conjunctiva, lacrimal caruncle and half-moon fold are, as a rule, reddening. At the prolonged course of the disease the lacrimal sac stretches (ectasia sacci lacrimalis), near the internal corner of eye a tumour becomes noticeable. In the course of time sac's mucous can atrophy and the track to canalicula can be closed, the locked sac filled with a liquid is formed as a result, that does not become empty at pressure — hydropsy of the lacrimal sac (hidrops sacci lacrimalis).

Quite often inflammation passes outside a sac on the surrounding tissue, causing its acute inflammation — phlegmon of the lacrimal sac. Redness and edema of the skin in the sac area appear, then spreading on eyelids, cheek and a corresponding side of the nose. In a few days a tumour grows soft and pus breaks through outside. A fistula, that appears after opening, is rarely closed, more frequently it remains (fistula sacci lacrimalis). Inflammation of the lacrimal sac is dangerous for the eye. because in case of the least damage of the corneal epithelium the corneal ulcer serpens can develop. Treatment. If discharge from the sac are insignificant, it is recommended to wash the lacrimal ducts 1-2 times, if they were stopped up by the clot of mucus. When washing does not give an effect, the operation should be performed. Dacryocystorhinostomy is usually used, that forms connection between the sac and nasal cavity.

Dacryocystitis of newborns. The lower end of the nasolacrimal duct in the antenatal period is closed by a thin membrane which disappears only shortly before birth. If it does not happen dacryo-cystitis occurs. A gentle pressure over the lacrimal sac produces the reflux of purulent discharge from the lower punctum.

Treatment. It is needed once or twice a day to squeeze content from the sac (descending massage) and wash an eye by solution of boric acid, instill 20% solution of sulfacyl natrium or antibiot-ics. If in a week suppuration is not stopped the probing of the lac-rimal ducts should be performed.

Consequently, the disease of the accessory apparatus of the eye can lead to the severe damages of the eyeball, especially the corne-as, therefore they should be cured in time and eliminated.

DISEASES OF THE CORNEA

In the past the cornea diseases were common and occurred as the complications of chicken-pox, gonorrhoea and trachoma. They were the main causes of blindness and poor sight. At present these diseases comprise about 1/4 of all eye diseases and may be quali-fied as the causes of reduction of vision and blindness in half cases. What is the cause of so high rate of the corneal diseases?

Anatomy of the Cornea

The cornea is the anterior part of the external eye membrane. It is the most projected part of the eye-socket. So, it may be easily traumatized by the external harmful factors such as pathogenic in-ferctive agents, physical, chemical and mechanical factors. Anato-mically the cornea borders are

closely connected with the conjunctiva and sclera through common blood supply and innervation. Due to this, different pathological processes may be easily transferred from the conjunctiva and sclera to the cornea.

The cornea is transparent because its tissue elements are in correct position, the water concentration in it is high, it includes about 18% of definitive collagen, its nerves have no myelin membrane and it does not have blood vessels.

The cornea consists of five layers.

1. The external epithelial layer (superficial flat squamous epithelium). It may be easily traumatized, desquamated and regenerated without impairment of transparency.
2. The anterior border-line membrane (the Bowman's membrane). This layer cannot be regenerated. In the case of its damage the cornea opacity takes place.
3. The corneal stroma (9/10 of the cornea thickness). It cannot regenerate too and may be substituted by the fibrous tissue.
4. The posterior border-line membrane (the Descemet's membrane). This can be partly regenerated without opacity.
5. The endothelium. This layer cannot be regenerated.

The cornea nutrition is accomplished with the help of osmosis and diffusion from the tear, the aqueous of the anterior eye chamber, marginal loop net of the vessels (superficial episcleral and deep scleral vessels) that formed by the anterior ciliary arteries.

The trophic innervation of the cornea is provided by the sympathetic nerves. The cornea has rich sensitive innervation (long ciliary nerves from the optic nerve, which is the first branch of the trigeminal nerve) the condition of which plays a great diagnostic role. The cornea sensitivity is low during the first year of life.

The main cornea functions:

- protective, because it is the part of the external eye membrane;
- refractive; its refractive power is 40-45 D.

In case of the cornea diseases only one visual function is impaired. In this case visual acuity is decreased. The characteristic features of the normal cornea are: transparent, spherical, convex, highly sensitive, without blood vessels; the refractive power is 40-42 D; the surface is smooth, reflective, glistening, moist. The cornea size (diameter) of a newborn is 9 mm, and since five years of age it is like the adult one — 10-11 mm. The limbus width is 1.5 mm.

The methods for the cornea diseases diagnosis are: the lateral light examination, biomicroscopy by the slit lamp, the investigations of the epithelium integrity (dripping of 1% fluorescein solution, reflectivity test), investigation of the cornea thickness (keratopachymetry), investigation of the

cornea sensitivity (the algez-imeter threads or the end of thin cotton wool filament may be used), bacteriological investigations.

The Corneal Diseases

Developmental Anomalies

1. Abnormalities in the cornea size.

Megalocornea (macrocornea) or large cornea — the increase of the cornea diameter over mm in comparison with the age norm.

Microcornea or small cornea — the decrease of the cornea diameter over mm in comparison with the age norm;

2. Abnormalities in the cornea form.

Keratoglobus — the cornea is more convex than the normal one.

Keratoconus — the central or paracentral part of the cornea is protruded. It may be congenital and acquired. The acquired progressive keratoconus arises at the age of -20 because of the hormonal disorders. In case of its progressive course the cornea becomes thinner in the center, its opacity grows and even its perforation may take place.

The characteristic features of visual acuity decrease are ametropia (more frequently astigmatism) and amblyopia. Treatment: glasses, contact lens for ametropia correction; amblyopia treatment (pleoptics); in case of keratoconus at the beginning stage — the conservative treatment (the preparations of vitamins A, E, emoxypine, tauphon, vitasik, tissue therapy); in marked thinness and the cornea opacity — penetrating keratoplasty is effective.

Inflammations (keratitis)

Keratitis are divided into 3 groups (according to A. I. Voloko-nenko).

A. Exogenous:

- cornea erosion;
- traumatic;
- bacterial (purulent);
- viral;
- fungal (mycotic).

keratitis mediated by the conjunctiva and eyelid diseases.

B. Endogenous:

- infectious (phthisic, syphilitic and herpetic);
- neurotrophic;
- due to avitaminosis.

C. Keratitis of unknown etiology.

The general clinical symptoms of keratitis. The "red eye" syndrome (pericorneal or mixed injection); the cornea syndrome (lac-rimation, photophobia, blepharospasm); decrease of visual acuity, pain, impairment in transparency and reflectivity. The infiltrate with irregular borders develops. The cornea surface above it is dim and rough. The cornea sensitivity in the infiltrate area is decreased. The infiltrate may be ulcerated. In many cases vascularization is observed (rooting of the vessels into the cornea). It may be superficial and deep. In case of superficial infiltration the blood vessels come from the conjunctiva, and ramify tree-like. In case of deep infiltration the blood vessels come from the limbus and are located in the deep cornea layers, do not ramify and look like a brush.

The blood vessel tract is often involved, and anterior uveitis develops.

Let's consider the clinical peculiarities of some types of keratitis.

Serpiginous (creeping) ulcer of the cornea. It troubles more often people of the old age with weakened immunity, the persons with chronic dacryocystitis and obstruction of the lacrimal ducts. In the past it was the occupational disease of peasants and took place during field works in summer and autumn. It was called "reaper's illness". In case of epithelial micro traumas by the cereal stems, leaves and tree branches, the microscopic flora penetrates into the corneal tissue and the infiltrate (ulcer in future) develops.

The clinical picture is very typical. The onset is sharp, the cornea syndrome and mixed or pericorneal injection develop. A circular yellow infiltrate appears in the center of the cornea, and then it quickly breaks up and the ulcer with the undermined progressive edge develops. The opposite ulcer edge is slope and clean, and the epithelization on its side takes place. The iridocyclitis with hypopyon soon joins it. The ulcer becomes deeper. When it reaches the Descemet's membrane it begins to protrude under the influence of intraocular pressure as black transparent vesicle (desceme-tocele). Descemetocele is the precursor of the cornea perforation. Complications of serpiginous ulcer. The corneal perforation, pan-uveitis, endophthalmitis, panophthalmitis and orbital phlegmon. Outcomes. The corneal keratoleukoma, symphysal corneal kera-toleukoma, secondary glaucoma, eye subatrophy. Treatment. Urgent hospitalization, bacteriological and bacteri-oscopic investigations are required. Locally: frequent dripping of the disinfect eye-drops, subconjunctival and parabolbar injections of antibiotics of wide spectrum of action and their ointments before sleep are recommended. Also the pupil dilatation with homatropin (1%), mesatone (1%), adrenalin (1%) and tropicamid (1%) are used. When the perforation is possible the tectonic keratoplasty should be performed. After epithelization the corticosteroids and resolution therapy are recommended.

General treatment consists in sulphanilamides, antibiotics (intramuscular injections) and immunotherapy.

Marginal keratitis. The marginal keratitis may be caused by bacterial conjunctivitis, blepharitis, meibomitis and dacryocystitis. It develops due to impaired cornea trophicity in compression of the marginal vascular retina by oedema or lytic action of the toxins and bacterial enzymes. It is usually observed in the old age. Clinical course. The eye suppuration and the corneal syndrome are observed. Near the corneal limbus the infiltrates develop and then they break up resulting in the oblong ulcer separated from the limbus by a thin layer of the transparent the cornea. The intensive vascular infiltration and pericorneal injection from the side of ulcer is observed. Visual acuity disorders are not observed. Prognosis is good but recovery is prolonged. Outcome. Peripheral opacity of the cornea. Treatment. Treatment should be the same as in case of the serpiginous (creeping) ulcer of the cornea but without mydriatics. The primary disease (conjunctivitis or meibomitis) treatment should be performed.

Viral keratitis. It is caused by herpes viruses, adenoviruses and chicken-pox virus. They differ from the bacterial keratitis by development against the background of the organism general reaction to viral infection (poor general condition, headache, elevation of the body temperature, etc.). The regional lymphadenitis may also occur. The sensitivity of the cornea is decreased, the suppuration is absent; the discharge is poor and seromucous. There are follicles on the conjunctiva and infiltrates in the cornea.

Adenoviral keratoconjunctivitis. At first acute adenoviral conjunctivitis with folliculosis develops, and punctate haemorrhages. In 5-7 days there develops the mixed infection, the corneal syndrome, subepithelial multiple punctate infiltrates that ulcerate and may be coloured by fluorescein. The visual acuity decreases.

Prognosis is favourable if the secondary infection does not occur. The tender, coin like punctate opacities remain at the places of infiltrates.

Treatment.

1. Anti-viral preparations. They should be injected in drops, ointments, subconjunctival, parabulbarly, with electrophoresis, phonophoresis and intramuscularly. These preparations are interferon, laferon, deoxyribonuclease, poludan, kerecid, IDU, Zovirax, oxolinic, tebrofenic and florenal ointments.
2. Prevention of secondary infection by antibiotics in drops.
3. Immunotherapy.
4. Therapy for desensitization.
5. The preparations to accelerate regeneration (quinine, taufon, vitasik).
6. Resolution therapy (after disappearance of inflammatory signs tarfot should be injected intraconjunctivally).
7. Anti-recurrent therapy (vaccination in the remission period).

8. Keratoplasty.

Fungal keratitis (keratomycosis) is a rare form of keratitis. It develops in case of general mycosis or as a secondary infection when other forms of keratitis occur. Clinical course. A big rounded infiltrate develops in the centre of the cornea. It looks like solid dry mass encircled by the demarcation line. In several weeks the infiltrate breaks up and ulcer with hypopyon develops. The outcome is the same as in case of cerpiginous ulcer of the cornea. The diagnosis is based on the characteristic features of the infiltrate, clinical picture and microscopic studies of the corneal scrape. Treatment.

1. Elimination of the dry mass with treatment of the infiltrate surface with iodine solution.
2. Fungicide preparations in drops, parabulbarly, intramuscularly and per os: akromicin, actinolysate, nistatin, amphotericin B.
3. Prevention of secondary infection (antibiotics, sodium sulfacil).
4. Keratoplasty should be used when there is no effect of the conservative therapy and in threat of the corneal perforation.

Endogenous keratitis is caused by general infections (syphilis, tuberculosis, malaria, brucellosis, leprosy, etc.). Here we will discuss the most common forms of endogenous keratitis.

Tuberculous keratitis. There are two kinds of tuberculous keratitis: hematogenic or metastatic (when mycobacterium metastases to the eye) and tubercular allergic develops after sensitization by mycobacterium toxins.

Tuberculous metastatic (parenchymatous) keratitis is long-term with alternation of acute conditions and remissions.

Etiology should be established according to general TB characteristic features — changes in the lungs, lymphatic glands, and positive specific serological reactions.

Characteristic features of TB metastatic keratitis are sharp decrease of vision, moderately developed corneal syndrome and mixed eye injection. There may also be observed the vascularization of the cornea and yellow infiltrates in the middle and deep layers. According to peculiarities of the cornea changes three types of metastatic keratitis are established.

1. Deep diffuse TB keratitis. The isolated yellow-grey infiltrates develop in the middle and deep corneal layers against the background of diffuse infiltration. The superficial and deep vessels infiltrate into the cornea. Descemetitis and precipitates also develop.
2. Deep limited or deep infiltrative keratitis. The clinical course is the same as in case of diffuse TB keratitis but the total corneal infiltration is absent. Some infiltrates in the posterior deep layers against the transparent cornea background are observed. Vascularization is much less.
3. TB sclerosing keratitis develops against scleritis background. It is characterised by oedema and hyperemia of the limbus and sclera near the limbus. A triangular infiltrate develops in the deep

layers of the cornea in the limbus and sclera from the inflammatory focus (its base is oriented at the limbus) as if the sclera grows into the cornea. There is small number of vessels in the cornea. There is epithelial oedema over the infiltrate without ulceration. Treatment of all types of TB metastatic keratitis is general phthi-siological TB therapy. Antibiotics, corticosteroids and resolution drugs are recommended parabolbarly; short active mydriatics should be injected in drops. Physiotherapeutic treatment (electrophoresis and phonophoresis with streptomisine, kanamicine and calcium chloride) is also recommended.

Tubercular allergic (scrofulous) keratitis. It occurs in children, teen-agers and young people. Symptoms. The symptoms include a scrofulous face with edema and skin eczema, a strongly developed corneal syndrome and eye ache. Pink-yellow or greyish infiltrates — phlyctenae develop in the superficial layers near the limbus or on the limbus. According to histological data they are nodules that look like tuberculoma but without mycobacteria.

The phlyctena peculiarity is its ability to migrate and presence of the bunch of the superficial dilated vessels. These vessels come from the conjunctiva to the cornea through the limbus. If the number of phlyctenae is high they form scrofulous panus moving through the cornea. There is a big pericorneal injection from the side of phlyctena. Phlyctena usually breaks up forming ulcer and then facet and scar. In severe cases there may be destruction of the corneal stroma, its perforation with formation of adherent leukoma or due to joined secondary infection endophthalmia or panophthalmia develop. Treatment of scrofulous keratitis should be performed in cooperation with a phthisiologist, who recommends general treatment. It includes antibiotics (according to the recommendations), corticosteroids, and resolution drugs that should be injected in drops parabolbarly and with the help of electrophoresis and phonophore-sis. The diet without carbohydrates is required. In case of threat of the corneal perforation, the lamellar tectonic keratoplasty is recommended.

Syphilitic parenchymal keratitis. It is usually manifestation of congenital syphilis; it develops at the age of 6-20. It is characterised by three periods.

1. Infiltration. Duration is 3-4 weeks. Numerous infiltrates like punctuates, lines and strokes develop extending from the limbus to the center of the cornea. The cornea becomes thickened, opaque and looks like opal glass.
2. Vascularization. The cornea looks like not fresh meat and sometimes like ripe cherry. The vessels grow into the deep corneal layers looking like a brush. This period lasts 6-8 weeks.
3. Regression and resolution. The transparency of the cornea reduces from the limbus to center (1-2 years). The empty vessels and opacity of the cornea may remain. The process is always bilateral.

Treatment should be performed in the venereologic hospital. Antibiotics, sulfanilamides, mydriatics, corticosteroids, dionin, yellow mercury ointment, physiotherapeutic treatment are recommended. In case of central leukoma the through keratoplasty should be performed. Herpetic keratitis may be superficial (vesicular or tree-like) and deep (disk-like or metaherpetic). Herpetic keratitis is characterised by a slightly manifested corneal syndrome, decrease of the corneal sensitivity, slow regeneration, it is usually unilateral and there may be a recurrence. In case of vesicular keratitis a lot of small transparent vesicles develop which turn into small ulcers. There may be superficial corneal opacities. In case of tree-like keratitis the vesicles and infiltrates combine and form a branch-like figure. The disease is long-term and iridocyclitis may accompany it. The infiltrates may be distributed deeply and in this case the opacities, that strongly decrease vision, develop. Disk-like keratitis is characterized by the formation of infiltrate in the cornea center. It looks like a light gray-colored disc. The infiltrate covers the whole corneal stroma. Metaherpetic keratitis is a combination of tree-like and disc-like keratitis. The infiltrate with a landcart-like form is accompanied by iridocyclitis. The corneal opacities always develop after deep keratitis.

Neuroparalytic keratitis. It usually develops in case of affection of the trigeminal nerve of Gasser's gland. The cornea loses its sensitivity, but the pain syndrome accompanies this process. The eye reactivity is decreased: it is manifested by small pericorneal injection and absence of the corneal syndrome. The disease starts with opacity and edema of the superficial corneal layers and erosion in the center. Then erosion spreads all over the corneal surface. In infection the pathology looks like the same as in case of creeping the corneal ulcer. The process is prolonged and may last a year. The corneal perforation is possible. Sometimes the alternation of the periods of remission and recurrence is observed.

Treatment should be given to improve the corneal nutrition. Vi-tasik, salcoseril and actovegin gels as well as thiamine ointment are recommended. For prophylaxis of the secondary infection sulfanilamides and antibiotics are recommended. In case of threat of perforation blepharorrhaphy should be performed. General treatment should be given in the co-operation with a neuropathologist (ATP, vitamins of B group, nerobol, hemodesis and rheopolyglukin are recommended).

Avitaminotic keratitis. They are caused by A, B1, B2 and PP avitaminosis. The most common and the most severe is keratitis caused by avitaminosis A. It may be alimentary or endogenous associated with liver or gastrointestinal tract pathology. It characterised by three stages: prexerosis, xerosis and keratomalacia. At the prexerosis stage the triangular whitish plaques develop on the conjunctiva (their bases are turned to the limbus). The cornea loses reflectivity and the epithelial desquamation develops. In xerosis the circular grey plaques or erosions develop in the centre of the cornea. Visual acuity decreases. In case of keratomalacia the corneal stroma break-up is observed.

In 1-2 days the absolute resolution of the cornea with membrane falling out may occur. The pain syndrome is absent. When a secondary infection accompanies this keratitis the endophthalmitis, panophthalmitis and then atrophy of the eyeball develop. Treatment. Vitamin A should be injected in drops and ointments parentally or intramuscularly. In case of E avitaminosis the cornea becomes thin, and keratoconus develops. Visual acuity decreases and wrong astigmatism takes place. Treatment. Vitamin A should be injected locally or intramuscularly. When keratoconus is progressive the through keratoplasty is recommended. In case of B1 avitaminosis the disc-like keratitis against beriberi background develops. In case of B2 avitaminosis parenchymal keratitis develops. In case of PP avitaminosis against pellagra background the epithelial desquamation, deep keratitis and the corneal ulcer may take place. Treatment. Local and general vitamin therapy, solcoseryl and actovegin are recommended.

The outcomes of keratitis

The outcomes of keratitis are opacity of the cornea and loss of its transparency.

The opacities should be differed from the corneal infiltrates (Table 2).

Depending on the degree opacities may be classified into three types.

Characteristic feature	Opacity	Infiltrate
Limits	Clear	Unclear
Inflammatory reactions (the corneal syndrome, injection)	Absent	Present
The corneal reflectivity	Present	Absent
Epithelial defects	Absent	Present
The corneal lustre	Present	Absent

Treatment The resolution therapy, dionin. proteolytic enzymes and torfort injections under the conjunctiva are recommended.

Spot (macula) is rougher corneal opacity. Visual acuity is much decreased.

Treatment. At first the resolution therapy should be given. If it is ineffective keratoplasty is recommended.

Leukoma is intensive opacity. The iris and pupil cannot be observed.

Treatment. Keratoplasty is recommended.

Keratoplasty may be lamellar, through, partial, almost total and total with the sclera border.

According to the aim it may be:

- tectonic (in case of threat or development of perforation);
- optic (in case of central spots, leukoma and keratoconus);
- therapeutic — antirecurrence (in case of herpetic keratitis);

- refractive — intralaminar for correction of refractive anomalies (keratomilosis in case of myopia, keratophakia in case of hypermetropia);
- meliorative (the lamellar transplantation of the cornea for the purpose to prepare for the next through keratoplasty);
- cosmetic is recommended for blind eyes.

Dystrophy of the Cornea

The corneal dystrophy is degeneration of the cornea in case of the corneal nutrition disorders. It may be primary and secondary.

Primary dystrophy is congenital and caused by impairment of protein metabolism. It may also be inherited.

Secondary dystrophy is often induced by pathological processes in the eye (traumas, keratitis, iridocyclitis, glaucoma, cataract extraction and IOL implantation) or develops in case of general diseases of the connective tissue (Still's disease, collagenosis). Dystrophy is characterised by absence of the inflammatory signs (the corneal syndrome, pericorneal injection), vascularization, and decrease of the corneal sensitivity. The opacity of different localisation, size and form develops in the cornea. Visual acuity decreases.

The Cornea Tumours

The corneal tumours are rare. They come more often from the limbus or conjunctiva.

Dermoid. It is a benign tumour that grows near the limbus and partially in the sclera. It is a white-yellow or pink tumour from the skin elements.

Treatment must be surgical. If the tumour takes 1/2 or 2/3 of the cornea, elimination should be combined with keratoplasty.

Papilloma. This tumour is flat or pedunculated. It is light pink, consists of papillae and may cover the whole cornea. Pain is absent.

Treatment includes cryodestruction and short focus R-therapy.

Melanosarcoma comes from the conjunctiva but does not inter-grow with the cornea.

Cryodestruction, enucleation or exenteration of the orbit, chemotherapy and X-ray therapy are recommended for treatment.

Scleral Diseases

The inflammatory scleral diseases are the most common. *Etiology* is associated with TB, rheumatism, collagenosis, chronic infections, syphilis, allergy, gout and viral infections. There are two types of scleral inflammations: episcleritis and scleritis.

Episcleritis. It is inflammation of the superficial scleral layers.

The *clinical* course is characterised by red sclera and eye ache. The corneal syndrome is absent. The bright red swelling without clear outlines is observed on the sclera. The pain is registered when

touching the eyelid by a glass stick or a finger. The clinical course is chronic, recurrent. The prognosis is favourable, without complications. Visual functions are not impaired.

Scleritis is affection of the deep scleral layers. Symptoms are the same as in case of episcleritis but more marked. A constant severe pain without pressing the sclera is observed. It usually develops like keratoscleritis or scleroiridocyclitis and may be complicated by secondary glaucoma.

Treatment is etiotropic; corticosteroids should be injected under the cornea and given in drops; desensitisation; dry warmth, UHF.

THE UVEAL TRACT

Anatomy of the Uveal Tract

The uveal tract consists of three parts: the iris, the ciliary body and the choroid. An arbitrary distinction has been made between the anterior uveal tract (iris and ciliary body) and the posterior uveal tract (choroid).

The **iris** regulates the amount of the light, which enters the eye through the pupil. Diaphragm is composed of the iris and lens, which divides the anterior chamber from the vitreous cavity and the posterior chamber. The iris consists of the anterior mesoderm layer (with numerous chromatophores in dark-eyed people) and a posterior pigment layer derived from ectoderm and contains sphincter and dilatator muscles of the pupil. The **ciliary body** is inaccessible for examination, can be inspected by palpation. It extends anteriorly to the scleral spur and looks like a ring with width of about 8 mm. The posterior zone is smooth and is known as *pars plana*. The ciliary body is composed of unstriped ciliary muscle fibres, stroma and blood vessels.

The **choroid** is a thin vascular membrane extending from the optic nerve to the ora serrata. It consists of five layers: suprachoroid, layer of large vessels, layer of small vessels, choriocapillaris and lamina vitrea elastica (the Bruch's membrane). It makes trophical function. The short posterior ciliary arteries supply the choroid. The long posterior ciliary arteries and the anterior ciliary arteries supply the iris and the ciliary body.

Diseases of the Uveal Tract

There are distinguished the following groups of the uveal tract diseases: 1) developmental abnormalities; 2) inflammations (uveitis); 3) dystrophies; 4) tumours.

Developmental Abnormalities

Albinism. The iris appears pink because of the absence of the pigment, and examination of the retina reveals that the choroidal vessels stand out prominently owing to the absence of the pigment in the retinal pigment epithelium. Patients have poor vision (0.01-0.05), often nystagmus and photophobia occur.

Treatment. Correction of refraction anomaly, pleoplastic.

Congenital colobomas of the iris. These are usually found in the inferonasal quadrant, and may sometimes be associated with colobomas of the ciliary body and choroid.

Treatment. Iridoplasty.

Persistent papillary membrane forms a net in front of the pupil as a result of incomplete reabsorption of the fetal capillary vascular system has no other pathological significance.

Aniridia (absence of the iris). Complaints to poor vision, photophobia.

Treatment, Iridoprothesis, contact lens.

Inflammations of the Uveal Tract (Uveitis)

Clinically uveitis may be anterior (iridocyclitis), posterior (choroiditis) and total (panuveitis) depending on which part of the uveal tract is affected.

Iridocyclitis. Acute and chronic forms are recognized. The patient complains of photophobia, lacrimation, dull pain and reduction of vision.

Clinical features:

- pericorneal vascular injection;
- hyperemia of the iris and the presence of the iris nodules;
- the pupil is constricted and react poorly to light;
- aggregates of cells adhere to the corneal endothelium (keratic precipitates).

In severe cases pus develops in the anterior chamber (hypopyon). There are also pain while touching and reduced accommodation. *Complications.* Posterior synechiae formation (adhesions between the posterior surface of the iris and the anterior lens capsule). If adhesions form all around the papillary margin aqueous humour secreted by the ciliary body cannot enter the anterior chamber. The iris root is therefore pushed forward to come in contact with the corneal periphery (peripheral anterior synechiae), blocking of the drainage angle, and the intraocular pressure raises (secondary pupil block glaucoma). Following the use of mydriatics the pupil margin may assume a scalloped contour in cases where posterior synechiae have formed. If mydriatics are not employed a membrane may occlude the pupil.

Differential diagnosis is conducted with acute glaucoma, acute conjunctivitis (Table 4).

Table 4. Differential diagnosis of acute iridocyclitis with acute glaucoma and acute conjunctivitis

Symptoms	Acute glaucoma	Acute iridocyclitis	Acute conjunctivitis
Clinical course	Sudden onset; severe pain referred to the region of temples, back of the head and teeth associated with nausea and sometimes vomiting	Gradual onset; steady dull pain; photophobia	Gradual onset; sensation of foreign body under the lids
Visual acuity	Much reduced	Slightly or moderately reduced	Normal
Intra-ocular pressure	High	Normal or reduced	Normal
Conjunctival vessels	Congestive hyperemia	Mixed or ciliary injection	Conjunctival injection
Cornea	Oedema	Keratic precipitates	No signs
Anterior chamber	Shallow	Normal depth	Normal depth
Pupil reaction to light	Absent, the pupil is dilated	Decreased, the pupil is narrowed	Normal
Iris	Sometimes oedematous	Oedematous	Normal

Choroiditis Inflammation of the choroids, which almost invariably spreads to the retina (choroidoretinitis).

Uveitis commonly occurs in the following systemic diseases: col-lagenosis, particularly the juvenile rheumatoid arthritis, ankylosing spondylitis, Reiter's disease; tuberculosis, secondary syphilis, sarcoidosis, Behcet's disease (hypopyon — iridocyclitis, aphtous ulceration of the mouth and ulcerative lesions of external genital organs), brucellosis, toxoplasmosis, etc. *Treatment.* To reveal the foci of infection it should be carried out the examination of the whole organism. Cycloplegics and mydriatics are used to relieve pain from ciliary spasm to dilate the pupil to prevent the formation of posterior synechiae. Antibiotics, sulphonamides are administered as well as corticosteroids, antiinflammatory and desensitising drugs.

Tumours of the Uveal Tract

Tumours of the uveal tract are divided into benign and malignant. Benign tumours are cysts, nevus, neuromas, leiomiomas.

Choroidal nevus. A benign pigmental tumour of the choroid, which mostly develops in adults.

Unlike malignant melanomas, there are no retinal degeneration, and visual field defects. Fluorescent angiography doesn't reveal the tumour.

Treatment is not needed. The tumour should be followed up in dynamics, because its malignization is possible.

Malignant melanomas of the iris. Despite the tumours of the iris may have slow growth, it is necessary to conduct surgical removal in time. If growth has been documented and there is danger of the tumour spreading into the ciliary body, removal of the tumour and the surrounding healthy iris is usually all that is required.

Malignant melanomas of the ciliary body are much more aggressive and require urgent treatment. When the tumour has not spread to the choroid or through into the anterior chamber, a localized removal may be considered (cyclectomy).

Malignant melanomas of the choroid. These pigmental tumours, which tend to occur in the older age group and may appear at the *age* of 30-40, but extremely rare before puberty. In transcleral illumination in the area of the tumour a shadow in the pupil is observed. Diagnosis is confirmed by growth of a pigmented tumour, the uptake of the radioactive phosphorus in the area of the tumour, by ultrasonography and by means of the fluorescent angiographic studies, which demonstrate the presence of a pathological circulation. As vision may not be involved until a late stage and no symptoms are noticed, the tumour can become relatively large before the patient is aware of any visual problems. The tumour, which is gray-brown in color, either spreads within the choroid, or breaks through the Bruch's membrane to become nodular or fungiform, so that eventually it can be seen through the pupil. Metastases are spread by blood flow, giving rise to secondary growths in many organs, principally the liver, bones, lung and brain. *Treatment.* Small tumours can be treated by X-ray therapy, light coagulation, cryotherapy or local excisions. Eyes with larger tumours should be removed providing no metastases can be found in other organs. Prognosis depends upon the degree of cellular differentiation of the tumour and its size. Small well-differentiated tumours have a good prognosis and may be observed for long periods without apparently altering the life expectancy of patient. With tumours larger than 10 mm in a diameter that are highly undifferentiated, or where there is evidence of extrascleral extensions or metastases the prognosis is poor.

Dystrophy of the Uveal Tract

Essential mesodermal atrophy of the iris. This is a unilateral progressive condition, beginning not earlier than the third decade of the life. The iris undergoes atrophy and becomes transparent. Polycoria appears, the pupil displaces, the eversion of the pigment leaf of the iris occurs; the total aniridia is possible. Secondary glaucoma is a frequent and serious complication.

Treatment is symptomatic, intended for improvement of trophism.

The **Fuchs' syndrome** is characterized by 3 signs: terochromia, precipitates, the secondary glaucoma.

Treatment is symptomatic.

Glaucomocyclic crisis. Young people mostly men are all Rise of IOP, decreasing of the visual acuity) oedema, precipitates of the corneal posterior surface are typical signs. The attack lasts from some hours to 1-2 weeks.

List of recommended literature:

1. Ophthalmology: textbook / O. P. Vitovska, P. A. Bezditko, I. M. Bezkorovayna et al.; edited by O. P. Vitovska. -2 nd edition. - Kyiv: AUS Medicine Publishing, 2020. - 648 p.
2. Ophthalmology: textbook / O. P. Vitovska, P. A. Bezditko, I. M. Bezkorovayna et al.; edited by O. P. Vitovska. - Kyiv: AUS Medicine Publishing, 2017. - 648 p.
3. Atlas of Glaucoma. Second edition: textbook / Neil T. Choplin, Diane C. Lundy. - Informa healthcare, United Kingdom, 2007. -364 p. ISBN-10: 1841845183.
4. Common Eye Diseases and their Management: textbook / N. R. Galloway, W.M.K. Amoaku, P. H. Galloway and A. C. Browning; -Springer - Verlag London Limited, 2006. – 208 p. ISBN 1-85233-050-32.
5. Ophthalmology at a Glance: textbook / JANE OLVER, LORRAINE CASSIDY; - by Blackwell Science Ltd a Blackwell Publishing company, USA, 2005. -113 p. ISBN-10: 0-632- 06473-0.

Additional:

1. Eye Diseases. Course of lectures: textbook / G. E. Venger, A. M. Soldatova, L. V. Venger; edited by V. M.Zaporozhan. - Odessa: Odessa Medical University, 2005. – 157p.
2. Ophthalmology: textbook. / Gerhard K. Lang, edited by J. Amann, O. Gareis, Gabriele E. Lang, Doris Recker, C.W. Spraul, P. Wagner. - Thieme Stuttgart. New York, 2000. - 604 p. ISBN 0-86577-936-8.
3. EYE Atlas. Online Atlas of Ophthalmology. / All rights Reserved, Oculisti Online. Copyright 2001. -408 p.
4. ABC of Eyes, Fourth Edition: textbook / P. T. Khaw, P. Shah, A. R. Elkington. - by BMJ Publishing Group Ltd, BMA House, Tavistock Square, London, 2005. - 97 p. ISBN 0 7279 1659

Electronic information resources

1. <https://info.odmu.edu.ua/chair/ophthalmology/>
2. <https://repo.odmu.edu.ua/xmlui/>
3. <http://library.gov.ua/>
4. <http://www.nbu.v.gov.ua/>
5. https://library.gov.ua/svitovi-e-resursy/dir_category/general/
6. <http://nmuofficial.com/zagalni-vidomosti/biblioteky/>
7. <https://guidelines.moz.gov.ua/documents>
8. www.ama-assn.org –American Medical Association
9. www.dec.gov.ua/mtd/home/
10. <http://bma.org.uk>
11. www.gmc-uk.org

Lecture № 3

Topic: Glaucoma. regulation mechanisms, intraocular pressure research methods. classification, clinic and treatment of glaucoma. Diseases of the retina and optic nerve.

Pathology of intraocular pressure, research methods here, open-angle glaucoma, closed-angle primary glaucoma, mixed primary glaucoma, treatment of primary glaucoma, acute attack of glaucoma, secondary glaucoma, disease of the retina, disease of the optic nerve.

Actuality of theme:

Glaucoma is a serious disease of the organ of vision, which is one of the leading causes of blindness and poor vision. Without treatment, it leads not only to complete blindness, but also to the death of the eye as an organ. Gradual loss of vision leads to a decrease or loss of working capacity and to profound disability. All this makes it very important to study the pathogenesis and clinic of this disease.

The development of methods for the prevention of glaucoma, its early diagnosis and treatment remain very relevant, in particular, mandatory tonometry of people over 40 years of age in district polyclinics when contacting any specialists, dispensation of glaucoma patients is foreseen.

Congenital glaucoma, associated with underdevelopment of the drainage system of the eye and the angle of the anterior chamber, with untimely diagnosis leads to blindness. With this disease, it is practically impossible to rely on such important research techniques as determining the visual acuity and field of vision, due to the young age of the children. Therefore, early recognition and treatment of congenital glaucoma significantly reduces the risk of developing blindness.

Diseases of the retina and optic nerve can be a manifestation of a number of general diseases of the body, such as hypertension, diabetes, inflammatory diseases (rheumatism, influenza, syphilis, etc.), a consequence of the toxic effects of alcohol and nicotine, and the result of complications of a high degree of myopia.

Changes in the retina and optic nerve in a number of diseases are detected in the early period and are so typical that an ophthalmologist can be the first to diagnose diabetes, GC, increased intracranial pressure. The course and outcome of these diseases largely depends on timely help, which can be done together with doctors of other specialties.

Timely preventive measures and treatment of patients suffering from chronic inflammatory diseases, a high degree of myopia, their rational employment will prevent disability and provide the opportunity for socially useful work.

Therefore, knowledge of the most common diseases of the retina and optic nerve, the ability to provide timely emergency care should be known to every doctor.

Goal:

Students should know:

- main symptoms and classification of glaucoma.
- the main symptoms of an acute attack of glaucoma.
- consequences and complications in case of untimely detection of glaucoma.
- principles of medication and operative treatment of glaucoma.
- classification and methods of treatment of secondary glaucoma.
- a picture of a healthy fundus.
- etiopathogenesis, clinic and treatment measures for retinal detachment, optic nerve atrophy.
- principles of prevention and social and labor rehabilitation of patients with diseases of the retina and optic nerve.

Students should be able to:

- measure intraocular pressure by palpation;
- recognize an acute attack of glaucoma;
- provide first aid in case of an acute attack of glaucoma;
- resolve the issue of hospitalization of the patient (planned, urgent);
- take measures to prevent glaucoma.
- carry out an examination of a patient with a disease of the retina and optic nerve (examine visual acuity, field of vision, sense of color).
- to interpret the oculist's record of the nature of changes in hypertension, diabetes, disc congestion.
- purposefully collect anamnesis from a patient with diseases of the retina and optic nerve.
- suspect an acute disorder of blood circulation in the arterial vessels and provide first aid.
- to conduct a medical and labor examination in diseases of the retina and optic nerve.

At the lecture, issues of duty, conscience, dignity, medical secrecy and responsibility are resolved.

Educational goals.

Professional training will be combined with ethical education. It is explained to the students that there is a kind of relationship between the doctor and the patient, which is regulated both by moral and ethical norms and by law. The norms of behavior of doctors in their relations with patients and

colleagues were developed over many generations and became the basis for the formation of medical ethics and deontology.

Basic concepts:

General goals: to get acquainted with the current state of glaucoma problems, to learn how to detect, to correctly and timely refer patients for treatment, knowing modern methods of treatment, achieving a high level of compliance with its main stages (diagnosis, treatment and valeo-medrehabilitation recommendations).

On the basis of knowledge of anatomy, physiology of the organ of vision, master the basic methods of eye examination, clinic, principles of treatment of diseases of the retina and optic nerve, be able to interpret the ophthalmologist's record, the nature of pathological changes of the fundus, recognize their connection with general diseases of the body.

Plan and organizational structure of the lecture:

№	The main stages of the lecture and their content	Time allocation (min.)
1	2	3
	<p>Preparatory stage:</p> <p>1 Determination of educational goals. 3</p> <p>2 Providing positive motivation 2</p> <p>The main stage:</p> <p>3 Presentation of lecture material.</p> <p>1. Pathology of intraocular pressure 10</p> <p>2. Research methods of IOP 10</p> <p>3. Open-angle glaucoma 5</p> <p>4. Primary closed-angle glaucoma 5</p> <p>5. Mixed primary glaucoma 5</p> <p>6. Treatment of primary glaucoma 10</p> <p>7. Acute attack of glaucoma 5</p> <p>8. Secondary glaucoma 5</p> <p>9. Retinal disease 10</p> <p>10. diseases of the optic nerve. 5</p> <p><i>The final stage:</i></p> <p>4 Summary of the lecture, general conclusions. 5</p> <p>5 The lecturer's answers to possible questions 5</p> <p>6 Tasks for student self-training 5</p>	

Content of the lecture material:**THE INTRAOCULAR PRESSURE PATHOLOGY**

At present glaucoma is considered as one of the main causes of blindness and visual disability. The frequency of glaucoma in individuals over 40 is about .5%. Every year, one in a thousand of this age group becomes affected with glaucoma. In developed countries about % of all blind people lost their vision because of glaucoma. Despite some progress, the problem of treatment of glaucoma has not been decided yet. The most effective treatment can be done at the initial stage of the disease; therefore, the early diagnosis remains an actual task of ophthalmology. It is very important to diagnose an acute attack of glaucoma and then to send the patient to the eye hospital immediately, as in the course of a few days — sometimes even hours — it can turn into blindness.

MECHANISMS OF THE INTRAOCULAR PRESSURE REGULATION

The foundation of the glaucoma pathogenesis lies in the breaking of regulation of intraocular pressure (IOP). From the course of anatomy, you know that the eye is closed space, the sides of which are three coats (fibrous, vascular and retinal) and the contents are the lens, vitreous, intraocular humour or aqueous humour.

The level of IOP is determined by two factors: rigidity of the eye coats, in primary, sclera; volume (capacity) of eye contents.

The first factor is a relative constant volume. The lens and vitreous are also relative constant size. The quantity of aqueous humour and blood in intraocular vessels changes. An aqueous humour produces by processes epithelium of the ciliary body. I his fluid serves as a source of nourishment lor the avascular transparent structures of the eyes. After fulfilling its function of nourishment providing, the aqueous humour enters the path of the outflow.

The IO fluid from the posterior chamber of the eye comes through the pupil into the anterior chamber, then pass through the corner of the angle of the anterior chamber. The angle of the anterior chamber forms in front by the cornea and sclera, behind — by roots of iris and ciliary body. The top of the angle is closed by the trabecular meshwork. The trabecula has 15 layers, plates with a great number of apertures, which forms system of the outflow (*fountain spaces*). Then the IO fluid passes into the scleral sinus or the Schlemm's canal, which is seen as a narrow split. It goes around the cornea like a ring. Then it passes through the collector channels, including the aqueous veins, into the episcleral and intrascleral vein plexus and anterior ciliary veins. The trabecular meshwork, Schlemm's canal and collector channels together are called the drainage system of the eye. Other posterior paths of aqueous outflow do not have an important meaning. The posterior pass through suprachoroidal space with vessels and nerves to posterior part of eye and coats of optic nerve, and a certain amount of aqueous humour passes out of the eye through the sclera. A constant level of IOP is supported by active and passive mechanisms:

- active mechanism is the secretion of IO fluid;
- passive mechanism is aqueous outflow through drainage system of the eye.

Increase of IOP level may be caused by:

- hyper- or hyposecretion of aqueous fluid;
- destruction of the outflow from the posterior to the anterior chamber (back sinechia, pupil blockade);

change in the drainage system, primary, in the trabecula and sinus.

METHODS OF INTRAOCULAR PRESSURE EXAMINATION

The **palpator method** of examination of IOP is used in those situations when it is impossible to use the instrumental methods (corneal diseases, postoperative period, after trauma, acute conjunctivitis).

The increase of pressure is indicated by:

- T+1 — the eye moderately hard, IOP is moderately increased;
- T+2 — the eye is hard, IOP is high;
- T+3 — the eye is hard as a stone, IOP is very high. A decrease of pressure is indicated by:
- T-1 — the eye is moderately soft, IOP is decreased moderately;
- T-2 — the eye is soft, IOP is low;
- T-3 — the finger feels no support when pressing the eye, IOP is decreased significantly;
- T-N — normal IOP.

Tonometry can be done with or without direct contact. Tonometry without contact:

pneumotonometer, fluctuating tonometer. In Ukraine the Maklakov's tonometer (contact) is used.

The normal IOP level (according to Maklakov) is 17-26 mm Hg.

Daily tonometry — IOP measurements in the morning and evening. In the norm the difference between two measurements (amplitude of fluctuation of IOP) should be no more than mm Hg.

Hour tonometry — IOP measurements every hours during two-day period.

Elastotonometry — measurements of IOP by four tonometers with weights of ; 7.5; 10; 15 g. Then we draw the elastic curve.

Normal dates of the elastic curve (EC):

1. Beginning (IOP with 5 g tonometer) — no more than 20 mm Hg.
2. Tonometric pressure (IOP with 10 g tonometer) — 17-26 mm Hg.
3. The end of EC (IOP with 15 g tonometer) — no less than 30 mm Hg.
4. Elastolifting — the length of EC (the difference between IOP with tonometer of 5 and 15 g) — in the norm - - 7—12 mm Hg.
5. The character of EC: direct in the norm, broken or discen-ding in pathology.

Tonography gives a quantitative data of secretion and aqueous humor outflow.

Normal data of tonography:

- Po (true IOP) -- 9-20 mm Hg (average 15-17 mm Hg);
- C (coefficient of outflow) — 0.14-0.6 mm³/min/mm Hg (average, 0.2-0.3);
- Po/C (the Becker's coefficient) — less than 100;
- F (min. volume of aqueous humor) less than 4.5 mm³/min (average 1.9-2.2).

Provocative tests (dark, water) are used only in a hospital.

The method of examination of angle of the anterior chamber is **gonioscopy**. A gonioscopic lens is used in combination with slit — lamp for gonioscopy. We see the cornea, the line of Schwalbe, trabecula. Schlemm's canal, scleral spur, ciliary body, and the root of the iris.

Examination of IOP in children is conducted under anaesthesia.

PATHOLOGY OF IOP _____

Not every IP increase results in glaucoma. Glaucoma is such increase of IP, which is accompanied by disturbance of visual functions and change of the optic disc (OD). The cardinal signs of glaucoma are increase of IOP, constriction of visual field from the nasal side and the atrophy of the optic nerve with glaucomatous excavation.

Glaucoma can be: congenital; acquired (primary and secondary).

Congenital Glaucoma or Hydrophthalmos

There are distinguished three clinical forms of congenital glaucoma:

1. The classical or simple form — develops as a result of an incomplete resorption of the mesodermal tissue in the angle of the anterior chamber.
2. Congenital glaucoma with anomaly of the anterior part of the eye (aniridia, coloboma of the iris). It is caused by the abnormal development of the angle of anterior chamber.
3. Congenital glaucoma with neurofibromatosis and angiomatosis of the head (encephalotrigeminal angiomatosis, *Sturge — Weber — Dimitry's syndrome*).

Sclera, cornea, limbus of the child's eye are very elastic, they stretch, the eye enlarges in the size and at the final stages reaches a considerably large size. Such condition is known as buphthalmos (a "bovine" eye). In 75% hydrophthalmos is bilateral. The disease is easy diagnosed within first days after birth in 90% of affected children.

Diagnosis methods of congenital glaucoma:

- lateral illumination, biomicroscopy (limbus, cornea, their size, transparency);
- keratometry;
- tonometry (under general anaesthesia or during deep physiological sleep);
- US-biometry (length of eye axis, depth of anterior chamber);
- ophthalmoscopy.

Clinical course. The following signs are typical for hydrophthalmos:

- stretched limbus (up to 2-3 mm, normal — 1 mm);

- large cornea (the norm — before 1 year — 9 mm, at 1 year — 10 mm);
- deep anterior chamber (the norm — 1.5-2 mm in newborns, 3-3.5 mm — in adults);
- enlargement of the eye size;
- increase of IOP;
- lowered vision;
- glaucomatous atrophy of the optic nerve (displacement of the vascular bundle, glaucomatous excavation, pale optic nerve).

Then the eye stretches and ruptures of discemetic membrane appear, thus permitting fluid to enter the corneal stroma with subsequent loss of transparency, lacrimation, photophobia, blepharospasm, redness. In later stages of glaucoma the leucoma of the cornea forms, it may be dystrophy of the cornea up to trophical ulcer.

Hydrophthalmos has 4 stages.

1. The initial stage.

Diameter of the cornea is 12-12.5 mm, length of eye axis is increased by 1.5-2 mm. Fundus of the eye is normal.

2. The developed stage.

Diameter of the cornea is 13-14 mm, the length of axis of the eye is increased by 3-4 mm. There are glaucomatous excavation of the optic nerve, partial atrophy of the optic nerve in the fundus of the eye.

3. The late stage.

Diameter of the cornea is more than 14 mm. Axis of the eye is more than 24 mm. There are atrophy of the optic nerve and glaucomatous cup in the fundus of the eye.

4. The terminal stage.

Absolute blindness, staphilomas of the sclera and the cornea, the corneal leucoma, complete atrophy of the optic nerve with glaucomatous cup (excavation).

Treatment is only surgical: goniotomy and goniotomy. laser surgery, sinusotrabeulotomy, reconstructive microsurgery of the angle of the anterior chamber.

An operation should be performed early, as soon as the diagnosis has been established.

Acquired Glaucoma

Primary glaucoma is the multifactorial condition with the threshold effect. It develops owing to combination of a few unfavorable to IOP regulation factors, each of which doesn't cause glaucoma, but association of some of them exceeds the compensative possibility of **IOP** regulative mechanism.

These factors are:

anatomical peculiarities of the anterior chamber angle:

- local vascular changes, which may be the signs of general vascular pathology (atherosclerosis, hypertensive disease, diabetes mellitus);
- diseases of the connective tissue, which cause sclerosis and degeneration of trabecular meshwork (collagenosis);
- neuro-vegetative alterations, which cause hypersecretion of aqueous humor.

Methods of diagnosis consist in functional and objective examinations.

Functional examination includes visometry, adaptometry, perimetry, campimetry.

Objective examination uses the method of lateral illumination, biomicroscopy, gonioscopy, ophthalmoscopy; measurement of **IOP** (diurnal and hourly tonometry, tonography, clastotonometry).

Classification of primary glaucoma

The forms of primary glaucoma are open angle glaucoma, narrow or closed angle glaucoma and mixed glaucoma.

Open-angle glaucoma

Pathogenesis — sclerosis of trabeculum or constriction of Schlemm's canal due to endocrine, vascular diseases (atherosclerosis, hypertension, diabetes mellitus). The angle of the anterior chamber (gonioscopically) is wide, open, can be pigmented.

Clinical course. As a rule, glaucoma is bilateral, but in 80% is asymmetric: damage of one eye is more than other ones.

It arises and progresses slowly, unwittingly for the patient. The symptoms are loss of visual acuity and visual field. At external examination the eye has normal appearance; only at a biomicroscopy it is possible to find dystrophical changes of the iris. An angle of the anterior chamber has normal sizes. There may be pigmentation of trabecula. The first objective sign is periodic or constant rise of IOP more than 27.0 mm Hg.

There are the typical changes of the optic nerve, revealed with using of ophthalmoscopy. The first manifestation is displacement of vascular bundle to a periphery edge of the optic disc. Then extension of physiological excavation up to the edge of the disc with an inflection of vessels. In the

last stage the optic disc pales up to the white -- grey colour with the edge excavation. The patients, as a rule, do not notice the first changes of visual functions, they are discovered only at inspection by the ophthalmologist. These are decrease of the dark adaptation, extension of a blind spot, appearance of paracentral absolute scotomas, contraction of peripheral borders. It is typical the contraction of the visual field from the nasal side. When considerable loss of visual field takes place, the central vision decreases. Without treatment the process results in complete blindness (VIS=0).

Closed-angle primary glaucoma

Pathogenesis consists of blockade, closing of the angle of the anterior chamber by the iris root owing to the forward location of the lens and the functional blockade of the pupil, or due to the total iris adhesion to the lens. *Clinical course.* Typical patients complaints are the iridescent circles around a light source, pain in the eye, periodic headache, foggy vision in the morning and after emotional tension, often change of glasses. Closed-angle glaucoma often starts with an acute attack. Objective signs of the acute attack of glaucoma are: congestive hyperaemia (wide episcleral veins — the "cobra's" symptom); a small anterior chamber; midriasis; narrow or closed angle of the anterior chamber at gonioscopy. There are the same changes of visual acuity, visual field, IOP, dark adaptation, fundus of the eye as in the open angle glaucoma.

Mixed primary glaucoma

At this form of glaucoma there are symptoms of both open-angle and closed-angle forms. The diagnosis of this form is made mostly on the data of gonioscopy, which shows, that the angle of the anterior chamber is partially closed and partially opened

Stages of primary glaucoma

1. The initial stage. Peripheral boundaries of the visual field are normal, but there are an extension of a blind spot, paracentral scotomas, decreasing of dark adaptation. There are displacement of a vascular fascicle, extension of physiological excavation of the optic disc on the eye fundus.

2. The developed stage.

Narrowing of the visual field from the nasal side: it is less than 55° , but more than $^\circ$. There is excavation of the optic nerve disc on the eye fundus.

3. The late stage.

Narrowing of the visual field from the nasal side is less than 15° . There is partial atrophy and excavation of the optic nerve on the eye fundus.

4. The terminal (absolute) stage.

The visual acuity is 0, there is complete atrophy of the optic nerve with glaucomatous cupping on the eye fundus.

Depending on the IOP level glaucoma may be with normal pressure (IOP is less than 26.0 mm Hg), with moderately increased pressure (IOP — 27.0-32.0 mm Hg), with high pressure (IOP — 33,0 mm Hg and more).

Depending on preservation of visual functions during enough long period of observation (6 months and more) glaucoma may be *stabilized* (if the functions do not change for the worse) and *non stabilized* (the visual functions progressively deteriorate).

Thus, the diagnosis of the primary acquired glaucoma includes 4 signs:

- the form of glaucoma (it is determined by character of the complaints, state of the angle of the anterior chamber);
- the stage of glaucoma (it is determined by state of visual functions and eye fundus);
- the level of IOP (a daily curve of IOP);
- dynamic of visual functions.

Treatment of primary glaucoma. It is impossible to cure a primary glaucoma. The purpose of treatment is normalization of IOP, stabilization of visual functions. The treatment may be conservative and surgical. The patients must be permanently under dispensary observation.

It is known such methods of glaucoma treatment: 1. Hypotensive medicamentous therapy: miotics (1, 2 or 3% solution of pilocarpin, 2 or 3 times a day); P-adrenergic blockers (0.25-0.5% timolol, aruthimol, optimol, clophelin, betoptic, once or twice a day). Lately new antiglaucomatic drugs — prostaglandin analogs (0,005% lathanoprost solution or xalathane, thravatan or ,004% thravoprost solution) are used -2 times a day. Regime of using and the drug are got out under the control of IOP in clinic. or preparations are used no more than times a day.

2. Surgical treatment. If miotics and adrenoblockers do not normalize IOP, the urgent antiglaucomatous operation is necessary. The type of operation depends on the form of glaucoma. In closed angle glaucoma (without goniosinechias), it is recommended iridectomy — removal of a part of the iris for support of outflow of aqueous humor from the anterior to the posterior chamber. It is performed from above on 12 hours near the root of the iris.

Surgical operations at scleral sinus are less traumatic and patho-genetical. They are sinusotomy — opening of the external side of the Schlemm's canal; trabeculotomy — cutting of the internal side of the Schlemm's canal; sinusotrabeculoectomy — a portion of the trabecular meshwork is removed.

Fistulization operations are intended for derivation of new outflow way for aqueous humor from the anterior chamber in the subconjunctival space. Their efficacy is about 80%. Their shortcoming is that it is not very easy to determine the hypotensive effect, owing to that hypotonia, secondary cataract may arise.

The secretion of aqueous humor can be decreased by cyclodiathermy, cyclocryopexy or cyclophotocoagulation.

Laser surgery is used in iridectomy, goniotomy (thermocoagulation of the iris root for shortening of its tissue and drawing out it from the angle of the anterior chamber) in closed angle glaucoma. There are the laser goniotomy and trabeculotomy to stretch the trabecula in the opened angle glaucoma.

The surgical treatment is effective for IOP normalization, but IOP normalization does not always stabilize visual functions. Distrophic changes of internal coat of the eye may progress even in normal IOP. Therefore patients with glaucoma must be under dispensary observation even after normalization of IOP. Twice a year they must take course of conservative treatment for improvement of metabolism, microcirculation in the tissues of the eye.

If the IOP is 24.0-26.0 mm Hg or the difference between the IOP of the right and left eye more than 5 mm Hg the diagnose "glaucoma suspected" is made. All the patients with such a diagnose must be examined in clinic with using the following methods of examination: perimetry, campimetry; daily, hour tonometry, elastotometry, gonioscopy, tonography; loading and unloading provocative tests. In dependence on the examination results the diagnosis "glaucoma" is confirmed or "glaucoma suspected" is removed.

Acute Attack of Glaucoma

Acute attack of glaucoma is the greatest degree of disorders of the IOP regulation in closed-angle glaucoma. Acute attack rises usually in the second half of the day or in the evening after emotional tension, taking of great amount of fluid.

Clinical course. The visual acuity lowers up to 0.09-0.01, sometimes to a photoperception. There is strong pain in the eye and in the corresponding half of the head with an irradiation to the trigeminal nerve and remote organs: in heart, epigastric area, stomach. Nausea, vomiting are typical.

Objective signs are: lacrimation, the corneal syndrome, oedema of the lids; stagnant injection of vessels of a front department of the eye ("the head of Medusa"); oedema of the cornea, lowering of its sensitivity, the surface of the cornea is rough, as if pricked by a needle; a narrow anterior chamber; a wide pupil (midriasis) with weak or no reaction to light; greenish colour of the eye fun-

dus reflex; if the eye fundus is visible, the atrophy of the optic nerve, pulsing of central artery of the retina are defined; IOP is 60-80 mm Hg; the eye is hard, as a stone (T+3).

Outcomes may be as follows:

- fast decrease of IOP leads to visual functions recovery, but the pupil usually remains wide;
- terminal (absolute) painful glaucoma develops with the strong steady pain in the eye, stagnant injection ("the head of Medusa"), bullous (vesicles) keratopathy, rubeosis of the iris. Visual acuity is 0;
- secretion of aqueous humour and IOP reduces, all clinical signs disappear gradually, but the vision, as a rule, never returns;
- acute attack of glaucoma may recur.

Differential diagnosis of the acute attack of glaucoma should be carried out with: acute gastrointestinal diseases, poisonings; hypertonic crisis; attack of stenocardia, if the attack of glaucoma is on the left eye; acute iridocyclitis (Table 3).

First aid in acute attack of glaucoma. 1-2% pilocarpin solution is instilled every 15-20 minutes; per os diacarb 0,5.

Table 3. Differential diagnosis of the acute attack of glaucoma and iridocyclitis

Symptoms	Acute attack of glaucoma	Iridocyclitis
Iridescent circles	present	absent
Irradiation of a pain	present	absent
Injection of the eye	stagnant	pericorneal
The cornea	oedematous, loss of sensitivity and transparency	transparent, mirror, sensitive
The anterior chamber	shallow	middle
Pupil	wide	narrow
IOP	very high	normal or hypotonia

or hypothiazid 25-100 mg) is given. A patient must immediately be referred to ophthalmological clinic.

Specialized help in acute attack of glaucoma. In ophthalmologic clinic 1% or 2% pilocarpin solution is instilled every 15 minutes during 2 hours and then every hour. 25 mg of aminacin or lytic mixture (1 ml of 2.5% aminacin + 1 ml of 1% dimedrol or 1.0 ml of 2.5% pipolphen + 1.0 ml of 2% promedol) is entered intravenously. Diacarb is administered orally in the dose of 0.25-0.5 g 2 times a day. The hyperosmotic agents are used (urea, glycerol, manni-tol, salt laxative). Diverted procedures (hot baths for legs, leech on temples, mustard plasters on a nape) are used too. The

antiglaucomatic operation (iridectomy) is necessary, when medical therapy has failed within 24 hours. If IOP decreases after medical therapy, it is recommended planned antiglaucomatic operation in a period of IOP normalization.

Secondary Glaucoma

The secondary glaucoma is a consequence or complication of other diseases of the eye. The following forms of secondary glaucoma are distinguished.

1. **Uveal**, postinflammatory glaucoma is caused by peripheral anterior synechiae or seclusio of the pupil.

Treatment. It is conducted therapy of uveitis, midriatics and massage of the pupil.

2. **Phacogenic** glaucoma: phacotopycal (subluxation, dislocation of lens); phacomorphic (enlarging of the lens size in traumatic cataract or in intumescent stage of senile cataract); phacolytic (in hypermature senile or traumatic cataract).

Treatment is surgical (extraction of cataract)

3. Neovascular. postthrombotic glaucoma (it is complication of central vein of retina occlusion).

Treatment consists in treatment of the basic disease and symptomatic! therapy.

4. Traumatic glaucoma: contusional. burny glaucoma; wound glaucoma.

Treatment includes antiinflammatory, surgical therapy.

5. Neoplastic glaucoma (glaucoma is associated with intraocular tumors: uveal melanoma or retinoblastoma).

Treatment. Enucleation of the eye or exzantation of the orbit.

6. Degenerative glaucoma (it is caused by diabetic retinopathies, uveapalhies).

In all forms of secondary glaucoma (except the neoplastic form) against a background of treatment of the basic disease, carbonic anhydrase inhibitors (diacarb) are administered, the surgical treatment is performed in case of need.

In summary it is necessary to tell, that the glaucoma cannot be treated, but it is possible to conduct prophylaxis of blindness caused by glaucoma. For this purpose it is necessary the early diagnosis of this disease. Early detection of glaucoma is performed by examination of all people older than 40 years old. The patients with hypertonic disease, diabetes meliitus and the persons, which have relatives suffered from glaucoma, represent a risk group for glaucoma and must be measured IOP 1 time a year. Other persons at the age above 40 are made tonometry 1 time per every 3 years. All the patients with glaucoma must be under a dispensary observation, in IOP compensation they are examined 1 time per 3 months.

General material and mass-methodological support

lectures: methodical development of lectures, multimedia presentation, models, tables, tool sets.

Questions for self-control:

1. Analyze the given daily tonometry curves.
2. With the help of Polyak's measuring ruler, determine the value of IOP from the prints of Maklakov's tonometer.
3. Treat a glaucoma patient.
4. Determine the type and stage of glaucoma based on the patient's complaints and the data of the examination conducted in the clinic.
5. Describe the fundus in patients with pathology of the retina and optic nerve based on thematic tables and the atlas of the fundus:
 - a) normal fundus
 - b) eye fundus in hypertensive disease of the 1st degree
 - c) fundus with a stagnant disc

List of recommended literature:

1. Ophthalmology: textbook / O. P. Vitovska, P. A. Bezditko, I. M. Bezkorovayna et al.; edited by O. P. Vitovska. -2 nd edition. - Kyiv: AUS Medicine Publishing, 2020. - 648 p.
2. Ophthalmology: textbook / O. P. Vitovska, P. A. Bezditko, I. M. Bezkorovayna et al.; edited by O. P. Vitovska. - Kyiv: AUS Medicine Publishing, 2017. - 648 p.
3. Atlas of Glaucoma. Second edition: textbook / Neil T. Choplin, Diane C. Lundy. - Informa healthcare, United Kingdom, 2007. -364 p. ISBN-10: 1841845183.
4. Common Eye Diseases and their Management: textbook / N. R. Galloway, W.M.K. Amoaku, P. H. Galloway and A. C. Browning; -Springer - Verlag London Limited, 2006. – 208 p. ISBN 1-85233-050-32.
5. Ophthalmology at a Glance: textbook / JANE OLVER, LORRAINE CASSIDY; - by Blackwell Science Ltd a Blackwell Publishing company, USA, 2005. -113 p. ISBN-10: 0-632- 06473-0.

Additional:

1. Eye Diseases. Course of lectures: textbook / G. E. Venger, A. M. Soldatova, L. V. Venger; edited by V. M.Zaporozhan. - Odessa: Odessa Medical University, 2005. – 157p.

2. Ophthalmology: textbook. / Gerhard K. Lang, edited by J. Amann, O. Gareis, Gabriele E. Lang, Doris Recker, C.W. Spraul, P. Wagner. - Thieme Stuttgart. New York, 2000. - 604 p. ISBN 0-86577-936-8.
3. EYE Atlas. Online Atlas of Ophthalmology. / All rights Reserved, Oculisti Online. Copyright 2001. -408 p.
4. ABC of Eyes, Fourth Edition: textbook / P. T. Khaw, P. Shah, A. R. Elkington. - by BMJ Publishing Group Ltd, BMA House, Tavistock Square, London, 2005. - 97 p. ISBN 0 7279 1659

Electronic information resources

1. <https://info.odmu.edu.ua/chair/ophthalmology/>
2. <https://repo.odmu.edu.ua/xmlui/>
3. <http://library.gov.ua/>
4. <http://www.nbu.gov.ua/>
5. https://library.gov.ua/svitovi-e-resursy/dir_category/general/
6. <http://nmuofficial.com/zagalni-vidomosti/biblioteky/>
7. <https://guidelines.moz.gov.ua/documents>
8. www.ama-assn.org –American Medical Association
9. www.dec.gov.ua/mtd/home/
10. <http://bma.org.uk>
11. www.gmc-uk.org