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Eduard BURIACHKIVSKYI
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METHODOLOGICAL DEVELOPMENT

TO THE LECTURES ON THE EDUCATIONAL DISCIPLINE

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Educational discipline: OPHTHALMOLOGY

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Head of Department _____ Liudmyla VENGER

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

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Head of the subject-cycle methodological commission for surgical disciplines

_____ Vasyl MISHCHENKO

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

Lecture №1

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, gonorrhea, 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 pro¬tection 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 palpe¬bral 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. Subcuta¬neous fat is porous that makes the eyelid skin very mobile. Oede¬ma of the eyelids appearing in various local and some general dis¬eases 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 immediate¬ly under the skin. The muscle is divided into two parts: a palpe¬bral part, which is nearer to the free eyelid edge (pars palpebra¬ls) and an orbital part, which is nearer to the orbital edges orbi talis).

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 semilu¬nar 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 semilu¬nar plates of the upper and lower eyelid are joined with the help of their medial ligament (lig. palpebrale mediate) 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 palpebrale 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 mus¬cle fibers lifting the upper eyelid has a great physiological impor¬tance, essence of which consists in simultaneous lifting of all eye¬lid 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 posteri¬or palpebral surface and anterior surface of the eyeball to the cor¬neal 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 (Zaiss' 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 el medialis are unit¬ed, and they form vessels of the arterial marginal arch (arcus tar-seus marginalisj: 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 abcess 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 palperbral 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, meimbomian, 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, appearence 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 oint¬ment, 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 dexa-sone, 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
provoked by paralysis of the facial nerve (n. facialis), which innervates the circular palpebral muscle. In this disease the eyless open, and it waters; when attempting to close the the palpebral remains open. Keratitis, ulceration of the lop due to it. Ectropion of the lower eyelid leads lation and conjunctive. Besides the paralysis of the facial nerve lagophthalmos can be caused by congenital shortening of the eyelids, cicatrical 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 rising 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 Midler's muscle innervated by the sympathetic nerve takes part in rising 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 tomours 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 ihe 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 (hanov'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 (Waldeier'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 mediates 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 conjuntival 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 meimbomian 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 m general diseases, autoallergic conjunctivitises.
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, pres¬sure 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 envolves the whole families.or

organized groups of people. The disease, as a rule, affe'ets' both eves, 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 hemosis 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 agenl is Weikselbaum's pneumococc. Pellides which are easily taken off by moist cotton wool appear on the mucous membrane of eyelids. Ii pellicle conjunctivitis (conjunctivitis membranacea). Pneumococcal conjunctivitis can be mixed up with gonoblennorrhea or diph-of the eye. therefore bacteriological analysis is necessarv. Treatment of sharp conjunctivitis. First of all secretion should be carefully deleted, washing eyes once or twice a day b> % solution of boric acid, by furacillinum solution or kalii permanganas :5000. Sulfonamides give good results, especially in conjunctivitis, caused be Koch — Wicks' bacillus: sulfacylum solubile is used each hours as powder or % solution. In severe forms of the disease sulfonamides act quickly and effectively at internal use. At the acute conjunctivitises, caused by the Koch — Wicks" bacillus, pneumococcus, stafilococcus. there are widely used application of % gentamycin solution as drops (each -3 hours), .25% laevomycetin solution -8 times a day, normax. tobrex — 4-5 times a day. Overnight in a conjunctival sac it is put % tetracyclini or erythromycini ointment. Medical films with sulfonamides are applied.

Angular conjunctivitis is caused by Morax — Axenfelds diplo-bacillus, always bilateral, has an acute, even chronic clinical course. The characteristic reddening of lids edges of near external and in¬ternal 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 dis¬appeared, otherwise the relapse comes easily.

Gonoblennorrhea is inflammation of the conjunctiva, caused by the Neisser's gonococci, belongs to very serious diseases of eyes. As well as at gonorrheal urethritis, gonococci can spread outside a conjunctiva and cause generalisation of infection with all ib own distinctive phenomena and complications. Therefore in gonoblennorrhea gonitis, myositis, endocarditis, general indispositions and increase of temperature are possible. The incubation period lasts for 1-2 days, lust signs of the dis¬ease: eyelids swell up strongly, become firm so that it is impossi¬ble 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 gonoblennorrhea 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¬phyloma 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 gonoblennorrheal 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 veneral diseases. Prophylaxis of gonoblennorrheal 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 conjunc¬tiva, accreted with the surface of the cornea. As a rule, pterygium appears near the internal edge of the cor¬nea, 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 cav¬ities. They are inclined to subsequent diffuse spread, therefore should be removed.

Birthmarks (naevi) of the eyeball conjunctiva are non-pigman-ted as white spots and pigmanted — as brown-black spots. Birth¬marks 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 in¬nervation. Likable fibres get to the gland with vessels from inter¬lacement near an internal

carotid. The secret of the lacrimal gland is a tear (lacrimae) — is trans¬parent, 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 con¬cha.

The lacrimal puncta are two openings lying near the posterior margin of each lid, near the inner conthus. The lacrimal canaliculus joins the punctum to the lacrimal sac. It first passes vertically, then horisontally 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 lega-ment. The lower end continues as nasolacremal 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 ii can result in formation ol abscess and even phlegmon of the orbit. As a rule, the disease is pained 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 (antibio¬tics, sulfonamides, analgetics, antihistamins). Physiotherapy is used locally (dry heat, UHF-therapy), washing of conjunctival sac with warm antiseptics and anesthetics, application of ointments with an¬tibiotics and sulfonamides. At abscess dissection is performed.

Inflammation of the lacrimal sac — dacryocistitis. When there are inflammatory processes in the mucous of nasolacremal duct, and also different processes in the nose, for example, hyperplasia of mucous tunic, atrophy processes in it and nasolacremal duct, nasal septum deviation, formation of polyps, nasolacremal 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 product a secret, a sac is full of mucous, mucous-festering or only festering discharge. Chronic inflammation of the lacrimal

sac develops (dacryocistitis 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, gonorrhea 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¬fective agents, physical, chemical and mechanical factors. Anato¬mically the cornea borders are

closely connected with the conjunc¬tiva 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 cor¬rect 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 !% fluoresceinc solution, reflectivity test), investigation of the cornea thickness (keratopachymetry), investigation of the

cornea sensetivity (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)

Keratitises 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 corneal 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 cor¬nea 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 parabulbar injections of antibiotics of wide spectrum of action and their ointments before sleep are recommended. Also the pupil dilatation with homa-tropin (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, meibomiitis 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 sep¬arated 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 meibomiitis) 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 suppu-ration 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 punctuate haemorrhages. In 5-7 days there develops the mixed infection, the corneal syndrome, subepithelial multiple punctuate 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 punctuate opacities remain at the places of infiltrates.

Treatment.

1. Anti-viral preparations. They should be injected in drops, ointments, subconjunctival, parabulbarly, with electrophoresis, pho-nophoresis and intramuscularly. These preparations are interferon, laferon, deoxyribonuclease, poludan, kerecid, IDU, Zovirax, oxolinic, teobrofenic 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 parabulbarly; short active mydriatics should be injected in drops. Physiotherapeutic treatment (electrophoresis and phonophoresis with streptomicine, 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 parabulbarly 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-Iike 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, Bl, 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 keratomalatia. 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 keratomalatia the cor-neal 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 endophthalmia, panophthalmia 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 Bl 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, solcoseril 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 torfot 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);

— refractional — intralamellar for correction of refraction 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 muscules 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 live 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) dystriphies; 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 owning 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, lridoprothesis, 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 mydriaticts the pupil margin may assume a scalloped contour in cases where posterior synechiae have formed. If mydriaticts 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 mode-rately 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 spondilitis, 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 alministered 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.** Dispite 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 it's 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 transparant. 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 ol the visual acuit) 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.nbuv.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 № 2

Topic: Glaucoma. Mechanisms of regulation, methods of intraocular pressure research. Classification, clinic and treatment of glaucoma. Gradual and sudden decrease in vision. Cataract: congenital, acquired. Diseases of the retina and optic nerve

Pathology of intraocular pressure, research methods here, open-angle glaucoma, angle-closure primary glaucoma, mixed primary glaucoma, treatment of primary glaucoma, acute attack of glaucoma, secondary glaucoma, lens disease, retinal disease, optic nerve disease.

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.

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

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.
- 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:

- 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.
- examine the front part of the eye and the lens by biomicroscopy;
- 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**
	Preparatory stage:	
1	Determination of educational goals.	**3**
2	Providing positive motivation	**2**
	The main stage:	
3	Presentation of lecture material.	
	1. Pathology of intraocular pressure	**10**
	2. Research methods of IOP	**10**
	3. Open-angle glaucoma	**5**
	4. Primary closed-angle glaucoma	**5**
	5. Mixed primary glaucoma	**5**
	6. Treatment of primary glaucoma	**10**
	7. Acute attack of glaucoma	**5**
	8. Secondary glaucoma	**5**
	9. Diseases of the Lens	**10**
	10. Retinal disease	**5**
	11. diseases of the optic nerve.	
	The final stage:	
4	**Summary of the lecture, general conclusions.**	**5**
5	**The lecturer's answers to possible questions**	**5**
6	**Tasks for student self-training**	**5**

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: rigidness 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 fulfiling 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 sinechiae, pupil blokade);

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+l — 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-l — 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 mm-Vmin/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 mm-Vmin (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 resorbtion 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);

— enlargment 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 goniopunctura. laser surgery, sinusotrabeculotomy, 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 unfa-ble to IOP regulation factors, each of which doesn't cause .ma, 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, hypertonic desease, 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 cannel 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 imptoms are loss of visual acuity and visual field. At external examination the eye has normal appearance; only at a biomi-py it is possible to find distrophical 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, con-straction 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 arc: 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, ІОР, dark adaptation, fundus of the eye as in the open angle glaucoma.

Mixed** primary **glaucoma

At this form of glaucoma there arc simptoms *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 angl 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 °. 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 sings:

- 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 imposible 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 — openning 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, gonioplastic (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 goniopuncture and trabeculopexy 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. Dis-trophycal 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, elastotonometry, 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 refered 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

antiglau-comatic 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,** postinflamatory 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 symptomatica! 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. Enucliation of the eye or exzanteration 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.

DISEASES OF THE LENS

Anatomy of the Lens

The diseases of the lens такс about % of eve diseases. The lens (lens crystalling 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 accomodative capability. Without accomodation, 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 nucleous, 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, rubel-la, 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 opacitv 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 arc "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 resorlves. 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 11 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 reclination 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 Dav-iel, 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 vitreoum, ditach-ment 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 discission.

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, ditachment 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 poiymethylmetacrelate. 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).

RETINA DISEASE

Pathological conditions of the retina and optic nerve are often caused by cardiovascular, neurological and other diseases, as well as endocrine disorders, which calls for the need for joint coordinated treatment of such patients by an ophthalmologist and a doctor of any specialty. In addition, changes in the fundus are of great diagnostic and prognostic importance.

It should be especially noted that diseases of the retina, primarily its dystrophic lesions, are currently one of the main causes of blindness and visual disability, which indicates the need for early diagnosis and timely complex treatment, both by an ophthalmologist and by general practitioners .

The retina is the peripheral part of the visual analyzer. It develops from the front part of the medulla, so it can be considered a part of the brain, taken to the periphery. In it, 10 layers are distinguished, in which 3 specific visual neurons are located:

1. Rods and cones (cellula optica bacilliformis et coniformis).
2. Bipolar cells (neurocytus bipolaris).
3. Ganglion cells (neurocytus ganglionaris).

Rods have very high light sensitivity and provide twilight vision and peripheral vision, there are a lot of them (about 150 million), they are located along the entire periphery of the retina to the border of its optical part (ora serrata).

Cones are located mainly in the area of the central fovea of the macula, there are about 5 million of them. They provide form vision and color perception.

The first neuron lies on a layer of pigment epithelium connected to the choroid, which ensures the continuous renewal of molecules of visual pigments (rhodopsin and iodopsin) necessary for the photochemical process of the act of sight. Thus, the function of the retina is closely related to the state of the choroid itself.

The second neuron is associative.

The third neuron has long processes that form the optic nerve.

The internuclear layers of the retina consist of fibrous structures and form the core of the retina. Outgrowths of ganglion cells form the optic nerve, which exits the orbit through the optic foramen.

In the middle cranial fossa, in the region of the Turkish saddle, there is a partial crossing of the fibers of the optic nerves of both eyes (only the medial fibers are crossed). After the intersection, the so-called optic tract is formed, containing fibers from the retinas of both eyes. The subcortical center of the visual analyzer is the external geniculate bodies, and the cortical center is the spur groove in the occipital part of the brain (fissura calcarina).
The blood supply to the retina is carried out from the central retinal artery. The retina has no sensory innervation, so its damage does not cause painful sensations.
The normal fundus looks like this: the optic nerve disc is pink, its borders are clear, the retinal arteries and veins are of uniform caliber, the ratio of the caliber of the artery to the caliber of the vein is 2:3, there are no foci.
The diagnosis of retinal diseases is based on the data of ophthalmoscopy, fluorescent angiography, functional and electrophysiological studies (visual acuity, visual field, color perception, dark and light adaptation, electroretinography, electrical sensitivity of the optic nerve to phosphene).
Complaints of patients are non-specific and consist of impaired central vision (photopsia, metamorphopsia, reduced visual acuity, central scotomas, impaired color perception) or peripheral vision (restriction and loss in the field of vision, reduced dark adaptation).

Ophthalmoscopic changes consist of 4 elements:

1. Changes in the caliber, walls and course of blood vessels.
2. Hemorrhages of various shapes, sizes and prevalence.
3. Diffuse or local clouding of the retina, so-called foci.
4. Pigment deposits (spots, freckles).

Pathology of the retina is extremely diverse. Among diseases of the retina, the following main forms are distinguished:
1. Diseases associated with general diseases of the body.
2. Inflammatory diseases.
3. Dystrophic changes.
4. Detachment of the retina.
5. Neoplasms.
6. Congenital changes.

Let's focus on the most common retinal diseases that doctors of all specialties should be familiar with.

Many cardiovascular diseases lead to various changes in the fundus. Thus, with hypertension, these changes reflect the pathogenesis of vascular disorders occurring in the body, and have great diagnostic and prognostic significance. According to the classification of A.Ya. Vilenkinoi, M.M. Krasnov, distinguish:

- hypertensive angiopathy,
- hypertensive angiosclerosis,
- hypertensive retinopathy,
- hypertensive neuroretinopathy.

With hypertensive angiopathy, there is expansion, twisting of veins, narrowing of arteries, their uneven caliber. Observed at I-II A century. hypertensive disease.

With hypertensive angiosclerosis, copper and silver wire symptoms, Salus-Gunn symptoms I, II, III appear in addition to the above.

Symptom Salus-Gunn-I: conical narrowing of the vein on both sides of the artery at the point of their intersection, the vein takes on the shape of an hourglass. Symptom Salus-Gunn-II: at the place of the arteriovenous junction, the vein bends arcuately and squeezes into the retina. Symptom Salus-Gunn-III: the vein at the intersection becomes indistinguishable because it is covered by a swollen retina. They take place during the IIA-III centuries. hypertensive disease.

With hypertensive retinopathy, foci and hemorrhages appear in the retina, vision decreases. Observed in the 3rd century. hypertensive disease.

Hypertensive neuroretinopathy is an unfavorable prognostic sign. The optic nerve is involved in the process. There is swelling of the optic disc, hemorrhages and swelling of the retina around it. Visual acuity decreases, the field of vision narrows. Observed in the III century. hypertensive disease.

However, there may not be complete parallelism between the clinical course of hypertensive disease and the picture of the fundus.

Treatment. The main disease is being treated. In the case of retinopathy, resorption therapy (fibrinolysin parabulbarno), angioprotectors, antioxidants (emoxipin, dizinon, doxium) are added to it, and in neuroretinopathy, diuretics and osmotic agents are also used.

With **renal hypertension**, there is narrowing of arteries, dilation of retinal veins without pronounced sclerotic changes, with a large number of exudative foci and plasmarrhagia. A typical star shape in the macular region. This is a bad prognostic sign, as the old authors said - a "funeral bell" for the patient. Life expectancy with the appearance of such changes on the fundus is 1-2 years.

Diabetes is a common cause of severe damage to the retina called **diabetic retinopathy**. They consist in the appearance of microaneurysms, hemorrhages, exudative foci, in the terminal phase - the development of proliferative processes, the appearance of newly formed vessels, the growth of connective tissue, the development of secondary retinal detachment.

Treatment consists in the use of angioprotectors, absorbents, anabolic hormones. In recent years, photo- and laser coagulation, cryotherapy have been used. The prognosis is unfavorable.
The general vascular pathology of the body leads to the development of such retinal diseases as acute occlusion of the central retinal artery, thrombosis of the central retinal vein.

Obstruction of the central retinal artery is caused by spasm (50%), thrombosis (45%) or embolism (5%) of the artery. It occurs, in addition to patients with hypertension, in young people suffering from endocarditis, in particular rheumatic, chronic infectious diseases.
Complaints of the patient about a sudden loss of vision up to the sense of light. A sharp narrowing of the arteries, swelling of the retina, and the "cherry bone" symptom are determined on the fundus. Atrophy of the optic nerve develops as a result of the disease.
Treatment: vasodilators (0.1% atropine retrobulbar, intravenous nicotinic acid, euphylline, trental), thrombolytic agents, anticoagulants.
The prognosis is unfavorable. Treatment is effective when applied in the first 4 hours of the disease.
Thrombosis of the central vein of the retina - usually occurs in elderly people suffering from hypertension and atherosclerosis. Patients complain of a sudden sharp decrease in vision, but complete blindness does not occur. On the fundus of the eye, multiple hemorrhages, plasmorrhages, expansion and twisting of veins, discontinuity of their flow, swelling of the retina, blurring of the borders of the optic disc, the so-called symptom of "crushed tomato" are visible.
The prognosis for vision is poor, but more favorable than with central retinal artery occlusion. After resorption of hemorrhages, atrophic foci are formed in the retina, some patients develop secondary glaucoma.

Treatment: anticoagulants of direct and indirect action, thrombolytic and resorptive drugs.

Inflammatory diseases of the retina

These include metastatic retinitis, chorioretinitis. They arise as a result of the blood stream carrying microorganisms from any purulent focus.

Complaints depend on the localization of the process. Lesions of the central parts of the retina are accompanied by a decrease in visual acuity, the appearance of blurred vision. With peripheral localization of the focus, there may be no complaints.

The diagnosis is established during ophthalmoscopy. Yellowish-white foci with indistinct contours rising above the retina are visible on the fundus. Exudation may spread into the vitreous body.

As a result, atrophic chorioretinal foci develop.

Treatment: anti-inflammatory and resorptive therapy, comprehensive examination of the patient to establish the etiology of the disease.

Dystrophic changes of the retina

Dystrophic changes of the retina are divided into the following groups:

1. Pigmentary degeneration of the retina.
2. Juvenile dystrophies of the retina.
3. Senile dystrophies of the retina.

Pigmentary degeneration of the retina (PDS). The etiology has not been clarified. The disease often has a family-hereditary character with a recessive type of inheritance.

Complaints of patients: weakening and loss of vision at dusk (hemeralopia), then a narrowing of the field of vision develops, in the terminal stage - visual acuity decreases, up to complete blindness. Hemeralopia can also be observed with avitaminosis A as a result of its deficiency in food, but more often with impaired absorption in the gastrointestinal tract due to liver disease. When vitamin A is prescribed intramuscularly, twilight vision is normalized.

With PDS, pigment foci appear on the fundus from the periphery in the form of bone bodies, which later occupy the central parts. Retinal vessels narrow sharply. The disc of the optic nerve becomes paler with a waxy shade, in the terminal stage, its complete atrophy develops. The prognosis is unfavorable.

Treatment: vasodilator, metabolic drugs, vitamins, tissue therapy, hormones, anabolic steroids, revascularization operations, retroscleroplombage, physiotherapeutic treatment (ultrasound, phonophoresis, electrophoresis, electrostimulation by "phosphene").

Juvenile dystrophies of the retina distinguish many clinical forms that differ in the picture of the fundus and the nature of the clinical course.

Diseases have a family-hereditary character, are transmitted in a recessive or dominant type and differ in a steadily progressive course. Macular degeneration appears in preschool or school age, sometimes in adolescence. However, it should be remembered that macular degeneration in children can occur in the first year of life with diseases such as Tay-Sachs, Niemann-Pick disease.

Tay-Sachs disease (familial amaurotic idiocy) is characterized by:

 blindness with typical changes in the macula (a grayish-white focus with a "cherry stone" in the center), strabismus and nystagmus;

 retardation in mental development up to complete dementia;

 progressive muscle weakness.

Death occurs before two years.

With Niemann-Pick disease (reticuloendothelial sphingomyelinosis):

 grayish-white focus with a "cherry stone" in the macula;

 yellowish atrophic disc of the optic nerve;

 exophthalmos;

 enlargement of the liver, spleen;

 lag in mental and physical development.

Lethal exit up to two years.

Senile dystrophies of the retina are peripheral and central.

Peripheral dystrophies can lead to tearing and detachment of the retina. Cryopexy and laser coagulation are performed prophylactically.

Macular dystrophies are extremely common, according to various authors, their frequency is 15-29% among people over 50 years old. Complaints about a gradual decrease in vision, as a result, vision decreases to hundredths, central absolute scotoma appears. Clinically, 2 forms of sclerosing macular dystrophy are distinguished: "dry" and exudative-hemorrhagic. With the I form, there are atherosclerotic changes in the retinal vessels, deposits of lipids, cholesterol, hyaline in the retina (druses), depigmentation, and atrophic foci.

With the exudative-hemorrhagic course of the disease, a disk-shaped yellowish-white focus, surrounded by hemorrhages, appears on the fundus. In the next one, a focus radiating into the vitreous body develops, which must be differentiated from a neoplasm of the choroid (melanoblastoma), the so-called pseudotumorous focus.

Treatment: with "dry" macular dystrophy - physiotherapeutic methods of treatment, vitamin therapy, metabolites, vasodilators, antioxidants, revascularization operations, ligation of the temporal artery, retroscleroplating. With the edematous form - angioprotectors, antioxidants, resorption therapy, laser coagulation, cryopexy.

Retinal dystrophy is a predisposing factor to the development of **retinal detachment**, especially when the eye is stretched (with high myopia). Detachment of the retina can also occur under the influence of cicatricial changes in the vitreous body. Most often, its direct cause is an injury or physical overexertion. The development of retinal detachment is related to the fact that it is anatomically connected to the underlying tissue in only two places: at the serrated line of the flat part of the ciliary body and at the optic nerve disc.

Complaints of the patient about the appearance of flashes of light or "lightning flashes" (photopsia) on the periphery of the field of vision - in the area opposite the retinal tear. Then there is a sensation of a "curtain" approaching from the same side, from the periphery of the field of vision to its center, a narrowing of the field of vision occurs, most often from above.

During ophthalmoscopy, the detachment zone looks like a grayish bubble or sail, against which the retinal vessels look dark, and the tears look bright red.

Before hospitalization, such a patient must be kept in bed, preferably with a binocular bandage. Urgent hospitalization is indicated. Treatment is surgical. Scleral depression operations with diathermocoagulation or cryopexy are performed to develop scar tissue that holds the detached retina. In recent years, laser coagulation has been widely used in the treatment of retinal detachment.

Retinoblastoma (glioma) - a malignant neoplasm of the retina. Occurs in the first months or years of a child's life. In its course, 4 stages are distinguished.

I stage is initial. Limited tumor node in the retina.

II stage - germination in the eye cavity, in the corner of the anterior chamber. A characteristic symptom is "amavrotic cat's eye", the pupil is dilated, yellowish in color, intraocular pressure is increased.

III stage - growth of the tumor into the orbit. Exophthalmos may appear. The tumor grows rapidly and looks like a cauliflower.

IV stage - metastasis to distant organs, germination in the skull cavity.

Differential diagnosis is made with retrolental fibroplasia, in which the intraocular pressure is normal; with the result of purulent septic endophthalmitis, which is usually accompanied by

hypotony of the eye. The most informative methods of diagnosis: ultrasound, computer tomography, radioisotope research, less informative diaphanoscopy.
Treatment: in I and II stages - enucleation; in III and IV - exenteration of the orbit followed by X-ray and chemotherapy.
The prognosis is unfavorable.

DISEASE OF THE OPTIC NERVE

Pathological processes in the optic nerve are also numerous and extremely diverse. They can be congenital or acquired, have an inflammatory, allergic, degenerative nature, be associated with the pathology of the vascular system, tumors of the optic nerve, anomalies of its development are found.
The most frequent diseases of the optic nerve are neuritis and atrophy of the optic nerve.
The optic nerve is extremely sensitive to toxic substances, for example, nicotine, methyl alcohol, and many capillaries in the septa surrounding the bundles of optic nerve fibers create conditions for its impression of infection. Therefore, optic neuritis can develop as a result of general infectious processes, focal infection, inflammation of the orbit, diseases of the central nervous system, and various intoxications.
Subjectively, the patient feels a decrease in visual acuity to hundredths, and sometimes to light perception. The direct reaction of the pupil to light becomes sluggish, like the pupil in a healthy eye. A central scotoma appears, the field of vision narrows, especially for red and green colors.
During ophthalmoscopy, a sharply hyperemic and swollen disc of the optic nerve is visible, its borders are blurred. Venules of the retina are expanded, arterioles are narrowed. Hemorrhages may appear on the disk and around it.
Treatment: the patient is indicated for urgent hospitalization. Broad-spectrum antibiotics, sulfonamide drugs, desensitizing, dehydration, vitamin therapy, nasal tamponade with a solution of cocaine and adrenaline, endonasal electrophoresis with diphenhydramine, calcium chloride are used.
Inflammatory processes in the optic nerve can occur in the part behind the eye. This is the so-called retrobulbar neuritis. The most common causes of retrobulbar neuritis are influenza, sinusitis, intoxications, alcohol, nicotine intoxication, and multiple sclerosis. I want to draw your attention to the last one, because retrobulbar neuritis can be one of the earliest signs of this serious disease. Therefore, all patients with optic neuritis need a consultation with a neurologist.

Methyl alcohol, in addition to general severe poisoning, causes retrobulbar neuritis with subsequent atrophy of the optic nerve and incurable blindness. Taking even 30 ml of methyl alcohol inside can make a person go blind.
Patients complain of reduced vision, the appearance of a central scotoma, impaired color perception, especially red and green colors. Ophthalmoscopically, at the beginning of the disease, no changes are detected, in the next, after 14-20 days, atrophy of the optic nerve develops.
Treatment: as with optic neuritis, as well as detoxification therapy.

Atrophy of the optic nerve develops as a result of many diseases, when there is inflammation, swelling, compression, damage, degeneration of the fibers of the optic nerve or the vessels supplying it. Hereditary optic nerve atrophy with a sex-linked recessive type of inheritance occurs - Leber's optic nerve atrophy.
Ophthalmoscopically, pallor of the optic nerve disc, narrowing of the retinal arteries is determined. The boundaries of the disc may be clear (with primary atrophy).
Treatment: it is carried out only in case of partial atrophy, it is ineffective in case of complete atrophy. To improve the viability of preserved conductors, local parabulbar and general vasodilators, vitamin and osmotherapy, ultrasound, laser stimulation, electrostimulation with phosphene are used.
It is also necessary to pay special attention to such a concept as **a congestive disc of the optic nerve**. This is a symptom of increased intracranial pressure. Patients usually do not have ophthalmological complaints, they are more often bothered by headaches. Sometimes there are complaints of short-term blurred vision.
During ophthalmoscopy, the disc of the optic nerve is enlarged and mushroom-like explodes into the vitreous body. The vessels of the retina seem to rise on it. The disk acquires a grayish-pink shade, its boundaries are unclear, the veins are sharply expanded, twisted, there may be hemorrhages on the disk and in the retina.
Atrophy of the optic nerve develops with the long-term existence of stagnant phenomena.

Congestive disc of the optic nerve must be differentiated from neuritis. The main difference is the preservation of visual functions with a congested disc and their sharp decrease with neuritis. Neuritis is also characterized by the appearance of a central scotoma in the field of vision, with a stagnant disc - an increase in the blind spot. With neuritis, bulging into the vitreous body is less pronounced than with a stagnant disc.

Treatment is aimed at the underlying disease. After eliminating the cause of the stagnation, the ophthalmoscopic picture is normalized, if the development of optic nerve atrophy has not occurred. All of the above indicates the need to know the main ophthalmic symptoms of common diseases to improve the quality of diagnosis and treatment, as well as the ability to provide first aid to patients with diseases of the optic nerve and retina and to refer them to an ophthalmologist in a timely manner.

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. Diagnosis of age-related cataract.
6. Differential diagnosis of different forms of cataract.
7. Diagnosis and correction of aphakia.
8. 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.nbuv.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