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OPHTHALMOLOGY

MANUAL FOR FOREIGN STUDENTS OF HIGHER MEDICAL EDUCATIONAL INSTITUTIONS OF THE III-IV ACCREDITATION LEVELS

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The manual is recommended by Academic Council of Higher State Educational Establishment of Ukraine «Ukrainian Medical Stomatological Academy» as a textbook for English-speaking students of higher educational institutions of the Ministry of Health of Ukraine (minutes No. 9 of the meeting of Academic Council May 23, 2018).

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The present manual is intended to help English-speaking students of higher medical educational institutions in studying Ophthalmology, preparing for practical classes and passing the final module control in this subject. The material of the manual is presented in 17 chapters, in compliance with the curriculum for training medical professionals.

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CHAPTER 1

ANATOMY AND PHYSIOLOGY OF THE ORGAN OF VISION

Ophthalmology is a branch of medicine which focuses on physiology, anatomy of the eye, diagnostics, treatment and prevention of eye diseases. The name is derived from two Greek words: *ophthalmus* − "eye" and *logos* − "study". Ophthalmology has been developing throughout the history of mankind. As every part of medicine, it was conceived in the ancient social units of civilization.

With the help of visual analyzer, a person receives more than 80% of all information about the world around. Every doctor of any specialization must know the structure of the visual analyzer. Thorough knowledge of the clinical anatomy of the visual organ will help the student to understand a large number of pathologies of the appendages of the eye, the eyeball, its membranes and the optic nerve. The importance of this section of ophthalmology cannot be overstated for doctors of any specialization and especially for eye specialists. Preventing the diseases of the organ of vision, timely diagnosis and treatment should be in the centre of attention when training a doctor of any specialization. Testing the visual acuity, visual field, colour perception and adaptation allows the doctor to draw the right conclusions in the process of occupational selection, military and labour expertise.

The visual analyzer is a complex system that consists of:

- the eyeball,

- the accessory structures of the eye (eyelids, conjunctiva, lacrimal glands, lacrimal ducts, oculomotor apparatus),

- optic pathways (optic nerves, optic chiasma, visual tracts, visual centres − external geniculate bodies, central neuron (as part of the Gratiolet's bundle),

- occipital lobes of the brain.

The eyeball is located in the bone formation − the orbit.

The eyeball has a nearly spherical shape. Its diameter is 22-24 mm, weight − 7-8 g. The eyeball consists of three membranes: fibrous, vascular and the retina.

The outer, or fibrous, membrane of the eyeball is represented by a dense elastic tissue. It consists of the sclera, the cornea and the limbus. 5/6 of it is an

opaque part (the sclera), and 1/6 − a transparent part (the cornea). The site of transition of the cornea into the sclera is called the limbus. The fibrous membrane performs the protective, formative and turgor functions; oculomotor muscles are attached to it.

The sclera forms the posterior part of the outer membrane. It is white in colour and consists of dense collagen fibres. Most of the sclera is hidden in the orbit, and within the eye fissure, two white triangles on both sides of the cornea are visible. The optic nerve passes behind through the sclera. At the site of its exit, the sclera is thinned and has the appearance of a thin connective tissue plate with numerous openings through which the fascicles of the optic nerve pass (lamina cribrosa). The sclera passes into the cornea not all over the entire surface at once. The deeper layers pass into the cornea earlier, the superficial ones − later, so that the deep peripheral layers of the transparent cornea are covered from the surface by the transparent layers of the sclera. This semi-transparent formation is called the limbus. The sclera is a place where the outer oculomotor muscles attach. These areas are least resistant to eye injuries, especially blunt traumas; sclera ruptures often occur at this site. The sclera is nourished by the marginal vascular network, the vessels that pass through the sclera and give off small episcleral branches, and also by the diffusion of nutrients from the liquid entering the suprachoroidal space which can permeate the sclera. Therefore, the sclera, being poor in blood vessels, is less prone to diseases of metastatic origin.

The cornea forms the anterior part of the fibrous membrane. The cornea is transparent, shiny and smooth. It has a convex shape, due to which it performs the role of an optical lens. The cornea is a highly sensitive membrane. It is avascular and has a certain size. The thickness of the cornea is 0.6-1.0 mm, the size is 11-12 mm, the optical force is on average 42.0 dioptres. The cornea is not permanently protected by the eyelids and in this connection it can be subjected to mechanical and chemical damage. The cornea receives nutrition from 3 sources: the marginal vascular network, formed by the anterior ciliary arteries, located in the limbus, the humour of the

anterior chamber and lacrimal fluid. The main nutrition is provided by the circular vascular network along the limbus. Oxygen enters the cornea directly from the air.

The cornea consists of 5 layers: anterior epithelium, anterior limiting membrane (Bowman's membrane), the corneal substance proper, posterior limiting membrane (Descemet's membrane) and posterior epithelium or endothelium. *The anterior layer* (epithelium anterius) is represented by the multilayer, squamous non-keratinized epithelium. It regenerates well when damaged. Epithelium performs the protective function and regulates the intake of moisture in the cornea from the conjunctival cavity. *The anterior limiting membrane, or Bowman's membrane*, is inelastic and smooth. It has low metabolic activity and is not capable of regeneration. The bulk of it (about 90%) is occupied by *the cornea substance proper*, which consists of recurrent uniform plate-like structures. These structures include collagen plates, which are interconnected by interstitial substance. *The posterior limiting membrane* (lamina limitans posterior), or Descemet's membrane, is highly elastic and resistant to damaging factors. *Internal epithelium, or endothelium*, serves as a membrane which protects the stroma from impregnation with the humour of the anterior chamber. Epithelium does not regenerate when damaged.

The cornea has three types of sensitivity − pain sensitivity, tactile and thermal. The branchlets of nerve trunks, which innervate the cornea, do not have the myelin sheath.

The vascular membrane of the eyeball, or vascular tract, is the middle membrane of the eyeball which consists of 3 sections: the iris, the ciliary body, and the vascular membrane proper.

The iris is the anterior section of the vascular tract, visible through the cornea. It is the diaphragm of the eye, which regulates the flow of light into the eye, depending on illumination. In addition, the iris takes part in ultrafiltration and outflow of intraocular fluid, as well as thermoregulation. In the centre of the iris, there is a circular opening (the pupil). By virtue of two muscles − the sphincter and the dilator, which are in the iris, the pupil dilates and narrows under the influence of a different amount of light. The change in the pupil’s shape indicates an inflammation

of the iris or disorders of sympathetic and parasympathetic innervation. The iris also performs an aesthetic function − it ensures the colour of the eyes, depending on the amount of pigment in it.

The ciliary body is the middle section of the choroid of the eye. It consists of a ciliary muscle which participates in accommodation, and processes (cilia) that produce the intraocular fluid. The aqueous humour fills the anterior chamber of the eyeball (the space between the posterior surface of the cornea and the anterior surface of the iris and the lens), and nourishes the lens and avascular structures of the eye. There are short and long processes of the ciliary body. They attach to the sclera at different levels and form the ora serrata. Blood supply of the ciliary body is carried out from the branches of the large arterial circle of the iris, located in the ciliary body a little anteriorly to the ciliary muscle.

The vascular membrane proper (the choroid) is the posterior part of the vascular tract, and it is visible only in ophthalmoscopy. It makes up 2/3 of the total vascular tract. The choroid takes part in the trophism of the avascular structures of the eye, the outer photoreceptor layers of the retina and in the maintenance of normal ophthalmotonus. The choroid is formed by the posterior short ciliary arteries, and consists of vessels of different calibres. The choroid has no sensitive innervation. Diseases of the choroid are usually combined with retinal diseases. They are manifested in the disorders of blood supply, dystrophy, and inflammation.

The retina is the inner membrane of the eyeball. It is a kind of "window into the brain" and a peripheral link of the visual analyzer. The retina is part of the brain that has separated from it in the early stages of development, but is associated with it through the optic nerve. Histologically, the retina consists of ten layers. The retina has only three histological layers in the central department (the macular zone). It perceives photostimulation and turns it into a nerve impulse, which through the visual pathway reaches the occipital lobe of the cerebral cortex where the visual image is formed. The retina is divided into two sections − *optically active* (lining the entire inner surface of the choroid up to the ora serrata) and *optically inactive* (from the ora

serrata it continues to the ciliary body and the iris). The central zone of the retina consists of nerve cells represented by three neurons:

- the first neuron − rods and cones;

- the second neuron − the bipolar cells, connecting the nerve cells of the first and third neurons;

- the third neuron − the ganglion cells. The axial cylinders of these cells from the entire retina gather in the optic disc and form the optic nerve. Blood supply to the retina is due to the central artery, the central vein of the retina and the vascular membrane. The branches of the central artery of the retina (CAR) have no anastomoses and complete obturation of one of them leads to irreversible degenerative processes of the corresponding retinal sector. The retina is attached to the frame of the eyeball in two places: around the disk of optic nerve (DON) and along the ora serrata (the border between the ciliary body and the iris).

Transparent intraocular media. Internal structures of the eye consist of transparent light-refractive media: the vitreous body, the lens and the aqueous humour, filling the eye chambers.

The anterior chamber (camera anterior) is a space bounded by the cornea from the front, by the iris from behind, and by the lens in the pupil area. The depth of the anterior chamber reaches on average 3-3.5 mm. In pathology, both the depth of the chamber and its irregularity acquire the diagnostic value.

The posterior chamber (camera posterior) is located behind the iris, which is its front wall. The outer wall is the ciliary body, the posterior one is the anterior surface of the vitreous body.

The lens is a biconvex transparent elastic structure. The optical force is 16-18 dioptres. It is located behind the pupil and the iris. The anterior membrane of the vitreous body is adjacent to the posterior surface of the lens. The lens has no vessels and nerve endings, and therefore, it is not predisposed to inflammatory diseases. The lens has a capsule. It is retained with the help of ciliary zonules, which are attached to the equator of the lens. The lens, together with the ciliary zonules, forms a septum (diaphragma oculi), dividing the cavity of the eye into two unequal parts − the

smaller one, the anterior, and the larger one, the posterior. With age, the elasticity of the lens decreases. This manifests itself beginning from the age of 40-45, and leads to the need to use glasses for work at a short distance. The pathology of the lens is expressed in its opacity (cataract) and in the change of its position (dislocation, subluxation).

The vitreous body (corpus vitreum) is a part of the optic system of the eye, filling the cavity of the eyeball. The vitreous body performs the shaping and optical functions. The volume of the vitreous body in adults is 4 ml. It consists of a set of thin, gentle fibrils and loose substance. The vitreous body is fixed in the region of the flat part of the ciliary body and near the optic disc. Outside, it is covered with a hyaloid membrane. It is 98% water and 2% protein and inorganic salts. The vitreous body has no vessels and nerve endings. It is nourished by the vessels of the choroid and the retina.

The optic nerve is a segment of the optic pathway. It begins from the processes of the ganglion cells of the retina, which form the disc of the optic nerve, and ends in the chiasma (after incomplete decussation). The optic nerve is divided into bulbar (intraocular) and retrobulbar (extraocular) parts. The retrobulbar part of the optic nerve consists of the orbital, canalicular and cranial (intracranial) divisions. The length of the orbital part is 4.5-5 cm.

The orbit is a bone structure of a pyramidal shape, containing the eyeball, the optic nerve, external oculomotor muscles, the ophthalmic artery and vein, all motor nerves of the eye, and the first branch of the trigeminal nerve. The length of the orbit is 4-5 cm, the height in the entrance area is 3.5 cm, and the width is 4 cm. The eye socket has 4 walls: internal, external, superior and inferior, three of which share borders with the paranasal sinuses. The internal wall is the thinnest and the most complex. The orbit joins with the maxillary, frontal, ethmoidal sinuses and with the cranial cavity. The inferior wall is formed by the zygomatic bone, the maxilla, the orbital process of the palatine bone. It verges on the maxillary sinus, which makes it possible for the inflammatory processes to spread to the tissues of the orbit. *The inferior wall* is often subjected to blunt injuries, which lead to the shift in the ocular

block to the bottom. The superior wall is formed by the frontal bone and the lesser wing of the sphenoid bone. There is a fossa of the lacrimal gland in the frontal bone from the outside and from above. *The superior wall* of the orbit verges on the cranial cavity. *The internal wall* is formed by ethmoid bone, lacrimal bone and the anterior part of the sphenoid bone. This wall is the thinnest one in the orbit. It is often damaged in blunt injuries which can lead to emphysema of the eyelids and orbit.

The external wall is formed by zygomatic bone, frontal bone and the greater wing of the sphenoid bone. This wall of the orbit is the densest.

In the walls of the orbit, there are apertures and fissures through which large vessels and nerves pass into the orbit cavity.

The optic canal (canalis opticus) is a bone channel with a round aperture through which the optic nerve (n. opticus) and ophthalmic artery (a. ophthalmica) pass.

The superior orbital fissure (fissura orbitalis superior) is located between the greater and lesser wings of the sphenoid bone. All oculomotor nerves, the first branch of the trigeminal nerve pass through the superior orbital fissure, whereas the superior opththalmic vein (v. ophthalmica superior) exits the orbit through it. When the superior orbital fissure is damaged, the symptomatic complex develops − ptosis (lower eyelid droop), mydriasis (dilated pupil), ophthalmoplegia totalis (absence of the eyeball movements), exophthalmos (eyeball bulging out of the orbit), tactile sensitivity disorder.

The inferior orbital fissure (fissura orbitalis inferior) is formed by the greater wing of the sphenoid bone and the maxilla. The fissure is closed by the dense fibrous membrane, including smooth muscle fibres. The lower infraorbital nerve enters the orbit and the infraorbital vein exits the orbit through it.

Blood supply of the orbit is provided by the ophthalmic artery. The main venous vessel is the superior ophthalmic vein. **Innervation** is due to the ciliary ganglion. It is located 7-8 mm behind the eyeball above the lateral rectus muscle and adjacent to the optic nerve. Its size is about 2 mm.

The muscular (oculomotor) apparatus provides movement of the eyeballs (external muscles). It consists of 4 rectus and 2 oblique muscles, elevator muscle of upper eyelid, the orbicularis oculi muscle that takes part in closing of the palpebral fissure. The external muscles of the eyeball are innervated:

- rectus muscles (superior, inferior, internal) and the elevator muscle of upper eyelid − by the oculomotor nerve;

- the lateral rectus muscle − by the abducens nerve (n. abducens).

- the superior oblique muscle − by the trochlear nerve (n.trochlearis).

The eyelids, the conjunctiva, the lacrimal organs, the lacrimal passages and the retrobulbar adipose tissue belong to **the accessory apparatus of the eye**.

The upper and lower eyelids are the complex anatomical structures, which protect the front surface of the eyeball. Owing to the blinking movements, they uniformly distribute the tear along the anterior surface of the eye, moistening the cornea and the conjunctiva. The upper and lower eyelids are connected by medial and lateral adhesions. In the inner angle, a recess − the lacrimal lake − is formed. The lacrimal caruncle is located at the bottom. The almond-shaped space between the open eyelids is called the *palpebral fissure*. The eyelids consist of two plates − the skin-muscular (external) and cartilage- conjunctival (internal). The skin of the eyelids is thin, it readily wrinkles, the subcutaneous tissue is loose, devoid of fat. Therefore, oedema and haemorrhage easily occur in the area of the eyelids by direct damage or pathological processes nearby the eyelids. The cartilages of the eyelids are slightly convex connective tissue plates. The eyelids contain a large number of sebaceous glands (meibomian glands). They are arranged in parallel rows, and their excretory ducts open into the intramarginal space of the eyelids (between the external and internal rib of the eyelids). The lipid secretion of these glands lubricates the intercostal space of the eyelids and does not let the tear roll down over the edge of the eyelids. The cartilage is tightly connected with the mucosa − conjunctiva. Dysfunction of the meibomian glands can cause stye (hordeolum), chalazion, blepharitis. The muscle of the eyelid comprises the orbicular muscle of the eyelids, elevator muscle of the upper eyelid, Riolan's muscle (narrow muscle strip on edge of

the eyelid at the root of the eyelashes) and Horner's muscle (muscle fibres of orbicular muscle covering the lacrimal sac). The orbicular muscle of the eyelid is innervated by the facial nerve. In paresis or inflammation of the facial nerve lagophthalmos (the inability to close the eyelids) occurs, which may lead to corneal dryness (xerosis) and adjunction of infection.

The elevator muscle of upper eyelid starts near the optic canal, goes under the upper part of the orbit and ends with three muscular fascicles. The outer fascicles is woven into the skin of the eyelid, the middle − into the thickness of the cartilage and the inner one − into the arch of the conjunctiva. Blood supply of the eyelids is carried out by the branches of the ophthalmic artery, included in the system of the internal carotid artery. Arteries correspond to veins which carry the venous blood toward the angular vein, the vein of the lacrimal gland and the temporal superficial vein.

The conjunctiva is a connective tissue transparent membrane. It is divided into three parts − the conjunctiva of the eyelids, the eyeball and transitional fold (the arch of the eyelids). The palpebral part covers the posterior surface of the eyelids. The conjunctiva of the eyeball reaches the limbus. The conjunctiva of the arch is a place of transition of the conjunctiva of the eyelids into the conjunctiva of the eyeball. The conjunctiva of the eyelids consists of columnar epithelium, which has a large number of goblet cells, producing mucus. Glands of Wolfring are located in the conjunctiva of the eyelids. The conjunctiva of the transitional folds contains the accessory lacrimal glands (Krause's gland) and lymphoid tissue. The conjunctiva of the eyeball is thinner. It is composed of stratified squamous non-keratinizined epithelium. Blood supply of the eyelids is due to two vascular layers − the superficial and profound. The superficial layer is formed by vessels that extend from the arteries of the eyelids and anterior ciliary arteries. The profound layer is made of branches of anterior ciliary arteries, which form the circular vasculature around the cornea. Veins correspond to arteries, which carry blood to the veins of the face.

Lacrimal apparatus. Tears constantly moisten the cornea and the conjunctiva. Lacrimal apparatus consists of:

- lacrimal apparatus (lacrimal gland and accessory lacrimal glandules (glands of Wolfring and Krause).

- lacrimal passages.

Lacrimal gland is located in the upper-outer portion of the orbit. It is divided into two unequal parts − the orbital and palpebral, which are connected with each other by a narrow isthmus. The orbital is larger than the palpebral one. The orbital part of the gland is not detectable through the skin, as it is located behind the bone edge of the socket. In the conditions of enlarged gland (e.g., tumour, oedema or eyelid droop), its palpation becomes possible. *Palpebral, or eyelid, part of the lacrimal gland* is located directly above the upper arch of the conjunctiva. In the condition of disjointed upper eyelid and the eye turning inwards and downwards, this part of the lacrimal gland is normally visible as a slight protrusion of yellowish lumpy mass. In the inflammatory conditions of the gland (dacryoadenitis), an expressed protrusion is found at this location due to oedema and densifying of the glandular tissue. The enlargement of the lacrimal gland can reach the extent of dislocating the eyeball. The tear, secreted by the glands, moisturizes the anterior surface of the eyeball. Lacrimal gland starts functioning by the third month of life. Prior to this, the children are crying without tears.

Lacrimal system starts with lacrimal points which pass into the lacrimal ducts, lacrimal sac and nasolacrimal canal, that opens into the inferior nasal concha.

Lacrimal points (punctum lacrimale) are the initial openings of the lacrimal apparatus. Lacrimal points are located at the posterior edges of the free margin of both eyelids, the upper one is approximately 6 mm, and the lower one is 7 mm from their internal adhesion. Lacrimal papillae face the eyeball and are almost adjacent to it, whereas the tear points are immersed into the lacrimal lake, at the bottom of which the lacrimal caruncle (caruncula lacrimalis) lies.

Lacrimal sac (saccus lacrimale) composes the upper, extended part of the nasolacrimal duct. Topographically it refers to the orbit and is placed in its medial wall in the bone recess − the fossa of the lacrimal sac. Lacrimal sac is the membranous tube 10-12 mm long and 2-3 mm wide. Its upper end terminates blindly.

This place is called the fornix of the lacrimal sac. Downwards, the lacrimal sac narrows and passes into the nasolacrimal duct. The passage of lacrimal fluid through the lacrimal passages normally lasts about 10 minutes. Approximately such an amount of time is required for 3% colloid silver or fluorescein 1% from the lacrimal lake to reach the lacrimal sac (5 min − tubular test) and then the nasal cavity (5 min − positive test probe).

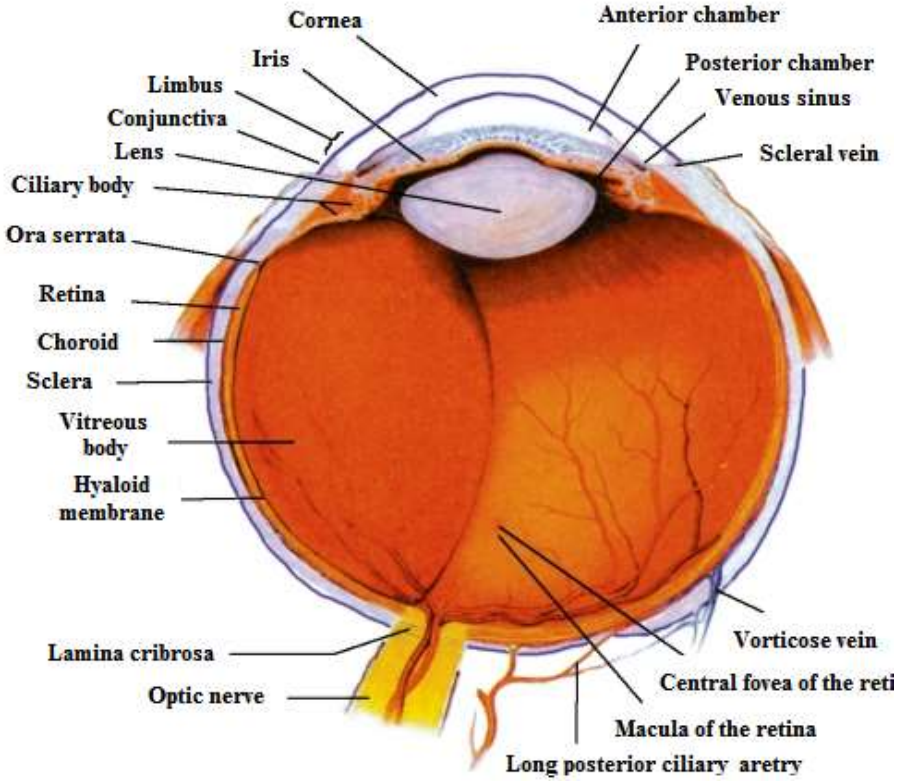


Fig. 1.1. Sagittal section of the eye.

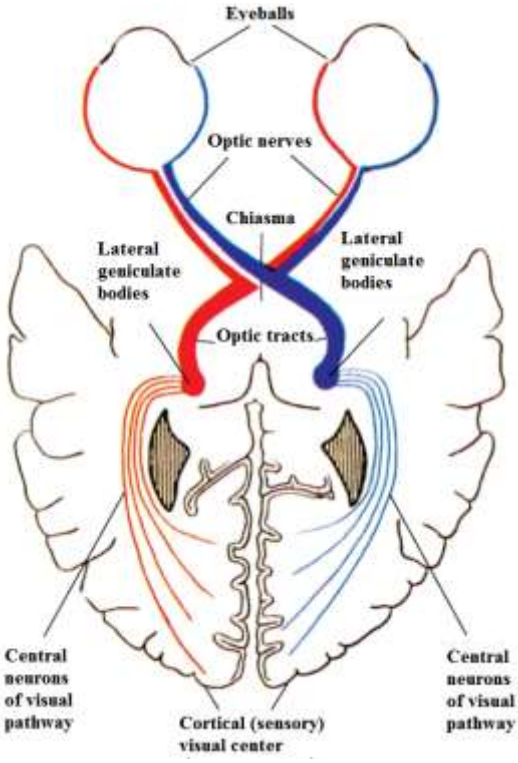


Fig. 1.2. The structure of the visual pathways.

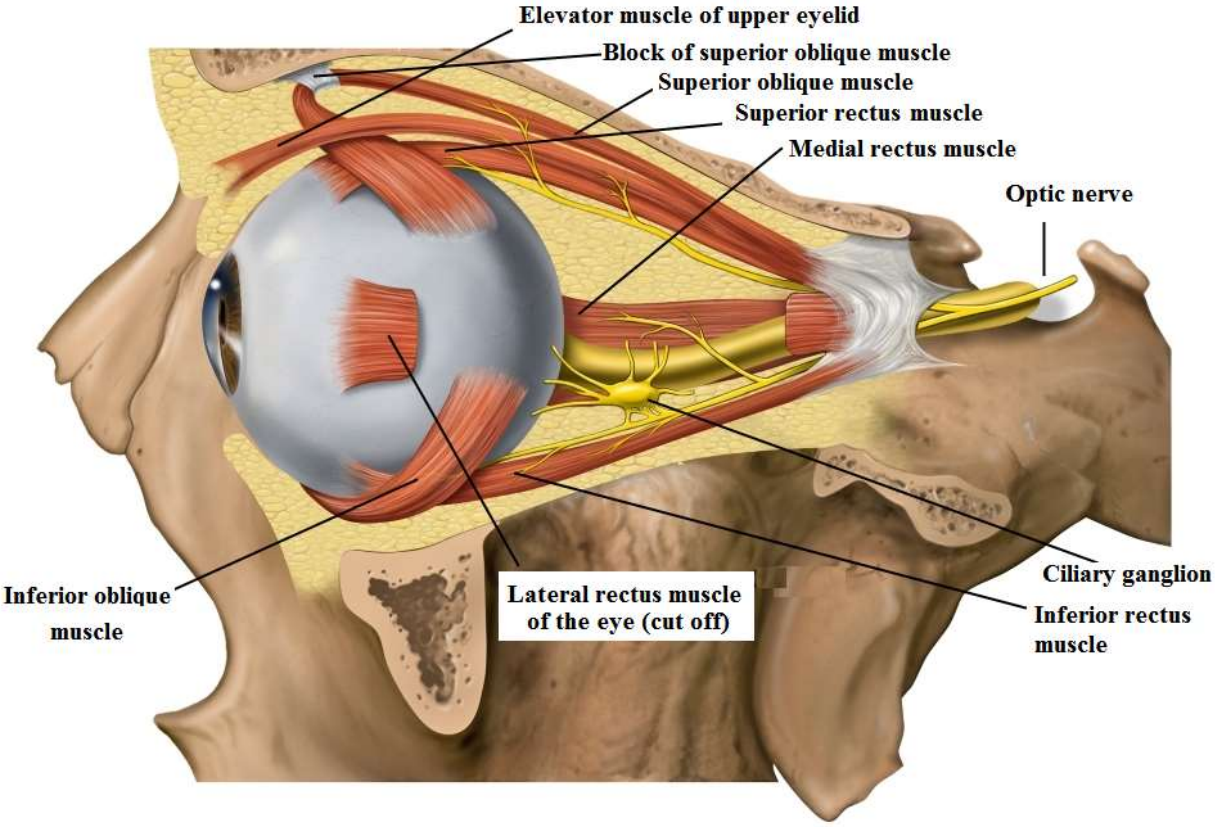


Fig. 1.3. The orbit and oculomotor muscles.

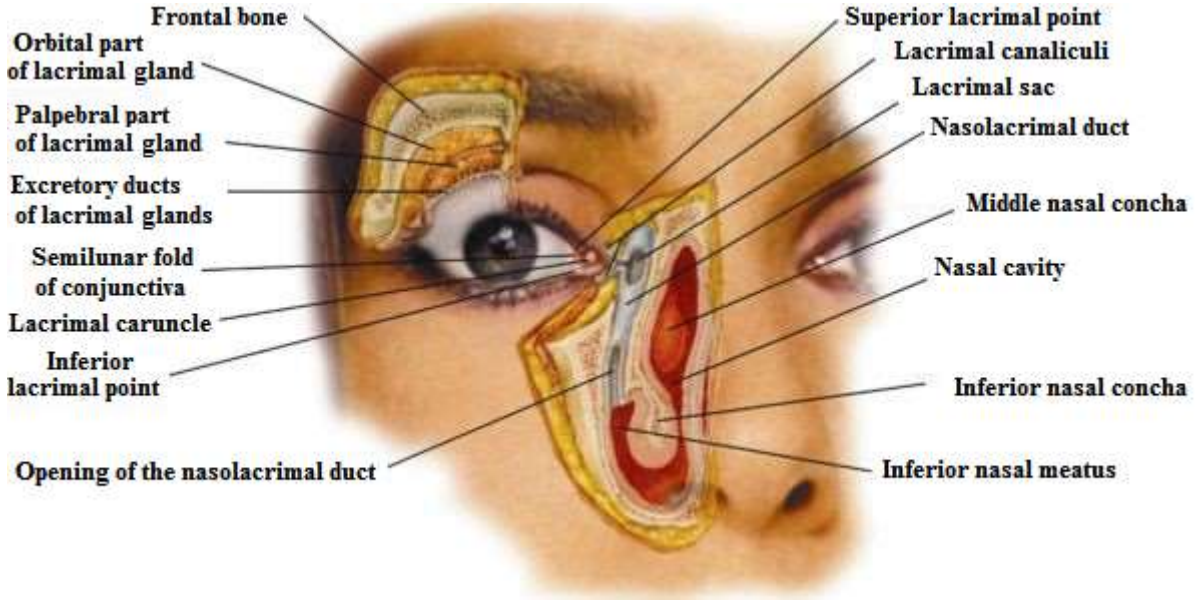


Fig. 1.4. The structure of the lacrimal apparatus.

CHAPTER 2

VISUAL FUNCTIONS

Visual analyzer is a single system consisting of three divisions:

1) peripheral, or receptor division;

2) conductor division with intermediate nerve centres;

3) cerebral, or central, division which is located in the cerebral cortex.

Rods and cones are the photoperceptive components of the retina and are located in different parts irregularly. Rods are located on the periphery of photoreceptor layer of the retina. Their total amount is 120 mln. Cones are found in the central parts of the retina. Their total amount is 7-8 mln. Both rods and cones contain the light-sensitive pigments.

Vision comprises five visual functions:

1. Central vision,
2. Peripheral vision,
3. Colour perception,
4. Photoreception (scotopic vision)
5. Binocular vision.

The list of key terms, parameters and characteristics which students should master while studying the topic:

Term	Definition
1. Visual functions	Central vision, peripheral vision, colour perception, photoreception, binocular vision
2. Field of vision	The space, perceived by the motionless eye
3. Scotoma (blind spot)	The portion of the field of vision, which falls out
4. Hemeralopia	Disorder of twilight vision
5. Membranes of the eyeball	Fibrous, vascular, net
6. Visual acuity	The ability to perceive two separate points that are spaced apart at a minimum distance
7. Adaptation	The ability of the eye to perceive the colours of

	different brightness
6. Normal trichromatism	The eye's ability to perceive three primary colours of the spectrum

Central vision provides a clear view of objects. Cones of the retina are responsible for central vision. Central vision is characterized by visual acuity. Visual acuity is the ability of the eye to distinguish two separate points located at a minimum distance from each other. Visual acuity is determined by means of special charts (S.S. Golovina, D.A. Sivtseva, V.E. Shevaleva, E.M. Orlova − this is the **subjective method**) in which the letters (optotypes) have different sizes. Optotypes are arranged in 10-12 rows. The principle of chart composition consists in arithmetic and geometric progressions. In each row, the size of the optotypes is the same, but it is constantly decreasing from the top row to the bottom. The testing is carried out in a well-lit room. The patient sits at a distance of 5 meters from the chart, their eyes are on the level of the middle of the field test, the head is straight. The table is illuminated by 60W lamp in mirror environments (Roth apparatus). The study begins with the right eye, the left eye is covered with opaque white plate. The testing begins with large optotypes of the chart. An optotype is showed for 2-3 seconds, then proceeding to the next one. The patient is asked to name and show optotypes from the first rows of the chart (2 outermost and 1 middle mark). If all signs are identified correctly, the smaller ones are shown. This continues until the patient makes a mistake. Visual acuity is scored by string of the smallest signs that the patient named correctly, or with an acceptable number of errors. Each row corresponds to a certain visual acuity, indicated next to it: first − 0.1; fifth − 0.5; seventh − 0.7, etc. The norm is considered to be reading line 10, which corresponds to the visual acuity of 1.0. If in lines 5-6 one letter is improperly named, and in lines 8-10 − 2 letters, one uses the concept of incomplete visual acuity of the corresponding row. If the patient does not distinguish even the first line (visual acuity is less than 0.1), then the doctor determines the distance from which the patient reads the optotypes of the first line. For this purpose, the patient is led to the chart until he/she sees the first line and this

distance is measured. Visual acuity is calculated by Snellen's formula: VIS = d / D (where "d" is the distance from which the patient sees the letter, D − the distance from which the normal eye sees this line). The distance D is indicated in the chart s next to each row. For the first line it is 50 m, for the second − 25 m, etc. It is also possible to use different charts with split optotypes (optotypes of B.L. Polyak), approximating to the patient, while distance D and the corresponding visual acuity is specified on the chart with optotypes. It is possible to demonstrate a different number of fingers, equating their size to the optotypes of the first row in the Golovin-Sivtsev chart and using the same Snellen's formula for calculation.

Thus, it is possible to calculate the visual acuity until 0.005. Further, it is expressed as the distance from the object. For example, the score of fingers from 20 cm distance; from 10 cm; near the patient's face.

If it is impossible to test the subject vision, that is, the patient does not see the optotypes of the charts or doctor's fingers, one needs to determine the presence and projection of light perception.

An objective method for determining the visual acuity is based on the occurrence of involuntary optokinetic nystagmus when looking at moving objects (nystagmus apparatus).

Peripheral vision. It allows a person to orient in space. It is characterized by a field of vision − this is a set of all points in space that are perceived by the motionless eye. The peripheral vision is provided by rods of the retina, which are located on its periphery. Methods for testing the field of vision are as follows: perimetry, campimetry, control method.

Perimetry (projection type, Forster's perimeter) is the method for testing the field of vision on a spherical surface in order to measure its boundaries and identify defects therein. White objects with the diameter of 3 or 5 or 10 mm are used − depending on the patient's visual acuity for measuring external boundaries and a diameter of 1 mm for identifying scotoma. For the study of the field of vision by colour, red, blue and green objects with the diameter of 5 mm are used. The testing is carried out in a dark room; adaptation is 3-10 minutes. The patient places the head on the frontal-chin

support of the perimeter; one eye is closed with a dressing that does not limit the field of vision of another eye. The examined eye is located in the centre of the perimeter arc, which is achieved by gradual movement of the frontal-chin support and head rest. The patient is asked to look still at the fixation point in the centre of the arc. By drum turning, the light object is transferred to the periphery of the perimeter arc; slowly rotating the drum, the object is moved to the centre at the rate of 2-3 mm/s by the degree scale; the moment is recorded, as soon as the patient notes the appearance of an object in the field of vision with the words "I see", and if on the path of its movement the object disappears or becomes less clear. The object is then transferred to the other edge of the arc and the study is repeated. The field of vision is consistently measured in 8-12 meridians at intervals of 45° or 30°. To determine the boundaries of the field of vision for white colour, the objects with the diameter of 3 mm are used, and to measure the central visual field defects − objects sized 1 mm. Perimetry for colours is conducted using the objects sized 5 mm in diameter (the borders of field of vision for colours are determined when the patient recognizes the colour correctly). One can also use the object on the pointer, by moving them along the perimeter arc. Combining these records in the formsheet with the field of vision scheme, one can determine the peripheral borders of the field of vision. Absolute scotoma in the scheme is marked with a solid colour, whereas the relative ones are bar shaded. Normal boundaries of visual field to white are: outside − 90°, upwards − 50-55°, inwards − 55°, downwards − 65-70°.

Loss of the visual field portion is called **scotoma**. The key features of scotoma are as follows:

- physiological and pathological,
- relative and absolute,
- positive and negative.

Physiologic scotoma is a blind spot, angioscotoma.

Blind spot is a projection in the space of the optic nerve disc. It is located in the temporal half of the visual field by 12-18˚ from the fixation point. Its diameter vertically is 8-9˚ and horizontally – 5-8˚.

Campimetry is the method of measuring the boundaries of the central parts of the visual field on a flat surface and defining therein visual function defects (it is used for studies of the visual field portions within 30-40° from the centre, in order to determine the sizes of the blind spot, the central and paracentral scotoma). The testing is carried out in a bright room with good natural light. The patient is seated back to the light at a distance of 1 m from the screen, the head is still, the eyes are on the level of the mid-plane. One eye is covered with a soft bandage. The doctor, preferably in a black coat and with black oversleeves, is next to the screen, from the side of the examined eye. The patient fixes with this eye the mark in the centre of campimeter. White objects sized from 1.5 to 10 mm, mounted on the long black rods, are slowly moving from the centre to the periphery along the meridians of the black screen. The doctor marks the points where the object disappears with the chalk or a pin. The results are transferred to the paper. Dimensions of scotomata are expressed in angular degrees. In normal conditions, physiological scotoma ("blind spot") has the form of an oval, which is located in the temporal half of the visual field between 12 and 18°. Vertical diameter of "blind spot" is to 8-9°, the horizontal − 5-6. 1/3 of the "blind spot" are located above the horizontal line passing through the centre of campimeter and 2/3 − below this line.

Amsler grid is used to assess the function of the central division of the retina (macula) or to establish the presence of the central and paracentral scotomata. Amsler grid (Fig.) consists of 5 squares * 2 cm. From a distance of 30 cm, each square is seen at an angle 1°. The grid has 20° in width and height. The testing is carried out in a well-lit room after the correction of refractive anomalies. The patient is asked to close the left eye and keep an Amsler grid at a distance of 30 cm in front of the right eye. The patient is asked what he/she sees in the centre of the page. Inability to see the central spot indicates the presence of central scotoma. When the patient fixes glance on the central spot (or on the centre of the page, if he/she does not see the spot), the doctor finds out if the patient sees all four corners of the chart, or some squares are absent.

When fixing glance on the spot, the patient is asked, whether all the lines are straight and not interrupted, or somehow twisted and broken. The patient is asked to circle any missing or curved sections on the grid with a pencil. Areas with uneven perception of Amsler grid are called metamorphomas (rough, winding, non-parallel lines). The procedure is done once again for another eye.

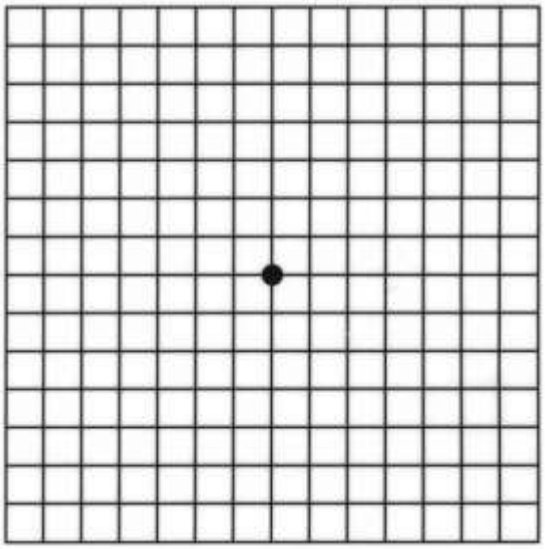


Fig. 2.1. Amsler Grid

The control method for determining the field of vision consists in comparing the patient's visual field with that of a doctor (which is considered to be normal). The patient is at a distance of 1 meter away from the doctor and closes one eye. Doctor, respectively, closes the opposite eye. The patient and the doctor look at each other's nose (fixing glance). The doctor then takes an object, and in the two main meridians (horizontal and vertical) moves it from the periphery to the centre. It is necessary to ask the patient whether he/she sees the object all the time, or if it falls out somewhere. This method makes it possible to identify the areas of visual field loss in patients.

Colour perception. With the help of colour perception we are able to discern the entire spectrum of colours of nature. The colour perception is provided by the retinal cones, which are located in the central zone of the retina. The eye normally sees the three primary colours of the spectrum: green, red and blue (*normal trichromatism*). Such people are called normal trichromats, and also have the ability to perceive up to

160 different colours. All variety of colours is perceived by mixing the three primary colours. Achromatic colours include white, gray and black. All achromatic colours are characterized by colour hue, brightness, i.e., the degree of their closeness to the white colour. Chromatic colours are characterized by brightness and saturation. There are three colour groups: 1) long-wave − red and orange; 2) medium-wave − yellow and green; 3) short-wave − blue, dark blue and purple. Outside of the chromatic spectrum, there are placed long wave, invisible to the naked eye − infrared, and shortwave − ultraviolet radiation. Colour perception is tested using *Rabkin's polychromatic tables*.

The patient is sitting with his/her back to the window, and is offered to name signs, which he/she sees keeping the table at a distance of 1 m at the level of the eyes. Exposure of each table is about 2-3 seconds. Answers are recorded in the test card, and then analyzed in accordance with the diagnostic table, located at the end of the book. If the patient gives vague answers, or does not see the individual figures and does not read XXIII, XXIV, XXV and XXVI of tables ("36", "14", "9" and "4"), it indicates the acquired colour vision disorder. The data obtained for each test are written to create a personalized card and are consistent with the instructions that are in the appendices to Rabkin's tables. Thus, the individual card is filled with responses according to the scheme: the number of image, the correct answer − "+", wrong − "-", doubtful − "?". The diagnosis is made according to the recommendations of the table for diagnostics of colour vision disorders, located in the appendix to the book by E.B. Rabkin.

Disorders of colour sensitivity may be manifested in abnormal perception of colours, called colour-anomaly, abnormal trichromatism or complete loss of one of the three components − dichromatism. In rare cases, there is only black and white perception − monochromasia.

Light perception (twilight vision). It provides the ability to see at low light and at dusk. At dusk, we do not distinguish colours ("at night all cats are gray"). Disorder of twilight vision can be congenital, resulting from vitamin deficiency or disease. This disorder is called hemeralopia.

Light perception is provided by the rods apparatus of the retina. The study of light perception: control samples, adaptometry. The simplest test is to monitor the actions of the subject in a dark room. It is possible to conduct Kravkov-Purkinje test. At the corners of a black piece of cardboard, sized 20x20 cm, 4 squares 3x3 cm in blue, yellow, red and green paper are pasted. The coloured squares are showed in a dark room at a distance of 40-50 cm from the eye. Normally the yellow square becomes distinguishable, then the blue one. In pathological photoreception, there is a bright spot in place of the yellow square, and the blue square remains invisible to the patient.

The light adaptation occurs within the first few seconds, and then it slows down and ends at the end of the first minute. The dark adaptation is slower: it increases for 20-30 minutes, and only by 50-60 minutes the maximum adaptation is achieved.

Binocular vision is the ability to see with two eyes. It is formed at the age from 2 months to 6-10 years and becomes stable by 15 years. For binocular vision, certain conditions are required:

- visual acuity not less than 0.3-0.4:
- full range of motion of the eyeballs;
- parallel position of the eyeballs when looking into the distance;
- corresponding convergence when looking at close range;
- the ability of fusion;
- getting the image on the correspondence points of the retina.

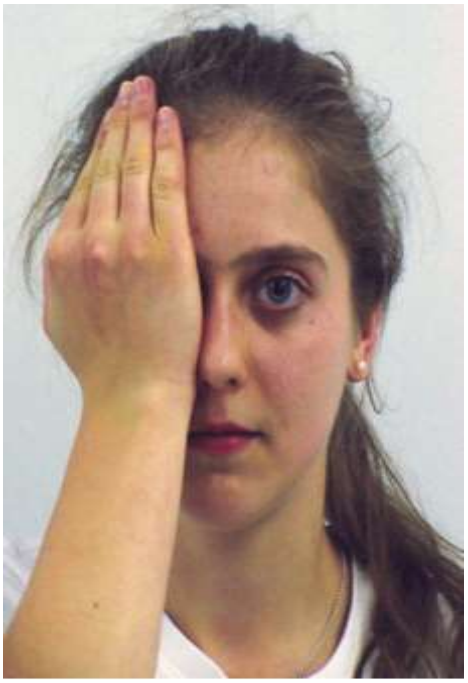
Examining the binocular vision − Sokolov's test.

This phenomenon can be explained by the fact that each eye has its own image and they are superimposed on each other. Hence, if one keeps a tube in front of one eye, and another eye is covered with hand, the image that we see through the tube opening is superimposed on the image of the palm in another eye. In the simultaneous vision, the "hole" does not coincide with the centre of the palm, and in the monocular − the phenomenon of the "hole" in the palm of the hand does not appear.

A patient holds a tube with one hand (for example, a folded sheet of paper) in front of the right eye. He/she puts the hand at the end of the tube, from the side of the left eye.

In the presence of binocular vision, there seems to be a “hole” in the palm of the hand, through which the image that the patient sees through the tube is perceived.

Fig. 2.2. Sokolov's test



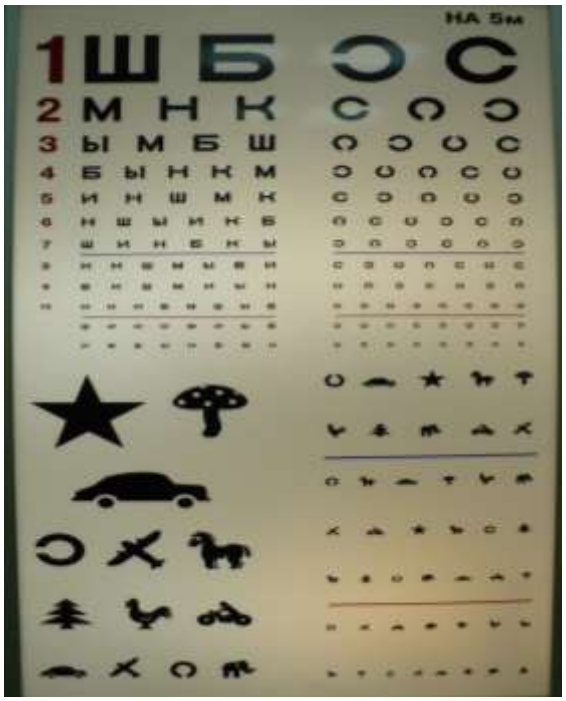


Fig. 2.3. Testing the visual acuity by subjective method

Fig.2.4. Testing the light perception.

Fig.2.5. Perimetry

Fig. 2.6. Static perimetry

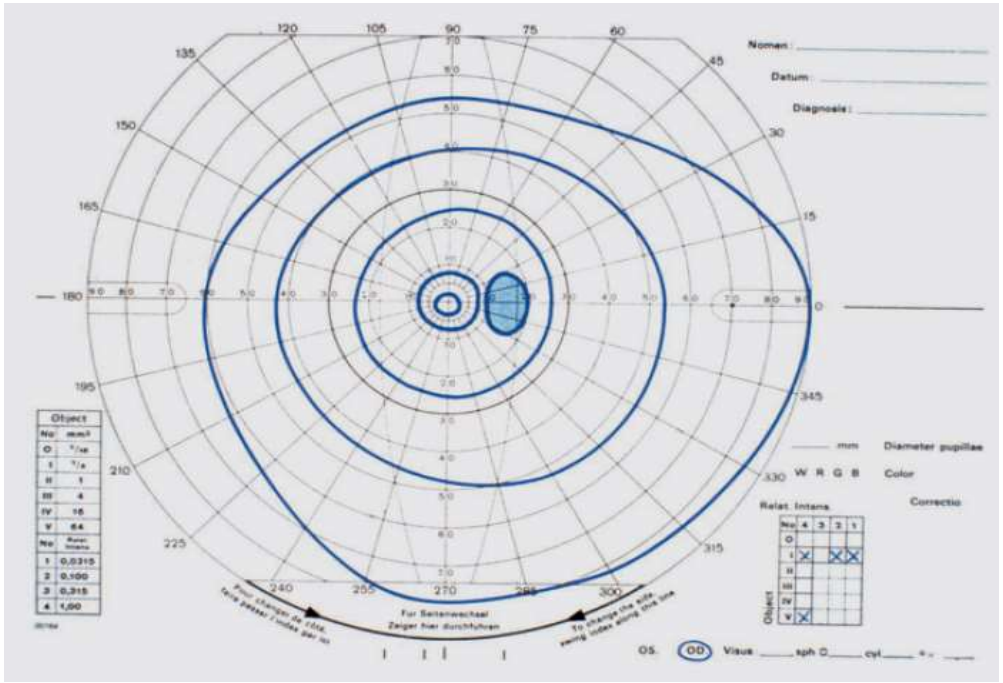


Fig.2.7. Normal boundaries of the field of view.

Fig. 2.8. Testing the field of view by the control method.

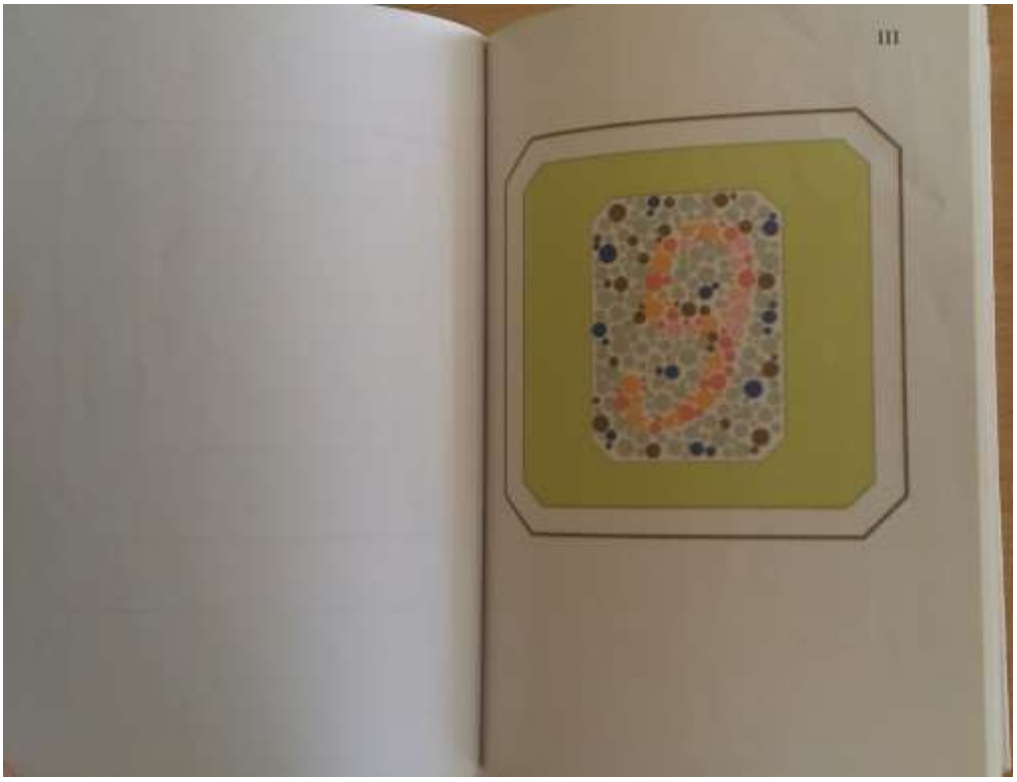


Fig.2.9. Polychromatic tables

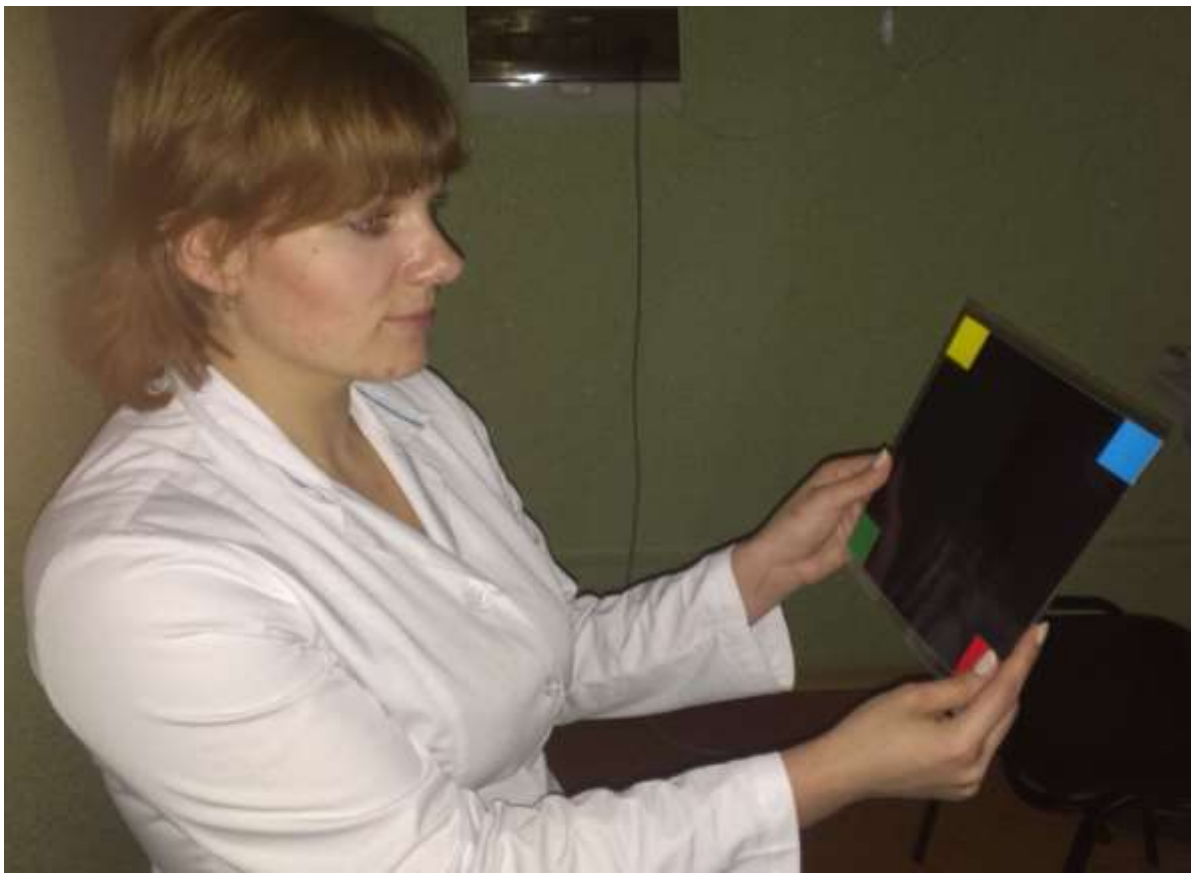


Fig. 2.10. Purkinje's square

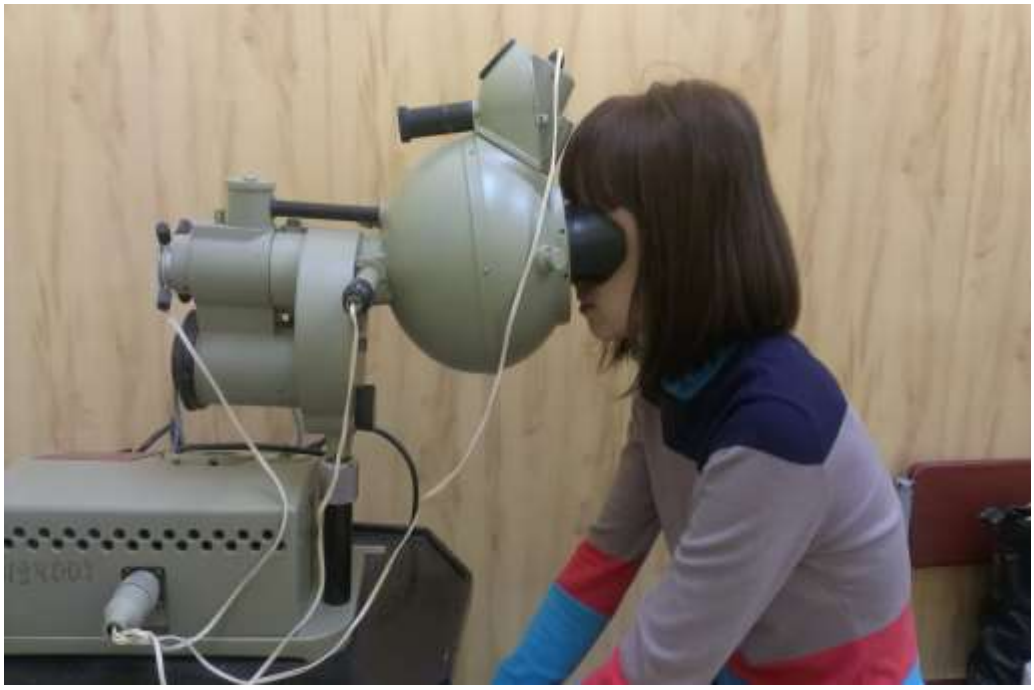


Fig.2.11. Adaptometry.

Theoretical questions to the chapter:

1. Anatomy of the retina and visual pathways.
2. The study of normal visual acuity.
3. Methods for examination of central vision.
4. The theory of colour vision.
5. Methods for testing colour vision, types of colour perception disorders.
6. The concept of the field of vision and types of peripheral vision.
7. Methods for determining the visual field, normal boundaries to white and chromatic colours.
8. The concept of photoreception, photoreception theory.
9. Testing and types of adaptation.
10. Methods for determination of dark adaptation, types of disorders and their treatment.

Practical skills which students should master:

1. Determine the visual acuity with the help of tables and formulas; recording visual acuity.
2. Determine the field of view, types of disorders.
3. Determine the colour perception with the help of Rabkin's table.
4. Define adaptation.

CHAPTER 3

REFRACTION AND ACCOMMODATION

One of the main conditions of the normal vision is a clear image on the retina of objects that surround us. Reduced vision into the distance or nearby, which is predetermined by refraction, that is, the refractive system of the eye, varies in different periods of life in almost all people. Knowledge of the laws of refraction of light rays by the eye's optical apparatus, its required ocular correction, allows one to determine how people see objects at different distances at different ages, how to help in case of reduced visual acuity and prevent its progress. With the help of visual analyzer, a person receives information about the world around us more than with any other analyzers combined. Every doctor of any specialization should know the structure of visual analyzer.

The list of key terms, parameters and characteristics which students should master while studying the topic:

Term	Definition
Refraction	The change in the direction of light in the optical medium
Accommodation	The eye's ability to see at different distances
Physical refraction	Power of the eye's optical system
Clinical refraction	Position of the main optical focus with respect to the retina
Emmetropia	Commensurable refraction
Ametropia	Incommensurable refraction
Astigmatism	Combination of several types of refraction in one eye
Dioptre	Unit measuring refraction

The human eye is a complex optical system, acting as collecting glass. Light rays, passing through its media, are refracted and collected inside the eye in the system focus, where the image appears. The eye has a refractive power (refraction) and is an optical device.

Refraction is a change in direction of light in the optical system of the eye.

The optical apparatus of the eye is made up of:

- the light refracting, light-conducting system, which is presented by the cornea (40dpr.), the lens (15-18dptr.), vitreous body, humour of the anterior and posterior chamber of the eye (2-5 dptr.). Total refractive power of the eye is referred to as ***physical refraction***.

- light-perceptive system − the retina.

Refraction is divided into ***physical*** and ***clinical*** and is measured in dioptres.

Physical refraction is a total refractive power of the optical system of the eye. On average, it is 60 dioptres in healthy adults. Physical refraction can also range from 57 to 72 dioptres. A dioptre is equal to an optical power of the lens with a length of focal distance of 1 meter.

The position of main optical focus of the optical system with respect to the retina is called the ***clinical refraction***. One can distinguish between static and dynamic refraction. The static subtype implies refraction with accommodation at rest, while the dynamic one − with the participation of accommodation.

Types of clinical refraction:

- emmetropia (commensurable).
- ametropia: myopia, hyperopia.

In **emmetropia**, main optical focus coincides with the retina. In this case, patients have clear images of objects in the distance and at close range. The main optical focus is a point on the main optical axis, where all parallel rays that come from distant objects converge, after going through all the optical media of the eye.

In **myopia (nearsightedness)**, the main optical focus is in front of the retina. Therefore, a blurred image is formed on the retina. By the degree of the change in direction, myopia is a strong refraction. In order that the image got on the retina, it is necessary to weaken the refractive power and put a diverging lens (concave) in front of the eye.

Hyperopia (farsightedness), the main focus is behind the retina. The optical power of the eye is weak. In order that the image got on the retina is necessary to increase

the optical power of the eye and the front of the eye to put the collective lens (convex).

Astigmatism is a phenomenon, when the power of the refractive lens of the eye is not the same in different meridians. It is the combination of different types of clinical refraction or different degrees of one refraction in one eye. Normally, the optical power of the eye is the same in all meridians.

Types of astigmatism:

1. Simple: there is emmetropia in one of the meridians, with ametropia in another one.

2. Complex: there is one of the types of ametropia in both meridians, but of varying degrees.

3. Mixed: myopia in one meridian, hyperopia − in another one.

Astigmatism can be:

- congenital and acquired
- correct and incorrect
- simple, complex, combined
- myopic, hyperopic, mixed astigmatism.

Complications: amblyopia, strabismus, accommodation spasm.

Astigmatism within 0.5 dioptres is considered to be physiological. In other cases, correction with cylindrical lenses is required.

Accommodation is the eye's ability to see objects clearly at a short and long distance. In the act of accommodation, lens, ciliary zonules and ciliary muscle are involved. When viewing close objects, ciliary muscle contraction, ciliary zonules relaxation and changing of the lens curvature (it becomes more convex) occur. With age (after 40), the lens loses its elasticity and the ability to accommodate the eye decreases (***presbyopia***). Correction with glasses should be prescribed for work at close range.

Methods for determination of clinical refraction:

1. Subjective − selection of corrective lenses.
2. Objective − pupilloscopy, refractometry.

SUBJECTIVE METHOD FOR DETERMINING CLINICAL REFRACTION

To conduct subjective method of refractometry, a set of lenses, a trial eye frame and eye charts are needed.

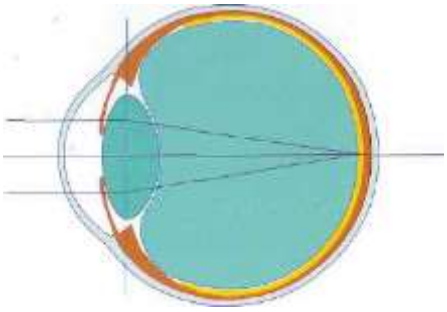
The patient puts on the universal frame, the interpupillary distance is adjusted. A glass of sph + 0.5 D is put in a lens holder. If the vision has improved, one continues to increase the power of the positive lenses at 0.5 D intervals, as long as the eyesight gradually improves along the chart rows and suddenly gets worse: then one should get back to the glasses, which gave the highest degree of vision. There may be several of such glasses. In this case, the degree of hyperopia (Hm) is determined by the "+" area itself, and gives the best possible visual acuity.

If visual acuity with spherical lens +0.5 D sph decreased, the spherical lens of -0.5 D with the sign "-" is put into the lens holder. Likewise, one continues to increase the power of the lens until the gradually improving eyesight suddenly decreases, and then one returns to the glass which gave the highest degree of vision, that is, until the patient gets the best visual acuity. The degree of myopia (M) is determined by the strength of the least "-" scope, and gives the highest visual acuity. If using spherical lenses, one cannot get complete correction of the generally accepted norm, then it is necessary to check for the presence of astigmatism (ast).

The screen with a slit is inserted into the trial frame. By rotating the screen, one looks for the meridian, which gives the best visual acuity and conducts the correction as described above. Then the slit screen is rotated 90° and refraction in this meridian is determined. The result is recorded.

After complete examination, visual acuity (VIS) without correction, the type and degree of ametropia and visual acuity with correction are recorded. However, it is better if the diagnosis of astigmatism is made on the basis of refractometry data

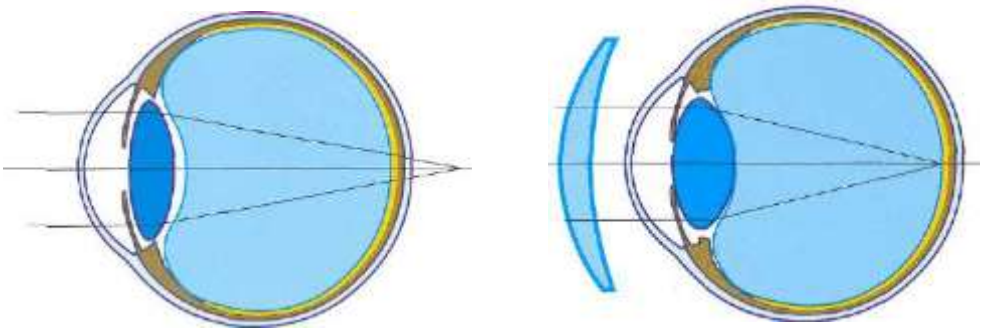
Refraction



Emmetropia (commensurable refraction)

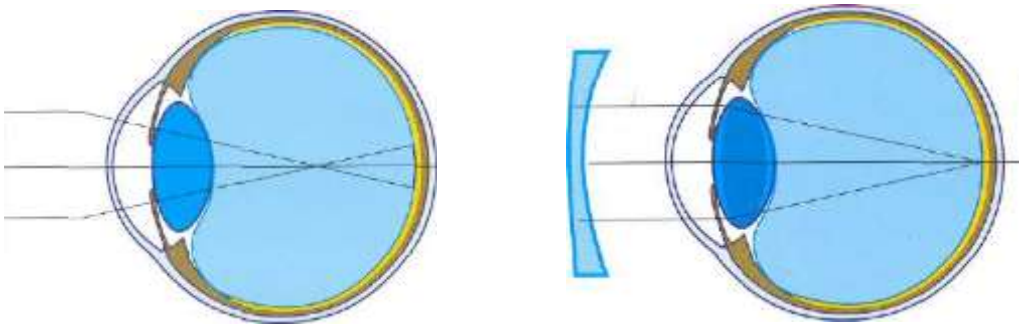
Parallel light rays are focused precisely on the retina. Positive lens with optic power of 1 dioptre focuses parallel light rays to a point which is from it at a distance of 1 m.

Hypermetropia



The point in which the parallel rays of light focus is behind the retina. In hypermetropia, the path of rays in correction with the collecting lens.

Myopia



Parallel light rays are focused in a point which is in front of the retina.

The path of rays in the myopic eye in correction with the dispersing lens.

OBJECTIVE METHODS FOR MEASURING CLINICAL REFRACTION

In order to conduct **skiascopy** (or **retinoscopy** − a term used in English-speaking countries) one needs:

1) ***mirror ophthalmoscope or skiascope*** (plane mirror with a hole in the middle);

2) ***skiascopy scales***;

3) ***light source*** − desk lamp.

Skiascopy scales are the set of lenses from 0.5 or 1 dioptre in the increasing order. One scale contains the collective lenses, the other − dispersing ones. The study is conducted in a dark room, the light source is to the left and slightly behind the patient. The doctor sits at a distance of 1 m and directs light reflected from the skiascope, to the examined eye. In doing so, the pupil manifests a bright "red" reflex. Slightly rotating the ophthalmoscope, the reflected beam is moved up − down or left − right through the hole of the skiascope, observing the motion of skiaskopic reflex in the pupil.

There are 3 types of motion:

1. Skiaskopic reflex is moved in the direction of motion of the mirror.
2. Skiaskopic reflex moves in the opposite direction to the motion of the mirror.
3. At the lowest mirror movement the pupil becomes dark.

In case of coincidence of the reflex and the mirror movement one can define hyperopia, emmetropia or myopia up to 1 dioptre. The second variant of skiaskopic reflex indicates myopia of more than 1 dioptre. Only in the third variant of the reflex movement implies myopia of 1.0 dioptre and in this case the measurement is stopped. When the eye reacted according to the first option, one puts the weakest collective lens of the skiaskopic scale and continues to move the mirror, watching the reflex in the pupil. If reflex continues to move in line with the movement of the mirror, one puts increasingly more powerful lenses up to the appearance of the skiaskopic reflex indefinite movement.

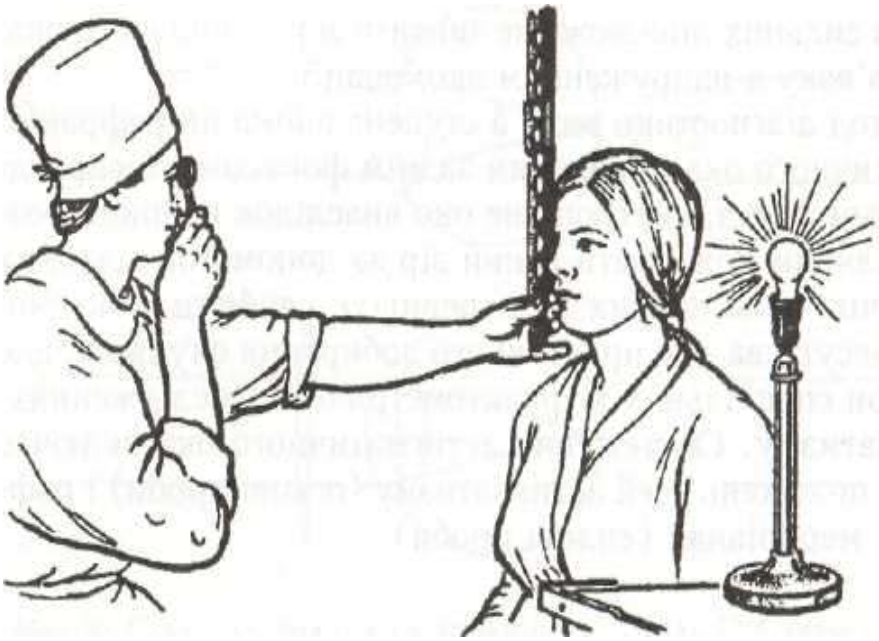


Fig. 3.1. Conducting pupilloscopy.

The optical power of this glass − Rs, is applied. For example, it is equal to 4 dioptres. With a stronger lens, the reflex will move in the opposite direction. The examined eye refraction R is calculated as: R = rs-1, in this case

R = + 4-1 = 3.

Consequently, hyperopia is 3 dioptres. If indefinite movement of the reflex appeared with a glass of +1 dioptre, emmetropia in the examined eye would be equal to: R = + 1-1 = 0 (Em).

The procedure is similar in case of the second variant with the reverse motion of the skiaskopic reflex. A stronger dispersing lens is applied, starting with the optically weakest one. Next, the doctor identifies the lens with which the reflex movement is indefinite. For instance, it has the optical effect of 6 dioptres. Refraction is calculated by the method described above: R = -6-1 = 7.

Therefore, myopia in the examined eye amounts to 7 dioptres.

In the examining of astigmatic eye, skiascopy is carried out in two main meridians. Clinical refraction is determined for each meridian separately.

Scotoscopy can be performed by a concave mirror. In this case, the reflex movement in all types and degrees of clinical refraction will have a direction opposite to that which was observed with a flat mirror.

When using scotoscope, which has its own source of light, a desk lamp is not needed. The following rules should be followed during scotoscopy:

1. The patient should look at skiascope.

2. The glass of scotoscopy scale which is used should be placed in a frontal plane at a distance of 12 − 15 mm from the eye.

3. The movement of the scotoscopic reflex should be evaluated not in the entire area of the pupil (particularly after instillation of mydriatic agents), but only in the area of 3-4 mm.

Refractometers. Optical refractometers provide an opportunity to measure the type and degree of refractive error, the degree of astigmatism and the position of its main axes.

ANISOMETROPIA

Anisometropia refers to the unequal refraction of both eyes. One can observe it quite often, but the difference between the eyes in refractive error as a rule is not significant. Such a difference does not affect the function of the eyes. However, there are cases when the difference is quite large (more than 2 dioptres). In case of slight anisometropia, binocular vision is preserved. In case of large difference, binocular vision is disrupted. Major complications of anisometropia are disorders of binocular vision depth and.

NEARSIGHTEDNESS

Nearsightedness, according to different authors, is noted in 20-60% of people and is the most common cause of visual acuity loss. There is no single scientific concept of the origin and development of myopia. The most common one is the theory of the origin of myopia by E.S.Avetisov, according to which three links play a role in its development: visual work at close range, combined with weakened accommodation; genetic predisposition; effects of intraocular pressure on the weakened sclera.

Classification of myopia.

There are three degrees of myopia:

• low − from 0.25 to 2.75 D

• moderate − from 3.25 D to 6.0 D;

• high − 6.25 D and above.

The clinical course:

• non-progressive (stationary)

• progressive.

Non-progressive myopia does not require treatment. Only correction is necessary. Myopia is considered progressive if during the year its degree increased by 1 dioptre or more. Such myopia requires both correction and treatment.

Clinically, nearsightedness is characterized by:

- decrease in visual acuity,
- good vision at close range,
- blurred vision at dusk,
- increased sagittal size of the eye by 2 mm or more,
- increased vision with the diaphragm.

Visual acuity in myopes is always lower than 1.0. At low degree and moderate severe myopia, the myopic cone can be identified in the fundus of the eye. It is a small ring in the form of a sickle at the temporal edge of the optic disc. Its presence is explained by the fact that in the extended eye, the pigment epithelium of the retina and choroid fall behind the edge of the optic disc, and the stretched sclera shines through the transparent retina. In myopia, the stretching of the eyeball occurs. The higher the degree of myopia, the longer is the antero-posterior axis of the eye (27 mm or more). Progressive myopia is dangerous for the formation of the *myopic cone* around the optic disc. It is a crescent at the temporal side of the optic nerve and causes functional impairment. However, if myopia increases further, one can observe the emergence of chorioretinal dystrophy in the fundus around the disk − posterior staphyloma, which tends to spread to the macular area. This in turn is accompanied by an irreversible decrease in visual acuity. Chorioretinal dystrophy does not always start around the optic disc. Myopia may be complicated by the appearance of gray (with black pigmentation) focus − Fuchs spots in the macular area. Retinal haemorrhages, predominantly in the macular area, are observed in the high degree progressive myopia.

In nearsightedness, an insufficient accommodation tonus can attenuate the convergence stress, creating conditions for the development of divergent concomitant strabismus. Patients with myopia should undergo annual screening. The progress of nearsightedness is observed at any age, but most often it occurs at the age of 14-17 years. By the end of the body's growth, stabilization of myopia may occur.

Treatment.

1. Identification of individuals with increased risk of developing myopia.
2. Normalization of accommodative ability. Patients are prescribed 2.5% Mesatonum solution or 0.5% tropicamide solution by 1 drop nocte during 1-1.5 months.
3. The regimen of visual stress. Every 40-50 minutes of reading or writing should be alternated with 5 minute rest.
4. In progressive myopia, sclera- strengthening surgeries are conducted.
5. Complete correction of myopia.

FARSIGHTEDNESS.

There are three degrees of hyperopia:

• low: up to 2 dioptres;

• moderate: 2.25 to 5 dioptres;

• high: more than 5.25 dioptres.

In the young age, with a low degree of hyperopia the visual acuity usually is not reduced due to accommodation tension. Thus, low refraction is compensated by strong accommodation in hyperopia. The latent hyperopia is the cause of spasms in the ciliary muscle. With the age decrease of accommodation, the latent hypermetropia progressively becomes apparent, which is accompanied by the decrease of the vision into the distance. Early development of presbyopia in hyperopia is also related to it.

During prolonged work at close range, the overload of the ciliary muscle often occurs. Clinically, this is manifested in headaches accommodative asthenopia or spasm of accommodation, which can be eliminated by proper correction, medical and physical therapy. In childhood moderate and high, hyperopia can lead to disruption of binocular vision and development of strabismus, usually convergent.

Treatment.

1. In normal visual acuity and low hyperopia, correction is not necessary.

2. In the reduced visual acuity and asthenopic complaints, the patient is prescribed permanent correction.

3. Children with hypermetropia more than 3.0 dptr. are prescribed constant correction with glasses.

Forms of correction of refractive errors.

1. Correction with glasses

2. Contact lenses

3. Phakic lenses

4. Refractive surgery.

Spherical and cylindrical lenses are used for ***correction with glasses***. Lenses can be collective (+ sign) and dispersing (− sign). Permissible tolerated difference between the glasses is no more than two dioptres. For astigmatism correction, cylindrical lenses are used.

Contact vision correction consists in the use of contact lenses. A contact lens (CL) is a transparent film or plate made of different polymers, which is in direct contact with the eye and is held in the eye by the capillary attraction forces. Contact correction has no age restrictions. Contact lenses are used for correction of myopia, hyperopia, astigmatism, anisometropia, presbyopia, for therapeutic purposes.

Surgical correction of refractive errors is known as "refractive surgery". There are corneal and lens surgeries, depending on the surgery area. In corneal refractive surgery, either the power of the cornea in the centre (in myopia) is weakened, or the refracting power of the cornea (in hyperopia) is increased.

Lens refractive surgery includes: removing of the eye lens and introducing the additional phakic intraocular lens.

ACCOMMODATION.

Accommodation is the ability of the eye to focus on the retina the image of objects placed to a different distance from the eyes. The physiological mechanism of

accommodation is the following: in contraction of the ciliary muscle, the ciliary zonules relax and the tension of the lens capsule weakens. The anterior surface of the lens becomes more convex and increases its refractive power. As a result, a person is able to see objects that are located at close range from the eyes.

Paralysis of accommodation occurs in lesions of the oculomotor nerve due to diseases, poisoning, injury or drug-induced exposure.

Accommodation spasm can result from prolonged stress of the ciliary muscle. It is characterized by the decrease in visual acuity, headache, fatigue when reading and false myopia or hypermetropia.

The ability to accommodate the eye is characterized by *the nearest point of clear vision* (punctum proximum). The function of accommodation depends on the type of clinical refraction and the patient's age. For instance, emmetrope and myope use accommodation when looking at objects that are closer than their distant point of clear vision. Hypermetrope is forced to constantly accommodate when looking at objects from any distance, because the farthest point is somewhat behind the eye.

Accommodation weakens with age. Age-related changes of accommodation are called presbyopia. This is due to densified lens fibres, disrupted elasticity and ability to change the lens curvature. Clinically it is manifested in the gradual aside of the nearest point of clear vision from the eye. Presbyopia occurs when the nearest point of clear vision is withdrawn by 30-33 cm from the eye and due to this the person loses the ability to work with small objects. It usually happens after the age of 40. For correction of presbyopia, patients are prescribed glasses for close range. At the age of 40, +1.0 dioptres are prescribed, and then 0.5 dioptres are added every 5 years. After the age of 65, as a rule, further correction is required. In hyperopes, age correction is accompanied by its degree. In myopes, the degree of myopia is subtracted from the rate of the presbyopic lens, required according to the age.

Age-related features of vision:

In newborns, the presence of the pupillary reaction to light in each eye is checked.

By the age of half year, the visual acuity increases from thousandths of unit to decimals.

By the age of 2-3 years, visual acuity is 0.5. Subsequently, it increases and corresponds to an average of 1.0, and in 15-25% of cases is equal to 1.5-2.0. Age farsightedness (presbyopia) develops after 40 years.

Colour perception develops in parallel with visual acuity. In children under the age of 1 year, it is possible to check colour perception by the infant's reactions to the same toy, but differently coloured.

The field of vision in children under the age of 3 years is tentatively checked. After the age of 3, the boundaries of the field of view are verified by instrument methods.

The presence of binocular vision contributes to the depth perception of objects. The lack of binocular vision leads to strabismus.

Fig. 3.2. A set of optical glasses

Fig. 3.3. Correction with glasses.

Fig. 3.4. Contact correction.

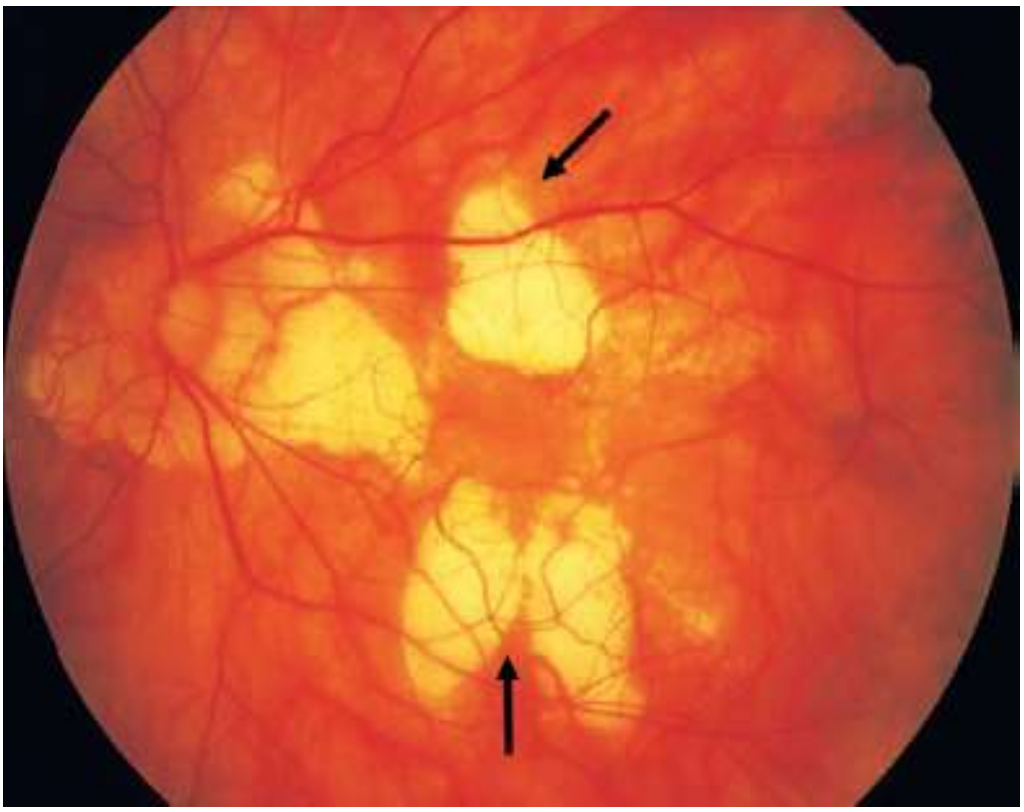


Fig. 3.5. Degenerative myopia

Fig. 3.6. Correction of astigmatism with phakic toric intraocular lenses

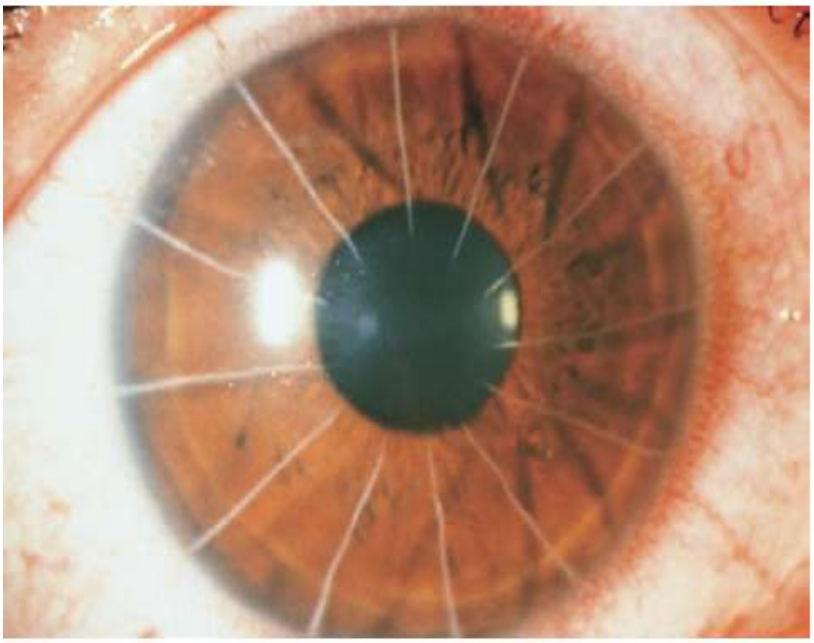


Fig. 3.7. Keratotomy.

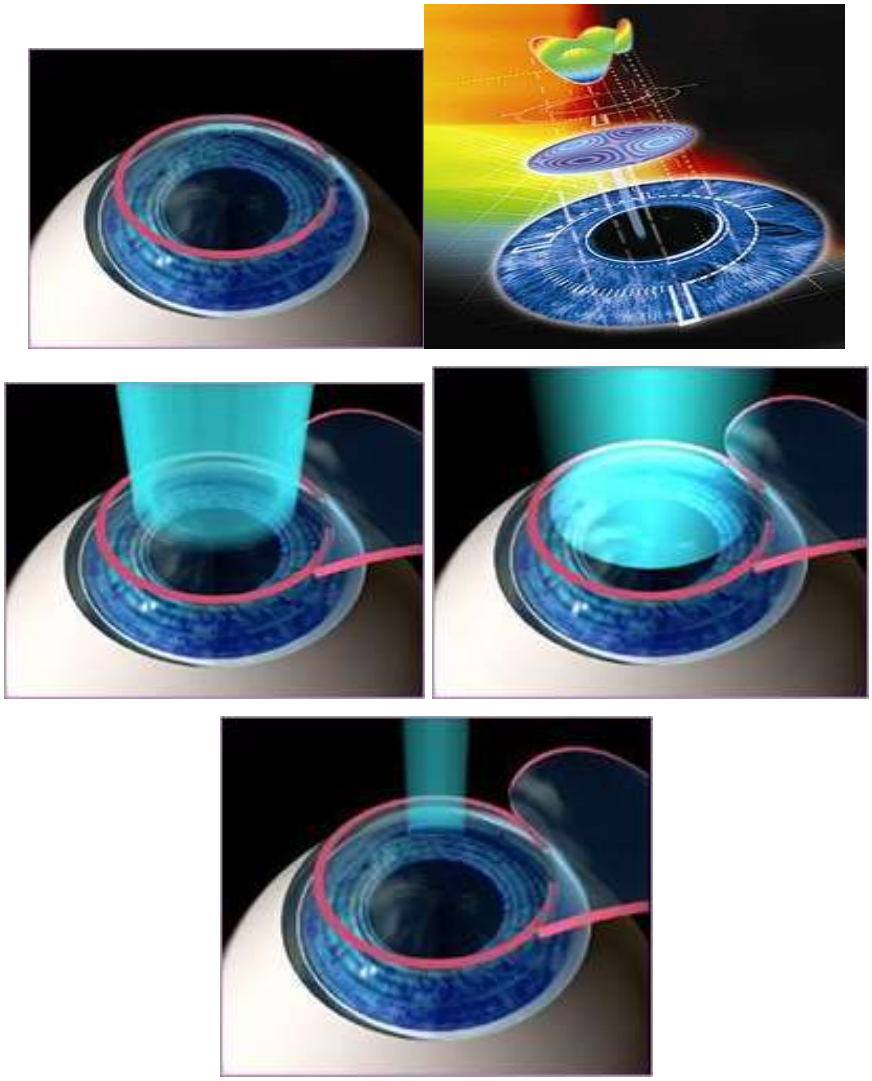


Fig. 3.8. Correction of astigmatism with excimer laser

Accommodation

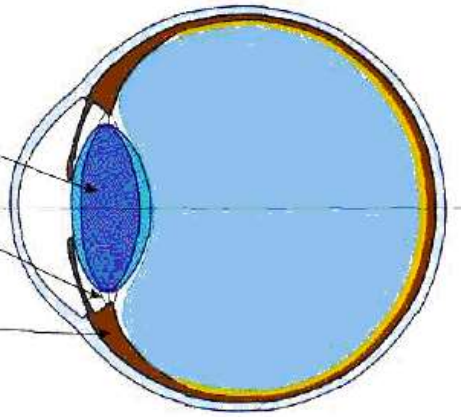
Presbyopia

Presbyopsia is manifested in the weakened ability to see properly at close range, which is experienced by almost all people after the age of 40.

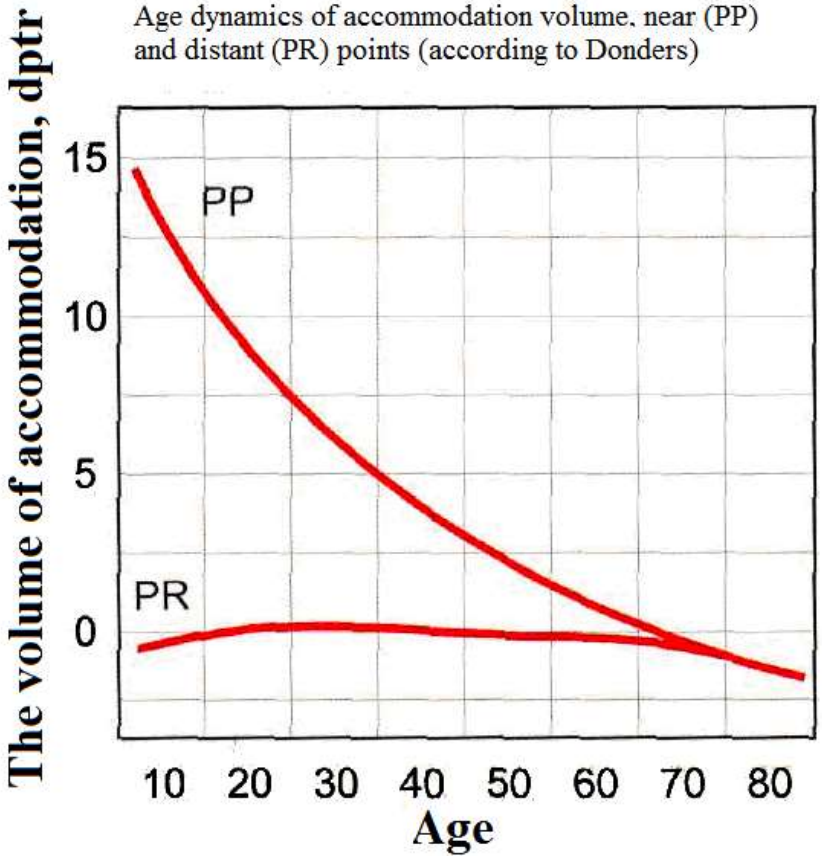
In presbyopsia, patients are prescribed glasses for reading with positive refraction lenses, and their optic power increases with age:
40-45 years - +1.00... 1.50 dptr
50 years - +1.50... +2.00 dptr
older than 55 years - +2.00... +2.50 dptr

Causes of presbyopsia:

1. Densifying of the eye lens
2. Changes of the ciliary zonule
3. Weakening of the ciliary muscle



Age dynamics of accommodation volume, near (PP) and distant (PR) points (according to Donders)



Theoretical questions to the chapter:

1. Physical refraction.
2. Units of optical power of lenses.
3. Characteristics of different types of clinical refraction.
4. The path of rays in the emmetropic eye.
5. The path of rays in the myopic eye, correction principles.
6. The path of rays in the hyperopic eye, correction principles.
7. Methods of determining the refraction (subjective and objective).
8. The concept of accommodation mechanism.
9. Characteristics of presbyopia.
10. Astigmatism, correction principles.
11. The concept of anisometropia, correction principles.

Practical skills which students should master:

1. Determining the clinical refraction by subjective and objective methods.
2. Determining the visual acuity.
3. Selection of corrective lenses.

CHAPTER 4

DISEASES OF THE EYELIDS

Eyelids and orbit protect the eyes from external influences and prevent conjunctiva and cornea from drying out. Morphological features of the eyelids, their innervation and blood supply determine the peculiarity of their pathology. In childhood, congenital and inflammatory pathologies of the eyelids and lacrimal organs predominate, whereas in adults inflammatory and tumoral diseases prevail. Early diagnosis of diseases of the eyelids and lacrimal organs facilitates their timely detection, proper selection of the treatment method, the prevention of tumoral processes.

The list of key terms, parameters and characteristics which students should master while studying the topic:

Term	Definition
Blepharitis	inflammation of the eyelid margin
Trichiasis	abnormal growth of eyelashes
Stye	acute purulent inflammation of the sebaceous gland or hair follicle around the sebaceous gland
Chalazion	chronic proliferative inflammation of the cartilage around the meibomian gland
Lagophthalmos	inability to close the palpebral fissure

The eyelids − the upper and lower − are complex anatomical structures, which protect the anterior surface of the eyeball. Owing to the blinking movements, they uniformly distribute the tear along the anterior surface of the eye, thus moistening the cornea and the conjunctiva. The upper and lower eyelids are connected by medial and lateral commissures. The inner angle is formed by the recess − the lacrimal lake. At its bottom, the lacrimal caruncle is located. The almond-shaped space between the open eyelids is called the *palpebral fissure*. The eyelids consist of two plates − skin and muscular (external), and cartilage and conjunctival (internal). The skin of the eyelids is thin, readily wrinkling; the subcutaneous tissue is loose and devoid of fat.

Therefore, oedema and haemorrhage easily occur on the eyelids as a result of direct injury or pathological processes nearby the eyelids. Cartilages of the eyelids are slightly convex connective tissue plates. The eyelids contain a large number of sebaceous gland (meibomian glands). They are arranged in parallel rows, and their ducts open in the intramarginal space (between the external and internal ribs of the eyelids). Lipid secretions of these glands lubricate the intercostal space of the eyelids and do not let tears roll down over the edge of the eyelids. Cartilage is tightly connected with the mucosa − conjunctiva. Dysfunction of the meibomian glands can be the cause of styes, chalazion, blepharitis. The muscle of the eyelid comprises the orbicular muscle, elevator muscle of upper eyelid, Riolan's muscle (narrow muscle strip on edge of the eyelid at the root of the eyelashes) and Horner's muscles (muscle fibres of orbicular muscle, covering the lacrimal sac). The orbicular muscle of the eyelid is innervated by the facial nerve. In paresis or inflammation of the facial nerve, lagophthalmos (inability to close the eye) occurs, which may lead to corneal dryness (xerosis) and adjunction of infection.

The elevator muscle of upper eyelid starts near the optic canal, goes under the upper part of the orbit and ends with three muscular fascicles. The outer fascicles is woven into the skin of the eyelid, the middle − into the thickness of the cartilage and the inner one − into the conjunctival arch. Blood supply of the eyelids is carried out by the branches of the ophthalmic artery, included in the system of the internal carotid artery. Arteries correspond to veins which carry the venous blood toward the angular vein, the vein of the lacrimal gland and the temporal superficial vein.

Methods of examining the eyelids.

1. External examination, palpation.
2. Examination at the side (focal) lighting.
3. Examination of the mucosa of the eyelids with eversion of the upper and lower eyelids
4. Biomicroscopy.

External examination. The patient (student) is seated facing the light. The doctor sits in front of the patient, withdraws the knees to the right. The parts of face that

surround the eye socket, the areas of the lacrimal gland and the lacrimal sac are examined. In the manifestations of oedema, hyperaemia, protrusions, palpation with forefinger is implemented. While examining the eyelids, one should pay attention to the colour of the skin, condition and position of the lacrimal points.

Side (focal) lighting. For a more detailed examination of the organ of vision, one can also use the combined method. It consists in examination of illuminated area through a strong magnifying glass at the lateral lighting of the eye (see Fig. 4.1.).

The study is conducted in a dark room. The patient sits with eyes wide open. The lamp is placed at the patient's left eye and in front at a distance of 50-60 cm. The doctor sits in front of patient, places the knees to the right, and the patient's knees to the left. The physician directs a focused light beam to the eye with the lens of 13.0-20.0 D, which the doctor in the right hand at a distance of 7-8 or 5-5.5 cm from the patient, or by a flashlight. The brightly lit part is clearly visible against the background of the shaded portion of the other eye. In left hand the doctor takes the second magnifying glass, which is held in front of the examined eye, or a binocular lens that is put on over the head.

Eversion of the eyelids for examination of the conjunctiva

The examination is carried out in a well-lit room. The patient is seated; the lamp is on the left. For examination of the conjunctiva of the lower eyelid, patient is asked to look up, the doctor at the same time pulls the lower eyelid down with the thumb, resting on the edge of the orbit (Fig. 4.3.). For examination of the conjunctiva of the upper eyelid, it is turned inside. To do this, the patient must be looking down, eyes open. The upper eyelid is held by the upper eyelid lashes with the index finger and thumb of the right hand. The upper eyelid is retracted from the eyeball. With the index finger of the left hand or with a glass rod the doctor pushes the top edge of the cartilage down, and at the same time it is necessary to turn the ciliary edge of the eyelid up with the right hand. The inverted eyelid is pressed against the eyeball. During the examination of the conjunctiva of the eyelid, it is essential to pay attention to the colour of the conjunctiva, its shiny surface.

Biomicroscopy. The examination is conducted using the slit-lamp. This method is used for examination of the anterior section of the eyeball. Biomicroscopy makes it possible to perform the intravital microscopy of the conjunctiva, the cornea, the iris, the anterior chamber, the lens, the vitreous body. The examination is carried out in a dark room. The patient is positioned with the forehead and chin on a special support stand. The illuminating source is adjusted at the outer side from the patient at an angle from 300 to 450. By moving the upper plateau of the coordinate table, one obtains a clear image of the light slit on the examined area of the eye. After that, the image of illuminated area is found under the microscope. By rotating the holding screw of the microscope, the maximum clarity of the microscopic images is achieved.

Fig. 4.1. The method of side lighting.

Fig. 4.2. Biomicroscopy.

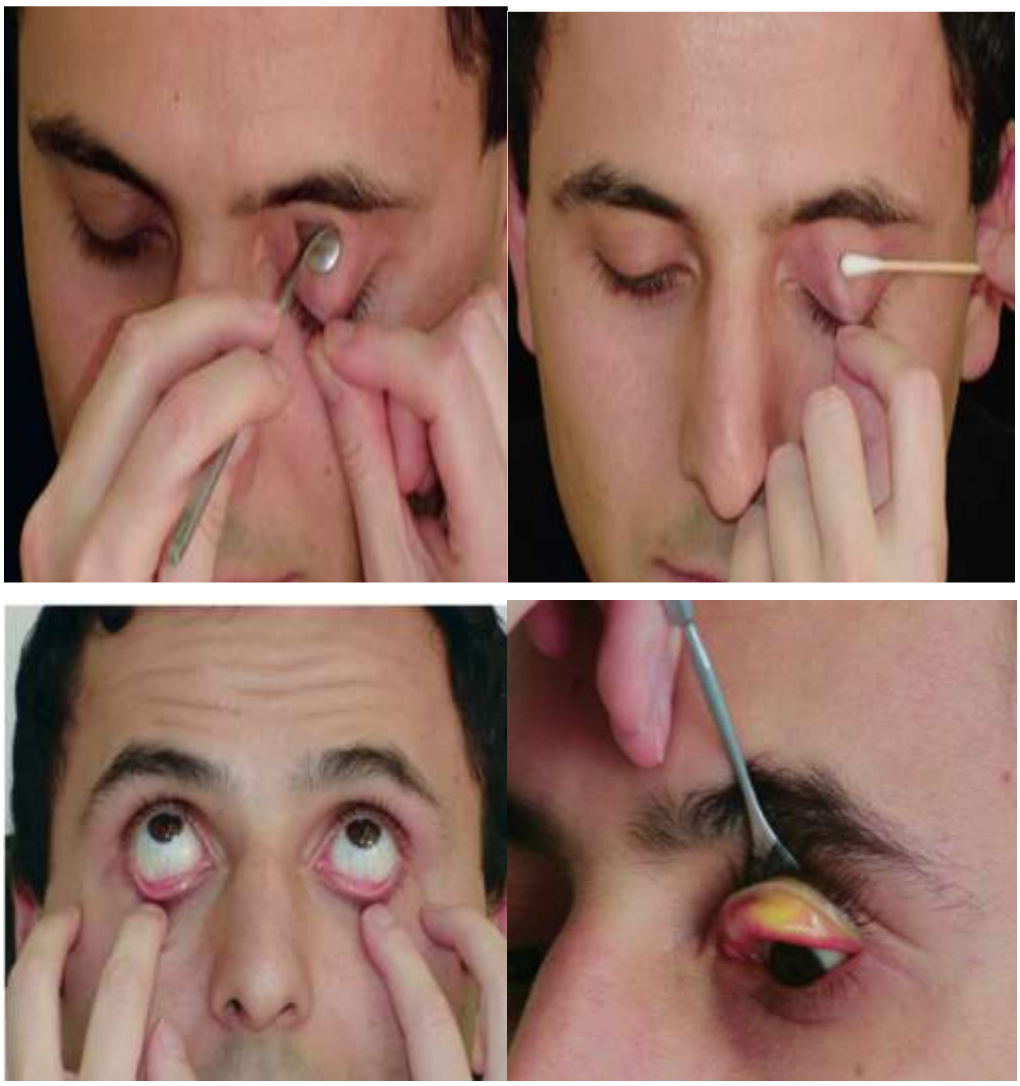


Fig. 4.3. Eversion of the upper and lower eyelids

Classification of diseases of the eyelids.

1. Developmental abnormalities
2. Inflammatory diseases.
3. Diseases of nervous and muscular apparatus.
4. Neoplasms of the eyelids.

Developmental abnormalities of the eyelids include: coloboma of the eyelids, epicanthus, lagophthalmos, ankyloblepharon.

Coloboma is a defect of the eyelids with a base at the edge of the eyelid. If there is a risk of corneal xerosis, the surgery is conducted.

Epicanthus is a semilunar vertical fold of skin between the upper and lower eyelids, which gives a person the "Mongoloid" expression. Surgical treatment is carried out only with the cosmetic purpose.

Ankyloblepharon is a partial or complete fusion of the upper and lower eyelids, often in the outer corner of the eye. Surgical treatment is the only option.

Lagophthalmos is the inability to close the eyelids. It occurs due to paralysis of the facial nerve that innervates the orbicular muscle of the eye. The surgical technique of stitching the lateral and medial eyelids is most commonly used.

Inflammatory diseases of the eyelids. These diseases include styes, chalazion, blepharitis, abscesses and cellulitis of the eyelids.

Stye is an acute purulent inflammation of the sebaceous glands around the eyelashes or at the root of the hair follicle. At the beginning of the disease, there is a sore point on the edge of the eyelid, then swelling and redness of the skin, and then the appearance of a yellow dot (suppuration of the stye). Recurrent styes indicate a decrease in the body's resistance (beriberi, anaemia, diabetes). The most common cause is Staphylococcus aureus.

Treatment. At the beginning, it is possible to prevent the development of the stye by lubrication of the affected area (the painful point) by alcohol solution or brilliant green. Topical antibacterial drops and ointments (floxan, ciloxan, tobrex, vigamox), sulfa drugs (sulfacyl sodium 30%) are applied. At the stage of infiltration, dry heat and UHF are used. Stye's extrusion and applying dressings in purulent discharge are contraindicated. In case of recurrent and multiple styes, the pus should be examined for flora and sensitivity to antibiotics.

Abscess, phlegmon of the eyelids is limited or diffuse infiltrative purulent inflammation of the tissues of the eyelids. The skin of the eyelids is characterized by redness and swelling of the entire surface, painful at palpation. There is caused by infection, injury of the eyelids, furuncle, diseases of the sinuses, the disorders orbit bones, sepsis.

Treatment. Intensive antibiotic therapy (broad-spectrum antibiotics, sulfonamides). In the presence of the formed purulent focus (fluctuation) − dissection, washing the

wound. Dissection is conducted in parallel to the edge of the eyelids, so as not to damage the muscles of the eyelids.

Chalazion is a proliferative inflammation of the cartilage around the meibomian glands. It is characterized by a limited round tight formation in the thickness of the cartilage of the eyelids. It is painless. Chalazion often results from of blockage of meibomian gland and proliferative inflammation.

Treatment. It is possible to administer betamethasone or triamcinolone (0.2 mg 1-2 times at weekly intervals) into the thickness of chalazion. This injection is possible from the side of the skin or the conjunctiva. When it is ineffective, surgical removal of chalazion with the capsule is necessary.

Blepharitis is a chronic inflammation of the eyelid margin. There are several forms: simple, scaled, ulcerative. Clinical manifestations are different:

1. Simple − is characterized by the thickening and reddening of the eyelid margin, there is a slight itching, irritation of the eyes.

2. Scaled − redness, swelling of the edges of the eyelids. The skin on the edge of the eyelid and eyelashes are covered with scales, the eyelid margins beneath them are hyperaemic.

3. Ulcerative − redness, swelling of the skin of the eyelids, crust on the edge of the eyelids, with ulcers beneath, point abscesses around the base of the eyelashes. Eyelashes are completely absent. It is caused most often by staphylococcal infection.

Causes of blepharitis:

- Air pollution (mechanical, toxic).
- General disorders (anaemia, diabetes, beriberi, gastrointestinal disease).
- Unconnected hyperglycemia, myopia, astigmatism.

Demodicosis. Inflammation of the eyelid margin, caused by Demodex mites. To diagnose Demodex, one needs to remove 5-8 eyelashes, which are placed on the cover-glass, moistened with water, covered with a glass slide and examined under the microscope. Demodicosis is a contagious disease. Transmission occurs through shared towels, pillows, mascara brushes. The clinical presentation of demodicosis is often characterized by lesions of the facial skin.

Treatment. The eyelid margin is treated with 1% brilliant green solution or alcohol, cleaned from scales. In ulcerative process, one should remove the scales after their softening with ointment. In demodicosis one can use metronidazole 10% on the eyelids edge. The eye drops of sodium bicarbonate 0.01% in 3 − 4 g for a day are prescribed.

Molluscum contagiosum. The disease is caused by a filterable virus (dermotropic virus). It is characterized by yellow disc-shaped nodules formed of the skin of the eyelids. In the centre of the nodule, there is a depression and point hole, from which the contents is extruded when pressed. Nodules can be both single and multiple. The size of a pinhead is up to 1.5 − 2.0 mm. Nodules are to be removed, cauterized by iodine or brilliant green.

Allergic dermatitis. Allergic reactions may develop by rapid type (urticaria, angioneurotic oedema) and slow type in 6-12 hours (skin eczema of the eyelids, toxicoderma of the eyelids). The causes of allergic skin lesions can medicines and cosmetics for eyelashes and eyebrows.

Diseases of the nervous and muscular apparatus include ptosis, ectropion and entropion of the eyelids.

Upper eyelid ptosis is the drooping of the upper eyelid is. It can be both congenital and acquired

Congenital ptosis is usually double-sided. It occurs against the background of underdevelopment of elevator muscle of upper eyelid or the nuclei of oculomotor nerve. Acquired ptosis may be neurogenic, myogenic or mechanical. Neurogenic ptosis occurs against the background of oculomotor nerve palsy. Myogenic ptosis occurs in myasthenia. Mechanical ptosis occurs in anophthalmia. Treatment depends on the cause and severity. In paralytic ptosis in adult, the conservative treatment with a neurologist is prescribed. In other cases, surgical treatment is necessary.

Entropion is the turning of the free margin of the eyelid towards the eyeball. Eyelashes are turned to the eyeball and injure the cornea and conjunctiva. Surgical treatment is essential.

Ectropion is the turning of the eyelids outwards from the eyeball. Along with the eyelid, lacrimal point is everted, and this may cause lacrimation. Surgical treatment is essential.

Neoplasms of the eyelids may be benign (papilloma, cutaneous horn, hemangioma, basal cell carcinoma) and malignant (basal cell carcinoma, melanoma). The treatment of benign tumours includes diathermocoagulation, cryotherapy, laser coagulation, surgical therapies. Treatment of malignant tumours of the eyelids combines surgery and radiotherapy.

Theoretical questions to the chapter:

1. Anatomical and physiological features of the structure of the eyelids.
2. Methods for examination of the eyelids (simple eversion, examination of the margins of the eyelids).
3. Inflammatory diseases of the margins of the eyelids, methods of treatment.
4. Allergic diseases of the eyelids.

Practical skills which students should master:

1. To conduct the external examination of the eyelids.
2. To conduct eversion.
3. To conduct the massage of meibomian glands.

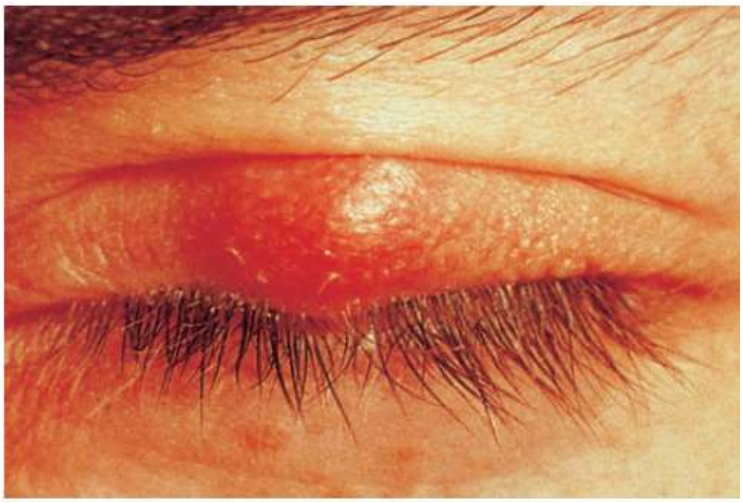


Fig. 4.4. Stye of upper eyelid

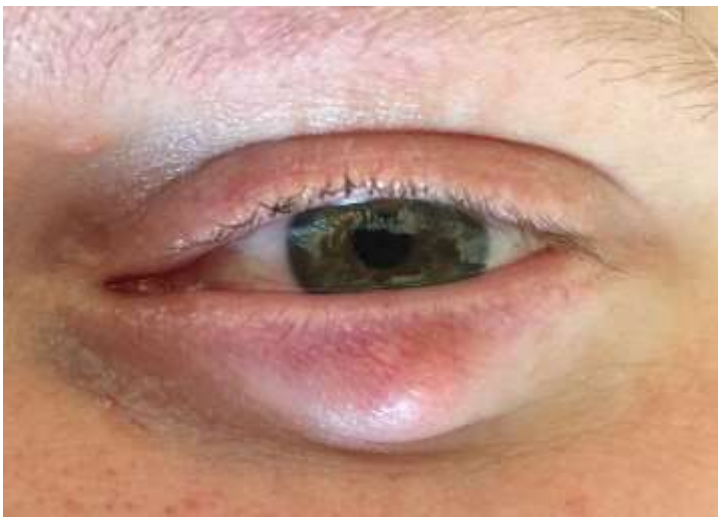


Fig. 4.5. Chalazion.

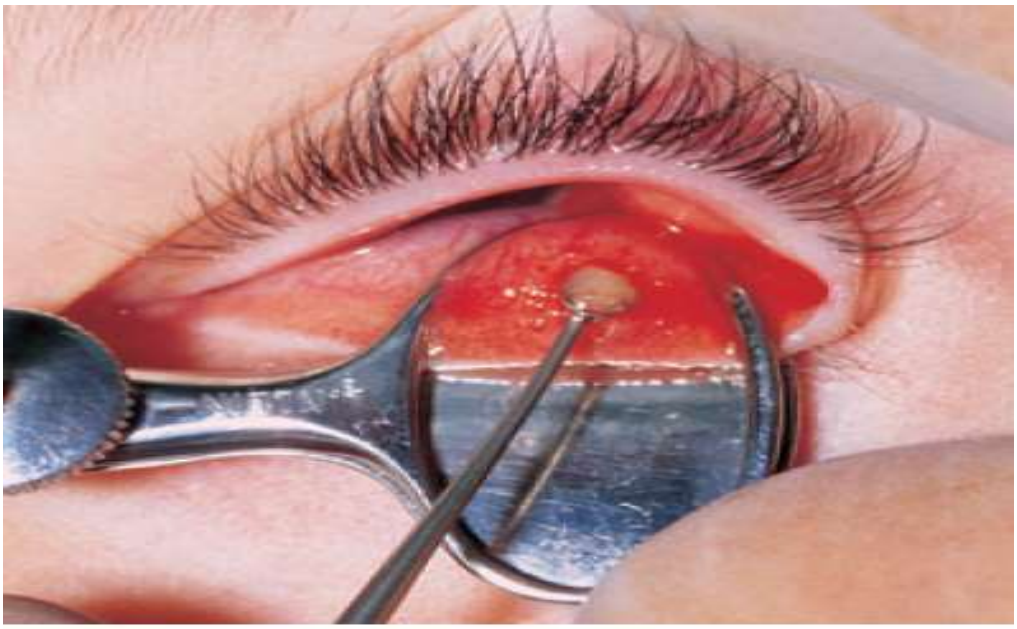


Fig. 4.6. Surgical removal of chalazion.

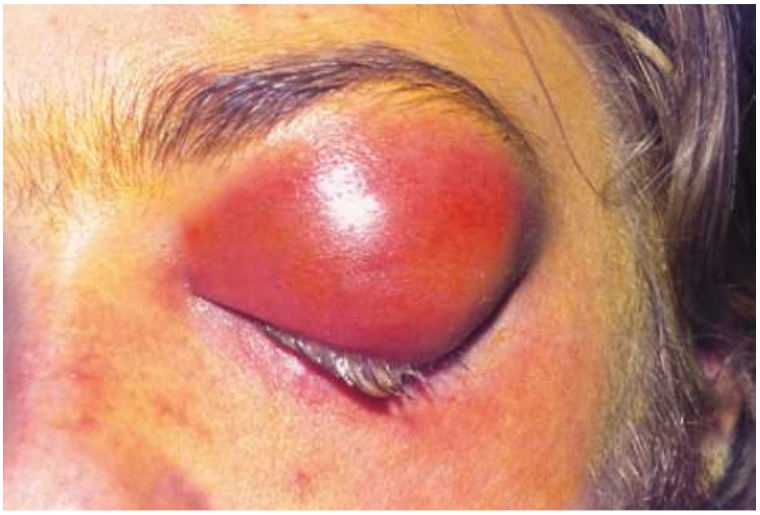


Fig. 4.7. Abscess of the eyelid.

Fig. 4.8. Necrotic phlegmon of the eyelid.

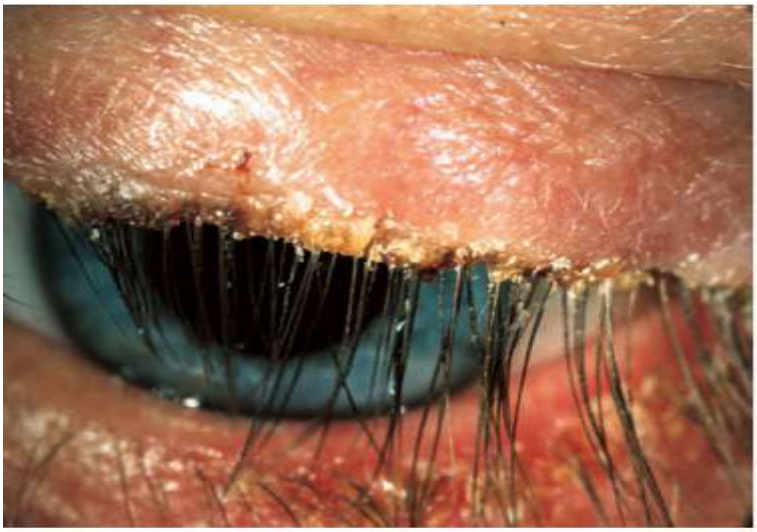


Fig. 4.9. Squamous blepharitis.

Fig.4.10. Coloboma of the upper eyelid.

Fig. 4.11. Entropion of the lower eyelid.

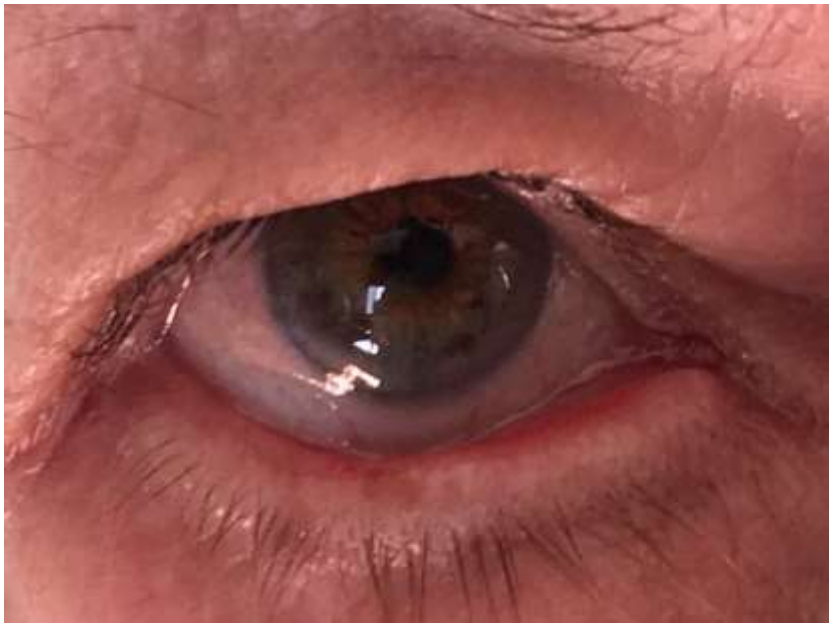


Fig.4.12. Senile eversion of the lower eyelid.

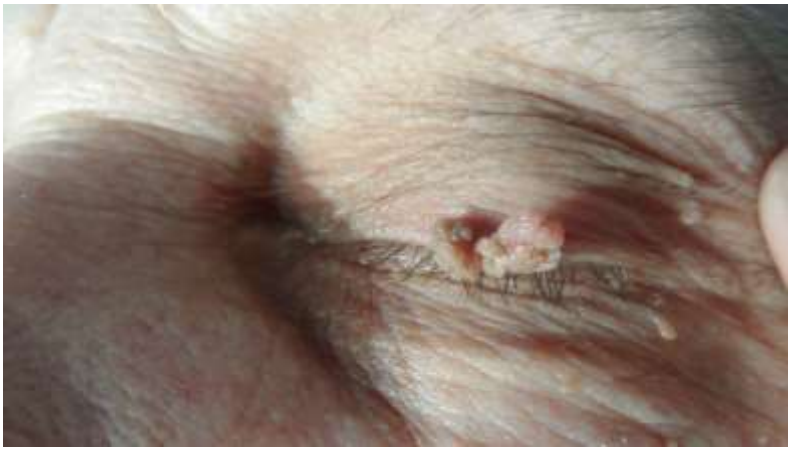


Fig.4.13. Papilloma of the upper eyelid

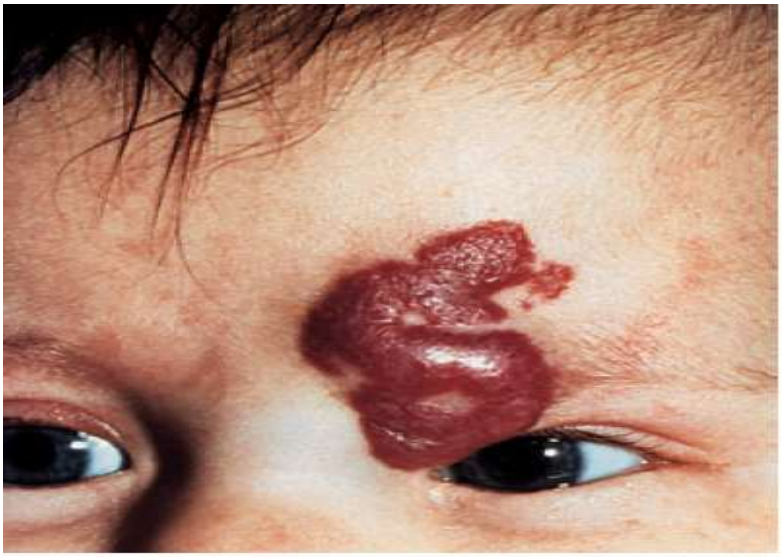


Fig. 4.14. Hemangioma of the eyelid

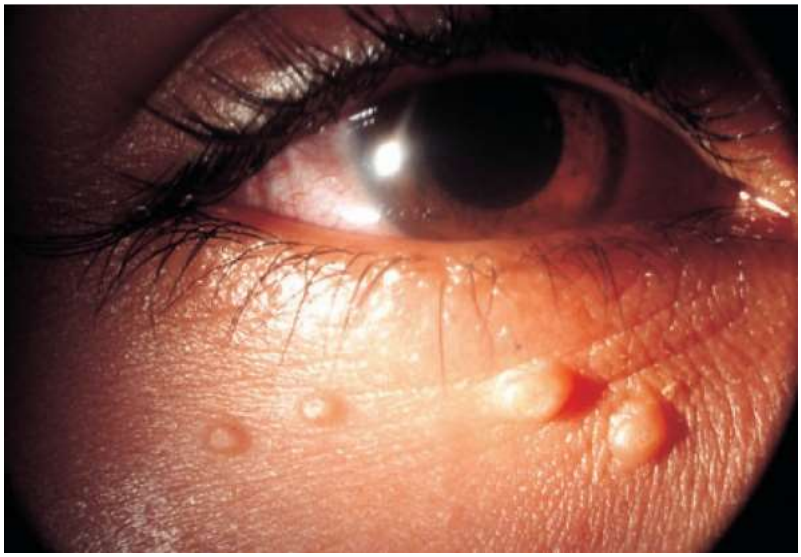


Fig. 4.15. Molluscum contagiosum.

CHAPTER 5

DISEASES OF THE LACRIMAL APPARATUS

Tear constantly moistens the cornea and the conjunctiva. Lacrimal organs consist of:

- lacrimal apparatus (lacrimal gland and accessory lacrimal glandules (Wolfring and Krause)
- lacrimal passages.

Lacrimal gland is located at the upper-outer portion of the orbit. It is divided into two unequal parts − the orbital and palpebral, which are connected with each other by a narrow isthmus. The orbital part is larger than the palpebral one. The orbital part of the gland is not detectable through the skin, as it is located over the hanging edge of the bone socket. When the gland is enlarged (e.g., tumour, oedema or ptosis), its palpation becomes possible. *The palpebral part of the lacrimal gland* is located directly above the upper arch of conjunctiva. When turning the upper eyelid outwards and the eye is turned inwards and downwards, this part of the lacrimal gland is normally visible as a slight protrusion of yellowish lumpy mass. In the inflammation of the gland (dacryoadenitis), this site develops the expressed protrusion due to oedema and the densified glandular tissue. The enlargement in the lacrimal gland can be so severe that it can result in the displacement of the eyeball. Tears, secreted by the glands, moisturize the anterior surface of the eyeball. Lacrimal gland begins to function by the third month of life. Prior to this, the infants are crying without tears.

Lacrimal system starts with the lacrimal points, which pass into the lacrimal ducts, lacrimal sac and nasolacrimal duct, that opens into the inferior nasal concha.

Lacrimal point (punctum lacrimale) is the initial opening of the lacrimal apparatus. Lacrimal points are located at the posterior edges of the free margin of both eyelids, the upper one is approximately 6 mm, and the lower one is 7 mm from their internal adhesion. Lacrimal papillae face the eyeball and are almost adjacent to it, whereas the tear points are immersed into the lacrimal lake, at the bottom of which the lacrimal caruncle (caruncula lacrimalis) lies.

Lacrimal sac (saccus lacrimale) composes the upper, extended part of the nasolacrimal duct. Topographically it refers to the orbit and is placed in its medial

wall in the bone recess − the fossa of the lacrimal sac. Lacrimal sac is the membranous tube 10-12 mm long and 2-3 mm wide. Its upper end terminates blindly. This place is called the fornix of the lacrimal sac. Downwards, the lacrimal sac narrows and passes into the nasolacrimal duct. The passage of lacrimal fluid through the lacrimal passages normally lasts about 10 minutes. Approximately such an amount of time is required for 3% colloid silver or fluorescein 1% from the lacrimal lake to reach the lacrimal sac (5 min − tubular test) and then the nasal cavity (5 min − positive test probe).

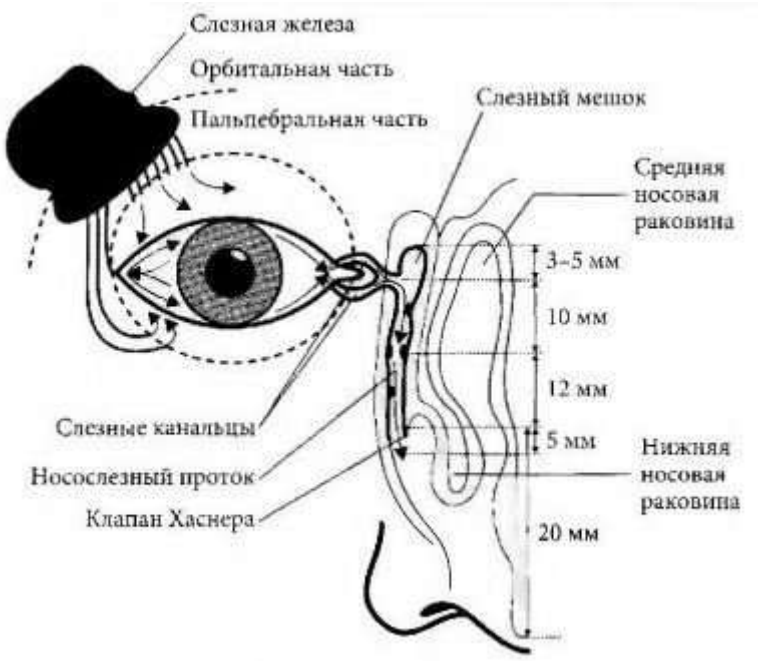


Fig. 5.1. The structure of the lacrimal apparatus

The list of key terms, parameters and characteristics which students should master while studying the topic:

Term	Definition
Dacryoadenitis	inflammation of the lacrimal gland
Dacryocystitis	inflammation of the lacrimal sac
Phlegmon of the lacrimal sac	acute purulent inflammation of the lacrimal sac
Canaliculitis	inflammation of the lacrimal duct
Hydrops	dropsy of the lacrimal sac

Examination methods.

1. Examination of lacrimal points.
2. Examination and palpation of the area of lacrimal sac.
3. Assessment of active lacrimation (colour sample).
4. Assessment of passive patency of lacrimal ducts (lacrimal probing).
5. Examination of lacrimal gland.
6. Quantitative evaluation of lacrimal production (Schirmer's test).
7. X-ray examination with the introduction of contrast medium into the lacrimal passages.

Examination of the lacrimal sac. Inspect the area of lacrimal sac (the portion below the inner ligament of the eyelids). In order to identify the content one needs to press the area of the lacrimal sac from the bottom upwards. The content (in the form of mucus or pus) will be discharged from the lacrimal points.

Schirmer's test. Schirmer's test is used to determine normal or reduced function of the lacrimal gland.

One needs to take a strip of filter paper, 35 mm in length and 5 mm is width. One end of 5 mm is folded and laid on the lower eyelid. The free end is hanging over the cheek. Normally, in 5 minutes the paper strip is wetted with tears with the width at least 15 mm. Lacrimal production is considered low when the length of the wetted portion of a strip is 15 mm and less.

Canalicular test. In the absence of changes in the lacrimal sac and the lacrimal points, one should conduct examination of canaliculi functions.

For this purpose, one needs to instill one drop of dye (3% collargol solution or 1% fluorescein solution) into the conjunctival sac and observe what time it takes substance to disappear from the conjunctival sac. In proper function of the canaliculi, it should disappear within two minutes.

When during the canalicular test the dye remains in the conjunctival sac for more than 2 minutes, it means that the suction function of the canaliculi is impaired.

Nasolacrimal test is performed in order to get an idea about the patency of nasolacrimal duct (obviously, if the function of the canaliculi is not impaired). A

small cotton wool ball is wound on the probe and is inserted into the inferior nasal meatus to a depth of 3-3.5 cm. The conjunctival sac is instilled with one drop of dye (3% collargol solution, or 1% fluorescein solution). The appearance of the dye on the cotton wool ball in 5 minutes indicates the normal patency nasolacrimal duct (positive sample), if the dye appears in 6-20 minutes, it is defined as the slow sample; the appearance of the dye in 20 minutes indicates the negative test.

The patient is anaesthetized by instillation of 0.5% alcaine solution in the conjunctival cavity. The doctor inserts the thin conical probe into the lower lacrimal point perpendicularly to the edge of the eyelids. Then the probe is advanced into the lacrimal canaliculus in parallel to the edge of the eyelids up to the entry into the lacrimal sac. The passage is then washed with sterile isotonic sodium chloride solution. The patient is asked to lower the head and pick up a tray. Liquid is supplied into the lacrimal passage and should pass in the nose.

Classification of the diseases of lacrimal organs.

1. Pathology of lacrimal apparatus (malformations, inflammatory diseases).
2. Pathology of lacrimal passages.
3. Neoplasms.

Anomalies of the lacrimal gland development may be related to its absence or insufficient development, hypofunction, hyperfunction, abnormal position (ptosis). The absence of the gland or its displacement leads to the drying up of the eyeball (xerosis). Surgical treatment is conducted.

Dacryoadenitis is an acute inflammation of the lacrimal gland. It has an aggressive onset. Dacryoadenitis is often a complication of infectious diseases − flu, sore throat, pneumonia. It is characterized by redness of the skin, swelling of the upper eyelid in the outer part, acute pain, the presence of tumour formation, deviation of the eyeball downwards and to the nose. The palpebral fissure is S-shaped. Diplopia is observed. Body temperature rises; lymph nodes are sharply enlarged. Abscess can also form.

Treatment. Intensive antibiotic therapy is prescribed: corticosteroids, non-steroidal anti-inflammatory drugs. In case of abscess formation − it is cut open and drained.

Chronic dacryoadenitis is the enlargement of the lacrimal gland. It is characterized by dense swelling in the outer upper corner of the palpebral fissure, the displacement of the eyeball and diplopia (double vision). Inflammatory effects are virtually absent. The process is two- or one-sided. It can be of different aetiology: tuberculosis, syphilitic.

The presence of constant lacrimation in the room (not in the open air, due to other stimuli) indicates the pathology of lacrimation passages. The following disorders are possible:

- eversion of the lacrimal point;
- narrowing or stenosis of the lacrimal canaliculus;
- inflammation of the lacrimal passages (canaliculitis − of fungal, chlamydial, syphilitic, herpetic aetiology);
- inflammation of the lacrimal sac due to stenosis or obstruction of the lacrimal channel.

Dacryocystitis is an inflammation of the lacrimal sac. It arises as a result of the obstruction of the nasolacrimal duct. It may be chronic and acute (phlegmon of the lacrimal sac). It is characterized by persistent lacrimation, swelling in the area of the lacrimal sac. By pressing on the region of the lacrimal sac, purulent content is discharged from the lacrimal points. Chronic dacryocystitis necessarily requires surgical treatment (formation of the anastomosis between the lacrimal sac and the nasal cavity − dacryocystorhinostomy).

Phlegmon of the lacrimal sac is characterized by redness, swelling in the lacrimal sac, reddening and oedema of the lower eyelid and the nasal area, lacrimation. It is accompanied by general reaction of the body. Formation of fistula (in almost all cases) is observed.

Treatment. Acute dacryocystitis is treated at the in-patient eye department. In acute dacryocystitis, cutting open and drainage of abscess is carried out. Antibacterial broad-spectrum medications, both topical and general, are prescribed. After acute dacryocystitis, dacryocystorhinostomy is routinely conducted.

Dacryocystitis of newborns. It arises when the gelatinous film which closes the mouth of the nasolacrimal channel fails to dissolve during the foetal development. Due to disrupted outflow of tears, a purulent inflammation of the lacrimal sac occurs. Swelling is observed in the area of the lacrimal sac. By pressing on it, purulent discharge effuses the lacrimal points. It occurs during the first days after birth. Formation of phlegmon is possible.

Treatment begins with the massage of the lacrimal sac. The lacrimal sac is pressed in the direction of the nasolacrimal channel and is gently massaged to restore the patency of the nasolacrimal channel. Massage is prescribed for 7-10 days. Antibacterial drugs topically in the form of drops are also prescribed. If the massage did not bring the expected result, probing is conducted, during which the film is destroyed and the patency of the nasolacrimal channel is restored.

Canaliculitis is an inflammation of the lacrimal canaliculi, which occurs as a result of the diseases of the eyelids, the conjunctiva and the lacrimal sac. Pathogens may be bacteria, pathogenic fungi (Aspergillus, Penicillium, Trichophyton, Actinomycetes) and viruses (herpes simplex virus). The causes of chronic inflammation of the lacrimal canaliculi may be tuberculosis, syphilis and chlamydia.

Patients complain of lacrimation. The skin in the area of canaliculi is hyperaemic, swollen, painful on pressure. Lacrimal points are expanded, hyperaemic and oedematous. When the area is pressed, mucopurulent or crumbly discharge (in fungal aetiology) appears.

Treatment. The contents of lacrimal canaliculi is removed by pressing and washing of the conjunctival cavity with furacilin solution 1: 5000 − 3-4 times per day, with potassium permanganate solution of 1: 5000 − 3-4 times per day. In the presence of bacterial infection conjunctival sac is instilled with antibiotic solutions (fluoroquinolones), sulphonamides, antiseptics for 7-14 days, 3-6 times per day. At night, antibacterial ointments are applied into the conjunctival sac. In mycotic canaliculitis, antifungal ointments are applied into the conjunctival cavity for 3-6 times per day. In severe cases, the dissection of the lacrimal canaliculus is carried out,

removing its contents, followed by treatment of the wound surface with 1-2% alcoholic solution of iodine.

Theoretical questions to the chapter:

1. Anatomical and physiological features of the structure of the lacrimal apparatus.
2. Methods for examination of the lacrimal apparatus.
3. Clinical presentation, diagnosis and treatment of acute and chronic dacryocystitis.
8. Features of treatment of neonatal dacryocystitis.

Practical skills which students should master:

1. To conduct the external examination of the eyelids and the lacrimal apparatus.
2. To conduct eversion of the eyelids.
3. To be able to carry out the Schirmer’s test, check the nasolacrimal patency.

Fig.5.2. Schirmer's test.

Fig.5.3. Washing of lacrimal passages

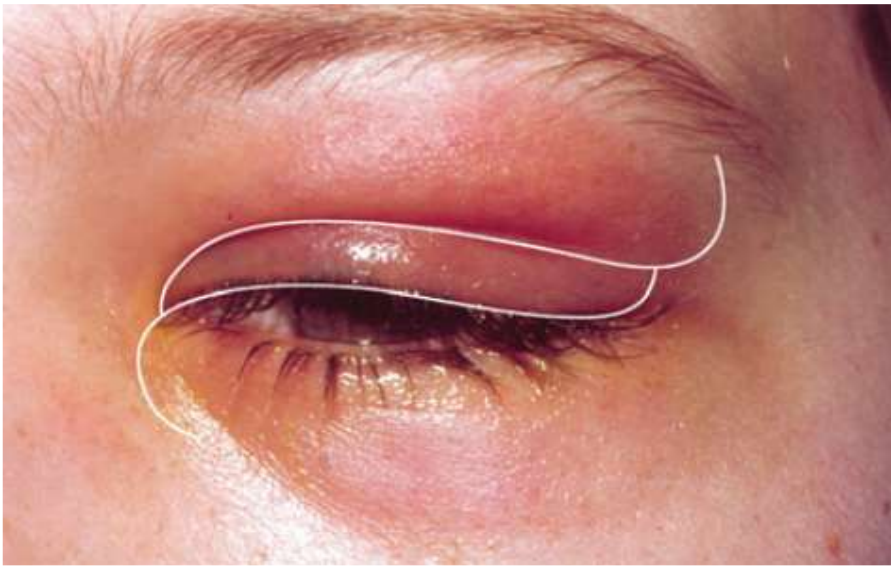


Fig. 5.4. Acute dacryoadenitis.

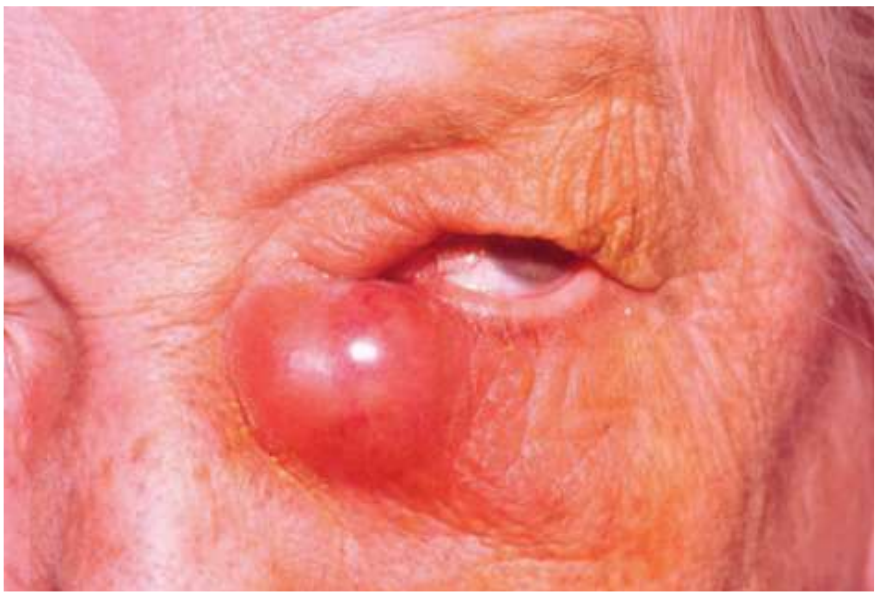


Fig. 5.5. Phlegmon of lacrimal sac.

Fig. 5.6. Atheroma of lacrimal caruncle

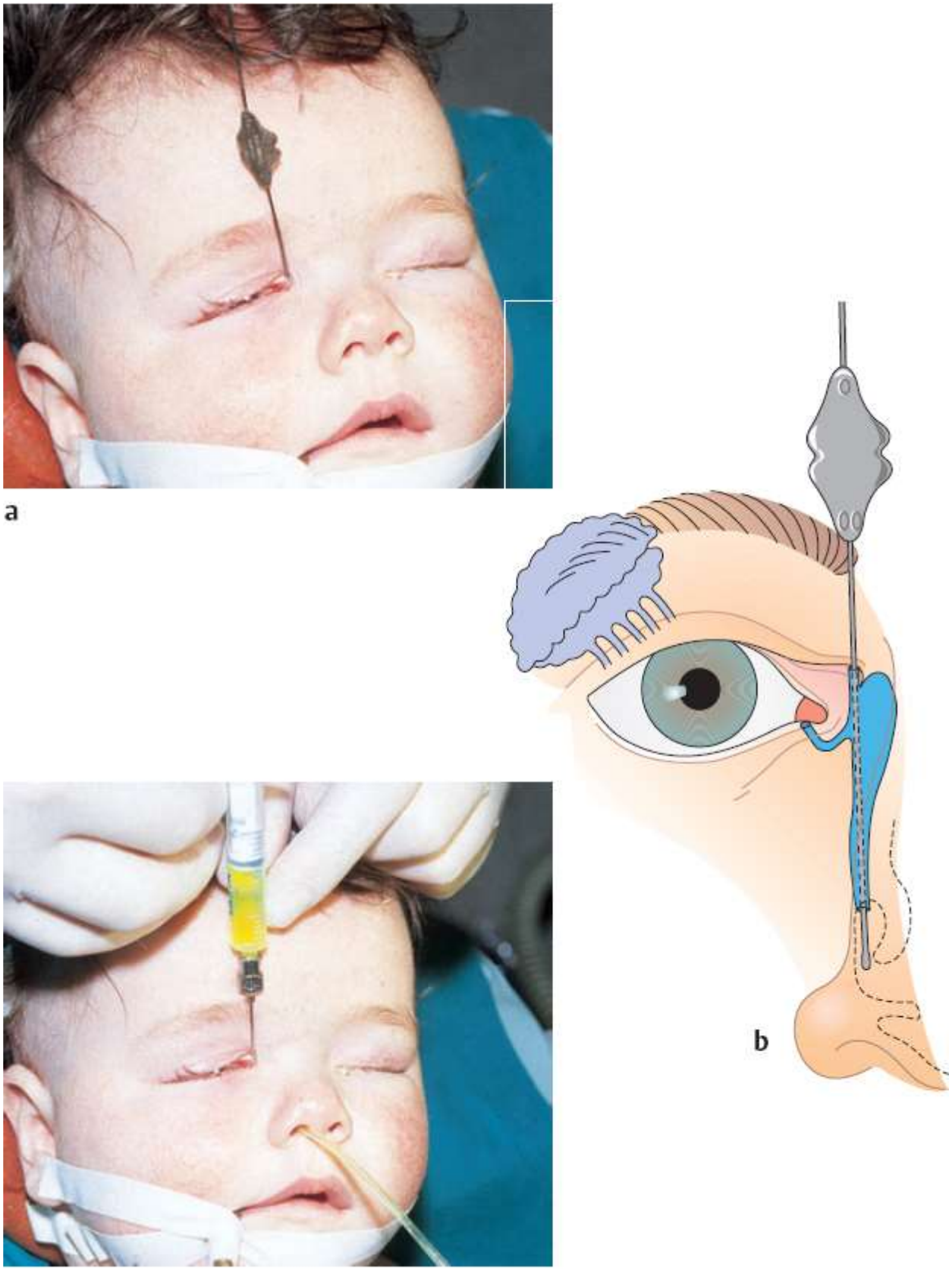


Fig. 5.7. Probing of lacrimal system.

CHAPTER 6

DISEASES OF THE EYE ORBIT

Diseases of the eye socket comprise one of the most difficult sections in ophthalmology. The complexity of the eye orbit is predetermined, first of all, by the fact that this small volume contains a lot of histologically different anatomical structures, which provide vital activities and functions of the eye. The eye socket is connected with the cranial cavity; it borders with the paranasal sinuses. Due to this, a variety of pathological processes, arising from these structures, may develop. The main symptoms of orbital disease are the displacement of the eyeball, its limited mobility. Displacement of the eyeball is manifested as a protrusion − exophthalmos or falling in − enophthalmos (happens much less frequently). The lateral displacements of the eyeball may also be observed.

The orbit is a bony formation of pyramidal shape in which the eyeball, the optic nerve, the external oculomotor muscles, and ophthalmic artery and vein, all motor nerves of the eye, and the first branch of the trigeminal nerve are located. The length of the orbit is 4.5 cm, the height in the entry area − 3.5 cm, the width − 4 cm. The orbit has four walls: inner, upper, outer, bottom. Three of these walls verge on the paranasal sinuses. The *internal wall* is the most complex and delicate. The orbit is connected to the maxillary, frontal, ethmoid sinuses and the cranial cavity. The *inferior wall* is formed by the zygomatic bone, the maxilla, the orbital process of the palatal bone. It is bordered by the maxillary sinus, which enables the spread of inflammation to the tissue of the orbit. The inferior wall is often subjected to blunt traumas that lead to the displacement of the eye block downwards. The *superior wall* is formed by the frontal bone and the lesser wing of the sphenoid bone. In the frontal bone on the outside and on the top, there is a fossa of the lacrimal gland. The *external wall* of the orbit verges on the cranial cavity. The inner wall is formed with the ethmoid bone, the lacrimal and the anterior portion of the sphenoid bone. This wall is the thinnest wall of the orbit. In blunt traumas, it is often damaged, which can cause emphysema of the eyelids and the orbit.

The outer wall is formed by the zygomatic, frontal bones and the greater wing of the sphenoid bone. This wall is the densest.

The walls of the orbit contain holes and fissures through which the large vessels and nerves enter the orbital cavity.

The optic canal (canalis opticus) is a bone canal with a round aperture through which the optic nerve (n opticus.) and the ophthalmic artery (a. ophthalmica) enter the orbit.

The superior orbital fissure (fisura orbitalis superior) is located between the greater and the lesser wings of the sphenoid bone. All oculomotor nerves, the first branch of the trigeminal nerve pass through it. The upper ophthalmic vein (v. ophthalmica superior) exit the orbit trough this fissure.

In case of damage of the superior orbital fissure, a complex of symptoms develops: ptosis (drooping of the upper eyelid), mydriasis (pupil dilation), complete ophthalmoplegia (lack of eyeball movements), exophthalmos (eyeball protrusion), disrupted tactile sensitivity.

The inferior orbital fissura (fisura orbitalis inferior) is formed by the greater wing of the sphenoid bone and the maxilla. The fissura is closed with a dense fibrous membrane consisting of smooth muscle fibres. The infraorbital nerve enters the orbit through it. The infraorbital vein leaves the orbital cavity through the inferior orbital fissura.

Blood supply of the orbit is carried out by the ophthalmic artery. The main venous vessel is the superior ophthalmic vein. **Innervation** is due to ciliary node. It is located behind the eyeball, 7-8 mm above the lateral rectus muscle and is adjacent to the optic nerve. Its size is about 2 mm.

Examination methods.

1. External examination, palpation.

2. Consultation of related specialists (ENT, dentist, neurosurgeon, endocrinologist, oncologist).

3. Exophthalmometry (protrusion of the eyeball − in the norm 12-18 mm, with the difference of not more than 2 mm between the eyes).

4. X-rays (plan radiography, CT, MRI).

Classification of diseases.

1. Congenital (malformations, tumours).

2. Acquired.

- Inflammatory (phlegmon, osteoperiostitis, thrombosis of the cavernous sinus).
- Tumours.
- Traumatic injuries (fractures of the orbital walls, foreign bodies).
- Endocrine disorders.

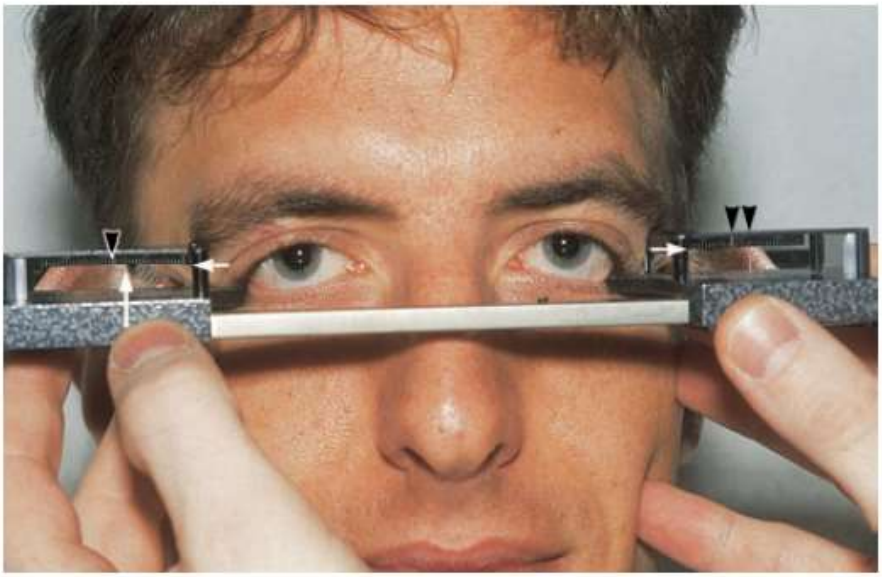


Fig. 6.1. Exophthalmometry

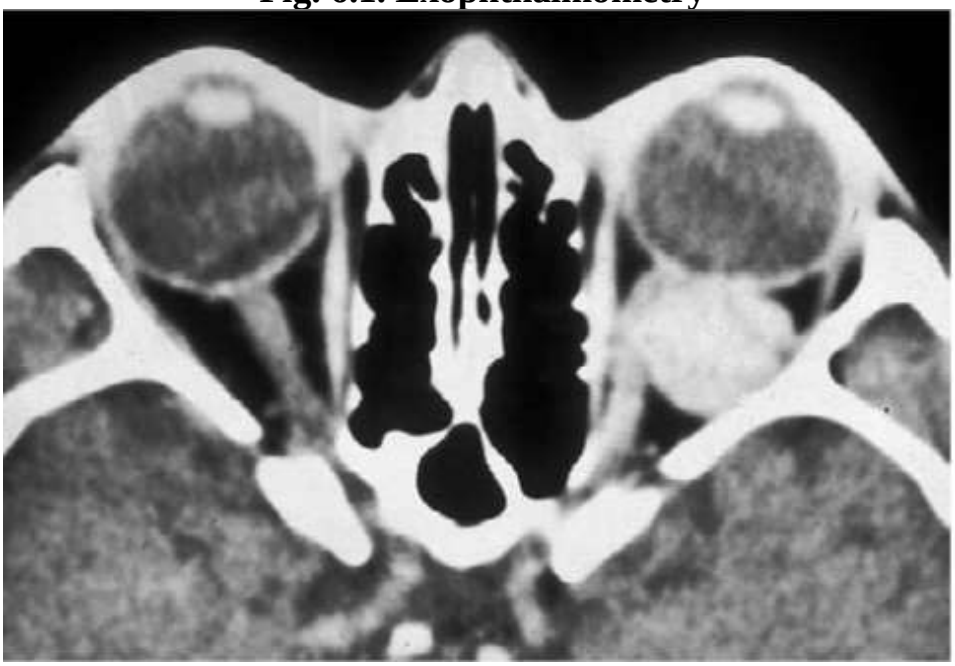


Fig. 6.2. Computer tomography of the orbit

The main symptoms of diseases of the orbit.

1. The displacement of the eyeball

Exophthalmos is a protrusion of the eyeball from the orbit. It occurs as a result of increased volume of the orbit associated with oedema, haemorrhage, tumour, diencephalic disorders, protrusion of the walls under inflammation, tumours of the paranasal sinuses. One can identify exophthalmos using Hertel's exophthalmometer. Normally, the protrusion of the eyeballs is 16-18 mm.

Enophthalmos is a retraction of the eyeball. It occurs as a result of reduced the volume of the orbit at fractures of its walls and the divergence of bone fragments, atrophy of orbital fat tissue, paralysis and paresis of sympathetic nerve.

Lateral displacement of the eyeball develops under the local processes in the orbital wall and soft tissues, at the displacement of bone walls. The displacement of the eyeball is often accompanied by double vision (diplopia).

2. Disorder of the eyeball mobility.

The degree of mobility disturbance may be different: from the mobility restriction in one direction or another, depending on the localization process, to complete immobility of the eyeball (ophthalmoplegia).

3. Pain. It is a characteristic feature of inflammation in the orbit.

4. Changes in the shape and size of the palpebral fissure.

5. Changes in the shape of the eyeball.

6. Changes in eye function, intraocular pressure.

7. Changes in the fundus.

PHLEGMON OF THE EYE SOCKET

Phlegmon of the orbit is an acute diffuse purulent inflammation of the entire orbit fibre with subsequent necrosis and dissolving of the orbital fibre.

Aetiology and pathogenesis. The disease is caused by staphylococci and streptococci. The infection usually extends into the orbit from the paranasal sinuses, in dental inflammatory diseases, in generalized infections, injury-related infections, as a

complication of purulent dacryocystitis, panophthalmitis, extrusion of styes and facial furuncles.

Channels of infection.

1) contact route: the shared bone wall; delicacy of the upper and inner walls; presence of aperatures and fissures; various variants of the norm, characteristic of the structure of the paranasal sinuses: in the significant distribution of the ethmoidal labyrinth cells, the large size of the frontal sinus, the optic nerve channels and optic chiasms can be closely placed to the wall of the main sinus.

2) haematogenous route (through the veins and perivascular spaces);

3) lymphogenous route (including along the perineural fissures).

Clinical presentation.

The disease develops acutely from several hours to 1-2 days. Patients complain of headache, fever (up to 39-40 degrees), chills, nausea and vomiting may be observed. Objectively, there is redness and swelling of the eyelids, the half face, exophthalmos, conjunctival oedema, restriction of the eyeball mobility, pain on the eyeball movement. Reduced visual function occurs due to compression or developed toxic neuritis of the optic nerve. There are retinal haemorrhages, retinal detachment. Prognosis in phlegmon of the orbit is serious enough not only for the visual organ, but for the patient's life. It is associated with the possibility of such complication as thrombosis of the cavernous sinus. Therefore, the treatment is carried out immediately at the hospital.

1. Broad-spectrum antibiotics, both general and topical, and sulfa drugs.

2. Cutting the orbit wide open to the depth of 4-5 cm in the presence of fluctuations. Topical use of cotton swabs with antibiotics and with hypertonic sodium chloride solution is prescribed.

Osteoperiostitis (periostitis) is an inflammation of the periosteum and bone formations of the orbit. There are simple and purulent; anterior and posterior types. *Simple osteoperiostitis* arises against the background of infectious diseases (influenza, measles, tuberculosis, syphilis). It is characterized by fever, headache, and weakness. There is swelling of the eyelids, conjunctival hyperaemia, exophthalmos

with displacement of the eyeball in the direction opposite of the localization process, chemosis, ptosis, ophthalmoplegia, diplopia. It may be accompanied by the decrease in visual function due to the swelling of retrobulbar adipose tissue. *Purulent periostitis* proceeds more severe and is characterized by the acute onset. Patients complain of high fever, general weakness, and headache.

A painful infiltration is formed on the one of the walls of the orbit, where detachment of the periosteum from the bone may occur to form a subperiosteal (periorbital) abscess. *Anterior periostitis* is characterized by localization of the process on the edge of the orbit to form infiltration and possible formation of fistulas on the skin of the eyelids. *Posterior periostitis* is characterized by infiltration in the area of the ethmoid bone, the main sinus. In such cases it is difficult to make the diagnosis.

Treatment of osteoperiostitis is carried out at the hospital. Systemic antibiotics, sulfonamides, and topical agents are prescribed. In vase of fluctuations and involvement of the paranasal sinuses, surgical treatment is required.

Neoplasms of the eye socket

Neoplasms of the orbit comprise 25% of all ophthalmic diseases.

Tumours of the orbit are divided into benign, malignant and locally destructive types.

Benign tumours are vascular, neurogenic, osteoma.

Clinical presentation. The main diagnostic features of orbital tumours are proptosis, limitation of the eyeball motion, the change in the width of the palpebral fissure, displacement of the eyeball to the side. In the fundus, congestive disc of the optic nerve, atrophy of the disc, haemorrhages are observed. There are changes in the field of vision in the form of restriction of boundaries and the emergence of scotomata. To clarify the diagnosis and differential diagnostics, CT scan with contrast and ultrasound scanning are carried out. Treatment is usually surgical.

Malignant tumours of the orbit may be primary (cancer, sarcoma, melanoma) and secondary (invasion into the orbit of the skin of the eyelids, the conjunctiva, the paranasal sinuses, the cranial cavity). The clinical presentation is characterized by pain, swelling of the eyelids, proptosis, diplopia, ophthalmoplegia. These symptoms quickly increase. For diagnosis, the doctors use the clinical presentation, computer

tomography, ultrasound scanning. Treatment of malignant tumours is carried out in combination of surgery, chemotherapy and radiation therapy.

Theoretical questions to the chapter:

1. Anatomical and physiological features of the structure of the orbit.
2. Examination methods for orbit diseases.
3. Clinical presentation, diagnostics and principles of treating the diseases of the orbit.

Practical skills which students should master:

1. To conduct an external examination of the orbit.
2. To determine the mobility of the eyeballs.

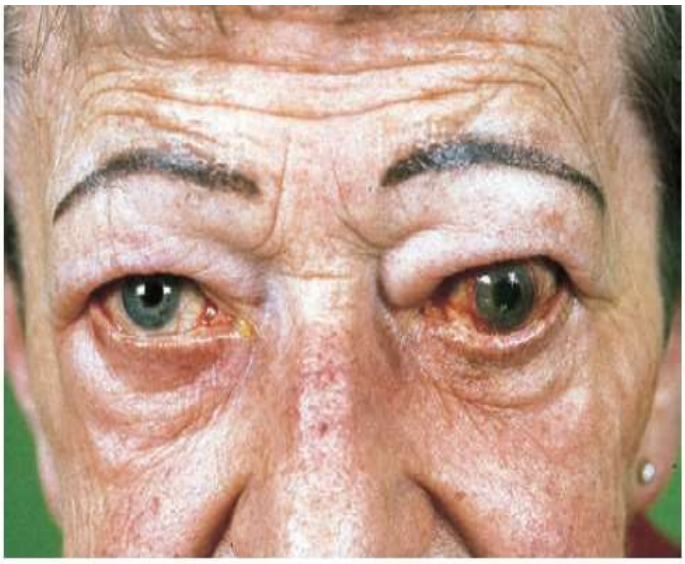


Fig. 6.3. Exophthalmos

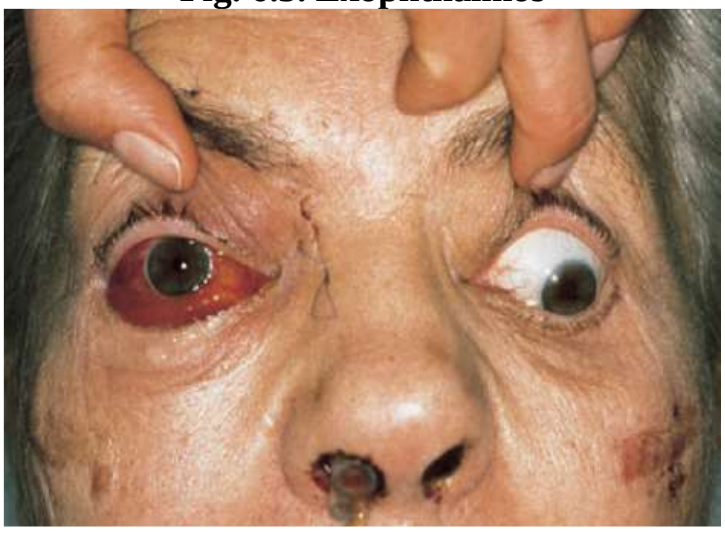


Fig. 6.4. Chemosis of the conjunctiva.

CHAPTER 7

DISEASES OF THE CONJUNCTIVA

Conjunctival diseases − conjunctivites − are among the most common pathologies among the inflammatory diseases of the eye, and comprise about 30% of ocular disorders. The most common conjunctivites are of bacterial and viral aetiology, allergic and dystrophic are observed less frequently. In recent years, the number of cases of allergic conjunctivitis has increased: they affect about 15% of the population. Acute conjunctivitis tends to occur in children, and less frequently − in the elderly people, and even more rarely − in middle-aged people. Chronic conjunctivitis is more common in middle-aged and elderly people. Of particular importance are conjunctivites in the form of epidemic outbreaks.

The conjunctiva is a connective tissue transparent membrane. It divided into three parts − the conjunctiva of the eyelids, of the eyeball and the transitional fold (the arch of the eyelids). The first part covers the posterior surface of the eyelids. The conjunctiva of the eyeball reaches the limbus. The conjunctiva of the arch is a place of transition of the conjunctiva of the eyelids into the conjunctiva of the eyeball. The conjunctiva of the eyelids consists of columnar epithelium, which has a large number of goblet cells producing mucus. Glands of Wolfring are located in the conjunctiva of the eyelids. The conjunctiva of the transitional folds contains the accessory lacrimal glands (Krause's gland) and lymphoid tissue. The conjunctiva of the eyeball is thinner. It is composed of stratified squamous non-keratinized epithelium. Blood supply of the eyelids is due to two vascular layers − the superficial and profound. The superficial layer is formed by vessels that extend from the arteries of the eyelids and anterior ciliary arteries. The profound layer is made of branches of anterior ciliary arteries, which form the circular vasculature around the cornea. Veins correspond to arteries, which carry blood to the veins of face.

The list of key terms that the students should master while studying the topic:

Term	Definition
The arch of the conjunctiva	the place of transition of the conjunctiva of the eyelids

	into the eyeball conjunctiva
Vascular injection	hyperaemia − can be superficial, deep or mixed
Chemosis	prolapsing of oedematous conjunctiva into the palpebral fissure
Blennorrhea	purulent conjunctival discharge
Conjunctival xerosis	dryness of the eye
Pannus	pathological vascularization with vessels ingrowth
Phlyctena	a nodule, consisting of lymphatic cells (epitheloid, plasmatic and lymphatic)
Pinguecula	hyperplasia of the connective tissue (lipoma)
Pterygium	a wing-shaped proliferation of the conjunctiva
Symblepharon	fusion of the conjunctiva of the eyelids with the conjunctiva of the eyeball
Pemphigus	blistering of the conjunctiva, severe chronic eye disease

Examination methods.

1. Examination of the mucosa of the eyelids by eversion method at side lighting.
2. Biomicroscopy.
3. Bacterioscopy of smears and scrapings from the conjunctiva.
4. Bacteriological examination of the discharge from the conjunctiva.

Classification of the conjunctival diseases.

1. Inflammations (conjunctivites).
2. Dystrophy (pterygium, pinguecula).
3. Neoplasms.

Inflammatory diseases.

Conjunctivites are divided into subtypes according to the course and etiological factor.

According to the course: acute and chronic.

According to the aetiology:

• bacterial − acute and chronic nonspecific catarrhal, diplobacillar, pneumococcal, acute epidemic, diphtheritic, gonoblennorrhea, gonococcal);
• chlamydial − trachoma, paratrachoma;
• viral − adenoviral fever, herpetic conjunctivitis, conjunctivitis at common viral diseases (varicella, measles, rubella), conjunctivitis, caused by molluscum contagiosum;
• fungal − candidiasis;
• allergic and autoimmune − vernal catarrh, drug-induced, pollinosis (hay fever), infectious and allergic conjunctivitis;
• conjunctivites in common diseases, metastatic conjunctivites.

Clinical signs and symptoms. Conjunctivites of different aetiologies have a similar clinical presentation: they begin acutely and are accompanied by expressed subjective sensations.

Subjective signs of conjunctivitis are smarting eyes, foreign body sensation, itching, redness, discharge from the conjunctival cavity, sometimes photophobia.

Objective signs of conjunctivitis are hyperaemia and oedema of the conjunctiva of the eyelids and transient folds, conjunctival injection of the eyeball, conjunctival discharge, the presence of the conjunctival haemorrhages, films, and follicles. Discharge can be of a different nature − mucous, mucopurulent or purulent. By the nature of discharge, one can preliminary define the aetiology of conjunctivitis. Purulent or muco-purulent discharge is characteristic of bacterial or viral aetiology. Mucous discharge is observed with allergic conjunctivitis. The appearance of the conjunctival petechial and extensive haemorrhages is possible, as well as easy detachable films. To identify the aetiology of conjunctivitis, it is necessary to conduct the laboratory studies of discharge from the conjunctiva to determine the flora and its sensitivity, as well as to evaluate the diagnostic titre of antibodies in the serum.

Bacterial conjunctivites.

Epidemic conjunctivitis is caused by Koch − Weeks bacterium. It is characterized by conjunctival hyperaemia and oedema with large and small subconjunctival haemorrhages, ischemia portions of the sclera conjunctiva in the palpebral fissure in

the form of a triangle, whose base faces the limbus. During the first days of the disease the mucous discharge is scanty. The patient cannot open the eyes because of the discharge, which glues the lashes. Subsequently, the discharge becomes abundant and purulent. Sometimes there are symptoms of intoxication. In children the process may spread to the cornea.

Pneumococcal conjunctivitis is characterized by the presence of the thin gray film on the conjunctiva. The disease begins acutely, affecting both eyes. There is a pronounced conjunctival injection, swelling of the transitional fold. On the conjunctiva of the eyelids and the arches, the delicate whitish-gray films appear that can be easily removed with a damp sponge. The conjunctiva does not bleed after removal of the film. In some cases, the process may spread to the cornea and it results in superficial marginal keratitis.

Gonococcal conjunctivitis. Gonorrhea is a veneral disease mainly affecting the mucous membranes of genito-urinary organs. It is a sexually transmitted disease and is caused by gram-negative Neisseria diplococcus (Neisseria gonorrhoeae). The source of infection is a person sick with gonorrhea. The transmission route is usually by contact infection. Gonorrheal conjunctivitis may develop in adult patients with gonorrhea of the urinary tract as a result of introduction of infection into the conjunctival cavity, as well as in people who are in direct contact with patients, due to failure to comply with hygiene rules. Infection of neonates occurs primarily during passage through the birth canal of a mother who has gonorrhea. It is clinically characterized by oedema of the eyelids, copious purulent discharge, severe conjunctival hyperaemia, formation of folds thereon. There is often an acute oedema of the conjunctival sclera (chemosis). Sometimes (5-40% of cases) there is a spread of inflammation to the cornea. At first, keratitis is superficial, then the corneal ulcer develops, which in 1-2 days may lead to corneal perforation as a result of compression of the limbus by the swollen conjunctiva of the circular vasculature.

Diphtheritic conjunctivitis is caused by diphtheria corynebacterium. This conjunctivitis is characterized by an acute and dense bluish-purple oedema of the eyelids, formation of necrotic films on the conjunctiva. When opening the eyes, the

serosanguineous nebulous liquid with flakes is discharged from the conjunctival cavity. After removing the films, the conjunctiva always bleeds. The isolated disease is extremely rare. Generally, it is accompanied by lesions of the mucous membranes of the nose, throat and pharynx.

Conjunctivitis caused by Morax − Axenfeld diplobacilli (angular conjunctivitis). As a rule, it is two-sided. The course is subacute or chronic. Patients complain of itching, redness in the corners of the eyes.

Treatment of bacterial conjunctivitis.

1. Compliance with the personal hygiene.

2. Washing the conjunctival sac with antiseptic solution (2% solution of boric acid, 0.01% myramistin, potassium permanganate solution 1: 5000, furacilin solution 1: 5000), disinfecting solutions.

3. Topical sulfonamides and broad-spectrum antibiotics are prescribed. Instillations are applied 4-6 times per day for at least 5-6 days. Morax − Axenfeld bacillus specifically responds to 0.5-1% zinc sulfate solution.

In diphtheritic conjunctivitis, patients are administered antidiphtheric serum and isolated. In order to prevent gonoblennorrhea in neonates, 20% sodium sulfacyl solution is instilled or antibacterial ointment is applied into the conjunctival cavity.

4. Non-steroidal anti-inflammatory drugs (indomethacin, diclof, diphtale topically as instillations).

Viral conjunctivites.

Adenoviral conjunctivitis is caused by adenovirus. Infection occurs by droplets or by contact. It is characterized by conjunctival hyperaemia, oedema and scanty discharge. Objectively, the follicles with point haemorrhages are observed on the conjunctiva of the eyelids. Subepithelial rash may appear of the cornea. Adenoviral conjunctivitis is characterized by general symptoms − fever, affected upper respiratory passages, headache and enlarged prootic lymph nodes. The disease can occur at any time of the year.

The causative agent of **epidemic hemorrhagic conjunctivitis** is enterovirus − 70. It is characterized by contact channel of infection. Hemorrhagic conjunctivitis is

marked by involvement of both eyes alternately. Objectively, there are acute conjunctival hyperaemia, extensive bleeding under the conjunctiva, oedema, mucous or mucopurulent discharge.

Treatment of viral conjunctivitis.

1. Antiviral drugs topically and generally as needed. As a rule, these are non-specific antiviral medications − interferon inducers, as well as synthetic low-molecular interferon inducer − cycloferon. It is administered at 2.0 ml of 12.5% solution intramuscularly once every day for 3 days. Oftalmoferon ointment, which is composed of human recombinant interferon alfa-2b, diphenhydramine and boric acid, is applied topically in the form of instillations 6-8 times per day.
2. The use of dexamethasone (at first 2 times per day, then 3-4 times per day).
3. Prevention of adjunction of secondary bacterial infections. Antibiotics: 0.3% tobrex solution, 0.3% gentamycin solution, 0.25% chloramphenicol solution, 0.3% ciprofloxacin solution.
4. If necessary, keratoplasty, vitamins, decongestants are applied.

Chlamydial conjunctivites. Chlamydiae are intracellular microorganisms which possess properties of viruses, as well as bacteria. **Trachoma** is a chronic infectious keratoconjunctivitis that occurs as a result of pathogen's contact with the conjunctiva. The incubation period is 1-14 days. At first, the conjunctiva becomes rough; then follicles appear, with their subsequent scarring. The process may end with recovery, but complications may occur in the form of corneal vascularization (pannus) and cicatricial deformity of the eyelids. Severe consequences of trachoma are as follows: formation of symblepharon; degeneration of lacrimal (accessory) and meibomian glands, resulting in corneal xerosis; cicatricial deformity of the cartilage of the eyelids, entropion of the eyelids, trichiasis.

Treatment of trachoma. Topical antibacterial and combined medications, non-steroidal anti-inflammatory drugs, antiallergic agents, improving reparation.

Allergic conjunctivitis.

According to the world summarized data, more than 30% of the population suffers from various allergic diseases. The disease occurs when a person is exposed to

allergens. It is characterized by oedema and hyperaemia of the conjunctiva of the eyelids and the eyeball, formation of the papillae on the conjunctiva. The surface of the conjunctiva resembles “cobble-stone pavement”. Patients complain of itching, burning and sensation of a foreign body. Allergic conjunctivitis is periodically aggravated.

Treatment of allergic conjunctivites.

1. Termination of contact with the suspected allergen.
2. Topical therapy includes application of antihistamines: opatanol, antazoline + tetryzoline; allergoftal; allergodil 0.05%; agents, inhibiting degranulation of mast cells: 2% lecrolyn, 2% cromohexal; 0.1% alomide, 2% cusicrom; corticosteroids: dexamethasone, maxidex.
3. Specific immunotherapy.

Pterygium is a wing-shaped proliferation of the conjunctiva. It is a triangular thickened and vascularized fold of the scleral conjunctiva in the inner corner of the eye. It develops usually in older people, who work in the environments contaminated with dust or chemical particles.

In case of progression and invasion of pterygium into the cornea, *surgical treatment* is conducted.

Pinguecula is a small prominent islet on the white background of normal scleral conjunctiva, made of thickened yellow tissue. It occurs usually at the inner margin of the cornea.

It does not require treatment.

Neoplasms of the conjunctiva.

1. Benign − nevi, papillomas, retention cysts.
2. Malignant − epitheliomas, melanomas.

Conjunctival **papillomas** are removed surgically. If the nevi are growing and increasing, they are removed by cryodestruction.

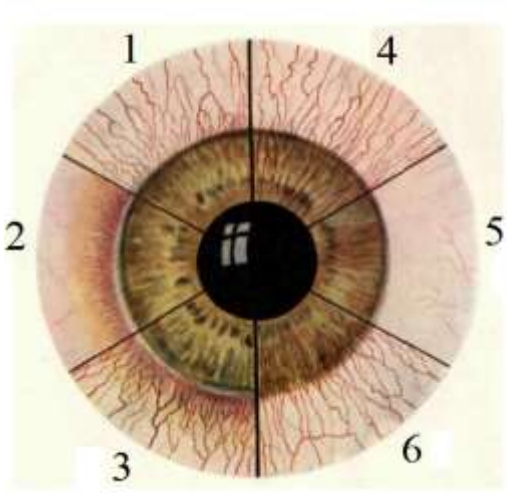
Epithelioma is a white-pink tumour on a broad basis. The tumour can spread to the conjunctiva of the sclera, and the cornea. Treatment consists in their early removal.

Theoretical questions to the chapter:

1. Anatomical and physiological characteristics of the structure of the conjunctiva.
2. Name the methods for examination of the conjunctiva.
3. Describe how to take a swab from the conjunctival cavity.
4. Describe how to make the analysis of the microflora.
5. What are the symptoms of Koch − Weeks conjunctivitis?
6. What are the types of viral conjunctivites?
7. Describe the clinical presentation of Morax − Axenfeld diplobacillar conjunctivitis.
8. Describe the aetiology, pathogenesis and clinical presentation of trachoma and its classification.
9. What are the complications of trachoma?
10. Describe the aetiology, pathogenesis and clinical presentation paratrachoma.
11. Describe the clinical presentation of vernal conjunctivitis.
12. Describe the clinical presentation and pathogenesis of tuberculous and allergic conjunctivitis.
13. Name the degenerative changes of the conjunctiva; describe pterygium and pinguecula.
14. Name and describe the benign and malignant tumours of the conjunctiva.
15. Describe the changes of the conjunctiva in case of systemic diseases.

Practical skills which students should master:

1. To conduct the external examination of the mucosa.
2. To conduct the eversion of the eyelids.
3. To check the secretory function of the conjunctiva.



Different types of eye injections

1) conjunctival injection
2) pericorneal injection
3) mixed injection
4) superficial vascularisation of the cornea
5) deep vascularisation of the cornea
6) mixed vascularisation of the cornea

Fig. 7.1. Types eyeball injection

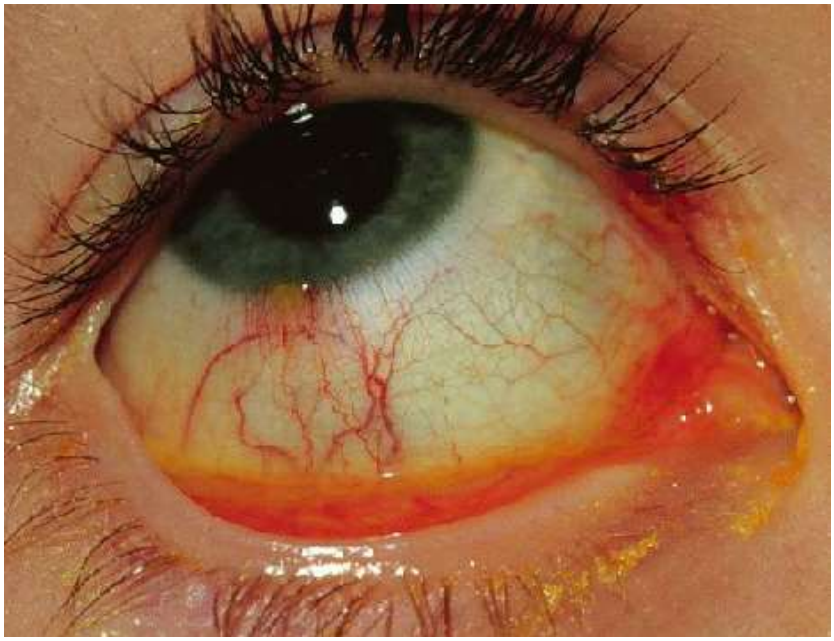


Fig. 7.2. Bacterial conjunctivitis

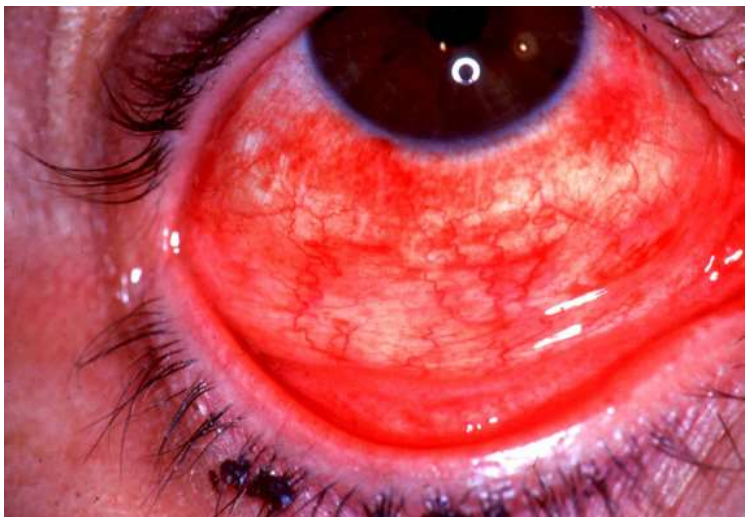


Fig. 7.3. Viral conjunctivitis

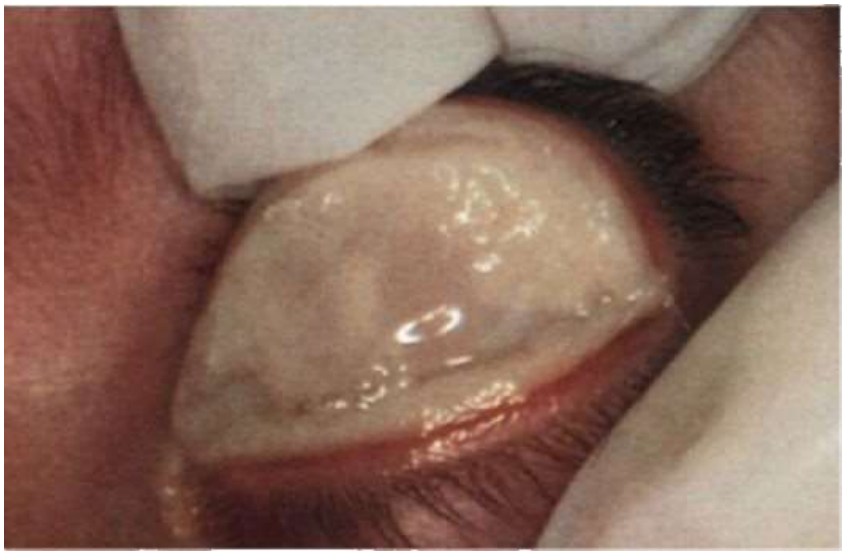


Fig. 7.4. Diphtheritic conjunctivitis

Fig. 7.5. Pneumococcal conjunctivitis

CHAPTER 8

DISEASES OF THE CORNEA

The cornea is the most important optical medium of the eye. The share of corneal diseases makes up more than 25% of the eye pathologies, and their consequences are often the cause of persistent loss of vision and blindness (almost 50% of cases). The cornea is a part of the fibrous membrane of the eye which occupies 1/6 of it, whereas the sclera − 5/6 of the entire outer membrane. It performs protective and optical functions. The limbus is place of cornea's transition into the sclera (1 mm).

The list of key terms that students should master while studying the topic:

Term	Definition
Cornea	Normally is of ellipsoid shape, the size is on average 10x11 mm
Microcornea	Undersized cornea
Megalocornea	Enlarged cornea
Keratoconus	Change of cornea's shape (cone-shaped cornea)
Keratoglobus	Change of cornea's shape (globe-shaped cornea)
Xerosis	Dryness of the cornea
Keratomalacia	Softening and disintegration of the cornea
Nubecula, macula, leukoma	A wall-eye, cloud, spot
Corneal syndrome	photophobia, lacrimation, blepharospasm, sensation of a foreign body
Pericorneal injection	Deep injections of vessels that results from inflammation of the anterior part of the eye
Vascularization	Invasion of vessels in the cornea
Phlyctena	Nodule of circular shape, which is a substrate of non-specific

	inflammation
Hypopyon	Pus in the anterior chamber
Keratomycosis	Fungal lesion of the cornea
Ectasia	Limited protrusion of the sclera
Staphyloma	Significant thinning with protrusion
Scleromalacia	Inflammatory nodules that can create the defects of the sclera

The cornea is the front part of the fibrous membrane. The cornea is transparent, shiny, smooth, it has a convex shape, due to which it serves as an optical lens; it is avascular and highly sensitive. It has a certain size. Corneal thickness is 0.6-1.0 mm, size − 11-12 mm, the optical power − on average 42.0 dioptres. The cornea is not permanently protected by the eyelids and therefore it may be subject to mechanical and chemical damage. The cornea is nourished by 3 sources: the looped marginal network, formed by anterior ciliary arteries and located in the limbus, the humour of the anterior chamber and lacrimal fluid. The main blood supply is carried out by the circular vascular network along the limbus. Oxygen enters the cornea directly from the air.

The cornea is composed of five layers: the anterior epithelium, the anterior marginal membrane (Bowman's membrane), the corneal substance proper, the posterior marginal membrane (Descemet's membrane) and the posterior epithelium or endothelium. The *anterior layer* (epithelium anterius) is represented by non-keratinized stratified squamous epithelium. It is easily regenerated if damaged. The epithelium performs the protective function and regulates the entry of moisture into the cornea from the conjunctival cavity. *The anterior marginal plate, or Bowman's membrane* is inelastic, smooth and with low metabolism; it is not capable of regeneration. *The corneal substance proper* occupies its basis weight, about 90% of its thickness and consists of recurrent and uniform plate structures. The structure of these plates includes collagen plates that are interconnected by interstitial substance. *Entocornea (lamina limitans posterior), or Descemet's membrane*. It is highly elastic

and resistant to damaging factors. *The inner epithelium or endothelium*. This layer functions as a membrane that protects the stroma from impregnation with the anterior chamber liquid. The epithelium is not restored when damaged.

The cornea has three kinds of sensitivity − sensitivity to pain, thermal and tactile. Branchlets of nerve trunks, which innervate the cornea, have no myelin sheath.

Examination methods.

1. The side lighting method.
2. The method in transmitted light.
3. Biomicroscopy.
4. Test with fluorescein.
5. Testing the corneal sensitivity.

Detection of the epithelial defects (fluorescein test)

Defects of the corneal epithelium are detected using 1% fluorescein solution. This solution is instilled into the conjunctival sac, and biomicroscopy study is conducted: epithelial defects are painted in green. The presence of these symptoms helps in differential diagnosis of immediate and cicatrical changes of the cornea.

Testing the corneal sensitivity. To check the corneal sensitivity, a cotton swab, rolled in a very thin flagellum is used. The patient sits with eyes wide open. The central corneal part is touched with cotton flagella, and then in 4 points at the periphery (12, 15, 18, 21 h respectively). The patient reports whether he/she feels the touch of the flagellum, and the physician observes the presence of the blinking reflex. Assessment: the method reveals severe sensory disturbances.

Fig. 8.1. Testing the corneal sensitivity.

Classification of diseases of the cornea:

• malformations (megalocornea and microcornea, keratoconus and keratoglobus).

• inflammatory (keratites, sclerites);

• dystrophies (congenital (primary) − macular, nodular and lattice; and acquired (secondary) − senile arc (arcus senilis) and endothelium-epithelium dystrophy.

• tumours.

Inflammatory diseases of the cornea.

Most of the pathologies of the cornea are inflammatory diseases, or **keratites**.

Classification of keratites.

1. By origin − exogenous and endogenous.
2. By the clinical course − acute and recurrent.
3. By the depth of the lesion − superficial and deep.
4. By the nature of inflammation − purulent or non-purulent.
5. By the localization − central, paracentral, peripheral.
6. By the extent − limited and diffuse.
7. By the form − punctate, keratitis nummularis, in the form of twigs and strokes.
8. By the outcome − with and without vascularization.

Exogenous keratites:

• erosion of the cornea;

• traumatic keratites caused by mechanical, physical or chemical injury (post-traumatic keratites);

• infectious keratites of bacterial origin;

• keratites caused by diseases of the conjunctiva, eyelids, meibomian glands;

• fungal keratites, or keratomycoses.

Endogenous keratites:

• infectious keratites:

• tuberculous: haematogenous and allergies;

• syphilitic;

• herpetic;

• neuroparalitical;

• avitaminous.

The majority of keratites are characterized by common symptoms, which includes **subjective** and **objective signs**. Photophobia, lacrimation, blepharospasm, foreign body sensation under the upper eyelid, redness of the eyeball, reduced visual acuity, in the central localization process in the cornea − are the **subjective symptoms**. Photophobia, lacrimation, blepharospasm comprise the *corneal syndrome.*

The **objective signs** of keratites include: pericorneal injection of the eyeball, the presence of corneal infiltrate. The shape, colour, size of infiltrate may be different. Hence, with a small cluster of leukocytes infiltrate has a grayish colour, with purulent melting − yellow, with severe vascularization − a rusty hue. Borders of infiltrate are always fuzzy due to swelling of surrounding tissue areas. The cornea in the area of infiltrate always loses its gloss, it becomes dull. In the infiltrate areas, sensitivity of the cornea is usually reduced or absent, depending on the aetiology of keratitis. Infiltrates may have different shapes (round, as branches of the tree, discoid) and localized in different parts of the cornea (centre, paracentral, at the limbus).

Herpetic keratites.

The disease is caused by herpes simplex virus. Herpetic keratitis is one of the most frequent diseases of the cornea and makes up 20-50% of all corneal diseases in hospitalized patients.

Herpetic keratites are of the following forms; vesicular, dendriform (tree-like) and disc-shaped

Vesicular and dendriform keratites are the surface forms, whereas the disk-shaped type is a deep form.

Vesicular keratitis is characterized by the appearance of vesicles on the corneal epithelium which burst and form shallow ulcers. The latter are gradually epithelized. Dendriform keratitis is characterized by infiltrate in the form of tree branches on the corneal epithelium. Most often, the infiltrate is located in the centre of the cornea.

Discoid keratitis refers to a deeper form, which is not dyed with fluorescein. It is characterized by the appearance of infiltrate in the form of a disc with clear boundaries in the centre of the cornea. It may be complicated by corneal ulcer.

Treatment.

1. Topical and general antiviral therapy. Interferon inducers in drops (ocoferon, oftalmoferon) in the form of instillations 6-8 times per day are topically administered. Medications are also applied both topically as drops or eye ointments and orally or intramuscularly: acyclovir orally, or valacyclovir (2 times daily), zovirax, viroleks, famvir, valtrex in tablets (5 times a day).

2. In superficial defects, epithelium is treated with 1% alcoholic solution of brilliant green or 5-10% alcohol solution of iodine, using the means reparative therapy (ocular drops emoxipine, vitacic, taufonum or carnosine, solcoseryl eye gels, actovegin, corneregel).

4. Anti-inflammatory therapy − the use of eye drops with nonsteroidal anti-inflammatory drugs (diclofenac, naclof, diclof, indocollir).

5. In the secondary bacterial infection, eye drops containing aminoglycoside or fluoroquinolone are prescribed.

6. Agents stimulating reparation of the cornea (corneregel, 4% taufonum solution, 1% tiotriazolin, 1% quinine, solcoseryl gel).

Bacterial keratites.

The causative agent of bacterial keratitis may be staphylococci, streptococci, pneumococcus. Inflammation usually develops against the background of blepharites, chronic dacryocystites, styes, conjunctivites, corneal injuries. The disease begins acutely. It is characterized by the appearance of the corneal syndrome, redness of the eyeball, loss of visual acuity. Objectively, infiltration is detected on the cornea, which quickly turns into an ulcer. The ulcer has edges and a gray-coloured bottom. Corneal ulcer serpens is characterized by presence of the progressive and regressive edges. Progressive edge is slightly raised, mined and surrounded by a strip of purulent infiltrate. The opposite (regressive) edge is clean. When the inflammatory process spreads to the deep areas of the cornea, destruction gradually approaches Descemet's membrane. The formation of descemetocoele − protrusion of Descemet's membrane is possible. At the bottom of the anterior chamber, the horizontal strip of pus (hypopyon) appears. Subsequently, perforation of Descemet's membrane may occur,

which is accompanied by outflow of the anterior chamber humour. The iris may be inserted in the perforated aperture or get constrained therein. In this case, the infection can penetrate the eye and cause purulent inflammation of the membranes of the eye − panophthalmitis.

Keratoplasty (tectonic, therapeutic) is required if there is a threat of ulcer perforation.

Treatment.

1. Antibacterial agents are prescribed topically in the form of drops and for systemic effect.
2. Sampling smears from the conjunctiva to determine the pathogen.
3. Prescription of mydriatics.
4. Non-steroidal anti-inflammatory drugs (diclof, indocollir).
5. If necessary, cauterizing of ulcer edges with 3% alcohol solution of iodine or brilliant green.
5. Prescription of immunomodulators, vitamins, and agents improving corneal reparation.

Fungal infections of the cornea.

Keratomycoses are caused by different types of fungi that are normally found in the conjunctival sac and lacrimal pathways. Long-term use of antibiotics and corticosteroids disrupts this balance and leads to the activation of fungal flora which becomes pathogenic. Keratomycosis occurs as a secondary infection in trachomatous pannus. Metastatic pathway of corneal infection is possible in case of general mycosis.

Clinical presentation. Keratomycosis is characterized by the association with microtrauma. At the site of erosion, a grayish-white focus with crumbly loose surface appears; it is surrounded by a yellowish border. The process slowly progresses; it deepens, the infiltrate decomposes to form a crater-like ulcer, hypopyon increases. Perforation of the ulcer may occur. Severe consequences are observed in untimely diagnosis and treatment with antibiotics and steroids. For diagnosis, it is necessary to conduct the microscopic examination of pathological material by fungal culturing, histological examination of corneal sequestrating sites.

Treatment. Nystatin and levorinum are used orally. Topical non-steroidal anti-inflammatory drugs and anti-fungal agents are prescribed.

ENDOGENOUS KERATITES

Tuberculous-allergic keratites

Tuberculous-allergic keratitis (phlyctenular or scrofulous keratoconjunctivitis) belongs to endogenous corneal diseases. It occurs most often in scrofula patients (children or adolescens). As a rule, the prerequisite for development of scrofulous keratoconjunctivitis is the TB infection, TB of the glands, the presence of active TB focus in the lungs. This gives rise to inflammatory sensibilization of the body, and in particular of the eye tissue, to the toxin of tubercle bacillus. Nonspecific factors also contribute to the development of the disease, such as metabolic disorders, vitamin A deficiency, systemic diseases, and adverse living conditions. On examination of patients, one should pay attention to the characteristic manifestations of this disease: acute photophobia (patients lie with face down in the pillow), blepharospasm. The eyelids are usually frantically compressed and it is very difficult to open them. On the cornea, phlyctenas (vesicles) are observed. Phlyctena is a nodule, which includes lymphocytes, epithelioid and giant cells. Phlyctena is different from a tubercle by the absence of tubercle bacilli in it. The structure of phlyctena indicates that this is not actually a tubercular inflammation, but only a kind of eye's reaction to the tubercular toxin.

Phlyctenas may be located on the conjunctiva of the eyelids, the eyeball, along the limbus, and finally, on the cornea. Their dimensions are different − from miliary to large, solitary phlyctenas. Usually they have an appearance of grayish protruding nodules, with adjoining superficial blood vessels. Phlyctenas of the cornea usually leave behind a thin and tender clouding in the surface layers. In some cases phlyctena decomposes and turns into an ulcer of the cornea. In phlyctenular keratitis, the iris is often involved in the process, and its inflammation arises (iritis), which is manifested by pupil's constriction, its flaccid response to light.

Treatment is conducted together with the phthisiologist.

1. Diet − hypocarbohydrate, hypochloride, rich in fats and vitamins.

2. Antituberculous chemotherapy orally (ftivazide, tubazide, para-aminosalicylic acid, streptomycin).

3. Topical instillations into the conjunctival cavity of mydriatics, steroidal anti-inflammatory drugs.

4. General desensitazition.

5. Vitamin therapy.

6. Specific congenital syphilis therapy with antibiotics and then preparations of mercury, silver and others under dermatovenerologic dispensary.

7. Topical use of antibiotics, sulfonamides, nonsteroidal anti-inflammatory drugs, vitamin therapy, agents improving the corneal reparation.

Neurogenic keratites.

Neurogenic keratites include *neurotrophic* and *neuroparalitical keratites*. They develop due to lesions of the trophic fibres of the trigeminal nerve at any portion of it. Neuroparalitical keratitis is caused by viruses (adenovirus, herpes simplex, herpes zoster, etc.).

Regardless of the cause, neurogenic keratites are associated with trigeminal nerve, which is manifested in the dramatic reduction or absence of corneal sensitivity. There are delayed regeneration of corneal defects, and a tendency to relapse.

The clinical presentation. In the superficial layers of the cornea, usually in its central parts, gray limited infiltrate of various sizes and shapes appears. The process gradually spreads. Subsequently, epithelium is rejected and sharply contoured flat ulcers of various shapes and sizes are formed. Corneal syndrome is virtually absent. This is due to the lack of sensitivity of the cornea. Subjectively, the disease causes almost no sensation. Only at the beginning, there is a slight pericorneal injection of the eyeball. The ulcer heals slowly, leaving behind a gentle clouding. A secondary infection may join, with development of purulent ulcers of the cornea.

Treatment is directed at improving the trophic properties of the cornea. Topical instillations of solutions of emoxipin, taufonum, oily solution of vitamin A are used. Actovegin gel, solcoseryl, corneregel are applied in the conjunctival cavity. Solcoseryl intramuscularly is administered to stimulate the reparation processes. For

prevention of secondary infection, instillations of antibiotics or sulfonamides solutions are prescribed. NSAIDs (ortophenum, diclofenac, indomethacin, voltaren and others), Vitamin B complex, ascorbic acid are administered intramuscularly or orally. To promote healing, laser stimulation and magnetic therapy with keratoplastic drugs are used.

Avitaminous keratites.

This type of keratites develops due to deficiency of vitamins in the diet or because of endogenous vitamin deficiency, which is observed in diseases of the gastrointestinal tract, vitamin metabolism disorders, liver disease and other conditions.

The clinical presentation. Depending on the severity, one can observe pre-xerosis, xerosis and keratomalacia.

Treatment. In the first place there are vitamin therapy and fight against secondary infection. For stimulation of reparative processes, actovegin, solcoseryl, corneregel are prescribed. For prevention of secondary infection, instillations of solutions of antibiotics and sulfonamides are applied. If necessary, parabulbar and subconjunctival injections of antibiotics are made. Intramuscularly NSAIDs are prescribed.

Outcomes of the diseases of the cornea

All processes in the cornea, depending on the aetiology of the disease, intensity of the process, its duration and recurrence result in its clouding of various degrees.

Corneal opacities are divided into nubecula, macula and leucoma.

Treatment: conservative and surgical.

Conservative comprises administering absorbable means in the form of drops or ointments, tissue preparations, enzymes, corticosteroids, etc., oxygen therapy.

Surgery involves lamellar and penetrating keratoplasty which may be conducted for the optical purpose − to restore visual acuity; medical − in keratites and ulcers that are not subject to drug treatment.

Theoretical questions to the chapter:

1. Anatomical and physiological structure of the cornea and sclera.
2. What are the methods of examination of the cornea and sclera?

3. Describe how to test the corneal sensitivity.

4. Describe the corneal syndrome.

5. What are the types of corneal abnormalities in shape and size?

6. Identify keratites of exogenous nature.

7. Identify keratites of endogenous nature.

8. Describe the aetiology, pathogenesis and clinical presentation of corneal ulcer serpens.

9. What are the complications of corneal ulcer serpens?

10. Describe the aetiology and pathogenesis of keratomycoses.

11. Describe the aetiology and pathogenesis of syphilitic parenchymal keratitis.

12. Describe the aetiopathogenesis of tuberculous allergic keratitis. Clinical features, diagnosis and treatment.

13. Describe the aetiopathogenesis of neuroparalytic keratitis.

14. Describe the clinical presentation and treatment of primary herpetic keratites.

15. Describe avitaminous keratites. Clinical presentation. Treatment. Prevention.

16. Identify the primary corneal dystrophies. Groenouw dystrophy, Fehr macular dystrophy, Dimmer lattice dystrophy, Messman dystrophy, François dystrophy, Schnyder dystrophy.

17. Describe the aetiopathogenesis of scleritis, episcleritis. Diagnostics. Modern methods of treatment.

Practical skills which students should master:

1. Conducting the external examination of the cornea.

2. Conducting the side or focal lighting.

3. Determine the corneal sensitivity.

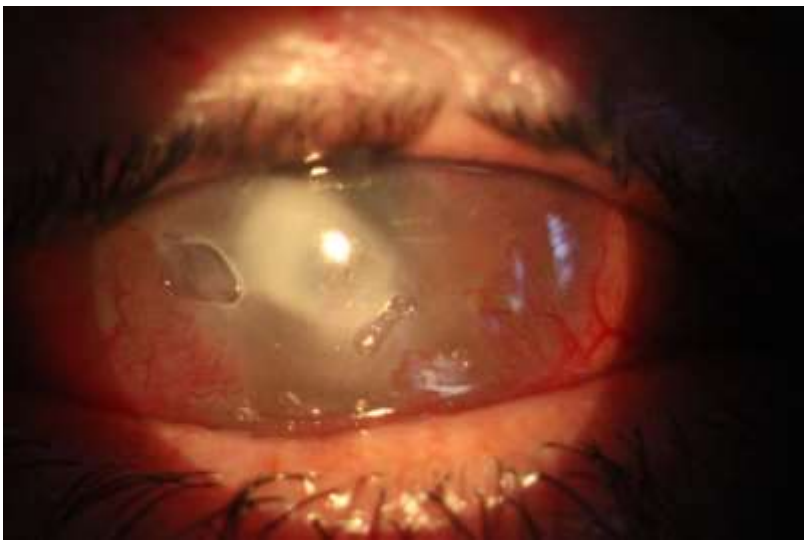


Fig. 8.2. Corneal ulcer.

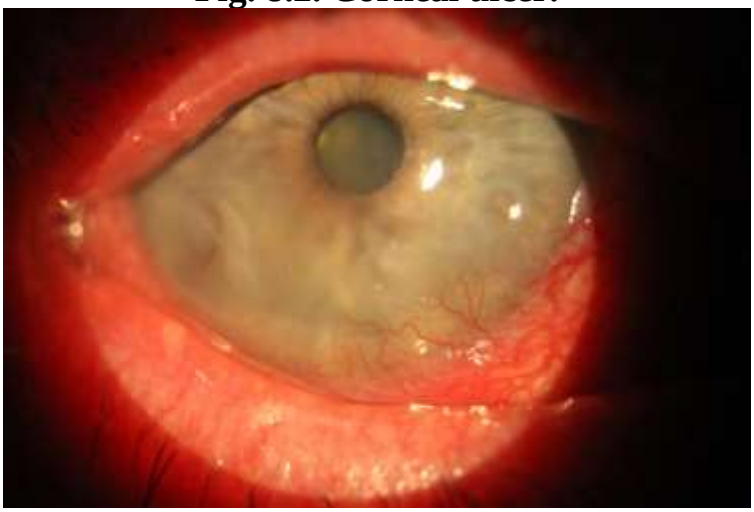


Fig. 8.3 Bacterial keratitis.

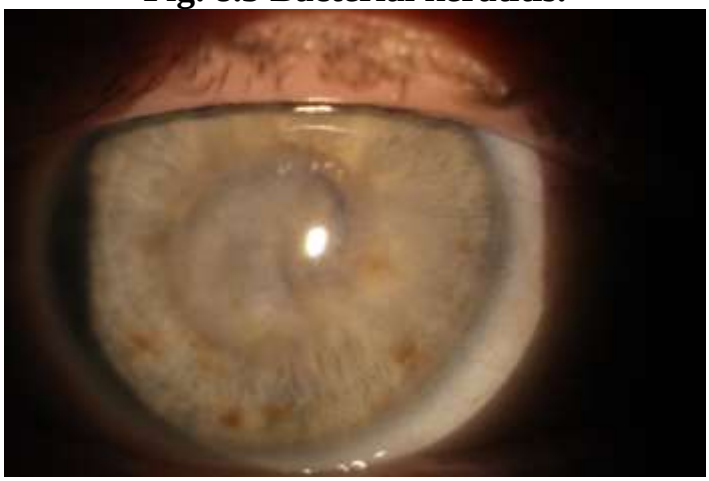


Fig. 8.4 Corneal clouding (macula).

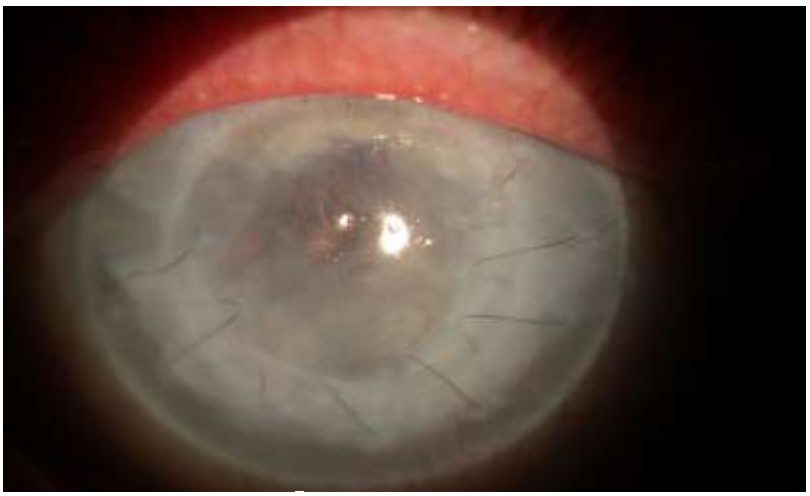


Fig. 8.5. Corneal graft disease

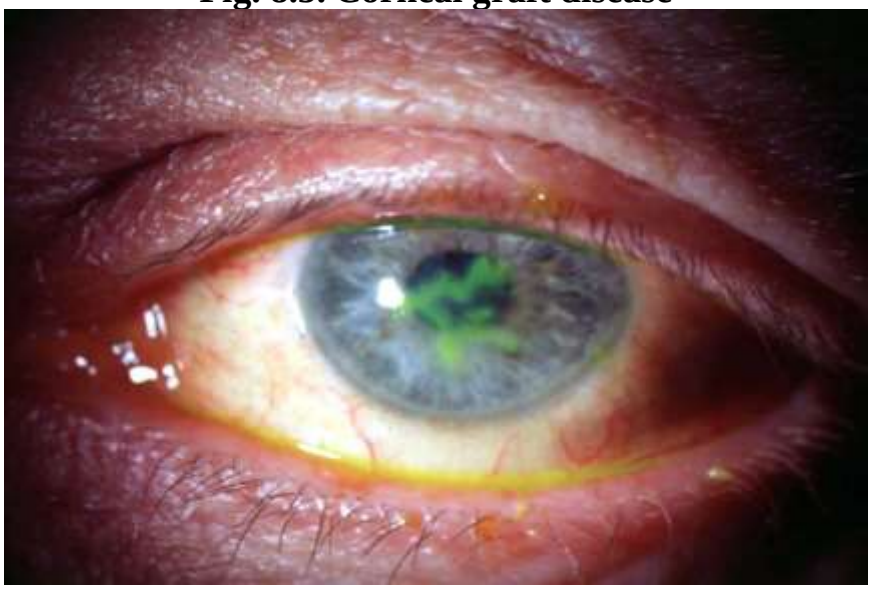


Fig. 8.6. Dendriform keratitis

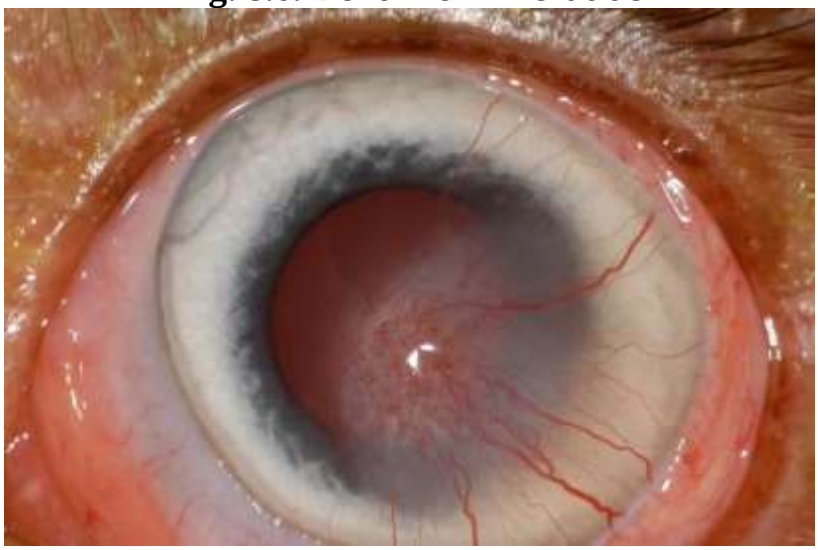


Fig. 8.7. Tuberculous (phlyctenular) keratitis.

CHAPTER 9

DISEASES OF THE SCLERA

The sclera forms the posterior portion of the outer membrane, it is of white colour and is composed of dense collagenous fibres. It accounts for 5/6 of the fibrous membrane of the eye. Most of the sclera is hidden in the orbit, and in the range of palpebral fissure, two white triangles are visible on both sides of the cornea. From behind, through the sclera, the optic nerve passes. In place of its exit, the sclera is thinned and has the form of a thin connective plate with numerous apertures through which the fascicles of the optic nerve pass (lamina cribrosa). The sclera changes over into the cornea not through the entire thickness at once. Deeper layers change earlier, the superficial ones − later. Therefore, the deep peripheral layers of the transparent corneal surface are covered with more transparent layers of the sclera. This semi-transparent formation is called the limbus. The sclera is the place of attachment of the external oculomotor muscles. These areas are the least resistant at eye traumas, especially the blunt ones, therefore, ruptures of the sclera often occur there. Blood supply of the sclera is provided by the marginal vascular network − the vessels transiting through the sclera and springing small episcleral branchlets, and by diffusion of nutrients from the liquid entering the suprachoroidal space, which can permeate the sclera. Therefore, the sclera, being poor in vessels, is little susceptible to the disease of metastatic origin.

The list of key terms which students should master while studying the topic:

Term	Definition
Ectasia	Limited protrusion of the sclera
Staphyloma	Significant thinning with protrusion
Scleromalacia	Inflammatory nodules, that can create defects of the sclera
Scleritis	Inflammation of the sclera

Methods for examination of the sclera.

1. Method of side (focal) lighting.
2. Biomicroscopy.

3. Diaphanoscopy.

4. Ultrasound study.

Classification of the diseases.

1. Congenital anomalies.

2. Inflammatory (sclerites, episclerites).

2. Dystrophic (ectasia and staphylomas).

3. Tumours.

Inflammatory diseases.

The most frequent diseases of the sclera are sclerites and episclerites.

Aetiology and pathogenesis. The main causes are systemic diseases (rheumatic fever, tuberculosis, sarcoidosis, systemic lupus erythematosus, syphilis), and viral infections. Re-exposure may be during the transition of the inflammatory process from the conjunctiva, cornea, periorbital tissues. Depending on the area affected, the process can be focal or diffuse. Depending on the depth of the lesion − superficial (episcleritis) and deep (scleritis).

Episcleritis is a focal inflammation of the superficial layers of the sclera. It is clinically characterized by one-sided eye redness, slight soreness, mild discomfort, limited swelling. Lacrimation and photophobia are slightly manifested.

Scleritis is an inflammation of deep layers of the sclera. It is clinically characterized by gradual development (for several days), limited redness and constantly increasing pain, radiating to the temporal area, the eyebrows and the jaws. One can distinguish the anterior and posterior sclerites, acute and chronic forms.

Anterior scleritis affects the area between the limbus and the equator of the eye. It is manifested by limited swelling of dark and violet colour, which s heavily protrudes over the surface of the sclera. The process is usually two-sided. It may also affect the cornea and the choroid.

Posterior scleritis is an inflammation localized in the posterior part of the sclera. It is characterized by swelling of the eyelids and the conjunctiva (chemosis), protrusion of the eyeball (proptosis). Tenonitis and limited mobility of the eyeball are observed. Sclerites have a long course, they are recurrent.

Purulent scleritis or scleral abscess is the most severe form of inflammation in the sclera. It begins acutely, there is a limited deep red colour swelling with a yellowish tinge and purulent infiltration. The infiltrate is sharply painful. There is a corneal syndrome, oedema of the conjunctiva and the eyelids. At the site of infiltration, the abscess forms. It may open and thus cause the development of endo- and panophthalmitis that often lead to blindness.

Treatment of scleritis and episcleritis depends on the aetiology of the pathological process. General and local treatments are prescribed. General therapy includes antibacterial, desensitizing, systemic medications. Topical corticosteroids and anti-inflammatory drugs are used; in the normal intraocular pressure, instillations of mydriatic agents are applied; in the increased intraocular pressure − antihypertensive drops are prescribed.

Dystrophic diseases.

This group of diseases is represented by changes in the shape and thickness of the sclera − these are ectasia and staphyloma. Ectasia and staphyloma may be congenital and acquired. Congenital diseases are associated with impaired intrauterine growth, increased intraocular pressure. Acquired diseases occur due to traumas, inflammatory diseases of the eyeball. They are clinically characterized by the appearance of spots and protrusions on the sclera. They may be of colours from bluish-black to slate black. Depending on the localization, one can distinguish intermediate, ciliated, equatorial and posterior scleral staphylomas and ectasias, capable of entailing changes in the corneal curvature, astigmatism development which leads to the decrease in vision. Scleromalacia may occur in the elderly people. The causes are collagenoses, metabolic disorders, vitamin deficiencies.

Surgical treatment is the only option. It is aimed at strengthening the sclera and closing the defect.

Neoplasms.

Cysts and *tumours of the sclera* are very rare. In most cases, they are secondary, developing as a result of invasion of epibulbar and intraocular tumours (melanoma,

retinoblastoma). In the large-sized scleral cysts, their removal and scleroplasty are prescribed. In malignant tumours, eye enucleation is conducted.

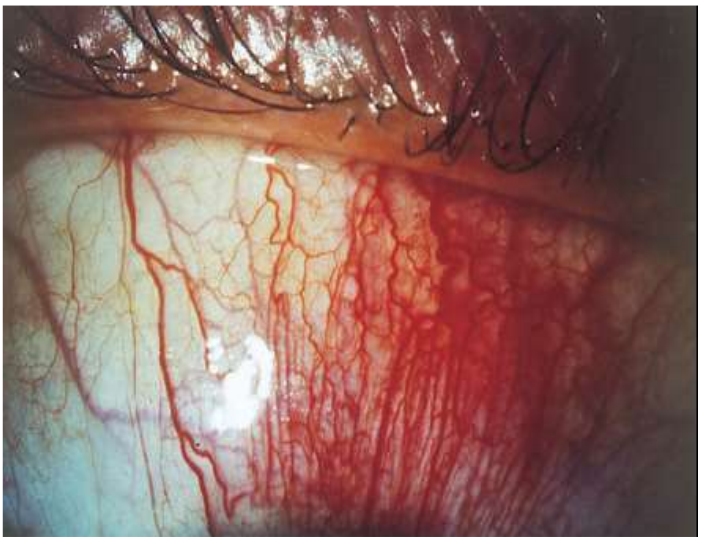


Fig. 9.1. Episcleritis

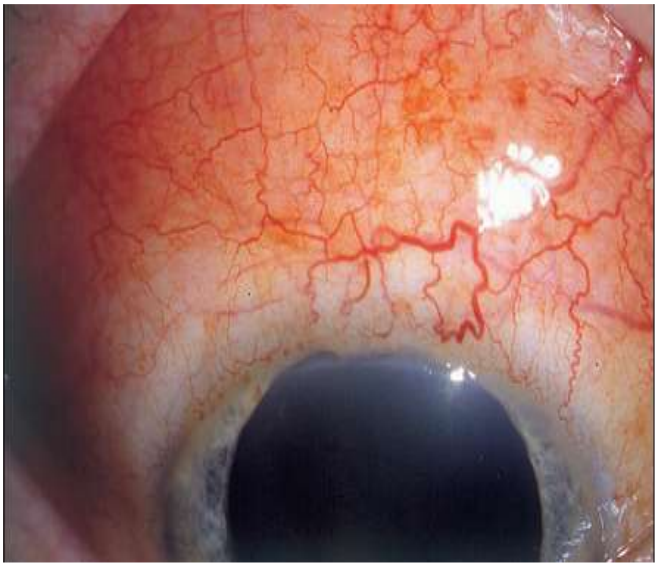


Fig. 9.2. Diffuse scleritis

CHAPTER 10

DISEASES OF THE LENS AND VITREOUS BODY

The lens is a biconvex structure of the human eye, which is located behind the iris, and in front of the vitreous body. The optical power of the lens is about 15 dioptres. However, it is not constant and it changes depending on the curvature of the lens, which in turn depends on the accommodation tension. After the age of 40, the accommodation ability is decreased by reducing the eye muscle strength, as well as the compaction and hardening of the lens nucleus − phacosclerosis. As a result, patients start seeing worse the objects at close range. This condition is called presbyopia. During this period, due to disrupted nourishment of the lens and deceleration of metabolism therein, the initial lens opacity can occur in its various layers. In the structure of the lens there are anterior and posterior capsules along the equator, intertwining with each other and forming a single capsular sac. The inner part of the lens, the densest one, is called the nucleus. The outer layers of the lens material are called the cortex. The cells off the lens constantly multiply.

The list of key terms, parameters, characteristics which the students should master while studying the topic:

Term	Definition
The main function of the lens	Transmission and refraction of light, participation in the accommodation.
The structure of the lens	The capsule (anterior, posterior), the cortex, the nucleus.
The source of nourishment of the lens	Intraocular fluid
Biochemical composition of the lens	Water, proteins, inorganic substances, amino acids.
Examination methods of the lens	The side lighting method, the transmitted light method, biomicroscopy.
Classification of the diseases of	Developmental abnormalities, cataract, lens

the lens	dislocation, aphakia.
Methods of treatment of cataract	Conservative (vitamin drops, vitamins orally), surgical (intracapsular extraction, extracapsular extraction, phacoemulsification of cataract)
Aphakia, its signs	The absence of the lens; signs − iridodonesis, deep anterior chamber, high hypermetropia, the lack of accommodation.
Methods of aphakia correction	glasses, contact, intraocular correction.

The lens contains no blood or lymphatic vessels and nerve fibres. It is nourished by the diffusion or active transport of nutrients and oxygen, which are dissolved in the intraocular fluid, through the capsule. The condition of the lens transparency is ensured by the balanced physical and chemical composition of its membranes, the content of water and ions, intake and release of metabolism products, as well as peculiarities of its structure. The human lens consists essentially of proteins and water (the latter accounts for about 65% of the weight of the lens).

From the birth and throughout a person's life, there is a gradual change in size, shape, consistency and transparency of the lens. Thus, in the newborn it is almost spherical, soft, and transparent. Further, with age, it assumes the shape of a biconvex lens, with a flat front surface, and acquires a yellowish tint, but its transparency is fully retained. With age, this yellowish saturation increases.

Examination methods.

1. The side lighting method.
2. Examination in the transmitted light.
3. Biomicroscopy.
4. Refractometry.

Classification of the diseases.

1. Developmental abnormalities.
2. Dislocation.
3. Cataract.

Cataract is a disease of the eye, the main feature of which is the reduced transparency of the substance or the capsule of the lens, which is accompanied by the decreased visual acuity. The causes of this clouding may be eye injuries, different diseases, and certain medications. However, most often it is caused by the age-related changes. In cataract, the vision worsens, the contours of objects are blurred, the colours become dull, and the dark vision deteriorates. In Greek, the word "cataract" means "falling down" or "a waterfall". In ancient Greece, it was believed that in cataract the gray colour of the pupil is determined by the presence of the film, which, like a waterfall, descends down on the pupil, taking away the person's vision. In senile cataract, the changes in the chemical composition of the lens occur. At the initial stage of cataract, the amount of fluid in the lens increases, resulting in the imbalance of ions, amino acids, reduced amount of water-soluble proteins, vitamins, decreased activity of some enzymes, increased use of oxygen, and disrupted lipid peroxidation.

As a result of free radical formation in the lens, the formation and accumulation of toxic compounds that lead to irreversible changes of proteins. The short-wave part of the light spectrum in the range of 200-300 nm stimulates the formation of free radicals. Of particular importance is the fact of decreased activity of antioxidant enzymes in the lens with age and reduced concentration of natural antioxidants (vitamins A, E, glutathione, etc.).

Cataracts are divided into congenital and acquired. In congenital cataract, clouding is limited in size and does not progress. One can distinguish congenital cataract by the location of the opacities in the lens. Hence, depending on the location of opacities in the lens, cataract is divided into the following types:

- anterior polar cataract;
- posterior polar cataract;
- fusiform (spindle) cataract;
- lamellar cataract (zonular) cataract;
- nuclear cataract;
- cortical cataract;

• posterior subcapsular cataract;

• total or complete cataract.

Acquired cataracts have a progressive course. By the cause of occurrence, acquired cataract is divided into several groups:

• senile cataract;

• traumatic cataract (which arose as a result of blunt trauma (contusion) or penetrating injuries of the eyeball;

• complicated cataract (in inflammation of the choroid, high myopia, glaucoma and other eye diseases);

• radiation cataract – associated with the damage of the lens with radiation energy such as infrared rays (professional cataract − e.g., cataract of glassblowers), X-ray, radiation exposure;

• toxic cataract (including medicated cataract in the long-term administration of certain drugs: corticosteroids, antimalarials et al.);

• cataracts, caused by systemic diseases (diabetes mellitus, hypothyroidism, metabolic diseases).

According to the degree of maturity, senile cataract is divided into four stages:

• initial cataract,

• immature cataract,

• mature cataract,

• hypermature (overripe) cataract.

Cataract typically begins at the cortex (cortical cataract), nucleus of the lens (nuclear cataract) or subcapsularly (subcapsular cataract).

Initial cataract. The earliest signs of cataract are the hydration processes in the lens. Fluid accumulates in the cortex of the lens between the fibres, according to the arrangement of raphes. The so-called “water gaps” are formed. Later on, characteristic (spoke-like) cortical opacities appear primarily at the periphery of the lens, in the region of the equator. During the transition of the anterior opacities to the posterior surface of the lens, they assume the typical shape of “riders”.

Immature cataract. The gradual development of cataract consists in the opacity movement in the direction of the central optical zone. In the initial cataract the clouding is localized in the equatorial region, not in the optical zone, and it does not affect the visual acuity. Meanwhile, in the immature cataract, the expressed lens clouding leads to a marked decrease in the visual acuity.

Mature cataract. The whole area of the cortex of the lens is occupied with opacities. Sometimes this stage is conventionally divided into the stage of almost mature cataract when there are significant opacities in the cortex and visual acuity ranges within the range of hundredths, counting fingers in front of the face, and the stage of mature cataract, which is characterized by complete opacification of the lens substance and a decrease in the visual acuity to light perception.

Hypermature cataract. Further development of cataract is accompanied by the decay lens fibres. The cortex of the lens dissolves and becomes uniformly milky white, whereas the lens capsule becomes folded. A denser nucleus, due to its weight, drops down and reminds a yellowish sac. Overripe cataract is also called Morgagni cataract. However, at the stage of overripe cataract the increase of the lens size is also possible (due to secondary hydration) and its blockage of the outflow of intraocular fluid paths from the posterior eye chamber, into the anterior, thereby increasing intraocular pressure and leading to secondary glaucoma and irreversible vision loss.

One of the first clinical signs of the disease may be complaints about the doubling objects, the emergence of “flies” in front of the eyes. Visual acuity in the early stages of cataract development deteriorates only into the distance, but improves by correction with myopic glasses. The duration of the initial stage can be varied from 1 to 15 years. With the development of cataract, decrement in visual acuity gradually progresses. In mature cataract, the subject vision is lost and only light perception remains, which should be at the correct projection of light in the absence of lesions of the retina and optic nerve.

Examination of the patient with cataract is conducted by means of biomicroscopy, which enables the doctor to receive the optical section of the lens and the anterior layers of the vitreous body, investigate its structure in detail, determine the location

and magnitude of opacities, and estimate the position of the lens. In the presence of significant opacities of the lens, a series of additional, specialised examination methods is necessary, in order to give the patient an answer about the possible results of the treatment of cataract. Hence, when discussing the surgery option, of primary importance are methods for calculating the power of intraocular lens, implantation of which is provided during the operation. For calculation, ophthalmometry is performed (examination of the refractive power of the cornea) and biometry (examination of the lens sizes, length of the anteroposterior axis of the eye). Ultrasound is also used in the B-mode for the diagnosis and localization of possible structural changes behind the lens, as well as determining the nature of their prevalence. In case of minor opacities and for the possibility to study the condition of the retina and the optic nerve, optical coherence tomography is performed. Laboratory methods are applied, usually before the admission to hospital. These include clinical analyses of blood and urine, blood tests for HIV, syphilis, hepatitis B and C, coagulation and glucose, an X-ray of the chest and paranasal sinuses. Furthermore, conclusions from the therapeutist, dentist, ENT specialist are necessary, and if needed − from other specialists (endocrinologist, nephrologist, etc.). All this is done in order to identify contraindications to surgery (decompensation of systemic diseases, detection and sanation of foci of chronic infection), which may complicate the course of the postoperative period.

Treatment of cataract. Attempts to treat cataract by conservative methods have been known since ancient times. For instance, in Egypt compresses with honey, wine, vinegar, milk, blood of various animals were used for this purpose. Hippocrates applied dieto- and physiotherapy. Starting from the 1930s doctors began to prescribe vitamin preparations to treat age-related cataract (only in the initial stage of cataract, with preserved visual acuity). Various eye drops are used: qiunax, oftan-catachrom, vita-jodurol, vitafacol, taufonum, viceinum, thiotriazoline etc. However, the use of conservative treatment does not lead to resorption of opacities, but only delays their subsequent development. The drops against cataract progression include vitamins, potassium iodide, antioxidants, amino acids, ATP, etc. Typically, a patient with

cataract is initially recommended a prolonged use (for years) of vitamin preparations at a frequency of instillations of 3-4 times per day. If the conservative therapy is ineffective, surgical treatment is conducted. There are several types of surgery to remove the clouded lens:

• Extracapsular cataract extraction
• Intracapsular cataract extraction
• Ultrasonic phacoemulsification
• Laser cataract surgery (laser phacoemulsification)

In extracapsular procedure, the removal of the lens nucleus and lens masses is carried out, preserving the posterior lens capsule that provides a barrier between the vitreous body and the anterior segment of the eye. The disadvantages of such operation are its excessively traumatizing effect, the need to perform a large incision and put a large number of stitches. The most modern technique of cataract surgery is phacoemulsification. In this method of treatment, the clouded lens is sonicated and removed by vacuum aspiration through a small incision using a thin instrument of dimensions comparable with the pen. After removal of cataract, the intraocular lens is implanted into the eye through the same incision. The operation usually lasts 10-15 minutes and requires no stitches.

Intracapsular cataract extraction is the removal of the lens capsule through a large incision. The operation is performed by a special device − cryoextractor − by icing the lens to the instrument tip. Currently, this method is almost never used because of its traumatizing effect. Nowadays, the ultrasonic phacoemulsification is the standard technique for cataract surgery. Laser extraction techniques with different types of lasers are also available (Er:yag, Nd:yag, excimer laser). Unlike ultrasonic phacoemulsification, the use of laser-based systems enables the safe removal of cataracts of maximum hardness in a short period of time, without causing injury to the internal structures of the eye.

One of the diseases of the lens is its dislocation (subluxation). The major causes of lens dislocation are inherited pathology (Marfan syndrome, Down syndrome, Edwards syndrome, Patau syndrome, etc.), trauma, connective tissue weakness

together with tenderness of Zinn ligaments, degenerative and dystrophic processes in the ligamentous apparatus of the lens, developing against the background of metabolic disorders. The main symptoms of the lens dislocation are: vibrating iris (iridodonesis), the equator of the lens is visible, the anterior chamber becomes non-uniform. For evaluation of patients with this diagnosis, biomicroscopy is conducted, examination in the transmitted light, ophthalmoscopy, ultrasonography. Secondary hypertension can be a threatening complication of the lens dislocation. Treatment is carried out only by surgery. A condition that develops as a result of removal of the lens is called aphakia and is characterized by iridodonesis, deep anterior chamber, high hypermetropia, the lack of accommodation. For correction of aphakia, glasses, contact lenses or intraocular correction are applied.

The vitreous body is a transparent gelatinous mass which fills the space between the posterior surface of the lens, the flat part of the ciliary body and the retina. The vitreous body is a permanent structure of the eyeball, it does not regenerate, and in case of loss at eye injuries it is filled with intraocular fluid. The vitreous body consists of a fibrous basis and a loose substance that fills the gaps between the fibres. The vitreous body contains special proteins − vitrosin and mucin that determine its viscosity, which is several times greater than the viscosity of water. Inside the vitreous body, since birth can be maintained, partially or completely, hyaloid (Cloquet) canal − narrow S-visible capillary gap which extends from the optic disc to the lens. The surface of the vitreous body is covered with a thin membrane, which borders directly on the internal marginal membrane of the retina and is called the limiting membrane of the vitreous body. The vitreous body is bound to the surrounding tissues only in several places: in the area of attachment along the flat part of the ciliary body, and the posterior surface of Zinn ligament fibres (ciliary zonule), in the portion of posterior surface of the lens capsule (closer to the equator), along the perimeter of the optic nerve and macula. The vitreous body is attached to the retina in the places of vessels passing, in areas that correspond to the equator of the eyeball and attachment sites of the internal eye muscles. The vitreous body contains no vessels and nerves. Thus, the vitreous body, filling the cavity of the eyeball, and

exerting the pressure on the retina, keeps it in a normal condition. Disorders of the vitreous body with the surrounding tissue cause its detachment. One should also keep in mind that the vitreous body is a part of the refractive system of the eye.

In most cases, *the diseases of the vitreous body* are secondary and develop as a result of inflammatory and degenerative processes of the choroid and retina, the eye injury, the presence of foreign bodies inside the eye, high myopia, intraocular parasites, diabetic and hypertensive retinopathy. Pathologic processes in the vitreous body are manifested with its disrupted transparency, clouding, haemorrhage, destruction, shrinkage. The vitreous body may be invaded with parasites from the subretinal space (cysticercus, dirofilaria, echinococcus). The main features of the vitreous body pathologies are its opacities. Their presence and nature are detected with the help of ophthalmoscopy, biomicroscopy, ultrasonic B-scanning.

Haemophthalmos is the haemorrhage into the vitreous body. Most commonly, the causes of haemophthalmos are eye injuries that disrupt the integrity of the vascular walls of the choroid or retina, as well as inflammatory processes, hemorrhagic glaucoma, diabetic and hypertensive retinopathy. Visual acuity in haemophthalmos is often reduced to light perception with incorrect projection of the light, sometimes complete blindness develops. In focal illumination and biomicroscopy, there is a dark brown, granular blood mass of a reddish hue in the vitreous behind the lens. In ophthalmoscopy, the fundus reflex is absent. Treatment is carried out in view of etiological factors. In the early days, rest and haemostatic agents are recommended. Subsequently, intensive resorbable therapy is conducted, in case of its failure − surgical treatment (vitrectomy). Vitrectomy is a surgery, which consists in the partial or complete removal of the vitreous body. Instead the vitreous body silicone or a special gas is injected in the eye cavity. It is also possible to carry out endolaser coagulation of the retina in order to strengthen the retina and prevent its detachment due to the fact that there will be no pressure of the vitreous body on the retina after the operation. In the late postoperative period, silicone is removed from the eye, the gas is resorbed on its own and is replaced by the intraocular fluid.

Theoretical questions to the chapter:

1. Anatomical and physiological characteristics of the lens.
2. The role of the lens in accommodation.
3. Methods of examination of the lens and the vitreous body.
4. Classification of diseases of the lens.
5. Classification of cataracts. Stages of development of senile cataract.
6. The main clinical symptoms and treatment of senile cataract.
7. Aphakia: signs, methods of correction.
8. Congenital pathology of the lens. Tactics of the ophthalmologist in congenital cataract.
9. Pathology of the vitreous body.
10. Haemophthalmos: clinical features, diagnosis, treatment.

Practical skills which students should master:

1. History taking from a patient with lens pathology.
2. Examination of the patient using the "side lighting" method.
3. Examination of the patient using the transmitted light method.
4. Mastering the techniques of biomicroscopy.
5. Checking the visual acuity and aphakia correction.

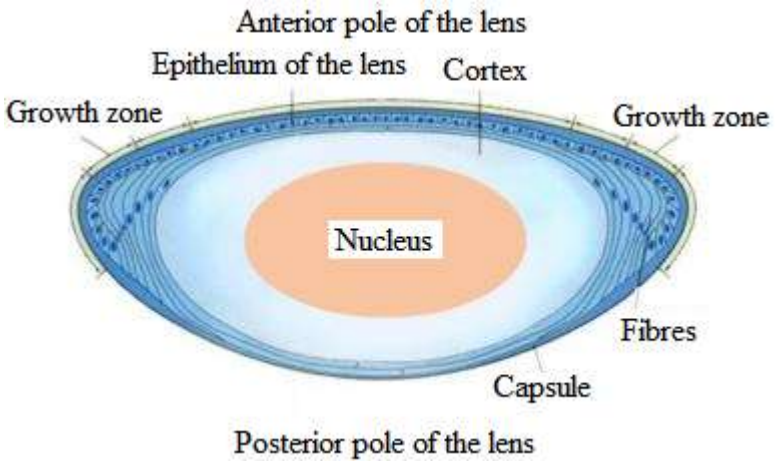


Fig.10.1. The structure of the lens.

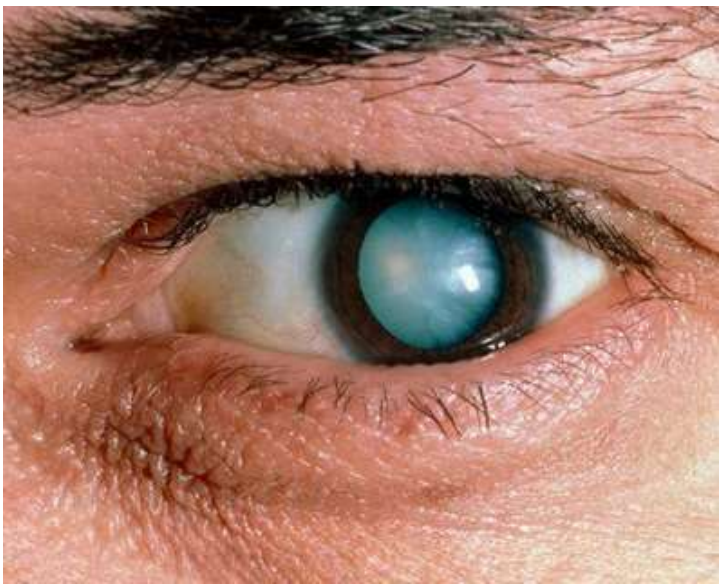


Fig. 10.2. Mature cataract.

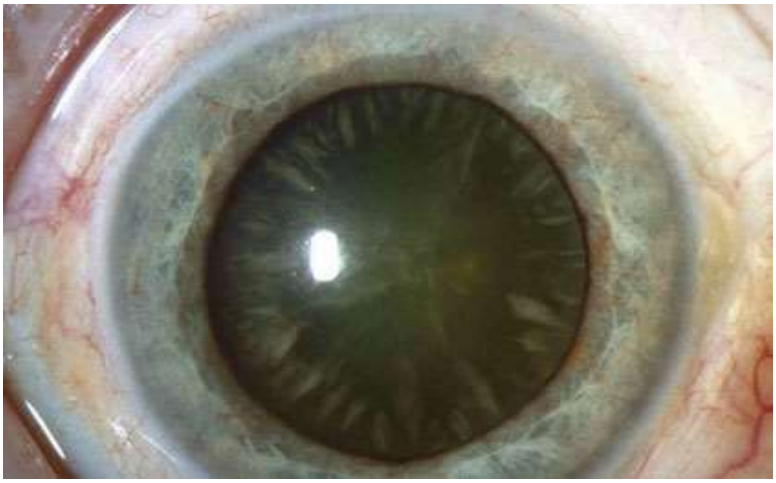


Fig. 10.3. Immature cataracts.

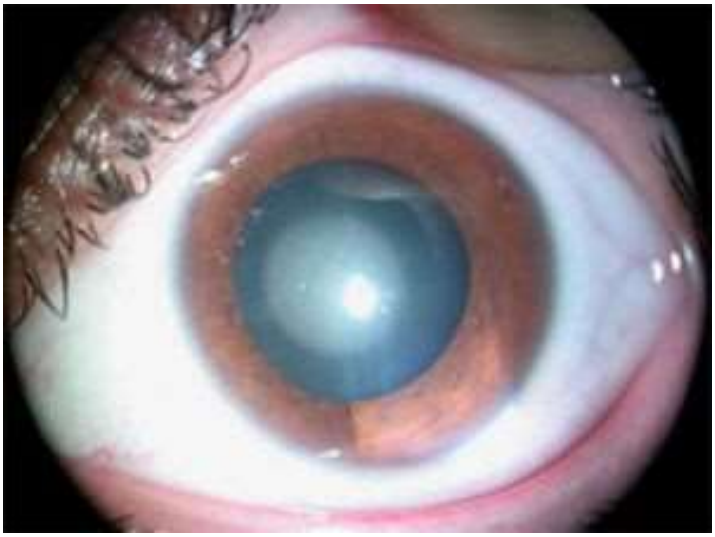


Fig. 10.4. Congenital cataract.

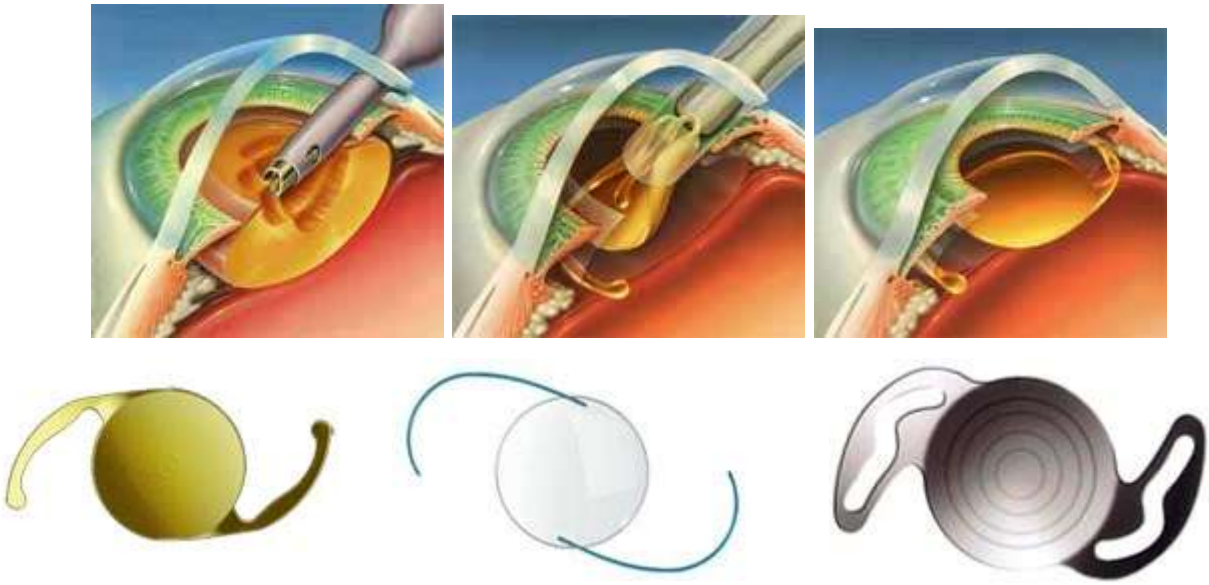


Fig. 10.5. Stages of cataract phacoemulsification.

Fig.10.6. Vitamin drops.

CHAPTER 11

DISEASES OF THE CHOROID

Among the diseases of the organ of vision, the disorders of the vascular tract account for 10% and often lead to impaired vision or blindness.

The choroid (vascular or uveal tract) consists of three parts − the iris, the ciliary body, and the vascular membrane proper. The functions of the vascular tract are as follows: trophism, participation in thermoregulation, production of intraocular fluid, the eye diaphragm (iris). The structure of each of the three divisions in the vascular tract has its own characteristics. Abundant vascularisation and the presence of pigment (melanin) is a common for all parts of the vascular tract. Vascular membrane is divided into two parts − the anterior (the iris and the ciliary body) and the posterior (choroid). This division is due to different perfusion and different innervation. Blood supply of the iris and ciliary body (the anterior portion) is carried out from the posterior long and anterior ciliary arteries; of the choroid (the posterior section) − from the posterior short ciliary arteries.

The anterior vascular tract has the sensitive innervations from the first branch of the trigeminal nerve, whereas sensitive innervations are absent in the posterior part. A large number of capillaries of the choroid can lead to the dramatic slowdown of circulation. Rapid evacuation of the blood is prevented by the intraocular pressure. In this regard, the vascular tract serves as a kind of “lagoon” for infectious agents and their metabolic products that can enter the capillaries with the bloodstream. These can be alive or dead bacteria, viruses, fungi, worms, protozoa, and their metabolic products. They can also be allergens. The choroid is closely associated with the retina and provides the normal functioning of photoreceptors. The choroid supplies the outer layers of the retina and the vitreous body; it is involved in the ultrafiltration and outflow of intraocular fluid.

The list of key terms, parameters and characteristics which students should master while studying the topic:

Term	Definition

Coloboma of the iris	The lack of the iris
Precipitates	Accumulation of cellular elements on the posterior surface of the cornea
Synechiae	Binding of the iris with the anterior lens capsule
Hypopyon	Level of exudate in the anterior chamber
Iridocyclitis	Inflammation of the iris and ciliary body
Choroiditis	Inflammation of the vascular membrane proper (the choroid)
Aniridia	The absence of the iris
Polycoria	Several pupils
Corectopia	Disrupted position of the pupil, where the it is shifted from the centre and occupies an eccentric position

Examination methods:

1. External examination.
2. Side lighting.
3. Biomicroscopy.
4. Testing the ciliary tenderness.
5. Gonioscopy.
6. Testing the pupillary reactions.

Fig. 11.1. Testing the sensitivity of the ciliary body.

Testing the sensitivity of the ciliary body:

The patient sits with the eyes closed and looking down. The doctor lightly taps with the index finger pad (or a glass rod) in the area of the ciliary body projection. Evaluation of results: the presence or absence of sensitivity.

Classification of diseases.

1. Developmental abnormalities (aniridia, coloboma, polycoria, corectopia of the pupil).
2. Inflammatory diseases.
3. Neoplasms.

Inflammatory diseases.

All inflammatory diseases of the vascular tract are called **uveites**. They are divided into anterior uveites (*iridocyclites*) and posterior uveites (*choroidites*). Inflammation of all parts of the vascular tract is called *panuveitis*.

Classification of uveites.

• by aetiology, uveites are divided into endogenous and exogenous,

• by clinical course − into acute and chronic,

• by morphological presentation − into granulomatous (metastatic haematogenous, focal) and nongranulomatous (toxic and allergic, diffuse).

Anterior uveites are divided by the nature of inflammation into serous, exudative, fibrinous-plastic and hemorrhagic. Posterior uveites, or choroidites, are classified by localization process into the central, paracentral, equatorial and peripheral uveitis.

Clinical presentation of anterior uveites (iridocyclites).

The *subjective symptoms* include corneal syndrome (photophobia, lacrimation, blepharospasm, and pain in the eyeball, which increases in the evening and at night), redness of the eyeball, and decreased visual acuity.

The *objective symptoms* include swelling of the eyelids, the presence pericorneal injection of the eyeball, appearance of precipitates on the posterior surface of the cornea, blurred of the anterior chamber humour, colour change and the pattern of the

iris, narrowing or changing the shape of the pupil, formation of the posterior synechiae, clouding of the vitreous body, and change in the intraocular pressure.

Acute iridocyclitis should be differentiated primarily with acute exacerbation of angle-closure glaucoma and acute conjunctivitis.

Clinical presentation of posterior uveites (choroidites).

The *subjective symptoms* include reduced visual acuity, photopsias (flashes), metamorphopsia (distortion of objects) by the central localization of the process and involvement of the retina in the inflammatory processes. Deteriorating night vision − hemeralopia in the peripheral localization of the processes. In the field of view, the relative or absolute central scotoma is determined.

The *objective symptoms* include the presence of inflammation foci in the fundus. Fresh inflammatory foci in the choroid resemble the cotton-like limited infiltrates with indistinct boundaries of yellowish-gray colour against the bright red background of the fundus. The inflammatory focus protrudes above the level of the retina and the retinal blood vessels ascent on it. At the end of the process, foci may fully dissolve, but are usually replaced by connective tissue, and a white scar with clear boundaries, surrounded with pigment, shines through the sclera. If the focus of the choroid is small and located on the periphery, the disease may become apparent accidentally during the routine examination. The central location of the focus always leads to decreased visual function.

Rheumatic uveitis. It occurs against the background of acute course (attack) of rheumatism. It is characterized by severe corneal syndrome and pain in the eyes. Mixed injection of the eyeball is determined. Multiple small gray precipitates are observed on the posterior surface of the cornea, abundant exudate in the humour of the anterior chamber, the iris is sanguine, blood vessels are dilated. Posterior synechiae are easily torn after instillation of mydriatics. The lens and the vitreous body are almost intact. Expressed vasculites can be determined in the fundus, which have the appearance of grayish “sleeves” on the vessels.

Clinical sign	Acute conjunctivitis	Acute iridocyclitis	Acute event of angle-closure glaucoma
Visual acuity	No changes	No changes or decreased	Dramatically decreased
Pain	None	Moderate	Very severe, with expressed irradiation
Ciliary tenderness	None	Expressed	None
Exudates from the conjunctival cavity	Mucous or mucous-purulent	None	None
Injection of the eyeball	Superficial	Deep	Displaced, congestive
Media	Transparent	Precipitates, exudation in the humour of the anterior chamber	Corneal oedema
The depth of the anterior chamber	Medium	Medium	Minor (slot-like chamber)
The iris	No changes	Oedema, hyperaemia, colour change	Sectoral atrophy may be observed
The pupil	No changes, response to light is intact	Miosis, change of shape (posterior synechiae), reduced response to light	Mydriasis, no response to light
Intraocular pressure	Normal	Normal or hypotension (sometimes hypertension)	High
Changes to the performance status	None	None	Headache, vomiting, increased blood pressure

Fig. 11.2. Differential diagnosis of iridocyclites.

All changes yield to regression with effective treatment and stabilization of rheumatism. The process recurs against the background of another attack of the disease. Treatment is topical and symptomatic.

Tuberculous uveitis occurs most often against the background of active pulmonary or mesenterial tuberculosis, and sometimes tuberculosis of bone. Inflammation usually occurs in one eye. Hyperaemia of the eye is slightly expressed, corneal syndrome is poorly defined. Characteristic features of tuberculous anterior uveitis are

“greasy” large precipitates on the corneal endothelium. In addition, there are grayish-pink nodules (granulomas-tuberculomas) in the iris and along the pupillary edge of the iris. Synechiae are usually poorly torn under the effect of mydriatics. New blood vessels are formed in the iris. Exudation may extend into the posterior chamber of the eye and the vitreous body, and as a consequence there is opacification (cataract) of the posterior lens capsule and vitreous body (golden rain).

Tuberculous foci of various sizes, without clear outlines, are found in different departments of the fundus. These foci do not merge, the pigment is deposited on their periphery, and in the centre they are of grayish hue. As a rule, the retina is also involved in the process, resulting in decreased visual function of varying degrees (depending on the location and size of lesions).

Syphilitic uveitis may occur in congenital and acquired syphilis.

Uveitis in acquired syphilis is characterized by moderate corneal syndrome, mixed injection, serous exudate in the anterior chamber and multiple tiny polymorphic precipitates.

Yellow-reddish nodules- papules with adjacent newly formed vessels are observed in the modified iris. Posterior synechiae are massive, broad, they are torn apart upon instillation of mydriatics. Accumulations of pigment are found in their place in the anterior capsule of the lens.

Floating opacity may appear on the vitreous body. Characteristic changes, resembling “spilled salt and pepper” are observed on the fundus. The changes in anterior and posterior eye departments in syphilitic uveitis may occur both together and independently.

Principles of treatment of uveitis.

Treatment is carried out in the ophthalmology in-patient department. Treatment is comprehensive and is aimed at the suppression of an infectious etiologic factor; blocking or regulation of local and systemic autoimmune reactions, prevention of complications.

1. **Anti-inflammatory therapy**.

• glucocorticosteroids. For treatment of anterior uveitis, topical (0.1% dexamethasone drops 4-6 times per day) agents or medications in the form of subconjunctival injections are generally used. In the treatment of posterior uveitis parabulbar injections are applied. In severe processes, they are used systemically;

• non-steroidal anti-inflammatory drugs are applied topically as instillations of 0.1% indomethacin solution, indomethacin, diclof, uniclof, as well as in the form of tablets and injectable forms.

2. **Mydriatics**. In the form of eye drops (atropine sulphate 15, irifrin, tropicamide) or subconjunctival injections.

3. **Fibrinolytic agents** (lidasa, gemase, fibrinolysin).

4. **Detoxification therapy** (hemodez intravenously, glucose solution with ascorbic acid).

5. **Desensitizing therapy**.

6. **Physiotherapeutic methods**.

7. **Etiotropic treatment** (depending on the causes of the disease) − antibacterial, antiviral therapy.

Complications of uveitis.

Complication of uveitis can lead to serious consequences, namely: band-like degeneration of the cornea, cataract, ocular hypertension, hypotension of the eye, endophthalmitis, panophthalmitis.

Theoretical questions to the chapter:

1. Serous iridocyclitis (the features of clinical presentation).
2. Clinical signs of fibrinous iridocyclitis.
3. Complications and consequences of iridocyclitis.
4. Treatment of iridocyclitis.
5. Clinical features of choroidites, treatment and consequences.

Practical skills which students should master:

1. Identify the projection of the ciliary body by palpation.
2. Conducting examination by the side lighting method.
3. Conducting ophthalmoscopy.

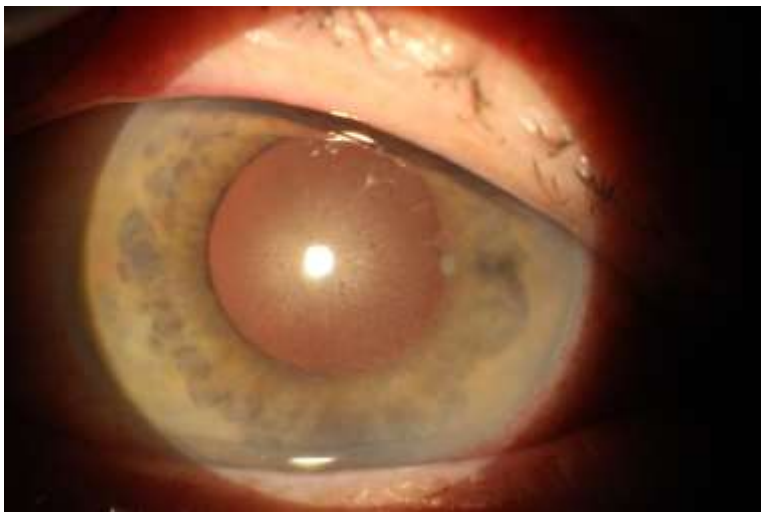


Fig. 11.2. Iridocyclitis.

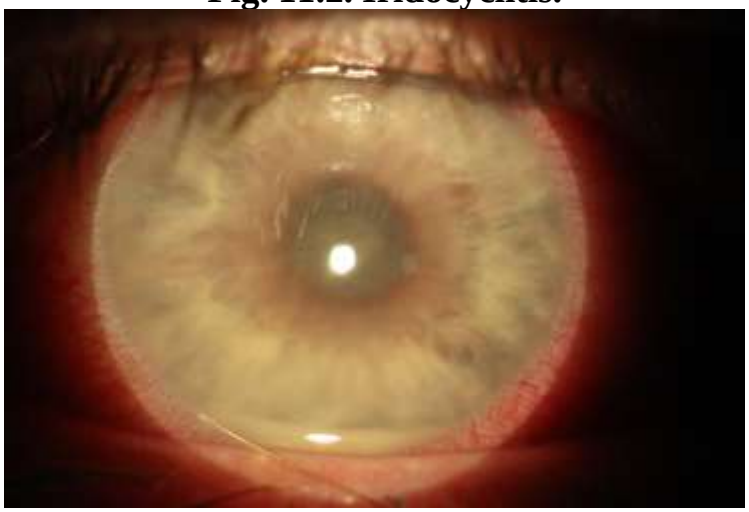


Fig.11.3. Hypopyon.

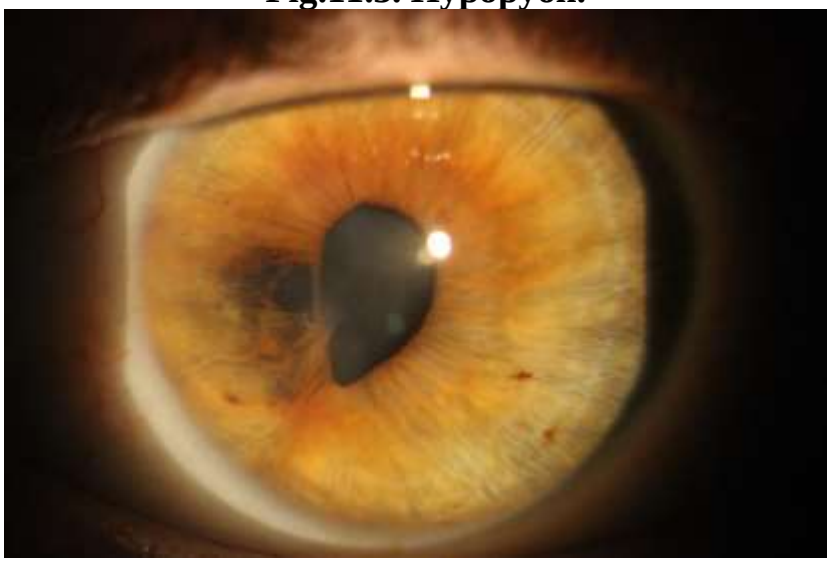


Fig. 11.4. Posterior synechiae.

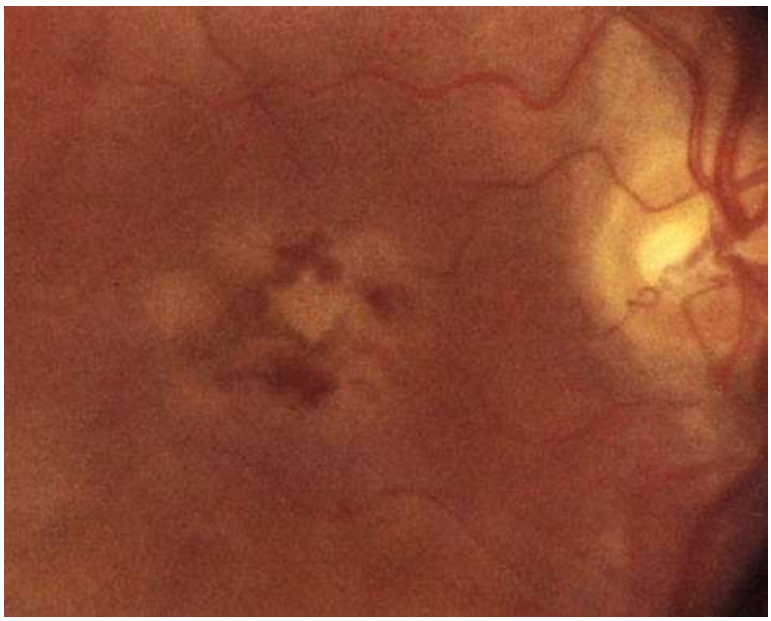


Fig. 11.5. Acute chorioretinitis.

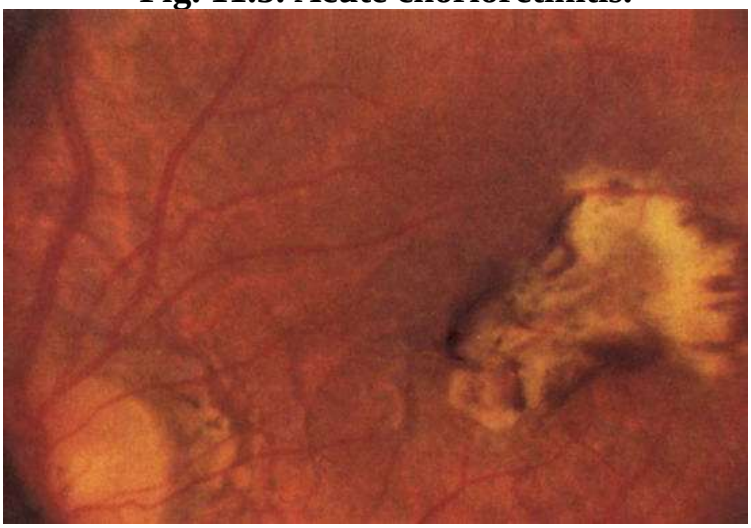


Fig.11.6. Clinical outcome of chorioretinitis.

CHAPTER 12

DISEASES OF THE RETINA

Diseases of the retina constitute an urgent problem due to the fact that a person gets 90-95% of information about the surrounding world through the organ of sight. Any pathological changes of photoreceptor department of the visual analyzer lead to visual impairment and irreversible blindness.

The list of key terms, parameters, characteristics which the students should master while studying the topic:

Term	Definition
Retinal degeneration	Disease, characterized by the pigmented and non-pigmented lesions with sharp edges in the fundus, cystic retinal degeneration in different parts of the fundus.
Pigment degeneration of the retina	The retinal arteries and veins in the fundus are narrowed, there are pigmented dendritic formations at the periphery of the fundus, the general background of the fundus is yellowish, the disc is pale, with a waxy tint and clear boundaries.
Central retinal vein thrombosis	Occlusion of the central retinal vein, which is characterized by a rapid decline in visual acuity, the appearance and of scotomata in the fundus, development of the crushed tomato symptom
Central retinal artery occlusion	A blockage which arises as a result of spasm or embolism, it is characterized by a sharp decline in visual functions, and development of the cherry-red spot in the fundus.
Retinitis	Inflammation of the retina

The retina is an inner membrane of the eyeball. It is a kind of "window into the brain" and a peripherical link of the visual analyzer. The retina is a part of the brain,

which was separated from it in the early stages of development, but is still connected with it through the optic nerve. Histologically, the retina consists of ten layers. The retina has only three histological layers only in the central department (the macular area). She perceives the light stimulus and converts it into the nerve impulse which through the visual pathways of reaches the occipital lobe of the cerebral cortex, where the visual image is formed. The retina is divided into two parts − the optically active (lining the entire inner surface of the choroid up to the ora serrata) and optically active (extending from the ora serrata to the ciliary body and the iris). Central retinal zone consists of nerve cells, represented by three neurons:

- the first neuron − rods and cones;
- the second neuron − bipolar cells, which connect the nerve cells of the first and third neurons;
- the third neuron − ganglion cells. The axons of these cells are collected in the optic disc from the entire retina and form the optic nerve. The retinal blood supply is provided by the central artery, central retinal vein and choroid. The branches of the central retinal artery (CRA) have no anastomoses and complete occlusion of one of them leads to irreversible degenerative processes in the corresponding sector of the retina. The retina is attached to the frame of the eyeball in two areas: around the optic disc and along the ora serrata (the boundary of the ciliary body and the iris).

Examination methods.

1. Visometry and perimetry.
2. Ophthalmoscopy (direct and indirect).
3. Fluorescein angiography.
4. Optical coherence tomography.

Indirect ophthalmoscopy:

This is one of the most important methods, since it provides information about the condition of the retina, its central departments (the macula), choroid and optic nerve.

The examination is carried out in a dark room. The doctor sits at a distance of 40-50 cm from the patient, takes the ophthalmoscopic mirror in the right hand, and in the left hand − lens of 13 dioptres. The switched-on lamp is on the left from the patient

and turned to him/her with a flap. The ophthalmoscopic mirror is held at the right eye, directing the beam of rays reflected by the mirror into the pupil of the examined eye, viewing it through the aperture of the ophthalmoscopic mirror. After obtaining a uniform light illumination of the pupil, the physician places the magnifying glass at a distance of 7-8 cm from the eye; wherein the ophthalmoscope aperture and the pupil should be aligned. Accommodating to the frontal plane, which is at a distance of 5-8 cm from the junction between it and the eye, doctor sees the opposite and enlarged image of the fundus as if hovering in the air. To improve examination of the fundus, the pupil of the eye is dilated in advance by means of mydriatics. The magnifier of 13 D is used for an estimated evaluation of the condition of the fundus. To examine the fundus in detail, direct ophthalmoscopy is applied.

Direct ophthalmoscopy is performed using manual electric ophthalmoscope. The examination is carried out in a dark room. The doctor presses the device tightly to his/her eye and moving closer to the patient (2-3 cm to the patient's eye), directs the light beam through the dilated pupil. Once the image of the fundus is visible, by turning a large refractive disc, the lens is selected, ametropia of the doctor and patient is corrected until obtaining a clear image. During direct ophthalmoscopy, the fundus can be seen in the large scale-up, but the field of view decreases. In redless light ophthalmoscopy, the blue-green filter is introduced into the optical system by rotating the disk. Ophthalmoscopy assesses the condition of the optic nerve. In normal condition (Fig. 12.7) it is round or oval, pale pink, aligned with the retina, the physiological excavation (indention in the centre) is 0.3. Vessels exit from the centre of the disc, they are divided into branches and are distributed over the retina. The ratio of the arteries to veins diameter is calculated at the edge of the disc and should be 2:3 (1:1.5). After examination of the optic disc, the central area of the retina (macula) which is responsible for central vision is observed. To do this, the patient is asked to look directly into the ophthalmoscope. The severity macular reflex and the presence of haemorrhage, oedema, degenerative changes are examined, and then the peripheral portions of the retina are assessed.

Fig.12.1. Indirect ophthalmoscopy.

Fig. 12.2. Mirror ophthalmoscope.

Fig. 12.3. Electric ophthalmoscope.

Fig. 12.4. Direct ophthalmoscopy.

Fluorescein angiography

This method is based on the fluorescein ability to absorb blue light spectrum and emit yellow-green light at normal blood pH (7.37-7.45). Prior to intravenous administration of fluorescein (5.0 ml of 10% solution) several images without filter are made. Then fluorescein is quickly injected into the cubital vein and after 5-7 seconds a series of shots starts. As the dye enters the bloodstream, it fills the choroid and choriocapillaries. It is an additional method to study the retinal and optic nerve.

Optical coherence tomography (OCT).

This method is based on determining the delay time of the light reflected from the tissue. The radiation source in the modern OCT instruments is a wide-band superluminescent diode. This method makes it possible to obtain the image of the retina and optic nerve in the form of a longitudinal “section” or “picture”. High spatial ability of OCT gives the possibility of intravital microscopy of the eye tissues.

Classification of retinal diseases.

1. Inflammatory disease (retinitis).
2. Dystrophic diseases.
3. Sudden loss of vision (retinal detachment).

4. Acute circulatory disorders in retinal vessels (acute occlusion of the central retinal vein and artery).

5. Retinal neoplasms (retinoblastoma).

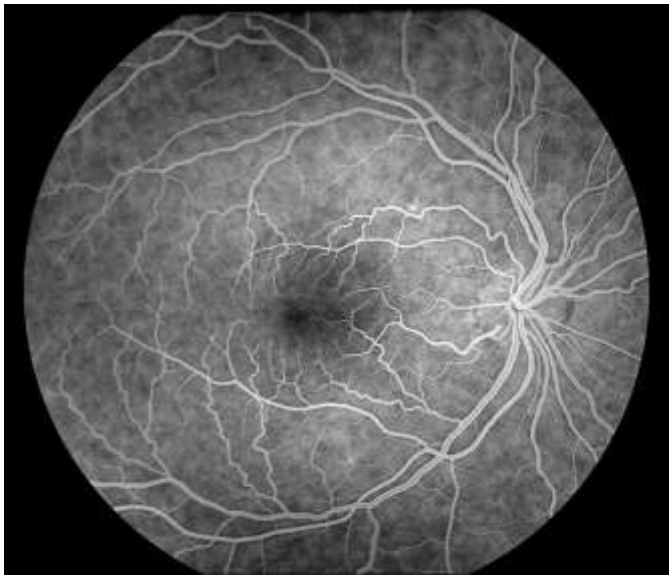


Fig.12.5. Fluorescein angiography (norm).

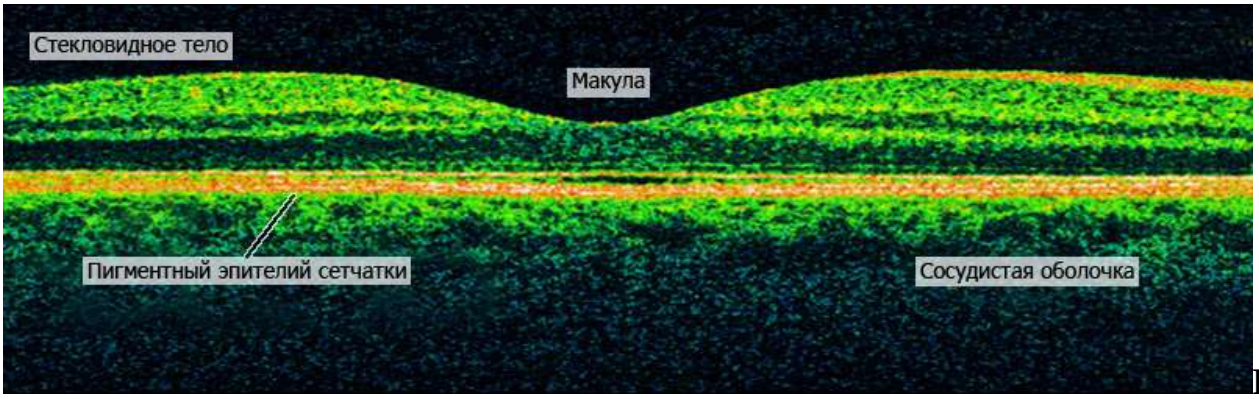


Fig. 12.6. Optical coherence tomography of the retinal (norm)

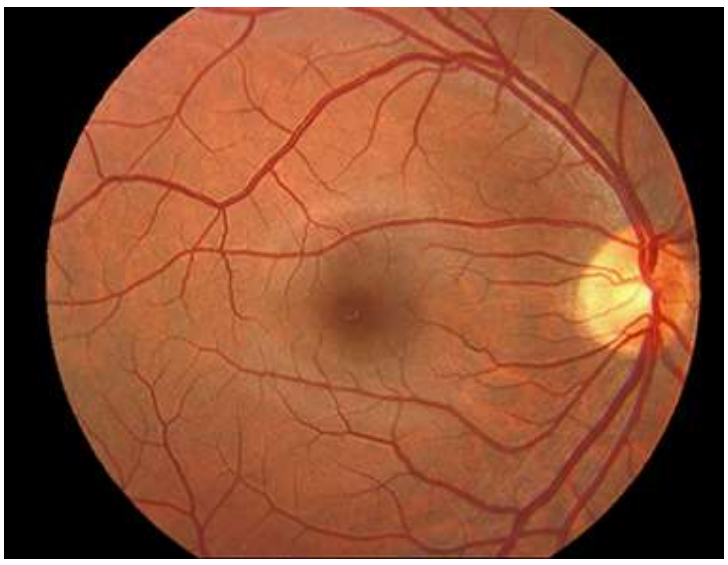


Fig. 12.7. Normal ophthalmoscopic presentation of the fundus.

Retinal dystrophies in a central region (macula − macular degeneration) are characterized by progressive course, reduction of central vision, impaired colour vision and loss in the central part of the field of vision (central scotomata).

Hereditary macular degeneration is a two-sided process. The clinical presentation and the time of occurrence of the disease in different members of the same family are the same. Early signs of hereditarymacular degeneration are photophobia and day blindness. The first ophthalmoscopic characteristics of inherited macular degeneration include the lack of macular reflex and appearance of graininess in the macula. In the field of vision, central scotoma appears. There is no pathogenetically based treatment of hereditary macular degeneration. It is recommended to wear sunglasses. *Pigmentary degeneration of the retina* is a hereditary tapetoretinal dystrophy, which is transmitted by the autosomal dominant type. It is characterized by lesions of the pigment epithelium and retinal photoreceptors. It is manifested by the appearance of hemeralopia (night blindness). Central vision is preserved for a long time. The ophthalmoscopic pattern is characterized by the appearance of the pigment of dark brown colour along the retinal vessels that resembles "bony corpuscles". They gradually capture more central areas. At the early stages of the disease, the optic disc is pale pink, and then it becomes pale and waxy. There is no pathogenetic treatment. Symptomatic therapy is prescribed.

Age-related macular degeneration is currently the main cause of visual loss in people over the age of 50. The disease is genetically determined. It is based on the loss of retinal pigment epithelium, Bruch's membrane and choriocapillary layer of the macular area of the retina. The process is usually bilateral. The risk factors are age, bad habits, poor diet, high blood pressure.

Several forms are distinguished − dry and wet macular degeneration. The dry form is more common. Patients complain of decreased visual acuity, metamorphopsia. In ophthalmoscopy, drusen are detected in the macular area of the retina, dry light-yellow dystrophic foci, whereas in the wet form − oedema and haemorrhage are

observed. In optical coherence tomography, detachment of the retinal pigment epithelium is visible.

Treatment is comprehensive and is aimed at slowing down the pathological process.

Acute occlusion of the central retinal artery occurs suddenly and is accompanied by the vision loss in the corresponding eye. The disease is caused by spasm, thrombosis, embolism of the artery. It occurs, not only in patients with essential hypertension, but also in young people suffering from endocarditis, valvular heart disease chronic infectious diseases.

The sudden cessation of blood flow in the retina leads to a dramatic disruption of cell metabolism, resulting in swelling of the ground substance. The retina loses its transparency and its swelling occurs. Transparency is preserved only in the area of the central fossa, where the retina is represented by only one layer of cones. There is no ground substance in the fovea. In this regard, ophthalmoscopic presentation of acute obstruction of the central retinal artery is very characteristic − against the white clouded background of the retina there is a clearly distinguished dark red central fossa, resembling a **"cherry stone"**. Arteries are sharply narrowed. In the minor arterial trunks, intermittent blood columns are visible. Veins are unchanged or are slightly narrowed. Blanching of the optic disc is observed.

In addition to the central retinal artery occlusion, obstruction of its branches can also be observed. Ophthalmoscopically, these patients present with retinal oedema, according to the spread area of the vessel. Visual functions are partially impaired, according to the affected area of the retina.

The disease is usually unilateral. The prognosis in case of central retinal artery embolism is usually unfavourable – the patient's vision cannot be restored. In the artery spasm, loss of visual function can be short-lived.

Treatment.

1. Urgent hospitalization.
2. Tablet of nitroglycerin under the tongue (0.0005g.).
3. Retrobulbar administration of 0.5 ml of 0.1% atropine sulfate.
4. 1% solution of nicotinic acid 1-5 ml or aminophylline solution intravenously.

5. Intramuscular administration of indirect anticlotting agents (fraxiparine).

Prognosis is poor − the process leads to irreversible loss of visual functions.

Central retinal vein obstruction. It occurs predominantly in the elderly people against the background of hypertension, athero- and arteriosclerosis, diabetes, infectious diseases (influenza, sepsis, pneumonia).

It is characterized by rapid onset, a sharp decrease of vision, the appearance of scotomata in the field of vision. Ophthalmoscopic thrombosis of the central vein looks quite common, resembling the "crushed tomato" presentation. The optic disc is oedematous, dark red, its boundaries are blurred, veins are dramatically dilated, convoluted. Arteries are narrowed, the retina is oedematous, there are multiple retinal haemorrhages of different sizes and shapes in the form of flames along the vessels and throughout the retina. The retina is oedematous. The optic disc is not visible due to haemorrhages. The process is usually unilateral. In thrombosis of the branch of central retinal vein, a similar presentation is observed only along the course of the respective vessel.

Treatment.

1. Urgent hospitalization.
2. 2.4% aminophylline solution 5-10 ml intravenously.
3. For high blood pressure − intramuscular injection of 25% magnesium sulfate solution 5-10 ml.
4. Parabulbar administration of heparin and fibrinolysin.
5. Intramuscular administration of indirect anticlotting agents (fraxiparine).

The prognosis is more favourable than in the central retinal artery obstruction. However, after thrombosis, neovascular glaucoma, retinal degeneration and atrophy of the optic nerve may develop.

Retinal detachment.

Throughout its area, the retina is loosely adherent to the choroid and is securely held only in the place of the optic nerve and the ora serrata. Being located between the choroid and the vitreous body, it is often involved in the pathologic process under either the influence of changes in the vitreous body (vitreoretinal adhesions,

diffluence, wrinkling of the vitreous body, haemorrhage into the subvitreal space), or due to the changes in the vascular membrane (choroiditis, dystrophy, tumours). Detachment can occur due to injuries, high myopia and eye injuries.

The pathogenesis of primary and traumatic retinal detachment is based on the retinal tear or its separation from the ora serrata.

Patients with retinal detachment complain of sudden loss of visual field – blurred vision, resembling a “curtain”, or a “veil”. This may be preceded by photopsias and metamorphopsia. Reduced visual function depends on the location and extent of the process, involvement of the macular area.

Objectively, during ophthalmoscopy, the veil-like gray film can be found against the background of red reflex. When the eye moves, this film sways like a sail in the wind. Retinal vessels become crimped, dark, leaning over the uneven area of retinal detachment.

Treatment of retinal detachment requires surgical intervention. It is aimed at blocking the gap and elimination of vitreoretinal adhesion, which pulls the retina into the vitreous cavity.

Retinal neoplasms.

The malignant retinal neoplasms include retinoblastoma. This disease occurs during the first few months (20%) or the first years (55%) of the child's life.

Retinoblastoma is a malignant congenital tumour which originates in the granular layers of the retina. The onset of the disease is symptomless, but in the nearest months the tumour reaches a considerable size, occupying most of the eye cavity. Ophthalmoscopic signs are the dilated pupil with a distinctive yellow fluorescence (amaurotic cat's eye). This phenomenon is detected by the infant’s parents. The tumour invades the orbit, the brain, bones and lymph nodes by haematogenous route. For diagnostics, ophthalmoscopy and ultrasound examination are used.

Treatment is aimed at preserving the eyeball and the child's life. In small sizes of the tumour, cryotherapy and laser destruction are applied. In large sizes, the removal of the eyeball is carried out.

Developmental abnormalities of the retina.

The congenital retinal disorders include myelinated fibres which in ophthalmoscopy have the appearance of brilliant white flames, located near the optic disc. The visual function in the presence of myelinated fibres is not affected.

The anomalies of the macular area in the form of *hypoplasia*, *aplasia*, complete absence of (coloboma) and cysts can be observed. Macular congenital abnormalities can occur as a local manifestation, but may be accompanied by microphthalmia, aniridia, albinism. As a rule, these pathologies involve visual impairment. Among other abnormalities, there are also retinal folds of the inner layers.

Theoretical questions to the chapter:

1. Anatomical and physiological features of the structure of the retina.
2. Methods of examination of the retina.
3. Classification of retinal diseases.
4. Acute occlusion of the central retinal vein and artery.
5. Retinal detachment. Aetiology, pathogenesis, treatment.
6. Retinal pigment degeneration: causes, clinical features, diagnosis, treatment.
7. Anomalies of the optic nerve (coloboma, fossa, myelinated fibres, pseudoneuritis): clinical presentation, diagnostic, treatment.

Practical skills which students should master:

1. Examination of the fundus by direct ophthalmoscopy.
2. Examination of the fundus by indirect ophthalmoscopy.
3. Formulation for treatment of chronic diseases of the optic nerve.
4. Formulation for treatment of retinal diseases which are accompanied by the progressive loss of vision.
5. Determining the visual field and colour perception in patients.

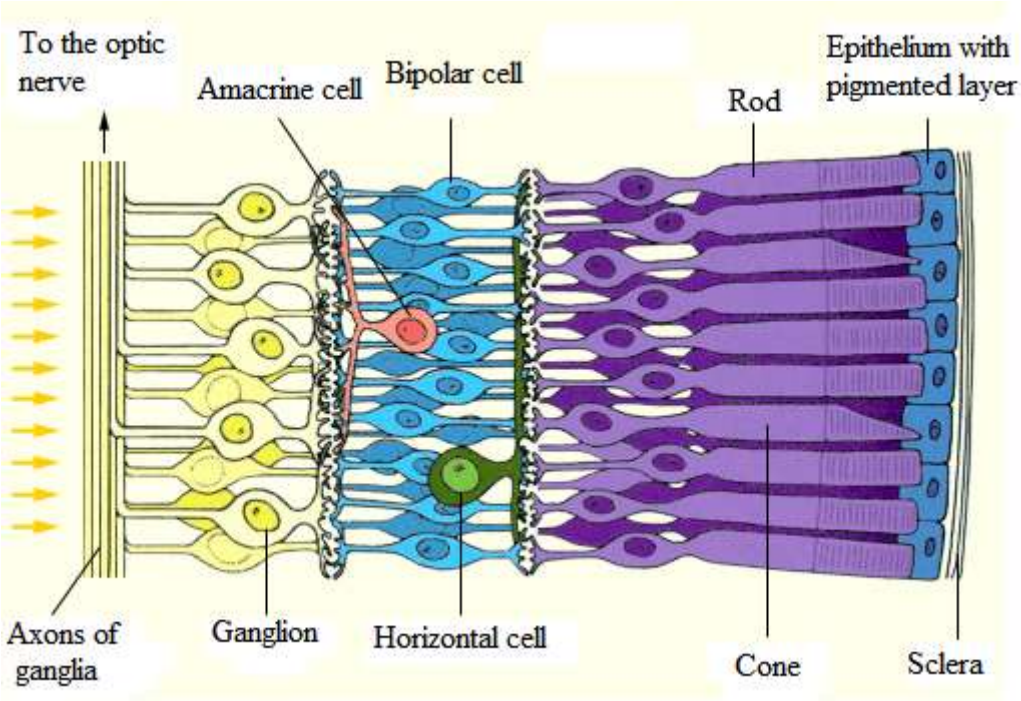


Fig. 12.8. Histological structure of the retina.

Fig. 12.9. Thrombosis of the central retinal vein

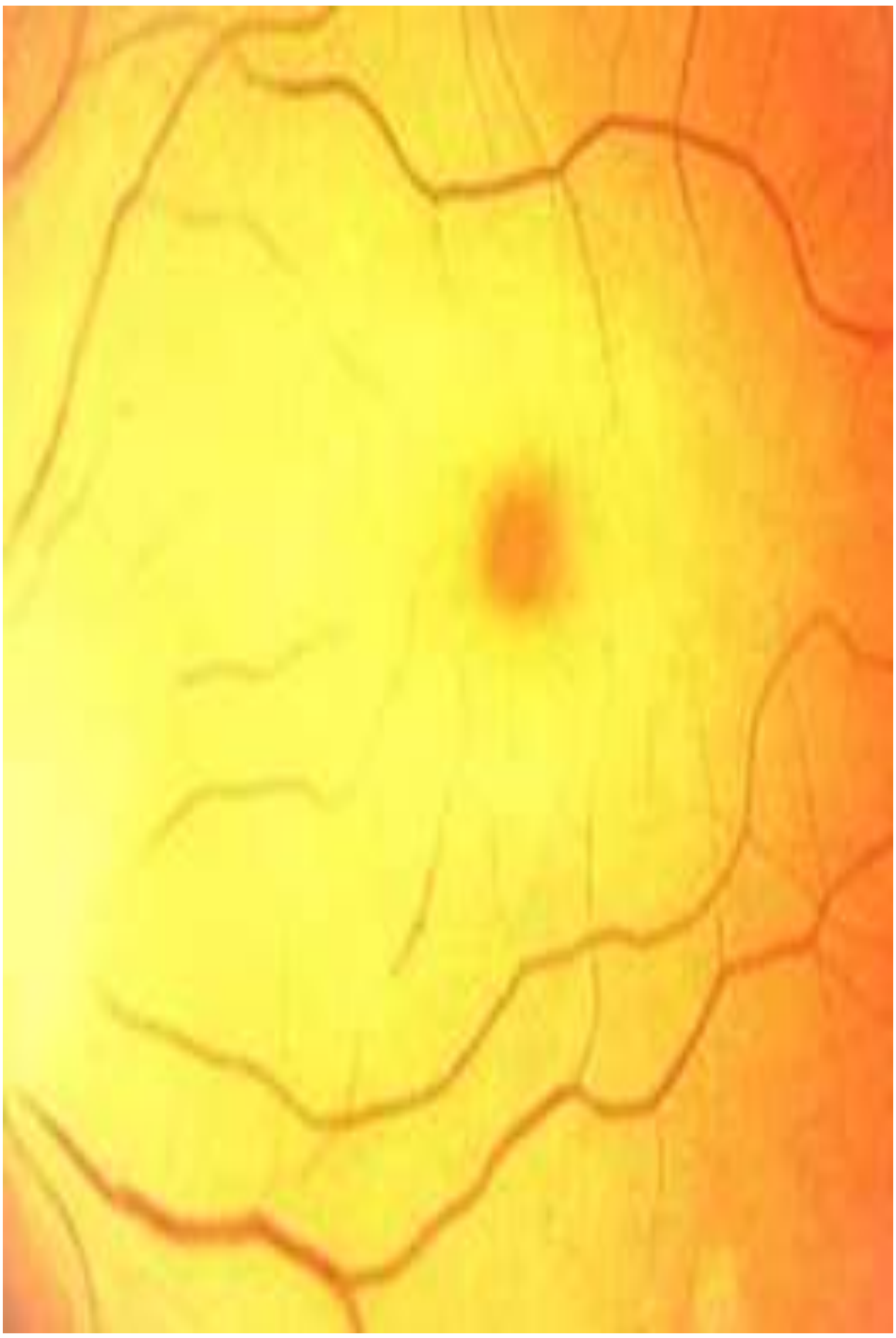


Fig. 12.10. Embolism of the central retinal artery.

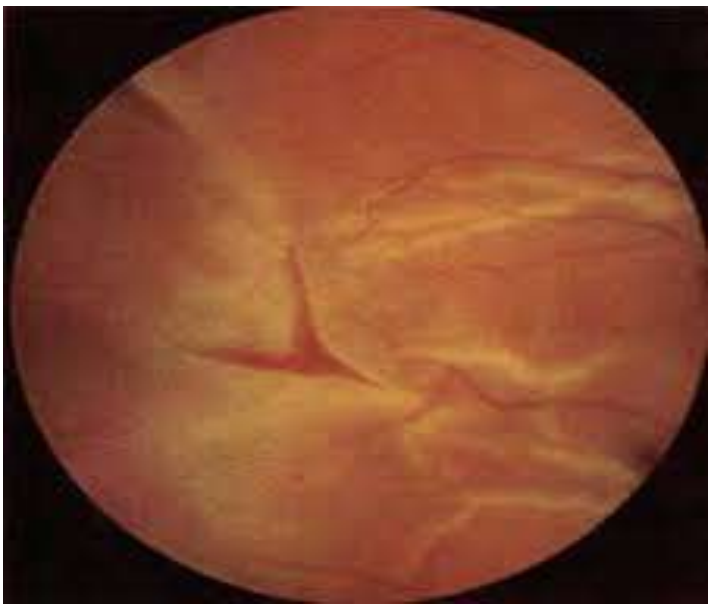


Fig.12.11. Retinal detachment.

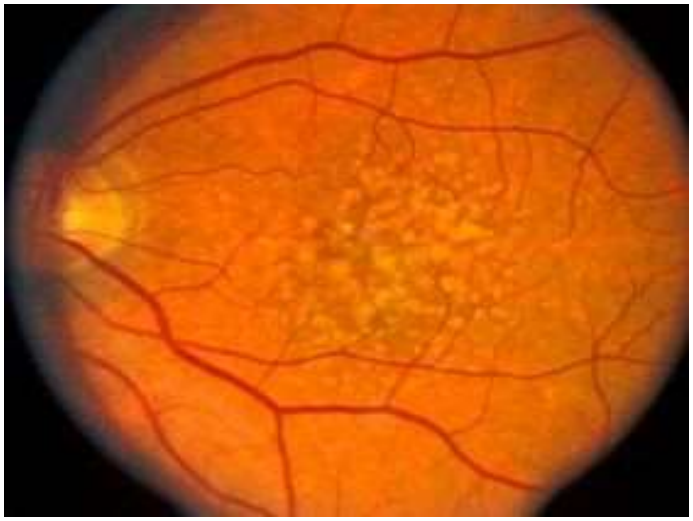


Fig. 12.12. Age-related macular degeneration.

CHAPTER 13

DISEASES OF THE OPTIC NERVE

The optic nerve is the second pair of cranial peripheral nerves. On one side, the optic nerve belongs to the retina, and on the other side − to the white matter of the brain. It determines its sensitivity to pathological processes that develop in the brain structures.

The optic nerve is a segment of the visual pathway. It begins with the processes of ganglionic retinal cells that form the optic disc, and ends in the chiasm (after a partial decussation). Optic nerve is divided into bulbar (intraocular) and retrobulbar (extraocular) parts. Retrobulbar part of the optic nerve consists of the orbital, canalicular and cranial (intracranial) departments. The length of the orbital portion is 4.5-5 cm. During ophthalmoscopy, the optic disc has a pale pink colour, clear borders, there is a funnel-shaped recess (excavation) in the centre. A part of the central retinal artery enters the excavation area and the central retinal vein exits. The length of the optic nerve from the optic disc to the chiasm is 35-55 mm. The optic nerve passes to the Turkish saddle, where its decussation is formed.

Only the medial fibres of the optic nerves intersect, while the lateral fibres pass along their side. Thus, after the chiasm, the optic pathway is formed in a part of which there are fibres, extending from the optic nerve of the right and left eyeballs. Further, the optic tract extends to the subcortical centres − optic radiation with the cortex of calcarine sulcus on the medial surface of the occipital lobe of the brain. Cortical centres of the visual analyzer correspond to the field 17 by Brodmann of the cerebral cortex.

The main sources of blood supply are ophthalmic artery branches (central retinal artery, posterior short ciliary artery), branchlets of the pia mater plexus.

Vessels that supply the optic nerve belong to the system of the internal carotid artery.

The list of key terms, parameters, characteristics which the students should master while studying the topic:

Term	Definition
Primary atrophy of the optic nerve	The optic disc is pale or grayish-white with clear edges, there is a flat shallow excavation, visual functions are reduced or completely absent.
Secondary atrophy of the optic nerve	The optic disc is pale gray with indistinct borders; there are signs of residual inflammation or congestion, haemorrhage, visual functions are reduced or completely absent.
Retinal atrophy of the optic nerve (in pigmentary degeneration of the retina)	The optic disk gray-yellowish, clear boundary, the retinal vessels, especially arteries narrowed visual function reduced.
Partial atrophy of the optic nerve	Partial blanching of the optic disk, usually of the outer half, wide flat excavation, clear boundaries, partial reduction or defects in the visual field, or its one-half loss.
Congestive optic disc	Increase of the optic disc and its bulging into the vitreous body, disc is of pink-gray colour, the borders are blurred. The retina around the disc is swollen with a grayish hue.
Chronic circulation failure in the optic nerve	Gradually decreased vision. Blanching of the optic disc is observed in the fundus, retinal vascular narrowing.

Examination methods.

1. Visometry and perimetry.
2. Ophthalmoscopy (direct, indirect).
3. Fluorescein angiography.
4. Optical coherence tomography.

Classification of diseases.

I. Congenital (malformations).

II. Acquired.

1. Inflammatory (neurites).
2. Non-inflammatory oedema of the disc (congestive disk).
3. Neoplasms.
4. Vascular disorders.
5. Injuries.

Inflammatory disease of the optic nerve.

Inflammation of the optic nerve is called optic **neuritis**. This inflammation can affect the intraocular portion of the optic nerve (intrabulbar) − **papillitis** and extraocular part − **retrobulbar neuritis**. This unequal division is due to the fact that the small front section of the optic nerve is clearly visible during ophthalmoscopy, and the rest of it − is inaccessible for examination. Among the causes of neuritis can be the inflammatory diseases of the brain, inflammation of the paranasal sinuses and systemic infection (influenza). The infection may also spread directly from the surrounding tissue or by haematogenous route. The optic nerve may be affected in different departments.

Papillitis.

Among the causes are papillitis are inflammatory diseases of the eyeball membranes, purulent processes in the orbit, paranasal sinuses, jaw, acute and chronic infections, toxic and allergic diseases.

Subjective signs. Patients complain of a dramatic decline in visual acuity, the emergence of central or paracentral scotoma in the field of view, impaired colour perception.

Objective signs. During ophthalmoscopy, there are hyperaemia and oedema of the optic nerve, its blurred borders, protrusion into the vitreous body, moderately dilated arteries and tortuous veins. Haemorrhage and plasmorrhages may be observed on the disc.

Treatment. There are two stages of treatment − immediate (before clarifying the aetiology), and etiological. At the first stage is general anti-inflammatory and

desensitizing therapy is conducted (corticosteroids retrobulbarly and orally, pipolphen, diphenhydramine, vitamins C, B1, B6, B12), as well as detoxication (rheosorbilact intravenously), dehydration (diacarb orally) and reflex therapy (electrophoresis with adrenaline). After defining the cause of the disease, etiotropic therapy is carried.

Retrobulbar neuritis may be caused by systemic infections, inflammatory diseases of the brain, sinuses, and teeth. However, most often the cause of retrobulbar neuritis is multiple sclerosis.

Subjective signs. Patients complain of blurred vision, pain behind the eyeball and with its movement.

Objective signs. During ophthalmoscopy, changes in the optic nerve are not observed at the onset of the disease. Gradually, in the late period, there is blanching of the optic nerve and the narrowing of blood vessels as a result of the development of descending atrophy.

The optic nerve is very sensitive to any intoxication in the body. Due to this, in poisoning (with methyl alcohol or the like.) optic neuritis develops, followed by atrophy of the optic nerve and irreversible loss of visual function.

Treatment of retrobulbar neuritis depends on the aetiology of the disease. As a rule, it is carried out according to the same principles as the conventional treatment of neuritis.

Congestive optic disc.

This is a swelling of the optic nerve of the non-inflammatory nature, due to increased intracranial pressure. Since the cerebrospinal fluid washes the optic nerve, the intracranial hypertension changes the conditions of circulation and trophism of the optic nerve. This leads to its swelling and blood congestion. Congestive optic disc develops in brain tumours, abscesses, inflammatory diseases of the cerebral meninges, associated with intracranial liquor hypertension.

Subjective signs. At the initial stages, patients do not complain of the visual organ dysfunction. They may complain of headache and dizziness.

Objective signs. Ophthalmoscopic pattern depends on the stage of the disease.

Stage I − hyperaemia. It is characterized by hyperaemia of the optic nerve, its blurred boundaries, the initial protrusion of the disk over the surface of the retina.

Stage II − oedema. An increase in the volume of the optic nerve due to its swelling. Oedema extends to the retina.

Stage III − ischemia. It is characterized by decreased blood supply of the disk. As a result, swelling decreases, vessels are narrowed, their spasm is observed. Hyperaemia of the disk is significantly reduced and pallor of the optic disc is visible.

Stage IV − atrophic. It is characterized by significant pallor of the optic disc with the development of atrophy.

Reduction of visual functions in patients, as a rule, is observed already from the third stage of the disease.

Treatment is based on elimination of the underlying causes of the disease. After eliminating the causes of the congestion phenomenon, ophthalmoscopic presentation (if atrophy of the disc does not develop) can be normalized in a period from 2-3 weeks to 1 − 2 months.

Atrophy of the optic nerve.

The reasons for the development of optic nerve atrophy may develop due to many diseases associated with inflammation, swelling, compression, damage, degeneration of the fibres of the optic nerve, or blood vessels supplying it. Optic atrophy often develops in the disorders of central nervous system, tumours, syphilis, brain abscess, encephalitis, multiple sclerosis, traumas of the skull, intoxication, poisoning with methyl alcohol.

Thus, many diseases of the optic nerve ends with its atrophy. It leads to reduced visual functions and disability.

One can distinguish *primary* (simple) and *secondary optic nerve atrophy*.

Patients with optic atrophy complain of decreased visual acuity, narrowed boundaries of the field of vision.

Ophthalmoscopic presentation is characterized by pallor of the optic nerve and the clarity of its borders during the *primary atrophy* and blurred boundaries in the *secondary atrophy*.

There are also full and partial atrophy of the optic nerves. Full atrophy of the optic nerve does not respond to treatment. In partial atrophy of the optic nerve, vasodilators, vitamins of B group, and physiotherapeutic methods are prescribed.

Ischemia of the optic nerve.

Ischemia of the optic nerve develops due to circulatory disorders in the arterial system that nourishes the optic nerve. This disease usually occurs in people over the age of 50, suffering from cardiovascular diseases (hypertension, atherosclerosis, diabetes, and vasculitis).

The disease begins acutely. Patients complain of a sudden loss of vision or its sudden decrease. In the field of vision, scotomata or sectoral areas of falling-out are defined. During ophthalmoscopy (objectively), expressed oedema of the optic nerve is observed, its boundaries are blurred, and it is increased and protruding into the vitreous body. It is of pale colour. A few days later, the swelling decreases and the signs of atrophy appear.

Treatment. Vasodilators, anticoagulants, neuroprotective agents, vitamins, corticosteroids are prescribed. Prognosis of the disease is always serious.

Drusen of the optic nerve.

Drusen are rare disorders of the optic nerve. Ophthalmoscopically they are characterized by the presence of acinar protuberances of grayish-white colour, consisting of circular formations covering up the surface of the optic nerve. This disease rarely leads to the decrease in visual functions. The occurrence of drusen is associated with degenerative processes in the damaged fibres of the optic nerve.

Tumours of the optic nerve.

These include glioma and meningioma. Meningioma develops from endothelium, which is located between the dura mater and arachnoid. The tumour can grow into the trunk of the optic nerve or extend beyond the dura mater. Gliomas develop from glial cells of the optic nerve. These tumours are characterized by slow growth, they do not metastasize. However, they can lead to complete blindness. The tumour usually develops in early childhood.

The first subjective signs of tumour of the optic nerve are decreased vision and change in the field of vision. Exophthalmos grows slowly and mobility of the eyeball is usually retained in full. During ophthalmoscopy of the fundus, one can observe the congestive optic nerve or its atrophy. Tumours of the optic nerve require surgical treatment.

Theoretical questions to the chapter:

1. Anatomical and physiological features of the structure of the optic nerve.
2. Methods of examination of the optic nerve.
3. Classification of the diseases of the optic nerve.
4. The clinical presentation of neuritis of the optic neuritis.
5. Atrophy of the optic nerve: aetiology, pathogenesis, clinical signs, and treatment tactics.
6. The secondary atrophy of the optic: aetiology, pathogenesis, differential diagnosis with congestive optic disc.
7. Aetiology, pathogenesis, ophthalmoscopic presentation of the congestive optic disk, the significance of the diseases of the central nervous system in the diagnosis.
8. Possible effects of the congestive optic disc.
9. Principles of treatment of primary and secondary atrophy of the optic nerve.
10. Treatment of the partial atrophy of the optic nerve.

Practical skills which students should master:

1. Examination of the fundus by direct ophthalmoscopy.
2. Examination of the fundus by indirect ophthalmoscopy.
3. Formulation for treatment of chronic diseases of the optic nerve.
4. Determining the visual field and colour perception in patients.

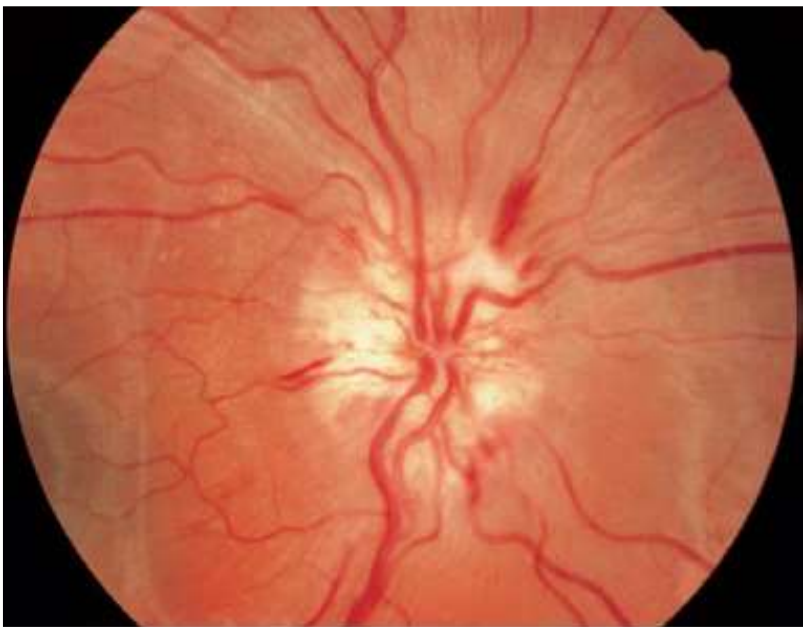


Fig. 13.1. Neuritis of the optic nerve.

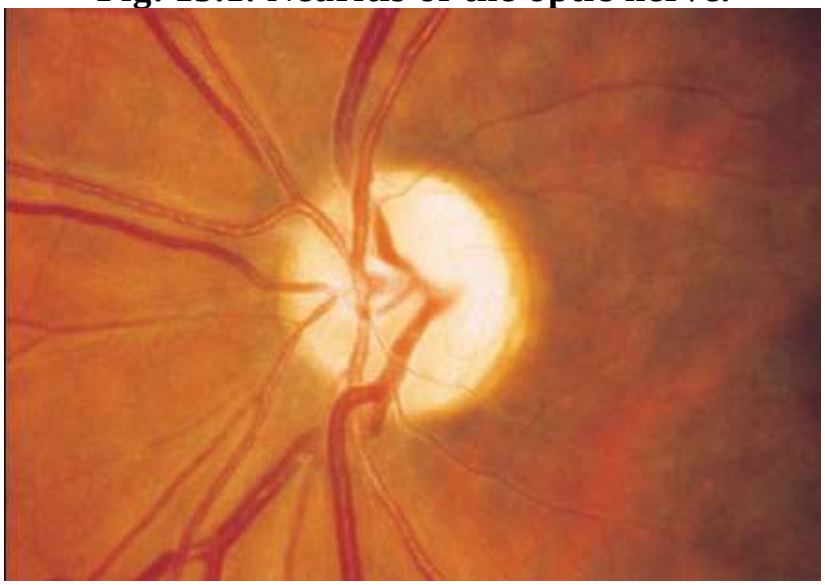


Fig. 13.2. Primary atrophy

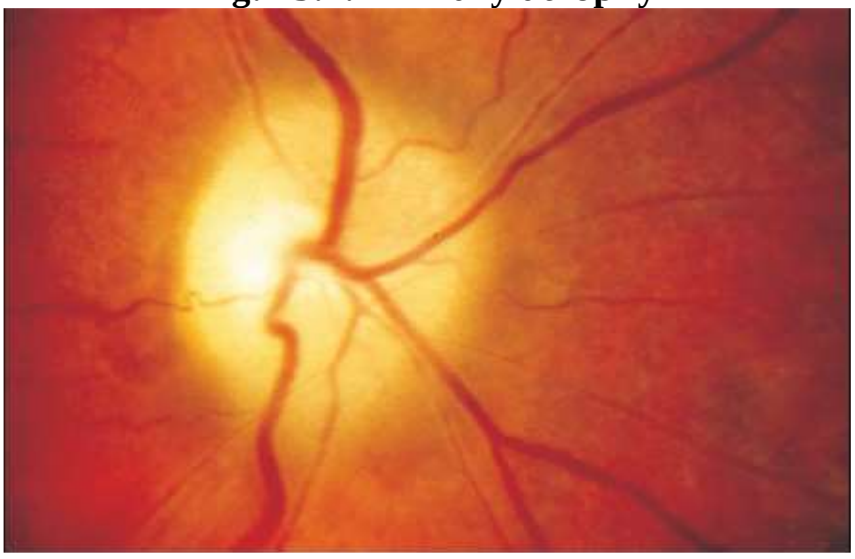


Fig.13.3. Secondary atrophy

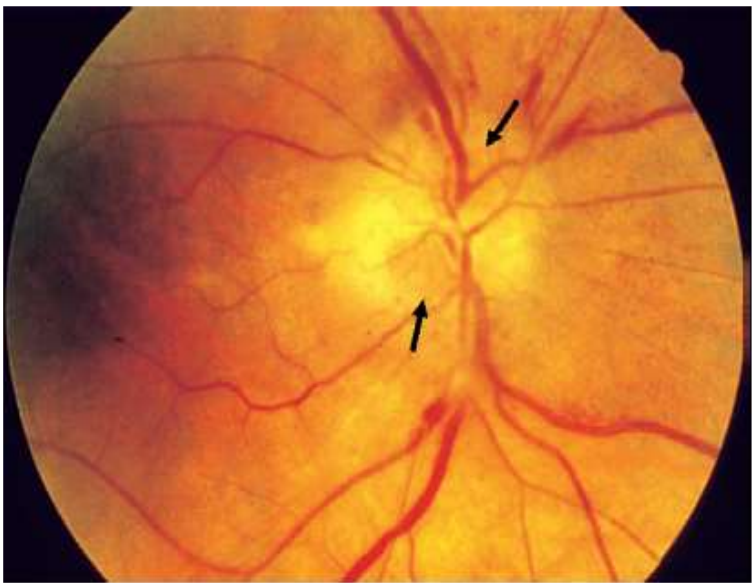


Fig. 13.4. Ischemia of the optic nerve.

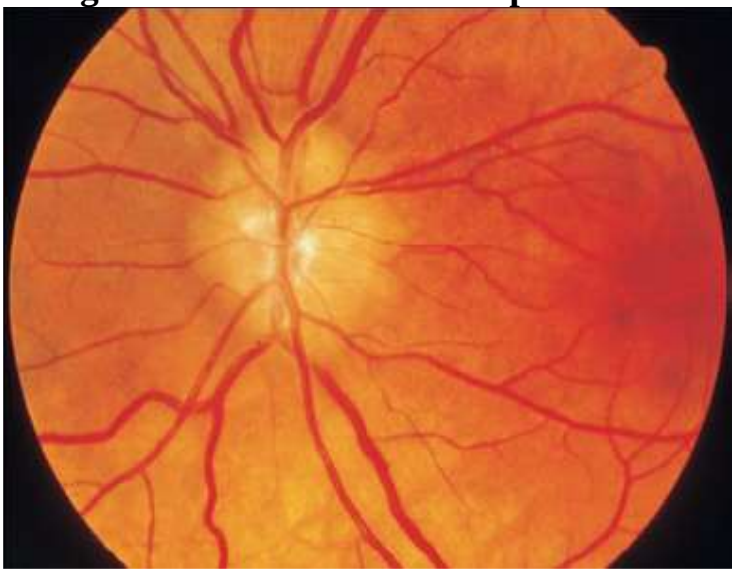


Fig. 13.5. Drusen of the optic nerve.

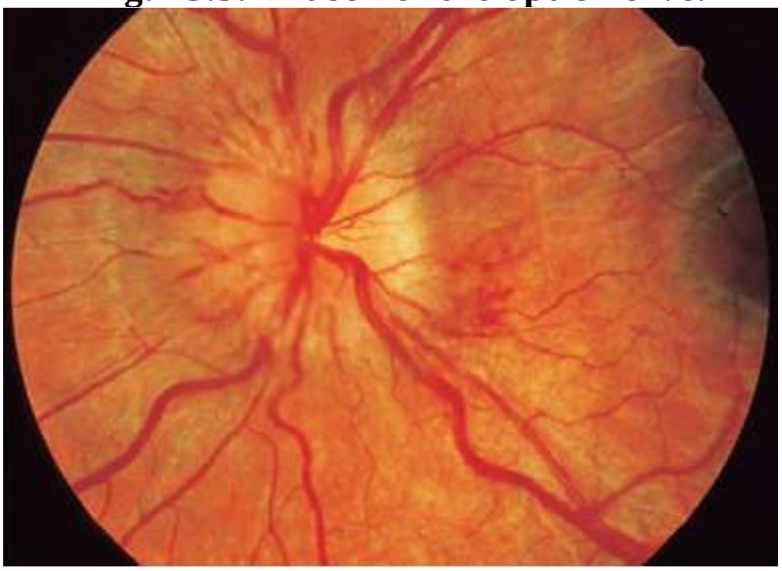


Fig. 13.6. Congestive optic disc.

CHAPTER 14

GLAUCOMA

Glaucoma is a chronic, progressive disease that affects the optic nerve with the development of a specific optic neuropathy, characteristic changes in the field of vision and impaired visual functions, which in some cases is accompanied by intermittent or persistent increase in the intraocular pressure (IOP) (definition from the Order of Ministry of Public Health, No.117 as of 15.03.07). Statistically, every fifth blind man lost the sight because of glaucoma.

Glaucoma can occur immediately or shortly after birth. If such the patient does not undergo the timely surgery, complete loss of vision may develop in 2-3 weeks. In adults, glaucoma develops after the age of 40. The frequency of glaucoma in adults is 1-1.5 cases per 100 people.

The list of key terms, parameters, characteristics that students should master while studying the topic:

Term	Definition
Glaucoma	A chronic, progressive disease that affects the optic nerve with the development of a specific optic neuropathy, characteristic changes in the field of vision and impaired visual functions, which in some cases is accompanied by intermittent or persistent increase in the intraocular pressure
Tonometry	A method for measuring the intraocular pressure (IOP). Types: palpatory, one-time, daily, hourly.
Normal IOP	Tonometric − 16-25 mm Hg, true − 9-22 mmHg.
Forms of the anterior chamber angle	Open − the angle in which all drainage structures are available for intraocular fluid; closed − the angle, blocked by the root of the iris.
Congenital glaucoma	Glaucoma, which occurs inherently or as a result of defects in foetal development and is manifested

	immediately after birth.
Open-angle glaucoma	Glaucoma, which occurs because of the changes within the drainage system of the angle of the anterior chamber. The angle of the anterior chamber thus remains open.
Closed-angle glaucoma	Glaucoma, which occurs because of the disturbance in the outflow of intraocular fluid, associated with the blockage of the anterior chamber angle by the iris root
Secondary glaucoma	Glaucoma, which results from eye diseases.
Stages of glaucoma development	Initial, advanced, far-advanced, end-stage.
Gonioscopy	A method for examination of the structure of the anterior chamber angle using a prism.

Anatomical features of glaucoma development. The adult's eyeball has a spherical shape and consists of three layers. The eye cavity contains the light-refracting and light-conducting media. The aqueous humour is produced by the ciliary processes of the ciliary body at a rate of 2.0 mm / min, it enters the posterior chamber of the eye, and from there through the pupil − into the anterior chamber. The outflow of fluid from the eye occurs mainly through the anterior channel, i.e. via the drainage system of the anterior chamber angle. The drainage system includes a porous corneal-scleral trabecula, the venous sinus (canal of Schlemm) and 20-30 collector vessels through which the filtered aqueous humour is removed from the scleral and venous sinus into the superficial veins of the sclera. Continuously circulating, the aqueous humour nourishes the avascular tissues of the eye (the cornea and lens) and excretes metabolism products of the eye tissues. The intraocular fluid includes chlorides, lactic and ascorbic acid, and small amount of protein. Production and outflow of the aqueous humour determine the level of intraocular pressure (IOP), which normally is in the range of 16-26 mmHg when measured by Maklakov's tonometer. Increased intraocular fluid production leads to the increase in intraocular pressure. However, this happens very rarely. In general, increased IOP is a consequence of the disrupted

fluid outflow. After production, the fluid flows into the posterior chamber of the eye, which is located between the lens and the iris. The fluid moves through the pupil into the anterior chamber, which is bounded by the cornea anteriorly, by the iris and lens posteriorly. The outflow of intraocular fluid occurs in the anterior chamber, where the angle of the anterior chamber is located at the boundary of the cornea and sclera. The area of the cornea's transition to sclera contains an indentation − canal of Schlemm. Its entrance is covered by connective tissue, called the trabecula. The trabecula bends under pressure into the channel and the intraocular fluid is filtered through it. Canal of Schlemm has a process of the scleral membrane − the spur, to which to iris and ciliary muscle are attached. From canal of Schlemm, the fluid outflow passes on into the venous system.

The physical essence of IOP increase is due to the imbalance between the production and outflow of the intraocular fluid. In most cases, the increase in IOP is predetermined by blockage of the anterior pathway of outflow with the aqueous humour from the eye − at the level of the anterior chamber angle. Persistent increase of IOP creates an imbalance with the pressure in the blood vessels that nourish the optic nerve and deflection of the ethmoid plate at the exit site of the optic nerve fibres, which in turn causes ischemia of the optic nerve and the deflection (recess) of the optic nerve. The optic nerve and other structures of the eye suffer from increased load and blood supply to the eye is disrupted.

One can distinguish open and closed shape of the angle. In case of closed angle of the anterior chamber, its structures are blocked by the iris root, which is attached to corneal-scleral formation above. Open-angle glaucoma develops gradually without expressed symptoms, which determines its severity. The affected eye seems unchanged, it has the usual colour, visual functions initially are not impaired, but their decline occurs already at the advanced stages with the development of atrophy (glaucomatous excavation) of the optic nerve, when there is a gradual decrease in visual acuity, specific changes in the visual field, and impaired dark adaptation.

Intraocular pressure is a relatively constant value, and in the norm it ranges from 16 to 25 mm Hg. Within 24 hours, the intraocular pressure can vary by 2-6 mm. It is

usually higher in the morning. It increases when blinking, and reduces till the evening.

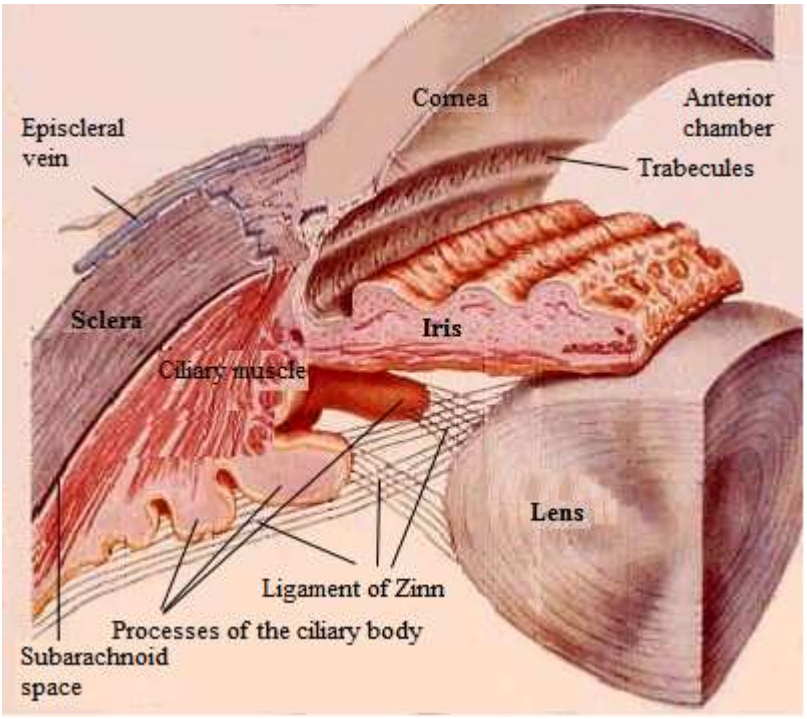


Fig. 14.1. The structure of the anterior chamber angle.

Examination methodology of intraocular pressure:

The easiest way to measure the intraocular pressure is by palpation. Although this technique is rather approximate, it gives an opportunity to detect the increased pressure under any conditions. The measurement is conducted with the forefingers through the upper eyelid of the closed eye. The eye is slightly pressed with the fingers, alternately, keeping them closely to the eyelids. The turgor pressure of both eyes is compared with the eye of a healthy person.

There are three methods of instrumental measurement of intraocular pressure: applanation (flattening), in which a load with a wide platform presses on the cornea; impression (pressing-in) − a pin is pressed under a predetermined pressure in the sclera or in the eyelid; and non-contact methods when the airwave hits the eye. In the CIS countries, Maklakov's method and Maklakov's tonometer are used for measurement of intraocular pressure. Maklakov's tonometer is a hollow cylinder weighing 10.0 grams. At the ends of the tonometer, smoothly polished layers of ground glass, 1 cm in diameter, are attached. Prior to the study, these sites should be

wiped with alcohol, then they are smeared with a layer of special dye. For measuring IOP, the eye is instilled with local anaesthetic solution (0.5% alcaine). The patient lies on the couch, face up, raises the arm up and looks at the index finger. The eyelids are open. Tonometer is lowered vertically on the centre of the cornea, the holder tweezers are unbraced. Under the pressure of the load, normally spherical cornea bends and flattens. The dye is washed off by the tear due to the contact with the cornea. The lower the intraocular pressure is, the more the cornea flattens, the greater the area contact is, and thus the more ink is washed off. After imprinting on a sheet of paper, only a trail of a washed away circle with a large diameter remains, and vice versa. Tonometer is turned over the other side and the intraocular pressure is measured again. IOP of another eye is measured in exactly the same way.

After the measurement, recording of the prints is carried out on the paper moistened with alcohol, and in accordance with Polyak's scale, in which the washed away circles "fit", determining the intraocular pressure.

A single detection of increased intraocular pressure cannot yet indicate the diagnosis of glaucoma, since a transient increase in intraocular pressure can be random, in excessive water consumption, etc. In case of identifying the repeated increases of intraocular pressure, subsequent examination of the patient is performed. In assumption of glaucoma, daily measurement of the intraocular pressure is conducted, in the morning and in the evening, for a week; hourly − 6 times a day (every 3 hours) for 2 days. When conducting a daily tonometry, attention is paid to the pathology of diurnal IOP curve if its limits are outside the normal parameters of tonometric intraocular pressure (16-25 mm Hg.), or it has high peaks. Considering the effect of increased pressure on the eye membranes, it is important to study their elasticity and possible reactions to the increased pressure. For this purpose, elastotonometry is used (drafting the correspondence diagram of the tonometer weight in grams and the value of the tonometric pressure − it displays the reaction of the eye membranes in response to increased pressure on the eye). To diagnose the hydrodynamics disorders of the eye (intraocular fluid outflow), Filatov's applanation tonography is applied.

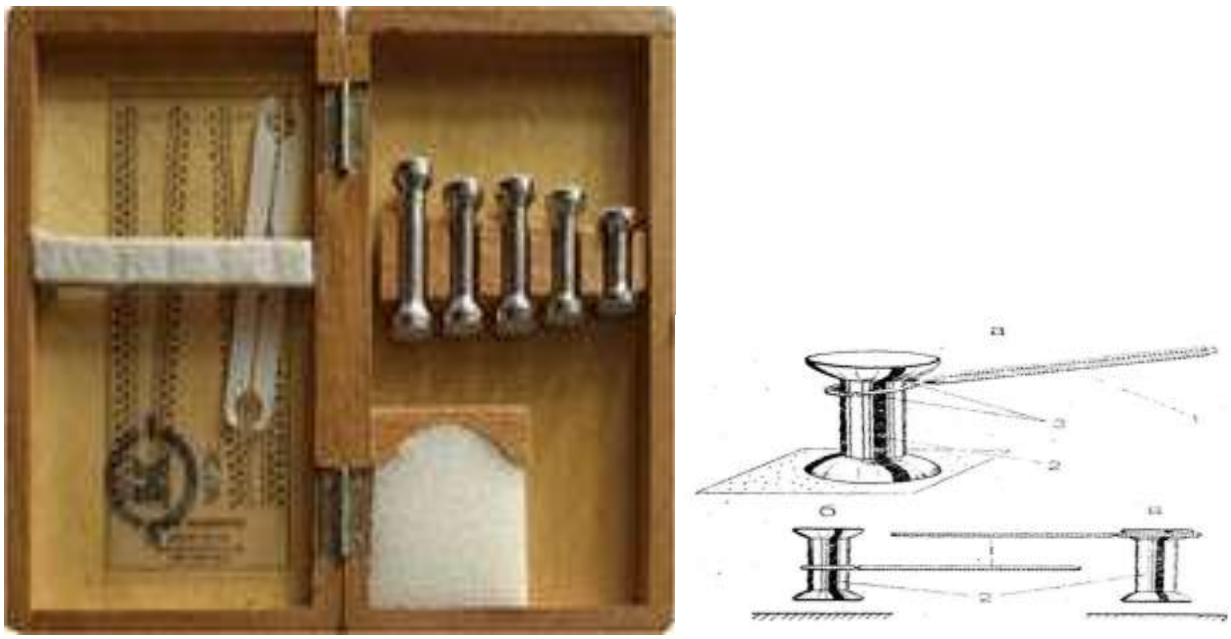


Fig. 14. 2. General view of Maklakov's tonometer.

Fig. 14. 3. The technique of dye application on Maklakov's tonometer

Fig. 14.4. The technique of Maklakov's tonometry.

Fig. 14.5. Palpatory measurement of the intraocular pressure.

Kalf-Plyushko method is carried out using the Filatov-Kalf set of tonometers weighing 5.0; 7.5; 10.0; and 15.0 g. A significant contribution to the development of applanation tonography technique was made by the former head of the Department of Ophthalmology of Poltava Medical Stomatological Institute Prof. D.G. Plyushko.

Electronic tonography consists in performing an extended tonography (4 minutes) using a special tool. On the tonograph's display, the doctor sees the data on the actual intraocular pressure (Po), then using special tables, the basic hydrodynamic parameters are calculated: the ease coefficient of the aqueous humour outflow C (normally 0.2-0.3 mm / min / mm Hg), minute volume of aqueous humour F (OK − 1.1-4.0 mm / min) and Becker coefficient Po / c (normally 30-100).

The form of glaucoma is diagnosed by gonioscopy. Gonioscopy is a method of examination of the anterior chamber angle using the gonioscope − a device with a system of mirrors that refract light. In open angle glaucoma, the entire anterior chamber angle is examined, and in closed-angle glaucoma − the angle covered by the iris root and it is impossible to examine its structure.

Changes of the optic nerve in glaucoma can be examined using ophthalmoscopy. The newest technology for examination of the optic nerve is optical coherence tomography (OCT), examination of the optic disk using the nerve fibre analyzer or

Heidelberg tomograph. The optical coherent tomography, scanning the nerve fibres, provides the clear characteristics of the damaged portion of the optic nerve fibres.

Further examination of the patient is carried out: collection of complaints and anamnesis, testing the visual acuity, field of vision on the perimeter and campimeter or on the static computer perimeter that replaces the two previous devices, examination of the eye with the side lighting and transmitted light.

Classification of glaucoma

Glaucoma is divided:

1. By origin:

- primary − disorders associated with the drainage system of the eye, and as a result of deterioration of the intraocular fluid outflow.
- secondary, which arises as a result of other eye diseases (inflammation, cataracts, diabetic changes thrombosis, injury, tumour, etc.), or after eye surgeries.

2. By the age the patient:

- congenital − is manifested before the age of 3 years
- infantile (from 3 to 10 years)
- juvenile (from 11 to 30 years)
- glaucoma of adults.

3. By the mechanism of ocular hypertension:

- open-angle
- closed-angle.

4. By the level of intraocular pressure:

- with normal IOP
- with increased IOP,
- with high IOP.

5. By the stages of glaucoma:

- initial
- developed
- far-advanced
- end-stage

6. By the course of the disease

- stable,
- unstable.

Congenital glaucoma is the most severe eye disease in newborns, which leads to early blindness more often than any other eye diseases. Congenital glaucoma is often called hydrophthalmos (“hydrated eye”) or buphthalmos (“bull’s eye”). The process is usually bilateral. It occurs in approximately 1 out of 10 thousand children. The disease can be hereditary (familial) nature, or develop because of disorder during the prenatal period. It is more common in boys. Late detection in children and failure to conduct a timely surgery lead to complete blindness as early as the preschool years. Surgical treatment of congenital glaucoma is carried out at children's ophthalmology department.

Clinical presentation of congenital glaucoma. Since congenital glaucoma is based on underdevelopment or abnormal development of the drainage system of the eye, blockage of the trabecular area of the anterior chamber angle by the foetal tissue, which is not absorbed until the end of foetal development and prevents the outflow of aqueous humour, the disease is characterized by increased intraocular pressure, progressive increase of the cornea and all dimensions of the eyeball, excavation of the optic disc and impaired visual functions. Due to the fact that the eye is overoccupied with plenty of intraocular fluid, thin membranes of the child's eye are quickly stretched, forming an enlarged eye − hydrophthalmos or buphthalmos (“bovine eye”). Treatment is only surgical (goniotomy, goniopuncture, trabeculotomy, trabeculectomy).

Acquired glaucoma is divided into primary and secondary forms. Primary glaucoma makes up about 80% of all glaucoma cases and is observed in 3 major clinical forms: open-angle closed-angle and mixed. Anatomical changes that can increase the IOP in primary glaucoma are located in the drainage system of the anterior chamber angle of the eye. The factors, contributing to the development of disorders in the drainage system of the anterior chamber, may be systemic diseases, diseases of the cardiovascular and nervous systems, metabolic disorders, the effect of irritants on the

body, such as the prolonged emotional stress. Primary glaucoma usually affects both eyes in turns. Primary glaucoma goes unnoticed for a patient which, in turn, allows the disease to progress and ophthalmologists have to deal with serious, advanced cases, when there is a question only of preserving the residual vision, and sometimes to maintain the eye as an organ and pain relief. Glaucoma develops in 4 successive stages − initial, advanced, far-advanced and end-stage. Each of them is determined by the state of the field of vision and the size of the optic disc excavation. One can also distinguish the condition of pre-glaucoma. In pre-glaucoma, the parameters of intraocular fluid are reduced, but there are no complaints. This condition requires constant monitoring of the ophthalmologist.

There are four stages in the course of primary glaucoma. Initial stage: at this stage IOP increase may be the only symptom. The patient may not have complaints; blurred vision may occasionally occur. The "annular scotomata", or "the blind spot expansion symptom" may be observed in the field of vision. At the advanced stage, there is narrowing of the visual field to the nasal side by 10-15 degrees. The patient complains of impaired visual acuity. Marginal excavation of the optic disc is observed in the fundus. Far-advanced glaucoma is characterized by a dramatic deterioration of visual acuity. The field of vision is narrowed to the tubular form, leaving up to 15 degrees from the fixation point. In the fundus: deep excavation of the optic disc. End-stage glaucoma is characterized by complete loss of vision. Optic disc becomes atrophic and gray.

While open-angle glaucoma develops in stages, the closed-angle form is characterized by attacks, during which the eye can undergo through several stages of development at once. *The acute attack of glaucoma*: occurrence of attacks is promoted by the factors that cause pupil dilation and aggravate the blockage of the anterior chamber angle. Psycho-emotional stress, physical overexertion, and other factors (abuse of tobacco, alcohol, lifting heavy weight, work with body bending, disrupted diet and so on) may contribute to the development of the acute attack of glaucoma. The clinical presentation of attack is as follows: sudden headache, which extends into the temple and jaw, sharp pain in the eye. The eye becomes red, hard as

a stone, the cornea is turbid due to oedema, the pupil is dilated, the pupillary response to light is absent, the vision is reduced (up to blindness). Intraocular pressure during the attack can be increased up to 50-60 mm Hg. Quite often, the acute attack of glaucoma is accompanied by nausea and vomiting that may be mistaken for gastrointestinal poisoning. Pain is always radiating in the area of the heart, which can also lead to a diagnostic error. Therefore, instead of immediate topical use of antihypertensive drugs, medicines for treatment of poisoning or angina can be administered, which worsen the course of glaucomatous process (gastric lavage, validol, atropine, nitroglycerin and so on).

The acute attack of glaucoma requires emergency care. Proper diagnosis and prompt comprehensive conservative treatment, usually within one day eliminate the acute attack of glaucoma, renewing the vision, and normalizing the condition of the eye. When normalizing IOP for more than a day, the patient may develop the irreversible blindness because of the death of optic nerve fibres. The acute attack of glaucoma occurs with the fairly pronounced changes on the part of the eyeball, which looks inflamed due to the dilated venous vasculature of the eyes and its overloading with blood. The anterior camera is rather shallow, the pupil is dilated, the cornea is oedematous. The acute attack of glaucoma is differentiated from acute iridocyclitis. The main difference is the size of the pupil: in the glaucomatous attack it is wide, in iridocyclitis − narrow; the anterior chamber depth: in the attack − it is small, in iridocyclitis − normal; in IOP: in glaucoma − sharply increased, in iridocyclitis − normal or even reduced; the corneal condition: in the attack − turbid, oedematous, in iridocyclitis − with precipitates; and vascular injection of the sclera: in the acute attack − congestive, in iridocyclitis − pericorneal.

First aid in the acute attack of glaucoma. Topically: 1% pilocarpine every 15 min for 1 hour, then every half-hour during the second hour, then every hour until lowering IOP, then 6 times a day depending on the degree of IOP reduction. In the period between the attacks, the administration rhythm of pilocarpine is 3 times a day. In the insufficient reduction in IOP 0.5% timolol twice a day and 2% azopt 3 times a day are added. Orally: diacarb 0.25 grams 2-3 times per day; osmotic agents (glycerin −

1-1.5 g / kg per day). Parenterally: 20% mannitol for 30 min. by 1.5-2 g / kg intravenously; 1% furosemide 20-40 mg / day intravenously or intramuscularly; lytic mixture − 2.5% chlorpromazine 1.2 ml, 2% diphenhydramine 1 ml, 2% promedol 1 ml. Distracting procedures: hot foot baths, leeches on the temporal area. If the attack cannot be eliminated after 12-24 hours, a surgery must be conducted.

Open-angle glaucoma is a chronic disease characterized by a stepwise course, and different values of intraocular pressure: normal − up to 27 mm Hg, moderately increased, − from 28 to 31 mm Hg, and high − 32 mm Hg and higher. Visual function in glaucoma can be assessed as stable (no changes in the field of vision and the optic nerve within six months), and unstable (when the field of vision is narrowed by 10 degrees at individual radii within six months). Open-angle glaucoma develops virtually unnoticed. Since the field of vision is narrowed gradually, sometimes a person accidentally discovers that he/she sees with only one eye, and another is blind. Such cases happen frequently. There are almost never complaints from patients with open-angle glaucoma, IOP is intolerant (not corresponding to the eye), the anterior chamber angle is open, reduced visual acuity, starting from the advanced stage, the field of vision changes, the optic disc immerses (excavation develops). Due to the gradual decrease in visual acuity in open-angle glaucoma, it often confused, even by ophthalmologists, with age-related cataract, although there is no clouding of the lens in glaucoma, and the loss of visual function is associated with the progressive development of glaucomatous atrophy of the optic nerve, which may be revealed both by ophthalmoscopy, and optical coherence tomography. Unfortunately, when confusing these two diseases, medical care is not adequately targeted at the underlying disease (glaucoma), which leads to disability and irreversible blindness. In the progression of cataract, the fundus is not visible, and therefore it is not possible to establish the status of the optic nerve. In this case, the only sign for differential diagnostics of glaucoma is the characteristic visual field loss, which is not possible in cataract, and increased IOP is also possible. In closed-angle glaucoma, the liquid accumulates inside the eye due to the fact that the iris root overlaps the anterior chamber angle, i.e., there is no access to the drainage system of the eye. In *closed-*

angle glaucoma, patient complains of pain, iridescent circles, continuously or intermittently elevated IOP, anterior chamber angle is closed, the anterior chamber is shallow, excavation of the optic nerve develops with recurrent acute attacks.

The first stage of detection of glaucoma includes preventive examinations of healthy people over the age of 40. Early detection of glaucoma is very important, since it leads to irreversible blindness. Given the possibility of late complaints of patients with glaucoma, doctors often have to deal with end-stage glaucoma. It should be remembered that its clinical manifestations develop as a result of IOP, inappropriate to the eye (intolerant). Late stage of glaucoma with low (below 0.08) visual acuity or sharply narrowed (less than 15˚ from the fixation point) field is an indication of the direction to medical and social assessment. In the case of complete absence of visual functions (visual acuity 0) and the presence of a pronounced pain syndrome, the presence of absolute pain glaucoma − the option of enucleation is discussed.

Secondary glaucoma is a disease that has developed against the background of other diseases of the eye and the body. Secondary glaucoma is characterized by large variety of clinical forms. Depending on the disease, which caused the disorder within the outflow of the aqueous humour, these diseases can be combined into seven major groups:

• inflammatory and postinflammatory glaucoma − induced by sclerites, uveitis or keratitis;

• phacogenic glaucoma − phakotopic, phakomorphous, phacolytic;

• vascular glaucoma − neovascular, phlebohypertensive;

• dystrophic glaucoma − in retinal detachment, in iridocorneal endothelial dystrophic syndrome or hemolytic;

• traumatic glaucoma − contusion, wound-induced, burn-induced, radiation, postoperative;

• neoplastic glaucoma − in intraocular tumours, in tumours of the orbit and endocrine exophthalmos;

• glaucoma, caused by corticosteroids.

Treatment of secondary glaucoma involves primarily the management of the underlying disease and elimination of its consequences.

Principles of comprehensive treatment of glaucoma primarily involve the development of administration regimen for topical antihypertensive agents, stabilizing the control of glaucomatous process and periodic (half-yearly) prescription of vascular and neuroprotective therapy. To preserve visual functions, it is necessary to keep the intraocular pressure within the individual (tolerant) standards. For this purpose, agents that reduce intraocular pressure are prescribed. These are miotics that narrow the pupil (in closed-angle glaucoma) and drugs that improve the outflow of intraocular fluid or reduce its volume (in open-angle glaucoma). Prostaglandin analogues are used to improve the outflow of intraocular fluid (xalatan, travatan, taflotan). The amount of generated intraocular fluid is reduced by drugs that block adrenoceptors (timolol, arutimol, cusimolol), carbonic anhydrase inhibitors (azopt, trusopt). The patient is contraindicated working at night, at high temperatures, to stay in the positions with inclined head for a long time. It is recommended to reduce the amount of fluids intake and limit the consumption of salty foods. In closed-angle glaucoma, it is contraindicated to stay in the dark, consume caffeine, alcohol, to take drugs that dilate the pupil (atropine, scopolamine, belladonna preparations).

In case of no or insufficient effect of conservative treatment, laser therapy is used, and if it is also inefficient − surgical methods of treatment. Trabeculospasis and gonioplasty can be conducted as the laser treatment. For surgical treatment of glaucoma, the operations of filter type are carried out − trabeculotomy sinusotomy, viscocanalostomy. In cases of end-stage pain glaucoma, especially in secondary neovascular glaucoma, pain treatment can be carried out, aimed at reducing the amount of production of intraocular fluid − diode laser diathermocoagulation or the ciliary body. The most common are fistulizing operations in which a channel for resumption of outflow of intraocular fluid is formed in the wall of the anterior chamber angle. In closed-angle glaucoma, iridectomy is conducted − excision of the iris portion which covers the structures of the anterior chamber angle. This operation is typically used in acute glaucoma attack. In absolute pain glaucoma, retrobulbar

administration of 96 degrees alcohol with 2% procaine is sometimes used for pain management. In case of pain relapse, enucleation can be carried out.

Patients with glaucoma should be under constant medical supervision of the ophthalmologist, with regular check-ups of IOP, the state of the fundus and visual functions once in 3 months. It is impossible to completely cure this disease − it is chronic. But with timely, correct and consistent treatment, the progression of glaucoma may be suspended and good vision can be preserved.

Theoretical questions to the chapter:

1. The angle of anterior chamber of the eye: anatomical and physiological features.
2. The mechanism of IOP regulation.
3. Methods of IOP examination.
4. Gonioscopy, types of the anterior chamber angle.
5. The classification of glaucoma.
6. Pathogenesis, clinical presentation and treatment of patients with congenital glaucoma.
7. The primary aetiology of glaucoma.
8. Classification of primary glaucoma and its detailed characteristics.
9. The volume of necessary studies in patients with glaucoma.
10. The principles of conservative treatment of glaucoma.
11. Clinical presentation of acute attack of angle-closure glaucoma, emergency management.
12. The methods of surgical treatment of patients with glaucoma.
13. Prevention of glaucoma.

Practical skills which students should master:

1. Measurement of IOP by palpation method.
2. Evaluation of tonometry data by Maklakov’s method.
3. Emergency care in the acute attack of glaucoma.

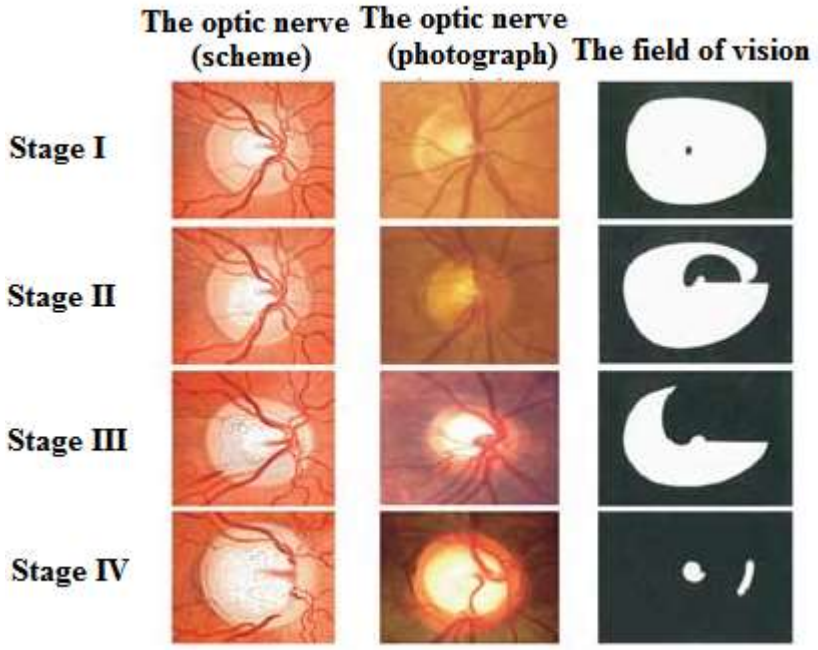


Fig. 14.6. Changes to the fundus of the eye in glaucoma

CHAPTER 15

EYE INJURIES

Eye injuries are divided into mechanical traumas and burns. Mechanical traumas, in turn, are divided into concussions and wounds. Contusion are inflicted by blunt objects (fist, snow, lump of earth, stone and the like), and wounds − by sharp, prickly or cutting instruments (scissors, needle, nail, etc.). Contusions mainly cause damage to all the parts of the organ of vision, but they are conventionally divided into contusions of appendages and contusions of the eyeball. Wounds are also divided into those of appendages and of the eyeball. Wounds of the eyeball, in turn, are divided into penetrating and non-penetrating. Penetrating wounds of the eyeball can be with and without intraocular foreign body. Burns are divided into thermal (caused by the action of boiling water, flame, molten metal, hot oil), chemical (caused by the action of acids or alkali) and radiation (caused by the action of ultraviolet, infrared and X-rays). Combined injuries to the organ of vision are observed quite often. Thus, for example, during a mine explosion or other combat devices, a person can sustain a burn, a contusion and a penetrating wound of the eye. Depending on the circumstances, under which the damage occurred, injuries are divided into employment (industrial and agricultural), household, school, athletic, military and criminal. All types of injuries have their own specific features.

The list of key terms which the students should master while studying the topic:

Term	Definition
Eye contusion	Eye injuries, caused by blunt objects
Penetrating wounds of the eye	Eye injuries with impaired external (fibrous) layer of the eye
Signs of penetrating wounds	Direct: the presence of a wound, the hole in the iris or in the anterior capsule of the lens, the presence of intraocular foreign body
Methods of diagnosis of intraocular foreign bodies	X-ray examination − X-ray localization by Comberg-Baltin and Vogt, ultrasound

	examination
Classification of eye burns	Thermal, chemical, radiation
Types of necrosis in chemical burns	Colliquative, coagulative
Ultraviolet ray ophthalmia	Cornea burn with ultraviolet rays (welding, quartz, etc.).
Sympathophthalmia	Malignant inflammatory process of the healthy eye resulting from posttraumatic inflammation

1. Mechanical damage to the organ of vision.

Contusion of appendages. Blunt trauma of great strength can primarily lead to fracture and displacement of the bones of the orbit. Depending on the nature of the bone fragments displacement, the orbit bone fracture may be accompanied by retraction of the eyeball (enophthalmos) or its protrusion (exophthalmos). If there is a compression in the upper orbital fissure, innervation may be impaired, which is manifested by limited eyeball movements, absence of skin sensitivity in the regions of the trigeminal nerve innervation, ptosis, mydriasis, exophthalmos. The palpation of the orbit edges at the fracture site is painful. In case of the bone fragments displacement, irregularities are palpable. Bone changes, corresponding to the fracture, are visible on radiographs. In the presence of fractures of the inner wall of the orbit, the air from the additional (ethmoidal) sinuses can enter under the skin of the eyelids (eyelids emphysema). After careful interviewing of the patient, it turns out that emphysema appeared not immediately after the injury, but after the patient exerted an effort − blew his/her nose or coughed. During examination, swelling of the eyelids, and air crepitus on their palpation are observed. In the area, adjacent to the edge of the orbit bone fragment, contusion of the skin may cause dilaceration. This dilaceration often reminds an incised wound.

Treatment. In cases of fractures of the orbit, the paranasal sinuses and the brain can also be damaged. Therefore, when treating such patients one needs to consult with the

otolaryngologist and neurosurgeon or neurologist. Treatment is carried out with the participation of these specialists.

Emphysema of the eyelids does not require treatment. It is recommended to refrain from blowing one's nose intensively.

Contusion of the eyelids often causes subcutaneous haemorrhage. In case of minor bleeding, the patient does not require therapy, haemorrhages resolve without treatment. In case of large hematomas, including retrobulbar ones, it is necessary to apply cold compress on the portion of the eyes for the first 2 hours after the injury, to prevent subsequent hematoma growth. Sometimes a pressure bandage is applied and haemostatic agents are used to stop bleeding. After the third day, enzymatic ointment preparations, thermal procedures are used to promote absorbable hematoma resolution.

Dilaceration of the eyelids in case of contusions requires primary surgical treatment.

Contusions of the eyeball. Contusions of the eyeball may cause a variety of injuries. Most often, subconjunctival haemorrhage, bleeding into the eye's anterior chamber (hyphema) and the vitreous body (haemophthalmos) occur.

Hyphema looks like a belt of blood at the bottom of the anterior chamber. Sometimes hyphema may fill the entire anterior chamber. In this case, due to the disrupted outflow of the aqueous humour of the anterior chamber, an increase of intraocular pressure (hypertension) often occurs. Vitreous haemorrhages are possible − haemophthalmos (its clinical presentation is described in Chapter "Diseases of the Lens and Vitreous Body"). A frequent consequence of eye contusion is the subluxation and dislocation of the lens, as well as traumatic cataract (see Chapter "Diseases of the Lens and Vitreous Body").

In the case of haemorrhage into the anterior chamber, the vitreous body, and the retina, haemostatics are administered and after a while, the resorbable therapy is carried out − 2%-3% potassium iodide solution in the form of eye drops, biostimulants (aloe, vitreous humour, FiBS, etc. as subcutaneous. injections, fibrinolysin solution as subconjunctival injections, lidasa intramuscularly and others).

Non-penetrating injuries also include foreign bodies, erosion, and non-penetrating wounds of the cornea. Non-penetrating wounds of the sclera are rare. Foreign bodies of the cornea are often fine particles of metal, emery stone, glass, that enter the eye at a sufficiently high speed. Least frequently, these include rolling pins of wood, thorns. The latter penetrate primarily into the deep layers of the cornea and break off. The presence of foreign bodies is accompanied by corneal syndrome: pain, lacrimation, photophobia, blepharospasm. For the most part, foreign body is easy to find during the examination on the slit lamp. If a foreign body remains in the cornea for a long time, the inflammatory infiltrate may appear around it. Around metallic foreign bodies, a belt of rust can form in 2-3 days.

Erosion of the cornea is caused by foreign bodies, stroke with a branch, or any other objects. Corneal erosion is characterized by pronounced corneal syndrome. In this case, the sensation of a foreign body can be so tangible, that the patient often cannot believe in its absence. Fluorescein test is effective for identifying erosions. After instillation of fluorescein, the erosion is painted in green colour and it can be clearly seen against the background of the uncoloured cornea.

Non-penetrating corneal injuries are relatively rare. In this a case, it is necessary to conduct a thorough examination by the slit lamp, which will help to ensure that the corneal wound is non-penetrating. Fluorescein test is also used: if there is a filtration of aqueous humour of the anterior chamber through the corneal wound, then a transparent “trickle” is clearly visible against the background of fluorescein.

Emergency care and treatment. Due to the rapid reproduction of epithelial cells, corneal erosion is quickly self-healing. However, for the prevention of bacterial keratitis antibacterial drops and ointments are applied. To stimulate epithelialization, the instillations of thiotriazoline drops, corneregel, actovegin or solcoseryl ophthalmic gels are used. Local anaesthetics, which are sometimes used to relieve pain, are contraindicated, since they inhibit epithelialization.

Treatment of non-penetrating injuries of the cornea is the same as in traumatic erosions. Stitches are applied on the wound only in case of valvular damage. Specialized surgical manipulations are required in scalp injury of the cornea, when

the surface layer of tissue or a flap detached from it and has a tendency of wrapping edges or shifting, and in case of presence of a foreign body in the deep layers of the cornea. Corneal foreign body must be removed immediately. To do this, after epibulbar anaesthesia, under the slit lamp, a foreign body is “raised” by the tip of a lancet-shaped needle and thus removed. Movement of the needle must be directed away from the corneal centre to the periphery with the following cleansing of the rim from rust or dust which is always found around the bed of a foreign body. Without doing this, the healing of corneal defects will be long.

Foreign bodies can be removed from the deep layers of the cornea only in the operating room using a surgical microscope. After removal of the corneal foreign body, it is necessary to conduct the fluorescein test, instill the antibacterial drops (0.25% chloramphenicol solution, gentamicin, tobrex, cyprolet, cyloxan, floxal, oftaquix, vigamox, or zymaxid and others), apply the antibacterial ointment (tetracycline, gentamicin, floxal and others) and an aseptic dressing for 1 hour.

The surface conjunctival lesions are often associated with minute foreign bodies (grit, a piece of bark, the wings of insects and the like), entering the conjunctival sac. Foreign bodies are often caught under the upper eyelid. Sometimes sharp foreign bodies (e.g., a spine, a prickle, a piece of glass, an eyelash) stuck into the mucosa. It is difficult to detect such foreign bodies in the conjunctiva. They cause tearing, photophobia, sensation of a foreign body, particularly when blinking. A common location is the mucous membrane of the upper eyelid. Therefore, in case of complaints of a foreign body in the eye, it is required to evert the upper eyelid and carefully examine the mucosa.

If a foreign body is located behind the lower eyelid, then it is washed away with tears due to blinking and lacrimation. If it is not washed away, one needs to pull away or evert the eyelid and remove it with a wet cotton wool ball or a piece of gauze. For removing the foreign bodies that are stuck in the mucous membrane, it is more convenient to use tweezers.

The symptoms of postcontusional mydriasis may occur, which are either compensated with time, or remain forever, not responding to the use of miotics.

Manifestations of such contusions are detachments of the iris from its root (iridodialysis), as well as ruptures or tears of the sphincter of the pupil. Such injuries are accompanied by hyphema, which were mentioned above, and also may affect the increase of intraocular pressure in severe iridodialysis or its decrease (hypotension) − under the action of the damaging factor on the ciliary body.

Changes in the retina in case of contusion may be the concussion of the retina, retinal haemorrhages, and even its rupture. Concussion of the retina (retinal commotion) is manifested by oedema, pallor and decreased visual acuity. Retinal oedema resolves completely in a few days. However, it may cause the development of retinal degenerative changes in the future. Retinal haemorrhage may lead to the development of cicatrical processes in the retina and a significant reduction of visual acuity. Large haemorrhages can leave degenerative foci. Retinal rupture causes its detachment.

Particularly severe cases of contusion can lead to the rupture of the sclera of the eyeball. Scleral rupture most often occurs near the limbus. In this case, the conjunctiva can remain intact − this is called the subconjunctival scleral rupture. Under such conditions, haemorrhage appears under the mucosa, which masks the scleral rupture.

In a strong strike directly on the eye, dilaceration of the optic nerve occasionally occurs, which results in sudden, complete and non-recoverable blindness of the eye. A strong stroke can cause damage to the lens − dislocation (subluxation) and postcontusional cataracts.

Emergency prehospital care. Patients with contusion of the eyeball are administered haemostatics (10% calcium chloride solution intravenously, 12.5% dicynon, 1% vicasol in injections or tablets), and a dressing is applied. The patient is immediately sent to the ophthalmologist at the polyclinic or hospital, depending on the severity of the injury. In case of retinal concussion, intravenous injections of hypertonic solution of sodium chloride, glucose with vitamin C are administered. In case of retinal rupture, laser coagulation of the retina is performed for prevention of its detachment. In the presence of retinal detachment, surgical treatment is carried out.

Ruptures of the sclera (both subconjunctival and extensive) require the primary wound treatment. A symptom of subconjunctival rupture of the sclera can be drop-out of the lens, the choroid, the vitreous body under the conjunctiva. Characteristic symptoms of the subconjunctival scleral rupture: chemosis of the conjunctiva, subconjunctival haemorrhage, the occurrence of coloboma of the iris, the displacement of the pupil, sudden and significant bleeding in the anterior chamber of the eye, folds of Descemet's membrane, ocular hypotony, translucence of the choroid beneath the conjunctiva.

Emergency prehospital care. Patients with subconjunctival scleral rupture are given haemostatics, antibacterial drops are instilled into the eye, a dressing is applied, and the anti-tetanic serum is administered. The patient is immediately directed to the ophthalmic injury care centre.

2. Wounds.

Wounds of the eye, just as contusions, are divided into those of appendages and of the eyeball.

Wounds of the eyelids can be in the form of incised wounds, tears and dilacerations. Wounds of the eyelids in the internal region often involve the lower lacrimal canaliculus. Due to a good blood supply, the regenerative process of the eyelids is fast, but the scar at the site of lacrimal canaliculi may lead to obstruction and lacrimation. Wounds of the conjunctiva are not dangerous. The danger resides in the subconjunctival haemorrhage which often arises in case of wounds of the mucosa; it may mask the sclera wound.

Emergency management. In the presence of wounds of the eye's appendages, as well as in wounds of any location, the anti-tetanic serum is administered. If the patient had not been vaccinated, antitetanic antitoxin is also administered. Wounds of the eyelids are washed with antiseptic solutions. The eye is instilled with antibacterial drops, a sterile dressing is applied and the patient is referred to the ophthalmologist. Wounds of the eyelids require primary surgical treatment. The stitches of a thin suture material are applied on the eyelids very carefully to prevent the cosmetic defect. Particular attention should be paid to the wounds with damage to the lower lacrimal

canaliculus. Primary surgical treatment of wounds is performed using the surgical microscope. It involves the matching and stitching of the wall of lacrimal canaliculus with a special probe. Without this, lacrimation will inevitably occur in the future. The conjunctival wound is also sutured. But in case of minor wounds with a good adaptation, it is not necessary to put stitches − conjunctival wounds heal very well. After the primary surgical treatment, antibacterial drops and ointments are applied. The wound of the eyelids is periodically treated with a solution of diamond green. The stitches are removed on the 5th-7th day.

Wounds of the eyeball are divided into non-penetrating and penetrating. In penetrating wounds, the integrity of all layers of eye's fibrous membranes (the cornea or sclera) is impaired. Complications of wound healing can be endophthalmitis, sympathetic inflammation and the like. Therefore, timely diagnosis and rapid medical care in case of penetrating wounds of the eyeball are extremely important.

In penetrating wounds, the patient notices pain in the eye, visual acuity is often reduced. These complaints depend on the degree of damage to the eye tissues and can vary to a wide extent.

During the check-up, the wound itself is examined in the first place (the entry hole of the wound tract). The edges of the wound can be more or less adapted. The wound of the cornea is often plugged by the iris. The iris prolapsing through the corneal wound is sometimes observed. In penetrating wounds, the sclera is predominantly dark coloured, because the choroidal pigment is visible in its depth. In case of extensive penetrating injuries, the vitreous body may fall out through the wound. Penetrating wounds are quite often accompanied by haemorrhage into the anterior chamber (hyphema) or the vitreous body (haemophthalmos). In case of penetrating wounds of the cornea, the anterior chamber may be shallow due to the outflow of humour therefrom. In case of penetrating wounds of the sclera and the loss vitreous body, the anterior chamber can be recessed. In the presence of penetrating corneal injuries, the damage to the iris can be often observed. The damage to the lens sac leads to traumatic cataract. Due to penetrating wounds of the eye, the intraocular eye pressure

decreases (hypotension). Penetrating wounds with intraocular foreign bodies are particularly dangerous.

Therefore, in case of penetrating wounds, especially if there is a suspected intraocular foreign body, it is necessary to conduct the spot-film radiography of the orbit. In case of detection of a foreign body, its X-ray localization by Comberg-Baltin is conducted. After topical epibulbar anaesthesia with alcaine solution, Baltin's prosthesis is set on the front surface of the eye. X-ray images are made in two projections and calculation of the foreign body location is performed, using special schemes. It should be kept in mind that only roentgenopaque foreign bodies are displayed on the radiograph, whereas glass, plastic, wood and the like cannot be displayed. In such cases, it is necessary to perform ultrasonic diagnosis, namely ultrasound biometry, or B-scanning.

Complications of the penetrating wounds of the eyes. In addition to the direct damage of eye tissues (structures), which occurs along the wound tract (iris rupture, traumatic cataract, stroke and the like), penetrating wounds may lead to a number of complications.

Retinal detachment most often occurs in the presence of penetrating wounds of the sclera. For the development of this complication, two conditions are required − the hole in the retina and changes in the vitreous humour, which are always present in case of penetrating wounds.

Metallosis as a complication arises in the presence of penetrating wounds with intraocular metallic foreign body. In case of prolonged stay of a metallic foreign body in the eye, it begins to rust. Secondary pathological changes occur in the eye membranes, cataract, iridocyclitis, uveitis are complicated.

Endophthalmitis is a severe complication, predetermined by penetrating wounds to the eye. Infection can get into the eye along with a foreign body or an object that injured the eye.

Damage to the inner membranes of the eye in case of penetrating wounds, even without the infection overlay, always causes inflammation of the choroid, or iridocyclitis. The severity of iridocyclitis varies in wide range. Sometimes this

inflammation has a very severe and prolonged course. The eye becomes inflamed, the patient suffers from photophobia. Exudate appears in the anterior chamber. The pupil adheres to the lens by the posterior synechiae and a fibrinous film is formed in its portion. On palpation, the patient feels soreness, the eye is soft. This form of iridocyclitis is called sympathetic. It is dangerous because this inflammation can cause a similar process in the healthy eye − sympathetic uveitis. It occurs not earlier than 2 weeks after the penetrating trauma, but its threat remains for many years after the injury. Inflammation can occur in the form of chronic fibrinous and plastic iridocyclitis or neurouveitis with periodic exacerbations. Despite the intensive treatment, sympathetic iridocyclitis often leads to blindness and subatrophy of the eyeball.

Emergency management. Patients with penetrating wounds of the eye must be referred immediately to the ophthalmological trauma centre. Antibacterial drops are instilled and a binocular dressing is applied, if the injury is in the posterior segment of the eyeball, and monocular − for injuries in the anterior segment.

Treatment. After radiography in the trauma centre, the emergency surgical treatment is conducted. Operation includes primarily the primary surgical treatment of the wound. If the iris has dropped out, it is re-inserted. In the case of dropout of the vitreous, it is cut off. If a penetrating wound of the sclera is farther than 6 mm from the limbus, cryopexy is carried around for the prevention of retinal detachment. In case of penetrating injuries with intraocular foreign body, the latter must be removed. A magnetic foreign body (steel, iron and the like) can be removed with a magnet. If the patient has traumatic cataract, its extraction may be simultaneously carried out. At pre- and postoperative stages, antibiotics are used. Antibacterial drugs are applied topically (0.25% laevomycetin solution, 0.3% tobramycin solution − tobrex, 0.3% ofloxacin solution − floxal, norfloxacin solution − vigamox, gatifloxacin solution − zymaxid and others), as well as systemically. Gentamicin solution 40 mg / ml, lincomycin 30% solution and others by 0.5 ml are administered subconjunctivally or parabulbarly every day. Anti-inflammatory therapy involves instillation of solutions of non-steroidal anti-inflammatory agents (diclofenac sodium 0.1% solution − naclof,

indomethacin 0.1% solution − indocollir and others) into the conjunctival sac 3-6 times a day, subconjunctival or parabulbar injections of glucocorticoids every day (dexamethasone 4 mg / ml solution by 0.5 ml). For prevention of posterior synechiae, mydriatics are topically applied (atropine sulphate 1% solution, tropicamide 0.5 − 1% solution). To improve the regenerative properties of the eye, the following medications are used: 20% ophthalmic gel of solcoseryl or actovegin, 5% corneregel. Treatment of penetrating wound complications is carried out in accordance with the nosological forms (hyphema, haemophthalmos, traumatic cataract, vitreous opacities, retinal detachment, iridocyclitis, endophthalmitis).

Particular attention should be paid to prevention and treatment of sympathetic uveitis. The only method to prevent this serious complication is enucleation of the injured eye. Therefore, if severe iridocyclitis develops in the blind or nearly blind eye with hypotension and significant inflammation after a penetrating wound, it is an indication for enucleation of the eye. These indications are almost absolute if the injured eye is blind. But when the sympathetic inflammation has already developed, enucleation does not help. Sometimes the affected eye, which preserved the vision, may remain the only sighted eye in the future. Therefore, enucleation after sympathetic inflammation is not conducted.

Treatment of sympathetic inflammation is the same as in uveitis. Special emphasis is placed on high doses of corticosteroids that are prescribed both topically and as a systemic treatment. If they are ineffective, cytostatics are used.

3. Burns.

Depending on the severity, burns are divided into mild, moderate, severe, and profound (I, II, III and IV degrees, respectively). In mild burns, hyperaemia and oedema of the skin of the eyelids, conjunctiva, epithelial erosion of the cornea are observed.

In case of moderately severe burns, blisters appear on the skin of the eyelids. Besides hyperaemia and oedema, there are areas of ischemia and superficial necrosis on the conjunctiva. The cornea is loose, stained with fluorescein. Not only epithelium, but also the surface layers of the corneal stromata are damaged. In severe burns, necrosis

of the skin of the eyelids, the conjunctiva and areas of the surface layers of the cornea is observed. The cornea assumes the appearance of ground glass. In case of profound burns, the complete necrosis of the eyelids, the conjunctiva, the cornea and the sclera portions may develop. The cornea is white as a porcelain plate.

Thermal, chemical and radiation burns have certain features. The most severe burns are caused by alkali (e.g., ammonia, ammonia water). This is due to their properties of acids to cause coagulation necrosis which prevents subsequent penetration of burn agents into the tissue. Alkali cause liquefactive necrosis − tissue softening. In this case, alkali continues to penetrate deeper and damage the tissue. Ultraviolet burns are called ultraviolet ray ophthalmia. First-degree burns have a favourable prognosis. Under the influence of the treatment, inflammation quickly subsides, tissues regenerate, and the functions are restored. Nebulous opacities may remain in the corneal surface. Third- and especially fourth-degree burns are characterized by severe and prolonged course. Concomitant iridocyclitis often occurs, bacterial inflammation joins, and the trophism disorder of the cornea develops. The process ends primarily with the formation of vascularized leukoma. Quite often, adhesions are formed between the sclera and conjunctiva of the eyelids. This condition is termed symblepharon. Particularly severe burns that damage the inner membranes of the eye, can not only lead to blindness, but also to the atrophy of the eyeball.

Regarding burns, it is very important to immediately assess their severity. In this case, one needs to know that thermal burns of moderate severity on the next day may become easier to manage than alkali burn, which, on the contrary, always gets worse on the next day. Therefore, patients with alkali burns, even if they look like first-degree, should always be referred to the ophthalmologist.

Particularly severe eye burns are caused by ammonia spirit. Ammonia spirit is given to patients to sniff up in order to prevent or treat fainting. There are cases when the patient unintentionally pushes the bottle with ammonia spirit aside due to its pungent smell, and the liquid enters the eye. Therefore, one must remember that patients should sniff up the cotton wool, soaked in ammonia spirit, and it must be brought to the patient’s nose (not over the eye!).

Emergency management. In case of thermal burns, antibacterial drops are instilled and antibacterial ointment is applied into the conjunctival sac. In chemical burns, it is important to immediately wash the eye. In case of alkali burns it is advisable to wash the eye with 2-3% boric acid solution and in acid burns − 2% sodium hydrogencarbonate solution. However, if there is no ready solution at hand, one should not waste time on its preparation: it is much more important to wash the eye immediately. It is convenient to use a rubber bulb for the forced washing out.

If solid substances are caught in the conjunctive (lime, cement, potassium permanganate and the like), one should carefully remove all particles with wet cotton swab. For this purpose, the lower and upper eyelids are everted, and a wet cotton swab, wound up on a glass rod, is introduced into the upper arch. This procedure is sometimes very painful. Therefore, prior to this, the eye is instilled with anaesthetics, or injected with analgesics. After washing out and removal of burn agents, antibacterial drops are instilled and antibacterial ointment is applied.

Treatment. In mild burns, antibacterial drops or ointments to prevent secondary bacterial inflammation are prescribed. For regeneration of the corneal epithelium, keratoprotectors are applied.

Patients with burns of moderate and severe degrees are referred to the ophthalmologist. In this case, besides antibacterial drugs for improving trophism, vitamin drops, solcoseryl ointment are administered; to improve epithelialisation − thiotriazoline eyedrops; with disintoxication aim − long-term instillations with 3% glucose solution, vitamins, antiseptic agents are applied. At certain stages of burn disease, corticosteroids are used. Among physiotherapeutic procedures phonophoresis with antimicrobials is used. In some cases, severe chemical burns require urgent corneal transplant surgery. If the burn is particularly severe and led to necrosis of all layers of the cornea, in order to preserve the eye, blepharorrhaphy is conducted (suturing of the eyelids). Later − in 6-12 months, if the inner membranes retain their functions, a series of plastic surgeries is conducted, forming the conjunctival cavity, keratoplasty or keratoprosthetics. A significant contribution to the development of

these operations was made by scientists of the Filatov Institute of Eye Diseases in Odessa.

4. The features of examination of patients with eye injuries.

Diagnosis of eye injuries is very often combined with injuries to other parts of the body. Therefore, in case of eye injuries, it is necessary to pay attention to general state of the patient and conduct examination of all organs and systems. In addition, in adults, it is necessary to take blood test for alcohol intoxication. It is required to find out all details of the injury (at home or at work, at what time and under which circumstances). It is important to clarify, whether the patient was vaccinated against tetanus. Acuity of vision in checked in both injured and healthy eyes. This may be important in the future to establish the degree of efficiency loss. Examination of the eye, in case of its damage should be done very delicately, so as not to cause additional trauma. To determine the nature of the injury (contusion, penetrating or non-penetrating wound, probability of intraocular foreign body and the like), anamnesis is of great importance. The presence of intraocular foreign body is suspected in case of traumas associated with metal strike against metal (e.g., hammer or chisel). Under such circumstances, a minute metal fragment can chip off, and it has the sufficient kinetic energy to break the eye membrane.

As mentioned above, particularly important is the diagnosis of penetrating wounds of the eye. For their detection, there are direct and indirect signs. Direct signs of penetrating traumas are the wound proper, especially the inner membranes of the eye dropped out through it, damage to the iris or the lens capsule along the wound tract, intraocular foreign body. Indirect signs are hypotension, the change in the anterior chamber depth, traumatic cataract. It is very important to conduct X-ray examinations.

In all cases, with the least suspicion of intraocular foreign body, spot-film radiography of the orbit is carried out. In order to display a small or low-contrast foreign body on the film more clearly, radiography is performed in the facedown position − closer to the filmpack. The patient's head should be in a position in which the tip of the nose and chin touch the table − in such a way, the projection of the

eyeball is not superimposed with the shadows of the frontal, maxillary and basilar bones.

However, X-ray films often contain minor shades (artifacts) which can simulate foreign bodies. Therefore, in order to exclude possible errors, the so-called polygram is carried out: two shots are made on one film. During the first shot, the patient looks down, while during the second − up. If the patient has an intraocular foreign body, the radiograph will contain two symmetrical shades − at the top and at the bottom.

For successful operation on removal of intraocular foreign body, it is necessary to precisely determine its location. For this purpose, Comberg-Baltin's prosthesis is used. The prosthesis is made of soft metal and has four lead mark points. The prosthesis is applied to the eye so that the mark point is accurately located on the meridians of 12, 3, 6 and 9 hours. Radiography is carried out in the anteroposterior and lateral projections. Focusing on the shadows of lead mark points, by means of special schemes, one can precisely establish, in which meridian and at what distance from the limbus, a foreign body is located. For the diagnosis of intraocular foreign bodies, ultrasound examination is also often used.

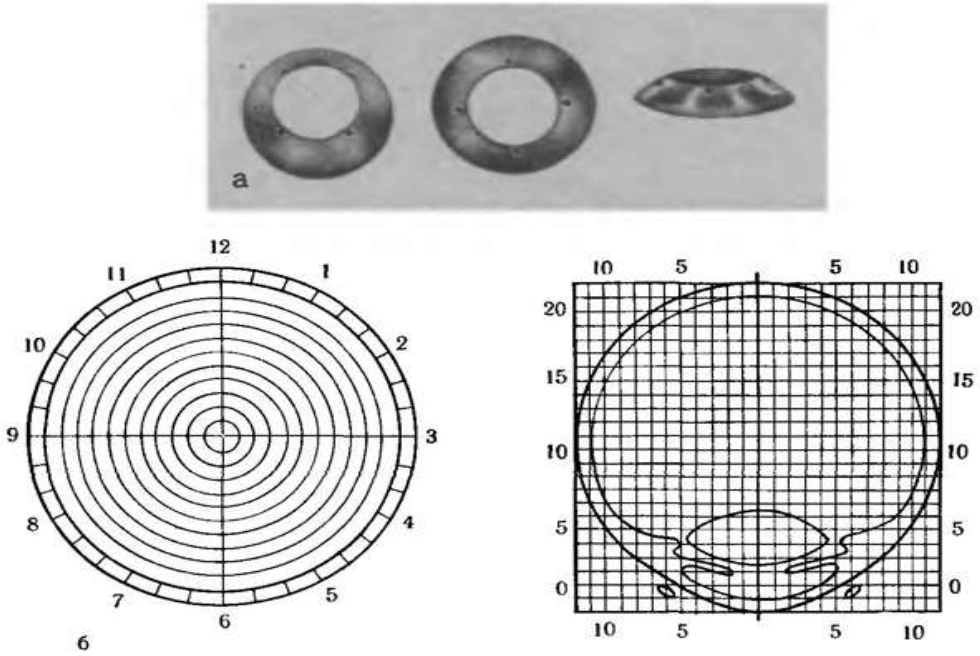


Fig. 15.1. Diagnosics of intraocular foreign bodies.

a – Baltin's prosthesis-indicator

b – schemes for determining the localization of foreign bodies

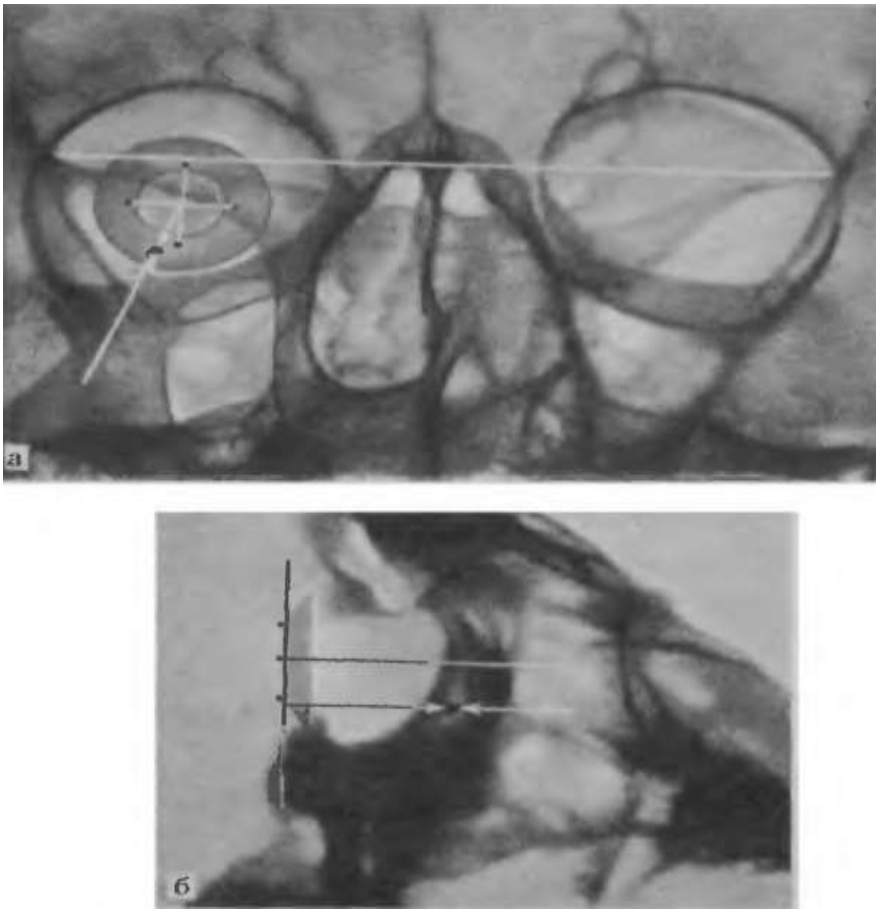


Fig. 15.2. X-ray image of the orbit in two projections.

5. Prevention of eye injuries.

Based upon all the above-stated, it is obvious that eye injuries are always very serious, and often lead to blindness. In addition, the traumas of the visual body usually occur in the most functional age, which is of great social importance. Therefore, prevention of eye injuries is of principal importance. The prevention of eye injuries includes primarily the compliance with industrial safety (safety glasses, shields, etc.). The lighting at the workplace must be adequate. One cannot perform work activities, not covered by the production procedure. Tired, sick and intoxicated workers must not be allowed to perform work activities.

At schools and kindergartens, prevention of eye injuries is based on children's discipline. Children must be explained, how dangerous can eye injuries be in case of careless handling of items such as scissors or a needle, and explosives. At

kindergartens, there must not be toys that can cause injury. During manual training classes, in the same manner as at the enterprise, all safety precautions must be observed.

Theoretical questions to the chapter:

1. Classification of contusions.
2. Clinical characteristics of contusions, treatment.
3. The classification of burns by severity.
4. First aid for burns of different origin.
5. Possibilities of conservative treatment and surgical treatment for burns and their consequences.
6. Classification and symptoms of penetrating wounds.
7. Clinical symptoms of the damage to the anterior segment of the eye.
8. Clinical symptoms of the damage to the posterior segment of the eye.
9. The volume of studies for patients with penetrating wounds. Methods of localization of the intraocular foreign bodies.
10. Principles of surgical treatment in wounds of the eyeball and appendages of the eye.
11. Complications of penetrating wounds.
12. Sympathophthalmia: clinical presentation, treatment, preventive measures.

Practical skills which students should master:

1. Identifying the nature of disorders that have arisen as a result of the visual organ damage.
2. Removing foreign bodies of the conjunctiva and emergency actions in the presence of corneal foreign body.
3. Emergency care in chemical and thermal burns of the eyes.
4. Emergency care in penetrating wounds.

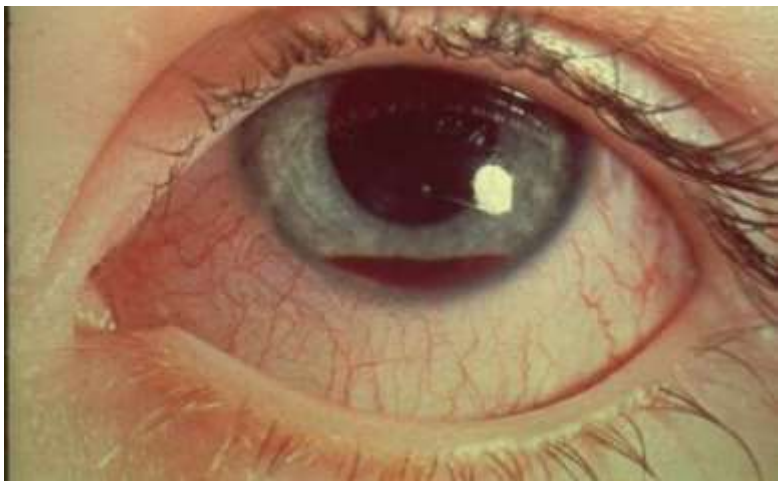


Fig. 15.1. Hyphema.

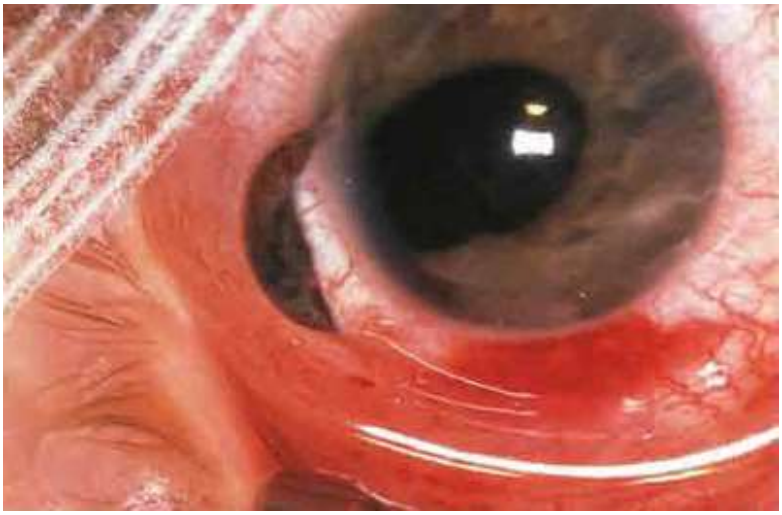


Fig. 15.2. Penetrating scleral injury.

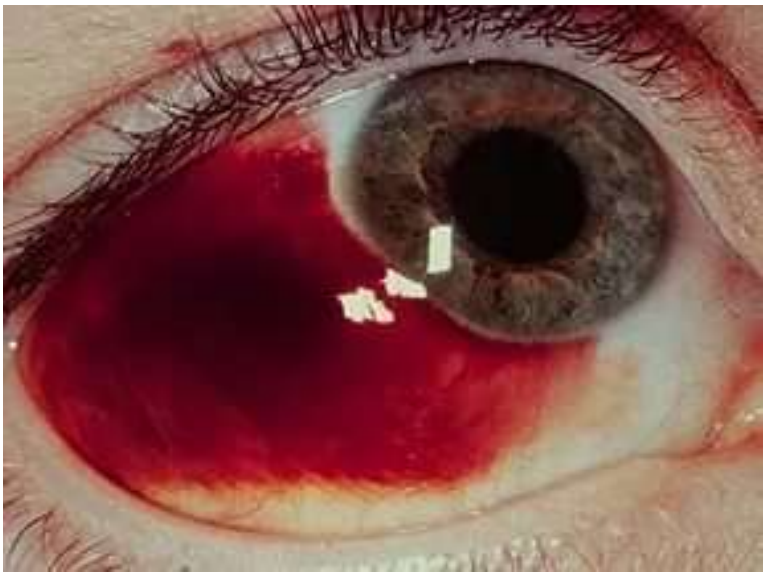


Fig. 15.3. Subconjunctival haemorrhage.

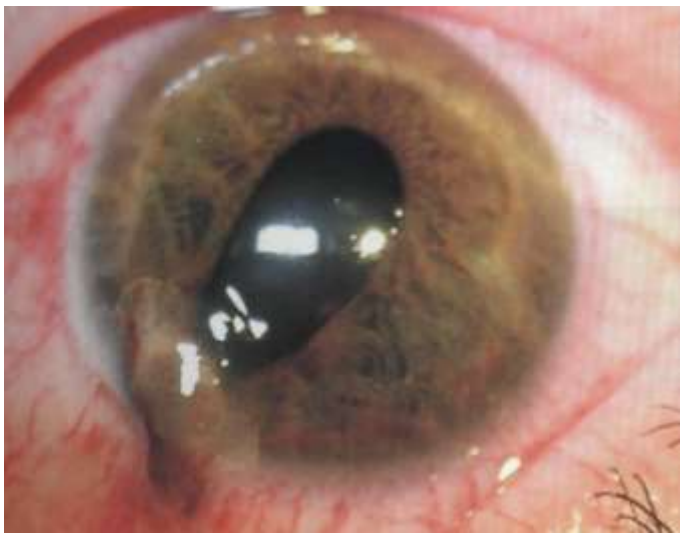


Fig.15.4. Penetrating corneal injury.

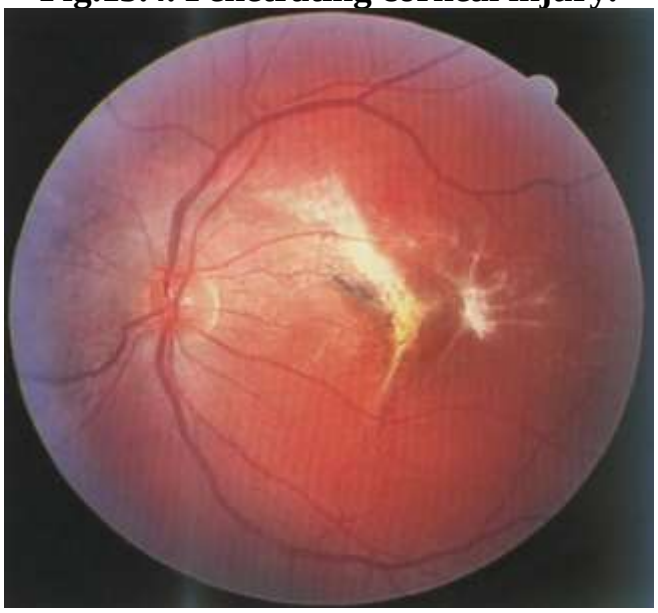


Fig. 15.5. Choroid rupture.

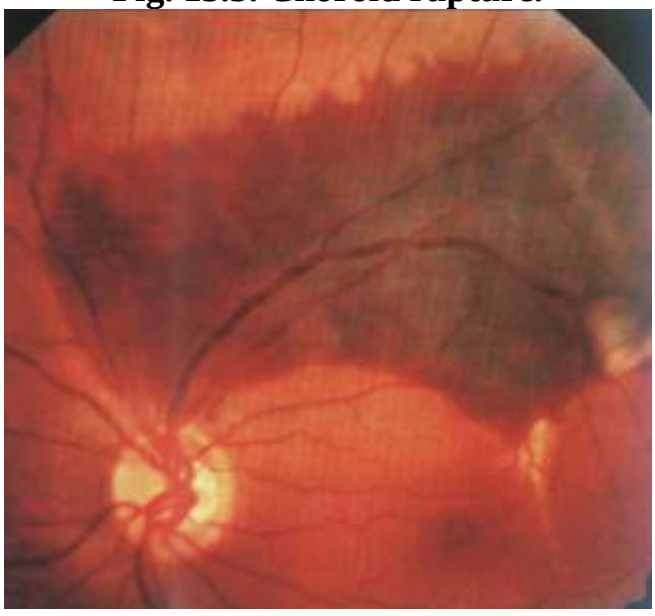


Fig. 15.6. Blunt trauma. Retinal haemorrhage.

CHAPTER 16

DISEASES OF THE OCULOMOTOR APPARATUS

The pathology of oculomotor apparatus is observed quite often − in 1.5-2.5% of children. Within the structure of eye pathologies, this disorder accounts for 7% of cases. The oculomotor apparatus develops until the age of 14. Disorders of the oculomotor apparatus give rise to strabismus, which is accompanied by disrupted binocular vision, development of cosmetic defects and limits the choice of profession.

Binocular sight implies the vision with both eyes. The formation and establishment of binocular vision occurs between the age of 2 months and 6-10 years, it is reinforced up to the age of 15. At birth, the infant has no conscious vision. By the beginning of the 2nd month, the development of joint movements of both eyes occurs, and the conditioned reflex connections between the stimulation of the retina and the eyes movement are reinforced. Already at 4-5 months, the prolonged fixation of object is observed. Fusion (merging) is formed from the second half year of life.

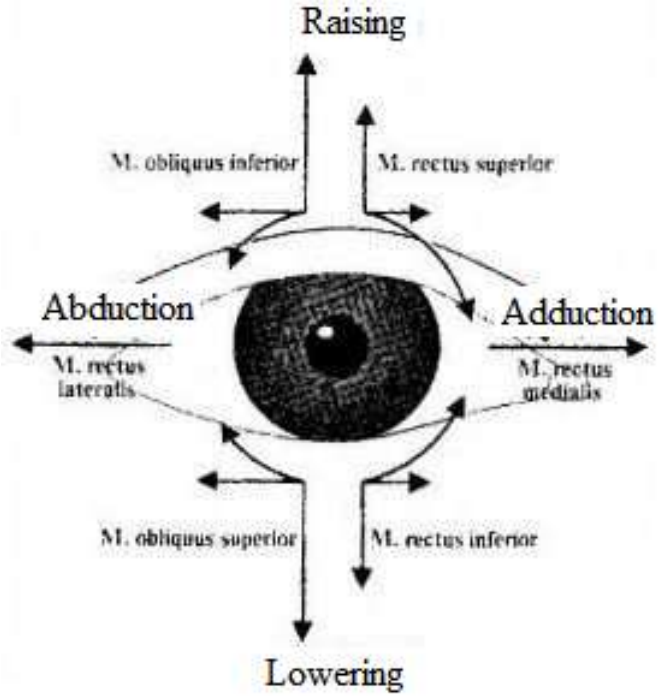


Fig. 16.1. The mechanism of the external muscles of the eye.

The mechanism of binocular vision. Assuming that a person looks at point A (Fig. 16.2), the image of this point is projected on the central fossa F1 and F2 − along the visual pathways the images are transferred to the cortical visual centre, where the

merging (fusion) of images occurs. This happens provided that the images are the same and fall on the same portions of the retina. These points are B1 and B2, which are located to the left from the central macular fossa on the same meridian, and at the same distance. The images that have come to the same (disparate) areas do not merge and are perceived as double (diplopia). If in closer looking at point A, point B gets on it, then the image goes to the right of the central fossa of the right eye and to the left − of the left eye. As a result, the merging of the images does not occur and the object doubles.

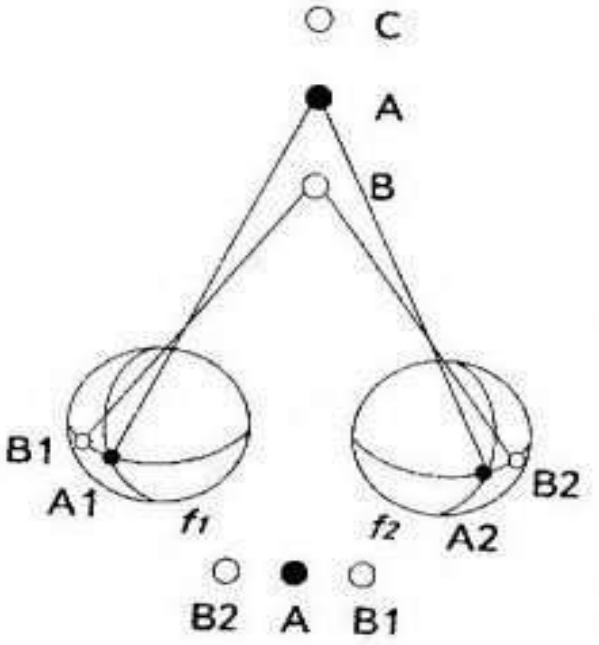


Fig. 16.2. Double vision of the object which is closer than the fixation object.

Necessary conditions for the formation of binocular vision

Visual acuity should be at least 0.4. The function of all 12 oculomotor muscles must be well-coordinated. A clear image of the viewed objects on the retina and equal size of these images in both eyes − iseikonia − are required. Anisometropia of more than 2 dioptres causes the development of aniseikonia. A good functional ability of the retina, pathways and higher visual centres are necessary. A clear association between accommodation and convergence, as well as their parallel innervations are essential.

In **monocular vision**, only one eye takes part in the binocular mechanism, the signals from the central part of the retina of the second eye are suppressed.

Simultaneous vision − both eyes are functioning jointly and equally, but there is no interaction between them.

Examination methods.

1. History taking.
2. Determining the visual acuity and correction for each eye.
3. Determining the nature of strabismus (concomitant, monocular, alternating, paralytic).
4. Determining the type and angle of strabismus.
5. Determining the nature of vision.
6. Examination of refraction by the objective method.
7. Examination of the visual fixation.

Examination of the binocular vision by Kalf's method (the two-pencils test): examination is carried out by using two identical pencils. The doctor holds a pencil vertically, and the patient, without looking with two eyes, must deliver the tip of the pencil to the doctor's pencil so that the two pencils form a straight line. In the presence of binocular vision, it is not difficult to do this, whereas in the absence of binocular vision, the patient does not succeed and misses the goal, as is easily seen, if one conducts the test with one eye closed. The patient with no binocular vision misses with both eyes open.

Examination of the binocular vision Belostotskiy-Friedman light test

The test is based on the principle of separation of the visual fields of both eyes with filters of red and green colours, which allows you to simultaneously display the physiologically equivalent tests to the right and left eye separately.

To conduct the study, the light test is used, which has a hollow box, with a light bulb situated therein. On the front wall of the case, there are 4 circles − two red, one green and one white. The patient looks at the device from a distance of 5 m. Without correcting the sight with glasses, the patient puts on red and green glasses: there is a red glass in front of the right eye and a green glass − in front of the left eye. Before starting the examination, the quality of filters is checked: by turns closing the left and right eye with a flap, the patient sees at first two red, then three green circles. The

study is conducted with both eyes open. In the presence of binocular vision, the patient sees four circles, and a circle of white takes on the colour of glass that faces the leading eye. In the monocular vision, one can see two or three green or red circles. In the simultaneous vision − five circles, arranged as homonymous or heteronymous doubling (Fig. 16.3.)

Examination of the binocular vision − Sokolov's test. This phenomenon can be explained by the fact that each eye has its own image and they are superimposed on each other. Hence, if one keeps a tube in front of one eye, and the hand − in front of another one, the image that we see through the tube opening is superimposed on the image of the palm in the other eye. In simultaneous vision, the "hole" does not coincide with the centre of the palm, and in the monocular − the phenomenon with a "hole" in the palm of the hand does not appear at all. The patient holds a tube with one hand (for example, a folded sheet of paper) in front of the right eye. At the end of the tube from side of the left eye, one puts the palm of the hand. In case of binocular vision, an impression of a "hole" in the palm of the hand appears, through which the image is perceived which the patient sees through the tube.

False strabismus. Normally, the optical axis of the eye that passes through the centre of the cornea does not coincide with the visual axis that connects the central fovea of the retina with the viewed object. Between them, the so-called Y angle is formed − positive or negative. With a large angle, there is an impression of the presence of strabismus. Generally, false divergent strabismus is associated with the positive Y angle. A perfect muscular balance of both eyes called *orthophoria*. However, much more often *heterophoria* is observed, which implies the unequal power of action of the eye muscles, caused by anatomical and neural factors. In heterophoria, visual performance, especially at close range, requires more than normal neuro-muscular tension in order to overcome the tendency to deviate in one of the eyes. Diagnosis of heterophoria is based on the exclusion of conditions for binocular vision. If the patient closes one eye with the palm of the hand, then the eye will deviate in one direction or another, depending on the type of heterophoria, and after the doctor removes the hand, an eye will make the setting motion in the direction opposite to

that in which it would be rejected. In order to distinguish heterophoria from the concomitant strabismus with little or periodic deviation, it is necessary to examine the binocular vision: in the first case it is present, in the second − it is absent.

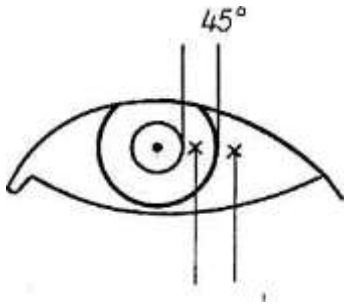
Concomitant strabismus.

As a rule, this is a pathology of early childhood, and a genetically determined disease. The cause of concomitant strabismus is the disrupted bifixation mechanism (the capacity of oculomotor system to simultaneously direct the fixations to the object and hold on it the visual axes of both eyes). The eye that sees worse does not fix the object and is deflected in one direction or another. One can distinguish accommodative and non-accommodative strabismus; monolateral and alternating. There is also the **convergent** form of concomitant strabismus, in which the visual axis of one eye moves away from the fixing point towards the nose, and **divergent**, when the visual axis extends to the temple.

Accommodative concomitant strabismus is associated with disruption of the normal relation between accommodation and convergence. The ratio is considered normal if in fixing the subject at a distance of 1 m, the degree of accommodation tension is equal to 1 dptr, and at a distance of 0.33 m − 3 dptr. (for emmetropes). Hypermetropia usually prevails in children. In this case, each distance requires a stronger accommodation. Therefore, in hypermetropia a greater impetus toward convergence predominates. In myopia, conversely: the need for accommodation decreases and the convergence impetus weakens. Therefore, in non-corrected hypermetropia, convergent strabismus develops, and in myopia − the divergent form. Accommodative strabismus can be corrected by the correction of ametropia.

Non-accommodative strabismus develops as a result of disrupted divergence. This occurs in the clouding of optical media, optic nerve disease, systemic diseases. Prescribing correction does not improve the condition. The angle of deviation completely disappears in accommodative strabismus; in the non-accommodative form it does not change; in partially accommodative − decreases. The deviation angle of the squinting eye is called primary, and the deviation angle of the healthy eye − secondary. To determine the deviation angle, Hirschberg's method is used: a beam of

light is directed into the patient's eye, and the location of light reflexes on the cornea is compared. In the fixing eye, the reflex is observed near the centre of the pupil or coincides with it, whereas in the squinting eye, it is determined at a position corresponding to a deviation of the visual line. One millimeter of its displacement on the cornea corresponds to the squint angle of 7°. The more the angle is, the farther from the centre of the cornea the light reflex is shifted. Hence, if the reflex is located along the edge of the pupil, the squint angle is 10°, and if on the limbus − 45°. Wide pupil complicates accurate determination of the distance between the light reflex and the centre of the cornea, while the eye's simultaneous deflection both horizontally and vertically complicates the application of Hirschberg's method. Providing the substantial γ angle, the squint angle is calculated with the required correction which is equal to γ angle.



Determining the strabismus angle by Hirschberg method

Persistent reduction in visual acuity of a constantly squinting eye is called **amblyopia**. By the reduction degree, amblyopia is divided into: low (visual acuity of 0.8-0.4), average (0.3-0.2), high (0.1-0.05), and very high (0.04 and below). By the fixation condition, one can distinguish amblyopia with correct and incorrect fixation. In the correct fixation, the viewed object is projected to the centre of the macula, in the incorrect − the image of the object is located outside the macula.

Treatment of concomitant strabismus. The ultimate goal of treatment of concomitant strabismus is restoration of the binocular vision.

1. **Optical (glasses, contact lenses) correction** of anisometropia or ametropia to ensure the highest level of visual acuity. Glasses are prescribed for continuous use. The control of functions is conducted once in 2-3 months.

2. **Pleoptic treatment**. It is a system of measures aimed at improving the treatment of amblyopia and enhancing the visual acuity. Such measures include the method of turning off one of the eyes (usually the one which sees better) from the act of vision with extra (only in farsightedness and in correct fixation!) visual load and light or laser stimulation, special computer programs, video-computer systems with biofeedback (Ambliocor).

3. **Orthoptics** is a system of measures aimed at restoring and strengthening the binocular vision. Orthoptics usually involves the use of synoptophore, Forbis apparatus, convergence trainer, the EYE software, contour prisms (diploptics). Orthoptic exercises are prescribed after the age of 2-3 years.

4. **Surgical treatment**. The purpose of this treatment is to obtain a symmetrical position of the eyes by means of the muscular balance. To enhance the action on a muscle, its resection is applied, to weaken − recession is used. The age of 3-5 years is optimal for this operation.

5. **Diploptics**. The method consists in restoring the functions in vivo. It is conducted primarily after surgical treatment. Diploptics exercises restore the eye's ability to overcome the physiological double vision and develop the mechanism of bifixation. The entire treatment takes about 2 years of hard, purposeful and systematic work of ophthalmologists together with parents and educators. Treatment of a young child must resemble an entertaining and diversified game.

Theoretical questions to the chapter:

1. The main conditions of binocular vision. The significance of binocular vision in choosing a profession.
2. Concomitant strabismus, its clinical presentation, diagnosis.
3. Latent strabismus, its clinical presentation, diagnosis.
4. False strabismus, its clinical presentation, diagnosis.
5. The principles of treatment of concomitant strabismus.
6. Paralytic strabismus, its symptoms, differential diagnosis, treatment.

Practical skills which students should master:

1. Determining the extent of the eyeball movement.

2. Determining the angle of strabismus by Hirschberg.

3. Determining the nature of fixation.

Fig. 16.3. The four-dot light test

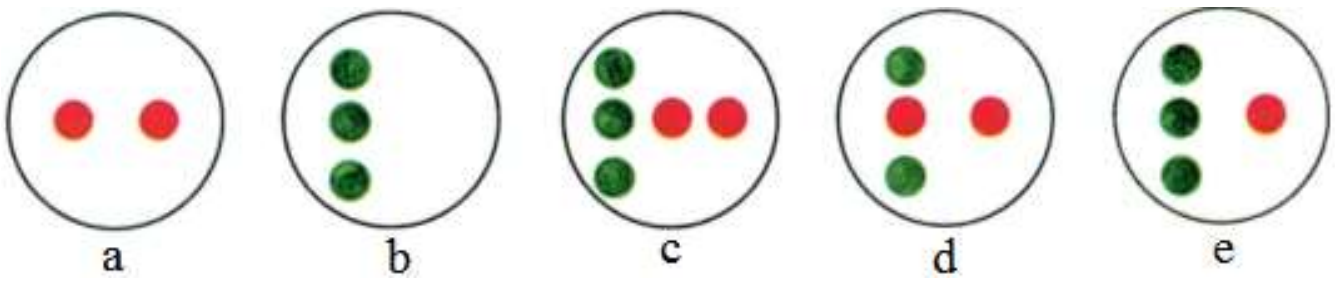


Fig. 16.4. Determining the nature of vision by the four-dot light test

a) right-sided monocular vision

b) left-sided monocular vision

c) simultaneous vision

d) binocular vision, dominant right eye

e) binocular vision, dominant left eye

CHAPTER 17

CHANGES TO THE ORGAN OF VISION IN SYSTEMIC DISEASES

The organ of vision is associated with many anatomical and physiological mechanisms of the body in general, and individual organs and systems in particular. Therefore, it is often involved in the systemic diseases of the body, or is the first to respond to them by functional or morphological changes. In fact, all pathologies of the organ of vision (except for injuries, individual exogenous inflammations of the eyelids, conjunctiva and cornea) are the manifestations of systemic diseases. Therefore, knowledge of ophthalmic symptoms in various systemic diseases is essential for doctors of all specialties for diagnosis, assessing the stage and dynamics, determining the prognosis of the underlying disease.

Changes to the organ of vision in general pathology are manifested as both functional and organic disorders of the central and peripheral vision, disturbances in the skin and muscular system − as paresis and paralysis of the oculomotor muscles and the accessory apparatus. With an increase of intracranial pressure, the development of the optic nerve oedema is often possible.

The list of key terms, parameters and characteristics which students should master while studying the topic:

Term	Definition
Tower-shaped skull	Congestive discs, simple or post-neuralgic atrophy of the optic nerve; nystagmus and strabismus are possible.
Basal skull fracture	“Raccoon eyes” symptom − the spread of blood under the skin of the eyelids and the conjunctiva of both eyes after a while after the cranial trauma. Sometimes there is a symptom of the upper orbital fissure. Congestive disks and haemorrhages of different character may be observed in the fundus.
Diabetic retinopathy	Characteristic changes of the retina in diabetes mellitus, which are noted for the appearance of microaneurysms,

	haemorrhages, hard exudates, proliferation foci.
Guist's symptom	Corkscrew-like curl of minor venous vessels around the macular area.
Salus' symptom	The symptom of crossing of an artery with a vein, in which the vein is pressed into by the artery.
Basal meningitis	The lesion of the oculomotor and trochlear nerves is typical.
Silver and copper wire symptom	Is due expressed sclerosis, the walls of retinal vessels become white or yellow.
Graefe's sign	Delay of the upper eyelid from the eyeball downward movement.
Cerebral syphilis	One-sided internal ophthalmoplegia (mydriasis and accommodation paralysis), congestive discs and optic neuritis, equilateral hemianopsia, and oculomotor nerve paralysis.
Brain tumour	Congestive discs, hemianopsia, oculomotor muscles paralysis
Sympathetic nerve paralysis	Ptosis, miosis and enophthalmos (Horner's symptom), sometimes hypotension of the eye, iris discolouration, skin redness, lacrimation, dilated retinal vessels on the affected side.
Thyrotoxic exophthalmos	The main symptom of Graves' disease. Dalrymple's sign, Stellwag's sign, Möbius sign, Jellinek's sign
Chronic myelogenous leukaemia	Myelomas are scattered in the retina at the periphery, yellowish-white rounded formations of the optic nerve.
Behçet's syndrome	It is characterized by septic purulent iridocyclitis, lesions of the skin and mucous membranes of the genitals and aphthous stomatitis.

The appearance of the so-called "raccoon eyes" symptom may indicate a basal skull fracture. The syndrome of upper orbital fissure sometimes develops in these traumas.

Thrombosis of the sinuses, especially the cavernous one, is characterized by exophthalmos and complete phthalmoplegia ophthalmoplegia. Paresis and paralysis of the oculomotor muscles develop in encephalitis, which leads to diplopia and deterioration of the pupillary reaction.

Pathological retinal conditions are observed in cardiovascular diseases and endocrine disorders.

Examination methods.

1. History taking.
2. External examination (the side lighting method).
3. Testing the visual acuity and visual fields.
4. Biomicroscopy.
5. Ophthalmoscopy.
6. Fluorescent angiography.
7. Optical coherence tomography.
8. Ultrasonic scanning.

RETINAL DISEASE IN CARDIOVASCULAR PATHOLOGY

Fundus changes in hypertension

The classification of hypertensive changes to the fundus by M.L. Krasnov is used: 1) hypertensive angiopathy; 2) hypertensive angiosclerosis; 3) hypertensive retino- and neuroretinopathy.

Hypertensive angiopathy is characterized by dilated and tortuous retinal vessels. Visual function is not impaired. The normal ratio of the arteries and veins of the retina (2:3) is increased (up to 1:4). There is a characteristic symptom of arterio-venous decussation (Salus-Gunn symptom), which manifests itself in the vein constriction by arteries. In the central parts of the retina, the corkscrew curls of minor veins appear (Guist's symptom).

Hypertensive angiopathy most often corresponds to the phase of unstable increased blood pressure and the initial stages of hypertension, including stage III.

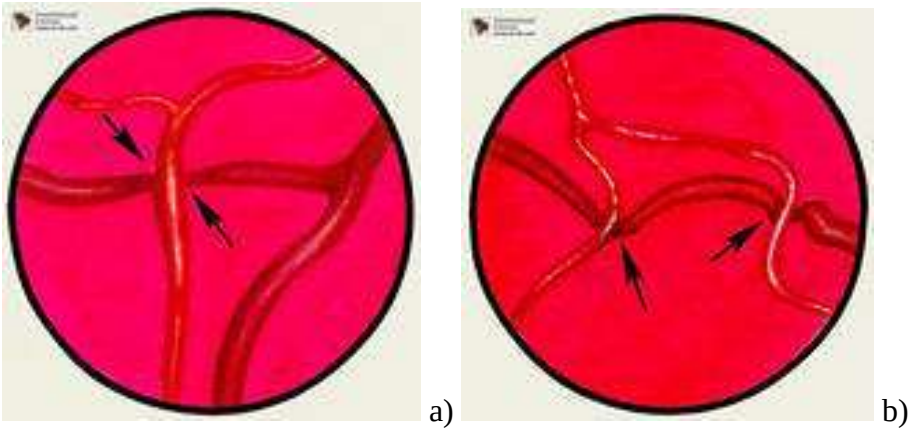
Hypertensive angiosclerosis is characterized by thickening of the artery walls and the appearance of uneven light reflex along them − the copper and silver wire symptoms. This occurs due to deposits of lipids in the walls of arteries and obliteration of individual arterial tubes.

Hypertonic angiosclerosis of the retina corresponds to the phase sustained increase in the systolic and diastolic blood pressure and is usually observed at IIA and IIB stages.

Hypertensive retinopathy is characterized by adjunction of the retinal tissue lesion to the above described changes. Plasmorrhages and haemorrhages, hard, whitish and yellowish exudates are observed. Visual function is impaired. Retinopathy of various severity is observed at IIIA − IIIB stage of hypertension.

Hypertensive neuroretinopathy usually develops at the late stage of hypertension. It is characterized not only by changes in the blood vessels and retinal tissue, but also by the involvement of the optic nerve. The disk of the optic nerve becomes swollen, increases in size. The oedema extends to the retina. Haemorrhages appear around the disk. Visual functions, colour perception are significantly impaired. Neuroretinopathy may be concluded by atrophy of the optic nerve. The differential diagnosis should be carried out with congestive optic disc.

In hypertensive disease, the complications to the organ of vision are: the central retinal vein thrombosis, obstruction of the central artery of the retina, haemophthalmos, front ischemic neuropathy, secondary neovascular glaucoma.



a) b)

Fig. 17.1. Changes to the eye fundus in a hypertensive patient.

a) − angiopathy; b) − angiosclerosis

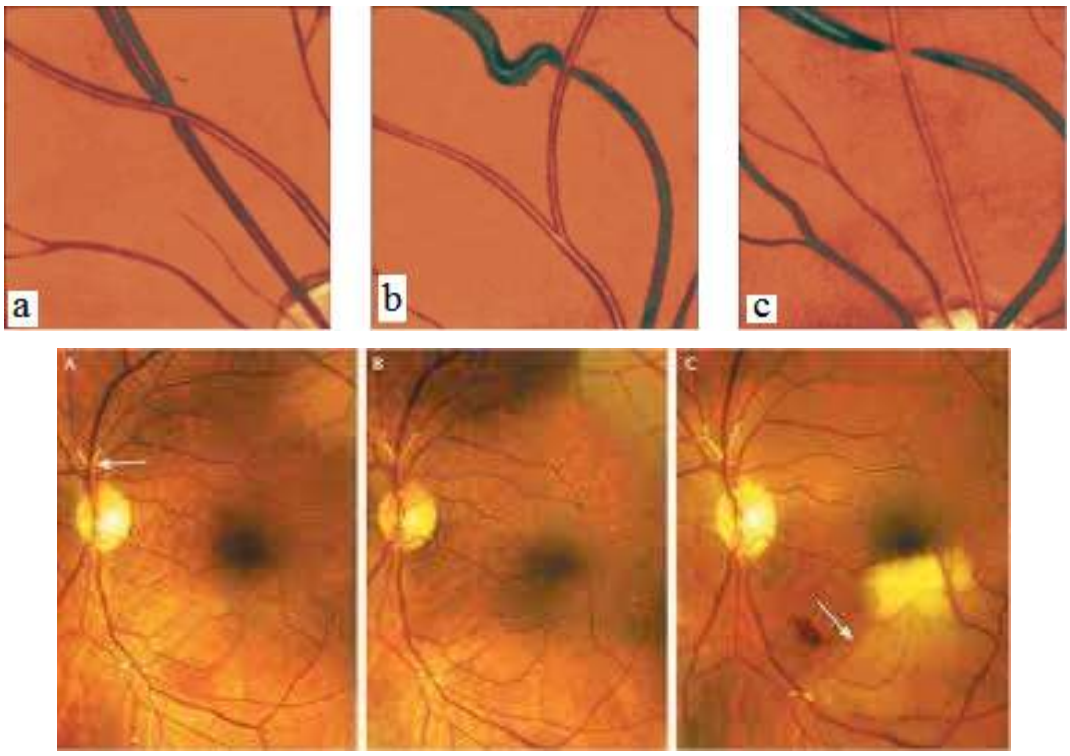


Fig. 17.2. Salus-Gunn symptom:
a) Salus 1; b) Salus 2; c) Salus 3

Renal hypertension is characterized by the formation of the "macular star". This is due to deposits of hard exudates of white (protein infiltration) and yellow colour (lipid infiltration) in the retina.

Treatment is carried out on an out- and inpatient basis together with the therapeutist. Hypertension balance is a prerequisite for treatment and prevention of complications.

CHANGES TO THE ORGAN OF VISION IN DIABETES MELLITUS

Diabetes mellitus

Diabetes has become an epidemic of the XXI century. Between 2007 and 2015, according to the world statistics, the number of people with diabetes has increased by 2.7 times, i.e., from 246 to 380 million people.

The latest researches on the diagnosis and treatment of diabetic retinopathy (DR) were presented at the 9th Euretina Congress in Nice. Professor of the University of Coimbra Jose Cunha Vaz defined two pathogenetic mechanisms of diabetic retinopathy. The first one is the increased permeability of the capillaries,

accompanied by microaneurysms and haemorrhages, which lead to macular oedema. The second one is the occlusion of capillaries of primarily thrombotic nature that leads to tissue ischemia and development of proliferative processes in the retina.

Improved understanding of the cellular and molecular processes in the pathophysiology of diabetic retinopathy will help determining the way of effective prevention of diabetic oedema and proliferative diabetic retinopathy.

In Europe, ophthalmological complications develop in 33.2% of diabetic patients. Indicators of these complications in Ukrainian patients are reliably higher, because patients seek medical care later; the price of treatment of diabetes complications is high enough for people who permanently spend money on hypoglycaemic agents or insulin; as well as due to the mindset of Ukrainian patients which is not directed at the prevention of a disease, but at the treatment of its terminal stage.

In all systemic diseases, it is easier to prevent the complications than to cure them. In diabetes, the complications are manifested by diabetic retinopathy, nephropathy, neuropathy and peripheral circulatory disorders − formation of ulcers on legs. One should not forget about diabetic cardiomyopathy and coronaropathy. According to many experts, the most important risk factors for DR is the level of glycemia, arterial pressure and blood lipids.

European Association for the Study of Diabetes (EASD) developed criteria to minimize the risk of diabetic complications. Thus, glycosylated hemoglobin should be at 6.5%, blood pressure not higher than 130/80 mmHg, blood cholesterol up to 1.8 mmol / l.

In real situation, only 7% of patients achieve the above recommended indicators, while 20% of patients with newly diagnosed diabetes already have evidence of non-proliferative diabetic retinopathy. If the course of diabetes is more than 20 years, the vast majority of patients have various ophthalmic complications.

Diabetic retinopathy (Fig. 17.3.) is a serious complication of diabetes, characterized by microangiopathy of the eye vessels with damage of pre-capillary arterioles, capillaries and post-capillary venules. A feature of such microangiopathy is increased

extravasation through the vascular wall and simultaneous occlusion of the capillaries, thickening of arteriolar walls and formation of arterio-venous bypasses.

Classification of DR by Kohner and Porta (1991):

- non-proliferative DR
- preproliferative DR
- proliferative DR

In the course of diabetes, the ETDRS research team distinguishes a condition when pathological changes appear on the wall. In ETDRS classification, manifestations of diabetic retinopathy are divided into non-proliferative and proliferative forms of the disease.

Diabetic maculopathy

Diabetic maculopathy is the damage of retinal centre, characterized by exudative, hemorrhagic and (or) ischemic retinal changes at a distance of 500 − 1500 μm from the fovea. Among the forms of diabetic maculopathy, one can distinguish exudative, exudative-ischemic and ischemic forms.

Exudative maculopathy is a swelling of the central portion of the retina in diabetes mellitus. It is characterized by the appearance of hard exudates and retinal thickening in the macula. The clinically significant macular oedema is a form of maculopathy that is subject to laser treatment and consists of the following parameters:

• Retinal oedema within 500 μm from the fovea.

• Hard exudates within 500 μm from the fovea, accompanied by retinal thickening.

• Retinal oedema with the area of 1500 μm no more than 1 DD (diameter of the disk) from the fovea centre.

Non-proliferative diabetic retinopathy

This form is characterized by the appearance of microaneurysms, haemorrhages, hard exudates, ischemic (cotton-like) areas on the retina.

Microaneurysms are the protrusions of the capillary walls, connecting the deep and superficial capillary plexus.

Microaneurysms are the first pathognomonic signs of DR. Hard exudates are the deposits of plasma and lipids in the outer plexiform layer due to extravasation

through the capillary walls. Retinal haemorrhages can be punctate and linear. The form depends on the location in the retinal layers. Large preretinal and intravitreal haemorrhages are caused by bleeding from large retinal vessels. Ischemic areas on the retina can appear as white cotton-like foci, which are caused by the infarction of nerve fibres.

Preproliferative diabetic retinopathy

This form reflects the growth of retinal ischemia, distribution of the areas of capillary occlusion and formation of intraretinal microvascular abnormalities (IRMA). The term IRMA refers to the vessels coming from arterioles to venules, bypassing the capillaries and the capillary bed. IRMA usually form in the areas of nonperfused capillaries; they resemble loops, segmented vessels − "bundle of sausages". In preproliferative DR, hard exudates, haemorrhages (punctate and linear) and cotton-like foci (areas of nerve fibres infarction) are also observed.

Proliferative diabetic retinopathy

This form of retinopathy is characterized by the appearance of fibro-glial-vascular proliferation in the optic nerve disc and paravasally on the retina. Abroad, this DR form occurs in less than 10% of patients with diabetes mellitus. In our country, this figure rises to 15-20%. In patients with type II diabetes, the incidence of proliferative retinopathy is 57-60% among patients above the age of 30. Factors that can trigger the development of proliferation in diabetes include high complicated myopia, posterior detachment of vitreous body and occlusion of the carotid artery. The development of newly formed vessels and fibrous glial proliferation is caused by a powerful release of vasoproliferative substances in the vitreous body. For triggering the formation of new vessels on the retina, at least a quarter of the area of the retina is required. When perfusion is not sufficient, the vasoproliferative factors are produced. The optic disc is the most common localization of new vessels. One reason for this detachment is the absence of internal marginal membrane on the surface of the nerve. The start of proliferation occurs in the venules, new vessels penetrate the defects in the internal marginal membrane and invade the posterior hyaloid membrane on a permanent basis.

Treatment should be carried out together with the endocrinologist. First and foremost, treatment is aimed at balancing hyperglycemia. For prevention of diabetic complications of the organ of vision, retinal laser coagulation is conducted. In diabetic macular oedema, corticosteroids and inhibitors of vasoproliferative factor are applied. In vitreoretinal proliferation, surgical treatment is needed.

Rheumatic retinitis

It involves the damage of retinal vessels (often including the choroid) in patients with rheumatism. It arises as a result of vascular wall permeability by protein substances with subsequent development of fibrinous changes in the vascular wall and the surrounding areas. Typically, small arteries are affected in the form of microangiopathy. Dilated and curled retinal arteries with uneven caliber are observed, with the appearance of tiny yellowish (punctate) foci therein. Pigment dispersion is observed in these areas. Sometimes along the arteries, there are the strips of plasmorrhages in the form of perivascular gray "sleeves". Less commonly, lesions affect the retinal veins. A small amount of tiny chorioretinal foci may appear in the front or in the periphery of the posterior pole of the eye fundus, indicating the lesion of the area of medium vessels and the choriocapillary layer.

Gestosis

In this case, ophthalmoscopic changes in the fundus are of particular importance. They are similar to the same changes in the conditions of renal retinopathy. In marked swelling in one or both eyes, transudative retinal detachment, thrombosis of its vein or branches of this vein can develop. Sometimes the changes in the fundus are the only indications for abortion. Absolute indications are as follows: a) retinal detachment due to toxicosis; b) retinopathy and neuroretinopathy; c) thrombosis of the central retinal vein.

Changes to the organ of vision in the pathology of hematopoietic system.

In **chronic myeloid leukaemia**, a yellowish-white rounded formation of 1/5-1/3 of the optic nerve disc (myeloma) with a haemorrhagic ring around usually appears in the periphery of the retina. The disc of the optic nerve and the retina around it are swollen.

In **anaemia**, retinal ischemia and oedema appear on the fundus, and due to this the optic disc is hardly visible. The tone of blood vessels is lost, arteries become indistinguishable from veins.

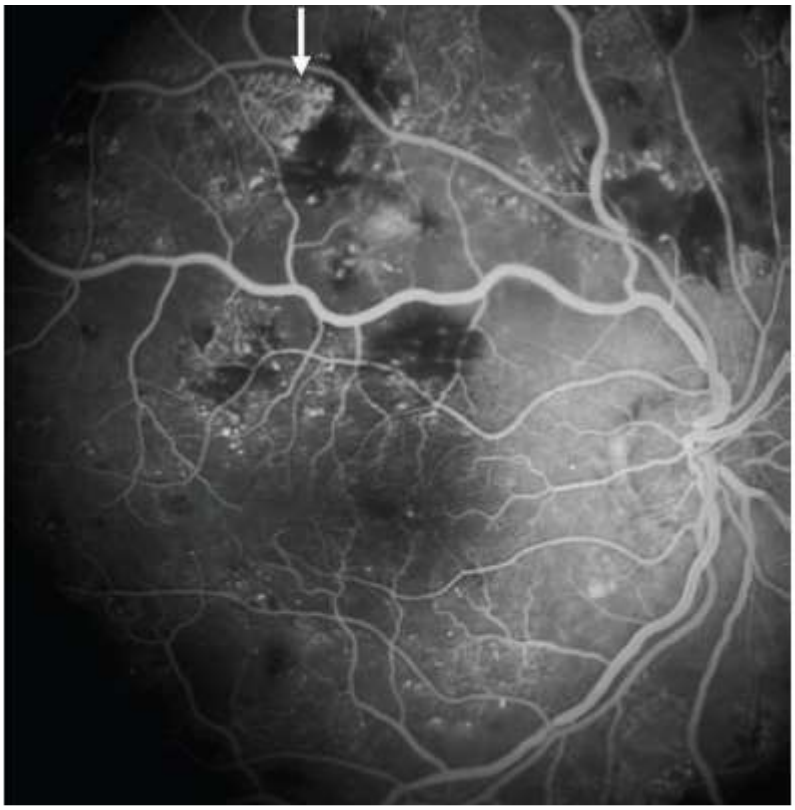


Fig. 17.3. Fluorescent angiography of a patient with diabetic retinopathy.

CHANGES TO THE ORGAN OF VISION IN THYROID DISORDERS

Thyroid disorders are wide-spread. Among other endocrine diseases, they are second only to diabetes by the frequency of occurrence.

Graves' disease (struma diffusa toxica; Perry's disease, Flajani's disease) is the most common disease of the thyroid gland, which is clinically manifested by the thyrotoxicosis syndrome. In diffuse toxic goiter, endocrine ophthalmopathy develops. Classification A.F. Brovkina et al. has a definite practical importance for characterizing the ophthalmic manifestations in diffuse toxic goiter. According to this classification, there are three forms of endocrine ophthalmopathy: thyrotoxic exophthalmos, oedematous exophthalmos and endocrine myopathy.

Clinical manifestations of thyrotoxic exophthalmos are slight protrusion of the eyeballs, rare blinking, retraction of the upper eyelid (Kocher's symptom), lagging of the upper eyelid in slow lowering the eyes (Graefe's symptom), tremor of the closed eyelids (Rosenbach's symptom), increased gloss of the eyes (Krause's symptom), convergence insufficiency (Möbius symptom), and partial failure to move the eyeballs outwards.

Edematous exophthalmos is characterized by swelling of periorbital tissues which leads to the eyes protrusion by 25-29 mm and more, the appearance of manifested symptoms of Graefe and Moebius. The mobility of the eyeballs is limited and diplopia develops. This leads to difficulty closing the eyelids, growing pain in the orbit, disrupted sensitivity of the cornea.

Endocrine myopathy is manifested by the weakness of one, two or more extraocular muscles. This leads to double vision and limited mobility of the eye. Strabismus develops (up to 15-60 °).

Endocrine ophthalmopathy requires **conservative treatment** which is directed at management of underlying disease.

Hypothyroidism (hypothyreosis) is a complicated complex of symptoms which develops due to the dramatic reduction of thyroid hormones in the blood.

The main ocular manifestations of hypothyroidism include reduced visual acuity, limited visual field for white and chromatic colours, deterioration of dark adaptation, impaired colour perception. The incidence of glaucoma and periodically occurring transient or stable rise in the intraocular pressure are observed. Complicated cataract often develops and, as already noted, endocrine ophthalmopathy may occur.

The main treatment of hypothyroidism is a replacement therapy with thyroid hormones.

Theoretical questions to the chapter:

1. What are the different forms of the optic nerve?
2. What is the normal caliber of the vessels of the optic nerve?
3. Describe the presentation of the normal fundus.

4. What are the changes to the organ of vision in different systemic diseases?

5. What are the changes to the organ of vision in brain tumours?

6. Explain the term “the raccoon eyes symptom”.

7. What are the changes in the retina in diabetes?

8. What are the characteristic features of oedematous exophthalmos and its treatment?

9. What are the characteristic fundus changes in patients with chronic myelogenous and lymphoid leukaemia?

10. What are the characteristic features of clinical presentation and treatment of toxoplasmosis?

11. What are the symptoms and treatment of Behçet's disease?

Practical skills which students should master:

1. History taking.

2. External examination of the eye.

3. Describe the presentation of the fundus of the eye.

4. Measuring the intraocular pressure.

5. Conducting the examination of the optic disc.

6. Paying attention to the shape of the optic nerve, its boundaries, the degree of its protrusion.

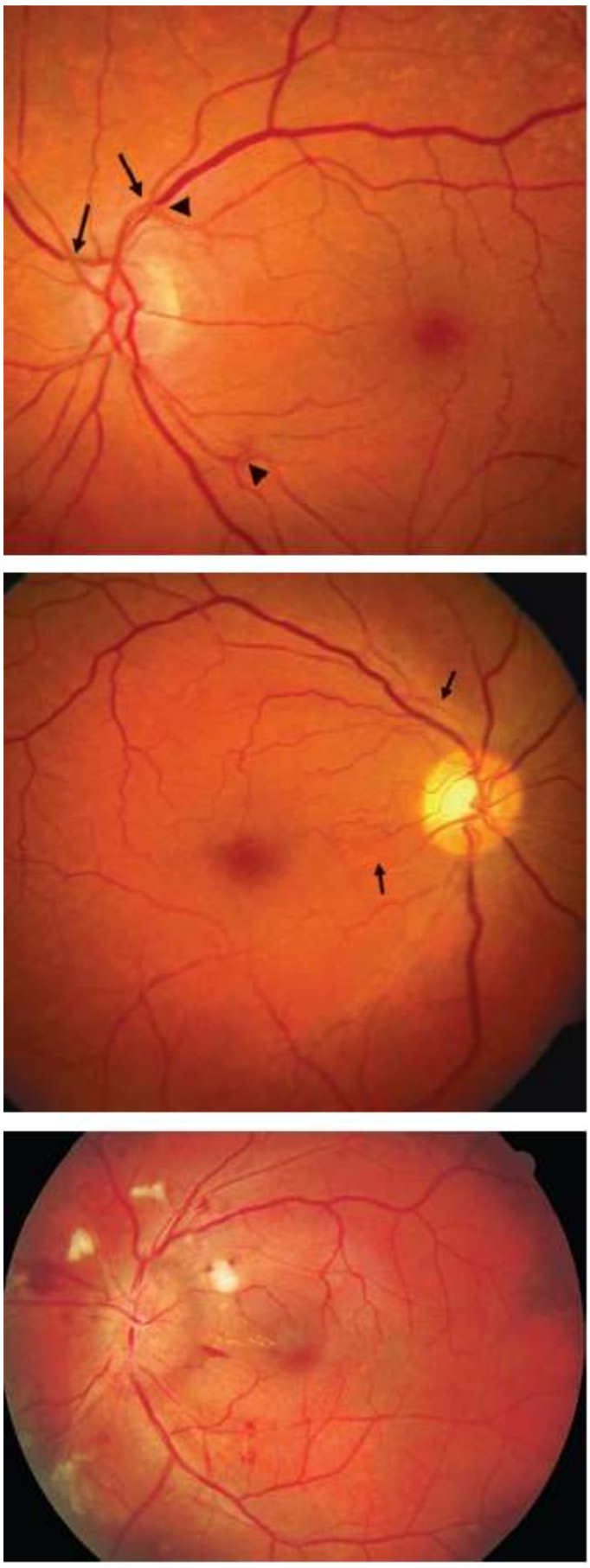


Fig. 17.4. Hypertensive neuroretinopathy.

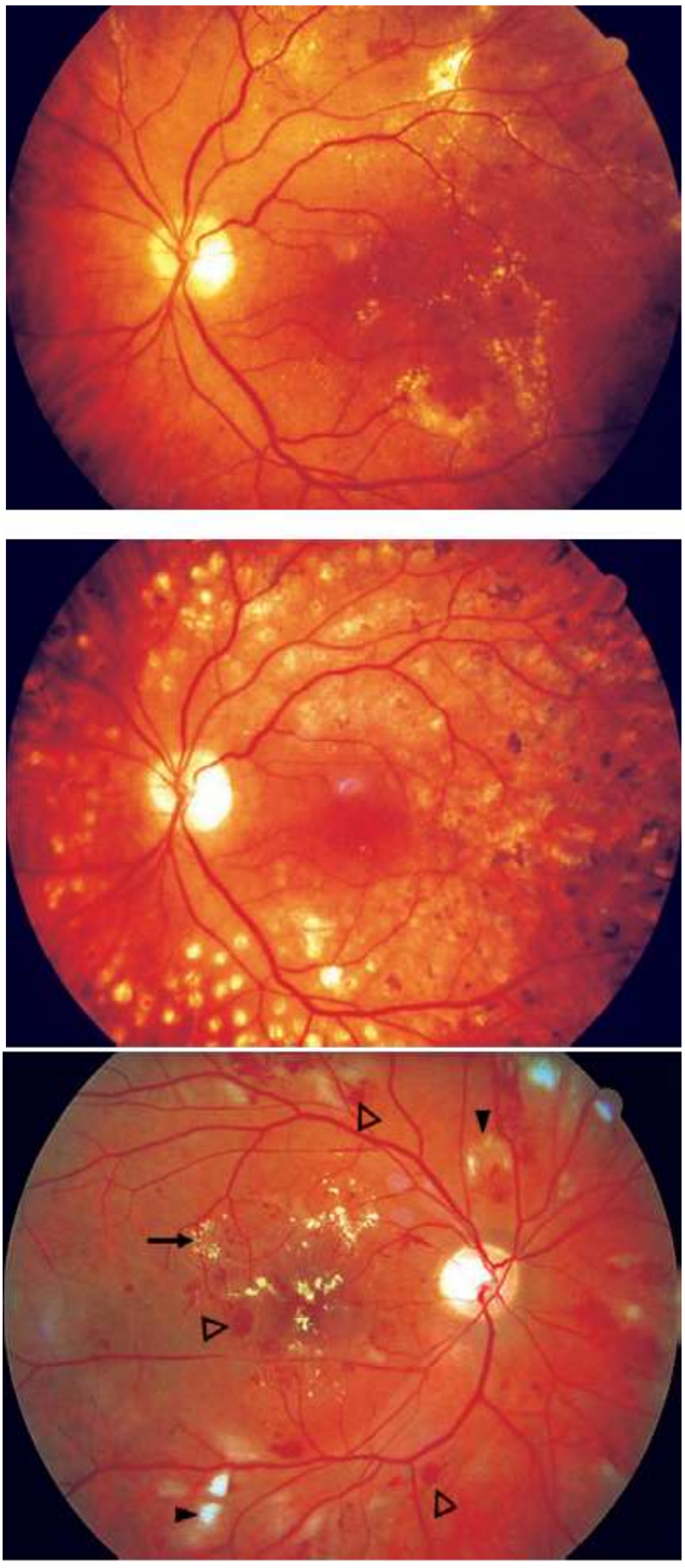


Fig. 17.5. Preproliferative diabetic retinopathy.

TESTS FOR SELF-CHECK

Anatomical and topographic features of vision

1. The function of the lens is:
refraction of light
binocular vision
colour perception
light perception
all of the listed

2. Optical system of the eye includes:
all of the listed
vitreous body and aqueous humour of the anterior chamber
retina
lens and cornea
iris

3. Protective apparatus of the eye includes:
the orbit and eyelids
lacrimal gland
external muscles
the conjunctiva
all of the listed

4. The functions of the external eye membrane are:
protection and refraction of light
forming its shape
refraction of light
protection
all of the listed

5. The functions of the ciliary body are:
accommodative and secretory
production of aqueous humour
refraction
accommodation
all of the listed

6. What vessels is the choroid formed by?
posterior short ciliary arteries
posterior long ciliary arteries
anterior ciliary arteries

central retinal artery
all of the listed

7. Nutrition of the lens is supplied by:
aqueous humour
tears
perilimbal arterial plexus
vitreous body
all of the listed

8. The internal membrane of the eyes is:
retina
choroid
sclera
ciliary body
lens

9. How many neurons does the retina have?
three
one
two
five
nine

10. How many histological layers of retina are there in the fovea?
four
ten
one
two
five

11. The subcortical visual centres are located at:
lateral geniculate body
chiasm
optic tract
optic nerve
all of the listed

The functions of vision

1. **Which part of the retina provides the highest visual acuity?**
the foveolar zone
yellow spots all over the area

optical disc area
vision in all parts of the retina is uniform
all of the listed

2. What is the blind spot?
projection in the field of view of the optic nerve
projection in the field of view of the macular zone
limited scotoma in any part of the field of view
visual field defects of retinal vessels
projection in the field of view of the ora serrata

3. What is the examination method for the field of view?
perimetry
gonioscopy
biomicroscopy
ophthalmoscopy
tonometry

4. What is the unit of measurement for the field of view?
degrees
centimetres
dioptres
minutes
all of the listed

5. The refractive power of the cornea is:
40.0 - 42.0 dioptres
18.0 - 20.0 dioptres
60.0 - 62.0 dioptres
28.0-30.0 dioptres
15.0-25.0 dioptres

6. The refractive power of the lens is:
18.0-20.0 dioptres
60.0-62.0 dioptres
40.0-42.0 dioptres
28.0-30.0 dioptres
15.0-25.0 dioptres

7. Photoreceptors of the retina are:
cones, rods
cones, Ganglion cells
cones, the cells of pigment epithelium
rods, the cells of pigment epithelium

all of the listed

8. What is the visual acuity of a newborn infant?
very low (about 0.01)
significantly lower than in adults (0.1-0.3)
sufficiently high (0.5-0.8)
high (1.0)
very high (1.5-2.0)

9. Which of the anatomical elements of the retina are colour receptors?
cones
rods
ganglion cells
the cells of pigment epithelium
all of the listed

10. What is scotoma?
focal defect of sight
impaired twilight vision
limitation of the field of view
lack of vision
all of the listed

11. The method for measuring visual acuity is:
Golovin-Sivtsev chart
Rabkin's table
Polyak's optotypes
by counting the doctor's fingers
all of the listed

Refraction and accommodation

1. Young children with refractive error are prescribed glasses for:
permanent use
at close range
into the distance
driving
glasses are not prescribed

2. Determining refraction in children is carried out by the method of:
skiascopy
corrective lenses
direct ophthalmoscopy

determining a further point of clear vision
perimertry

3. Presbyopia develops at the age of:
40 years
20 years
30 years
60 years
50 years

4. The average distance between pupils in adults is:
64-66 mm
56-58 mm
60-62 mm
60-68 mm
70-72 mm

5. What type of convex glasses does a 70-years-old emmetrope need for reading?
+ 4.0 D
+ 2.0
+ 3.0 D
+1.0 D
+2.5 D

6. With age, the power of accommodation:
decreases
increases
does not change
astigmatism develops
all of the listed

7. What lenses improve myopia?
negative
positive
cylindrical
spherocylindrical
all of the listed

8. What kind of refraction is strong:
myopia
emmetropia
hypermetropia

astigmatism
ametropia

9. An objective method of determining clinical refraction is:
skiascopy
biomicroscopy
ophthalmoscopy
method of corrective lenses
all of the listed

10. A further point of clear vision in the emmetropic eye is:
located at infinity
in front of the eye
absent
located behind the eye
located in 5 metres

11. A further point of clear vision in the myopic eye is:
in front of the eye
located behind the eye
absent
located at infinity
all of the listed

Diseases of the eyelids, lacrimal organs and orbit

1. The upper orbital wall borders with:
frontal sinus
pterygopalatine fossa
maxillary sinus
nasal cavity
all of the listed

2. The external orbital wall borders with:
pterygopalatine fossa
maxillary sinus
frontal sinus
nasal cavity
all of the listed

3. The inferior orbital wall borders with:
maxillary sinus
pterygopalatine fossa

frontal sinus
nasal cavity
all of the listed

4. The internal orbital wall borders with:
ethmoid sinus
maxillary sinus
frontal sinus
nasal cavity
all of the listed

5. In case of acute dacryoadenitis, pathological process is localized:
in the outer part of the upper eyelid
in the inner part of the upper eyelid
in the outer part of the lower eyelid
in the inner part of the lower eyelid
all of the listed

6. When washing the lacrimal passages, where is the fluid released?
trickles from the nose
drops from the nose
through another lacrimal point
through the same lacrimal point
all of the listed

7. Anatomical structure of the lacrimal system includes:
the lacrimal gland, the lacrimal point, nasolacrimal ducts, lacrimal sac, lacrimal rivus, lacrimal lake
lacrimal sac, nasolacrimal duct, lacrimal rivus, lacrimal lake
lacrimal sac, nasolacrimal duct, lacrimal gland, lacrimal lake
lacrimal point, nasolacrimal ducts, lacrimal sac
none of the listed

8. Where is the lacrimal point located?
in the inner corner of the eye
in the outer corner of the eye
in the middle of the eyelid
on the upper eyelid
all of the listed

9. Abnormality of the eyelids is:
coloboma
ptosis
blepharitis

lagophthalmos
iritis

10. Inflammatory disease of the eyelids is:
all of the listed
hordeolum
abscess
blepharitis
demodicosis

11. Distichiasis is:
accessory row of eyelashes
misdirected eyelashes
downward drooping of the upper eyelid
outward protrusion of the lower eyelid
all of the listed

Diseases of the conjunctiva

1. How many parts is conjunctiva divided into?
3
1
4
5
10

2. What are the physiological functions of the conjunctiva?
all of the listed
trophic
moisturizing barrier
protective
secretory

3. Which of the following conjunctivites is of viral aetiology?
all of the listed
epidemic keratoconjunctivitis
herpetic conjunctivitis
epidemic haemorrhagic conjunctivitis
pharyngoconjunctival fever

4. What is the degree of infectivity of epidemic keratoconjunctivitis ?
high
low

unstudied
middle
very low

5. Pterygium is:
dublicator of the conjunctiva
inflammation of the conjunctiva
benign tumour of the conjunctiva
scar of the conjunctiva
all of the listed

6. Pinguecula is:
yellow colour neoplasm of the conjunctiva near the limbus due to hyperplasia of the connective tissue
inflammation of the conjunctiva
scar of the conjunctiva
malignant tumour
all of the listed

7. Clinical features of allergic conjunctivitis are:
redness, itching, conjunctival "cobblestones"
purulent discharge
no complaints
only mucus discharge
all of the listed

8. What medications are topically applied in bacterial conjunctivitis?
antibiotics
vitamins
corticosteroids
antivirals
all of the listed

9. What diseases of the conjunctiva can occur?
all of the listed
infectious
viral
acute
allergic

10. The conjunctiva – is:
the mucous membrane
a part of the external membrane of the eye
a part of medium layer of the eye

the internal membrane of the eye
all of the listed

11. Methods for examination of the conjunctiva are:
biomicroscopy and side lighting
perimetry
the transmitted light method
gonioscopy
all of the listed

Diseases of the cornea and sclera

1.The average value of the refractive power of the cornea in adult is:
43 dioptres
23 dioptres
30 dioptres
50 dioptres
33 dioptres

2. The combination of signs: photophobia, lacrimation, blepharospasm, pain in the eye - is typical for:
keratitis
cataract
retinal detachment
atrophy of the optic nerve
all of the listed

3. The following is NOT typical of keratitis:
increased intraocular pressure
reduced tactile sensitivity of the cornea
the presence of infiltrates in the cornea
pericorneal injection
mixed injection

4. The adverse effect of keratitis can be all of the listed, except for:
macular degeneration of the retina
corneal leukoma
vascularisation of the cornea
ulcers of the cornea
scar of the cornea

5. Blood supply of the cornea is performed by:
lacrimal fluid, the capillary network of the limbus zone, ocular fluid
posterior long ciliary arteries, nasociliary artery, lacrimal fluid

intraocular fluid, anterior ciliary arteries
central artery of the retina
all of the listed

6. The features of the normal cornea are:
transparent, shiny, high-sensitive, of spherical shape, avascular
shiny, conical, sensitive, having a certain size
transparent, ellipsoidal, having some shape
white, conical, devoid of sensitive innervations
all of the listed

7. What histological layers does the cornea have?
epithelium, endothelium, stroma, Bowman's membrane, Descemet's membrane
anterior and posterior epithelium, the proper substance (stroma)
anterior and posterior pigment epithelium, anterior and posterior vitreous body plates, stroma
epithelium, stroma, Bowman's membrane

8. The main property of the anterior epithelium of the cornea is:
high regenerative ability
participation in the development of lacrimal fluid
mechanical protection of underlying tissues
ensuring the exchange processes between the cornea and the intraocular fluid
all of the listed

9. The main functions of the cornea are
protective, light-conducting, photorefractive
protective, light-conducting
photorefractive, production of moisture
light-conducting, photorefractive
all of the listed

10. The principal methods for examination of the cornea are:
method of side lighting and biomicroscopy
the transmitted light method and method of side lighting
biomicroscopy and ophthalmoscopy
skiascopy
perimertry

11. Band-shaped keratopathy is commonly caused by deposition of:
calcium salt
magnesium salt
ferrous salt

copper salt
all of the listed

Disease of the lens of the eye

1. The refractive power of the lens is:
18.0 – 20.0 D
28.0 – 30.0 D
40.0 – 42.0 D
60.0 – 62.0 D
30.0 – 40.0 D

2. The lens in case of accommodation tension:
becomes flat
becomes more convex
does not change
removes downward, away from the cornea
all of the listed

3. The lens opacification is referred to as:
cataract
leukoma
aphakia
glaucoma
iritis

4. Aphakia is accompanied with the following changes of the anterior chamber:
anterior chamber becomes deeper
anterior chamber becomes of irregular depth
anterior chamber becomes shallow
anterior chamber becomes thin
all changes are possible

5. What are the methods of lens examination?
the slit lamp examination and the transmitted light method
direct and indirect ophthalmoscopy
transillumination
gonioscopy
perimetry

6. These types of cataract are related to age, EXCEPT FOR:
mixed
zonular

nuclear
brown
all of the listed

7. The term “pseudophakia” means:
absence of the lens
the presence of an artificial lens in the eye
condition of the eye after the lens removal
dislocation of the lens
all of the listed

8. Clinical signs of age-related cataract are as follows:
in the study by transmitted light, there are dark bands in the form of "spokes in the wheel", IOP is normal, reflex of the fundus of the eye is absent
the eye is at rest, the pupil is black, atrophy and excavation of the optic nerve
increased IOP
congestive injection of the eyeball, the anterior chamber is shallow
the pupil is wide, high IOP

9. What is a non-progressive type of cataract?
congenital
senile
traumatic
diabetic
all of the listed

11. The most common causes of congenital cataract are:
all of the listed
rubella in the woman’s anamnesis during pregnancy
metabolic disease in the woman’s anamnesis during pregnancy
influenza in the woman’s anamnesis during pregnancy
smoking and alcohol

Diseases of the uveal tract

1. Identify the parts of the vascular membrane:
iris, ciliary body, choroid
iris, cornea, choroid
lens, sclera, iris
iris, ciliary body, lens
sclera, choroid, retina

2. Blood supply of the iris and ciliary body is provided by:
anterior and posterior long ciliary arteries

short ciliary arteries
ophthalmic artery
Zinn's arterial circle
all of the listed

3. Blood supply of the choroid is provided by:
anterior and posterior long ciliary arteries
short ciliary arteries
ophthalmic artery
Zinn's arterial circle
all of the listed

4. Innervation of the iris and ciliary body is provided by:
trigeminal nerve
long and short ciliary nerves
oculomotor nerve
the sympathetic nerve
optic nerve

5. Identify the type of uveitis according to anatomical characteristics:
all of the listed
anterior uveitis
posterior uveitis
panuveitis
none of the listed

6. The administration route of medications in the eye in uveitis:
all of the listed
instillation of drops
application of ointments into the conjunctival sac
electro- and phonophoresis of medications
injection under the conjunctiva, parabulbar injections, retrobulbar injections

7. The symptoms of posterior uveitis are:
all of the listed
decrease of vision
micropsia
photopsia
scotoma

8. Identify the signs of acute iridocyclitis:
all of the listed
pericorneal injection

contraction of the pupil
posterior synechia
ciliary sensitivity

9. Identify the type of uveitis according to the nature of exudate:
fibrinous
haemorrhagic
purulent
mixed
all of the listed

10. Identify the type of uveitis according to the morphological presentation of inflammation:
all of the listed
granulomatous
agranulomatous
mixed
none of the listed

11. One of the earliest features of anterior uveitis includes:
aqueous flare
keratic precipitates
hypopyon
posterior synechiae
cataract

Diseases of the optic nerve

1. Congestive disks of optic nerves are characterized by:
all of the listed
oedema of the disk tissue
blurred borders
expansion of veins of the retina
haemorrhages

2. The causes of the development of congestive disks of optic nerve can be:
all of the listed
tumour of the brain
tumour-like diseases of the brain
brain cysts
brain injury

3. Neuritis of the optic nerve is characterized by:
all of the listed

a sharp decline in vision
hyperaemia of the optic disc
swelling of the optic disk
oedema of the optic disk

4. Optic neuritis is attributed to the following group of diseases:
acute
chronic
subacute course
latent course
subacute

5. If the patient has optic neuritis, what changes of vision can be observed?
considerably and quickly reduced
does not change
decreases slightly and slowly
any of the listed options
blindness

6. When patient has optic neuritis, what is the colour of the disk?
hyperaemia
pale
grey
no changes
all of the listed

7. When pituitary tumours are detected, there is:
bitemporal hemianopsia
concentric narrowing of the visual field
absolute central scotoma
bilateral hemianopsia
all of the listed

8. What changes can be observed on the fundus in retro-bulbar neuritis?
no change
hyperaemia
blurred borders
haemorrhages
oedema

9. Where lesion can be located in patient with right-sided hemianopsia?
left optic tract
chiasma
right optic tract

the occipital lobe of the cerebral cortex on the left
all of the listed

10. Topographically, the optic nerve can be divided into the following segments:
intraocular, orbitalis, intercanalis and intracranial
anterior segment, middle segment, posterior segment
the inner part, the outer part
anterior part, posterior part
all of the listed

11. Primary optic atrophy results from:
neurological disease
retinal disease
chronic glaucoma
papilloedema
diseases of the lens

Disease of the retina

1. Amaurotic cat's eye reflex is observed in:
retinoblastoma
papilloedema
papillitis
retinitis
all of the listed

2. The most common lesion which impairs vision in diabetic retinopathy is:
macular oedema
microaneurysm
retinal haemorrhage
retinal detachment
cataract

3. Commotio retinae is observed in:
concussion injury
papilloedema
central retinal vein thrombosis
central retinal artery thrombosis
all of the listed

4. Night blindness is caused by:
dystrophies of retinal rods
central retinal vein occlusion

dystrophies of the retinal cones
retinal detachment
cataract

5. In central retinal artery occlusion, a cherry-red spot is due to:
the contrast between pale retina and reddish choroids
haemorrhage at macula
increased choroidal perfusion
increase in retinal perfusion at macula
all of the listed

6. The most common primary intraocular malignancy in adults is:
choroidal melanoma
retinoblastoma
squamous cell carcinoma of the conjunctiva
iris naevus
all of the listed

7. A patient with long-standing diabetes mellitus noticed sudden muscae volitantes. On examination, the red reflex is dim, no details of fundus can be observed. The patient may have:
vitreous haemorrhage
non proliferative diabetic retinopathy
cystoid macular oedema
central retinal vein occlusion
retinal detachment

8. Occlusion of the lower nasal branch of the central retinal artery results in one of the following field defects:
upper temporal field defect
lower nasal sector field defect
upper nasal sector field defect
lower temporal sector field defect
blindness

9. A young patient with sudden painless loss of vision. On examination: a cherry-red spot with clear AC. The likely diagnosis is:
central retinal artery occlusion
central retinal vein occlusion
diabetes mellitus
branch retinal vein occlusion
retinal detachment

10. In retinal detachment, fluid accumulates between:
neurosensory retina and layer of retinal pigment epithelium
outer plexiform layer and inner nuclear layer
nerve fibre layer and the rest of retina
retinal pigment epithelium and Bruch's membrane
all of the listed

11. How many layers does the retina consist of?
10
5
7
1
8

Glaucoma

1. The main symptoms of glaucoma are:
all of the listed
excavation of optic disk
specific changes in the field of view
intolerant IOP
loss of vision

2. What produces aqueous humour?
ciliary body
anterior chamber
choroid
lens
cornea

3. What factors determine the magnitude of IOP?
all of the listed
elasticity of the corneoscleral membrane
blood vessels of the uveal tract
production of aqueous humour
outflow of aqueous humour

4. What is the normal value of intraocular pressure?
16-25 mmHg
12-16 mmHg
20-28 mmHg
9-22 mmHg
15-25 mmHg

5. One of the signs of acute attack of glaucoma is:
oedema of the cornea
conjunctival injection
clouding of the lens
deepening of the anterior chamber
all of the listed

6. The main type of surgery in acute attack of glaucoma is:
filtration operation
iridectomy
operations decreasing the secretion of aqueous humour
phacoemulsification
all of the listed

7. What groups of medications are used to reduce IOP?
mydriatic agents
parasympathomimetics
neurotropic agents
angioprotectors
all of the listed

8. What method is used to examine the angle of the anterior chamber?
gonioscopy
biomicroscopy
ophthalmoscopy
skiascopy
all of the listed

9. The optimal frequency of instilling myotic drops to patient with glaucoma is:
once a day
3 times a day
2 times a day
4 times a day
5 times a day

10. Determine the stage of glaucoma if the visual fields, physiological excavation of the optic disk and IOP are normal?
there is no glaucoma
developed glaucoma
initial glaucoma
advanced glaucoma
all of the listed

11. All of the following is associated with open angle glaucoma, EXCEPT FOR:
loss of central fields
enlarged blind spot
generalized depression of isopters
tubular vision
all of the listed

Strabismus

1. The signs of concomitant strabismus are:
all of the listed
equality of primary and secondary deviation angles of the eyes
absence of diplopia
maintaining the mobility of the eye to the full extent
none of the listed

2. The symptoms of paralytic strabismus are:
all of the listed
secondary deviation angle of the eye is more than the primary one
the presence of diplopia and dizziness
limitation of eye movement in the direction of the paralyzed muscle
none of the listed

3. The forms of concomitant strabismus are:
all of the listed
alternating
accommodational, partially accommodational, nonaccommodational
with and without amblyopia
monolateral – right or left eye

4. What are the signs of concomitant strabismus?
the direction of eye deviation
condition of accommodation, presence of amblyopia
single or bilateral strabismus
constancy of strabismus
none of the listed

5. What are the types of amblyopia according to the degree of reduction of vision?
all of the listed
medium degree from 0.3 to 0.2

high degree from 0.1 to 0.05
low degree from 0.8 to 0.4
none of the listed

6. What are the surgeries that increase the action of muscles in strabismus?
shortening of the muscle
tenorrhaphy
tenomyoplastics
partial myotomy
none of the listed

7. What are the surgeries that weaken the action of muscles in strabismus?
recession
tenomyoplastics
partial myotomy
tenorrhaphy
none of the listed

8. Identify the types of amblyopia by the nature of fixation:
all of the listed
with central fixation
with non-central fixation
with intermittent fixation
none of the listed

9. Identify the methods of determining the angle of strabismus
all of the listed
by using perimeter
by synoptophore
Hirschberg's method
none of the listed

10. The principle and sequence of treatment in accommodational strabismus include:
all of the listed
treatment of amblyopia (pleoptic)
resumption and consolidation of binocular vision (orthoptics)
prescription of glasses in accordance with the detected ametropia
none of the listed

11. All the following are extraocular muscles of the eye EXCEPT FOR:
ciliary muscle
superior rectus muscle
inferior oblique muscle

superior oblique muscle
all of the listed

Damage of the vision organs

1. Types of eye injuries are:
all of the listed
penetrating injury
blunt trauma
perforating wound
none of the listed

2. Which injuries are possible in case of blunt trauma?
all of the listed
corneal abrasion
haemophthalmos
subcutaneous eyelid emphysema
disruption of choroid

3. What are the reliable signs of penetrating trauma?
corneal and iris wounds
iris wound
hyphaema
corneal wound
none of the listed

4. Identify the change of the lens position in blunt trauma:
dislocation of the lens
microphakia
cataract
iridodyalisis
all of the listed

5. What is the main method for X-ray localization of foreign intraocular bodies:
Comberg-Baltin method
Kuhnt's method
with fluorescein
all of the listed
none of the listed

6. Ways of extraction of intraocular foreign bodies are:
diascleral
corneal section

through the flat part of ciliary body
back access
all of the listed

7. Prevention of sympathetic inflammation includes:
removal of the injured eye
absorptive therapy
epithelialising therapy
removal of the foreign body
all of the listed

8. What is enucleation?
extraction of the eyeball after intersection of the optic nerve
extraction of the inner tissues leaving the eyeball, sclera and optic nerve
extraction of the eyeball without intersection of the optic nerve
extraction of all eye inner tissues after intersection of the optic nerve
none of the listed

9. For the diagnosis of subconjunctival disturbance of sclera, the following is used:
examination of the wound
ophthalmometry
exophthalmometry
refractometry
perimetry

10. What is the perforating wound of the eye?
double penetration of the eye tissues
penetrating trauma of the cornea
penetrating trauma of the sclera
penetrating trauma of the limbus
all of the listed

11. What is the main sign of corneal penetrating wound of the eye?
corneal injury
iris wound
hyphaema
haemophthalmos
none of the listed

Emergency conditions in ophthalmology

1. Haematocornea is accumulation of blood in:
layers of the cornea

conjunctival sac
vitreous body
layers of the retina
none of the listed

2. For diagnosis of subconjunctive rupture of the sclera, the following is used:
transillumination
ophthalmometry
exophthalmometry
refractometry
perimetry

3. What type of injection is typical for contusions of the eyeball?
mixed injection
pericorneal injection
conjunctival injection
surface injection
none of the listed

4. In the syndrome of the upper orbital gap, the following is affected:
the oculomotor nerve
the optic nerve
the second branch of the trigeminal nerve
the lower orbital vein
all of the listed

5. For treatment of subconjunctival scleral rupture, the following is applied:
urgent surgery
inpatient conservative treatment
outpatient conservative treatment
planned surgery
none of the listed

6. Tuber tissue that forms a crust, deep opaque corneal opacity are typical for:
fourth-degree burn
first-degree burns
second-degree burns
third-degree burns
all of the listed

7. For diagnosis of intraocular foreign bodies, the following is used:
X-rays
refractometry
ophthalmometry

exophthalmometry
perimetry

8. What is the perforating wound of the eye?
double penetrating wound of the eye membranes
penetrating wound of the cornea
penetrating wound of the sclera
penetrating wound of the limbus
none of the listed

9. What are the absolute conditions for penetrating trauma:
all of the listed
the presence of intraocular foreign body
the loss of the inner membranes of the eye
penetrating wound in the outer membrane of the eye
penetrating wound of the cornea

10. Enucleation is:
extraction of the eyeball after the intersection of the optic nerve
extraction of the inner membranes leaving the eyeball, sclera and optic nerve
extraction of the eyeball without intersection of the optic nerve
extraction of the inner membranes of the eyeball after the intersection of the optic nerve
none of the listed

11. Emergency aid for abscessed hordeolum includes:
surgery, antibiotic therapy
dry heat, UHF
antibacterial drops
anti-inflammatory drugs, dry heat
vitamins

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