The ministry of health of the Republic of Belarus

Educational institution «Gomel State Medical University»

Department of Otorhinolaryngology with a course of ophthalmology

Discussed at the meeting
department
Document № 8.1 dated 16.06.2023

MANUALS

for 6th year students of faculty of foreign students on ophthalmology

TOPIC №3. GLAUCOMA. TRAUMA.

The time 6 hours

Gomel, 2023

MOTIVATION FOR LEARNING TOPICS, TRAINING AND EDUCATIONAL PURPOSES, THE REQUIREMENTS TO THE ORIGINAL LEVEL OF KNOWLEDGE

Glaucoma has been recognized as a clinical disease for 150years. Until recently, intraocular pressure (IOP) was considered to be the only pathogenetic factor, but its significance has now been qualified to some extent. The glaucomas are a diverse group of disorders that damage the optic nerve, resulting in characteristic optic nerve head cupping and visual field loss. The intraocular pressure (IOP) in such persons is presumed to be inconsistent with normal optic nerve function and is usually elevated above normal values. Nevertheless, IOP elevation need not be present to make the diagnosis of glaucoma. Patients with characteristic optic nerve cupping and visual field loss in the presence of normal IOP are diagnosed as having normal-pressure (low-pressure) glaucoma. Conversely, patients with elevated IOP in the absence of detectable secondary abnormalities in ocular structure and function are classified as ocular hypertensives.

The eye is a highly sensitive organ that is well-protected by the bony orbit and eyelid. Common traumatic eye injuries occur through blunt or sharp objects or chemical burns. Closed globe injuries usually follow blunt trauma and have a varied clinical presentation (superficial corneal abrasion to retinal hemorrhage). Open globe injuries usually follow sharp or high-velocity blunt trauma and present with ocular volume loss or a prolapsing uvea in addition to the sequelae of closed ocular injuries. Orbital floor fractures are a type of periocular injury following high-velocity blunt trauma to the globe and upper eyelid which present with unilateral periorbital pain, edema, and/or ecchymosis, enophthalmos, and an orbital rim "step-off" which is confirmed by CT. Chemical burns of the eye present with ocular pain, erythema, and blepharospasm. Treatment of traumatic eye injuries depends on the precise underlying injury. Chemical burns require immediate and adequate irrigation with water beginning prior to hospitalization. Urgent stabilization, antibiotics, and immediate ophthalmologic consultation is often required to rule out serious injury (e.g., severe chemical burns, open globe injuries, retinal detachment, extraocular muscle entrapment) and determine the need for surgery.

The purpose of the class: To study the etiology and pathogenesis of primary glaucoma, the classification of glaucoma, the criteria underlying the clinical classification of primary glaucoma. Conservative principles and indications for surgical treatment of primary glaucoma. To familiarize with tactics of management of patients with an acute attack of glaucoma. To study the technique of measuring IOP by palpation and tonometry according to Maklakov, to study the width of the angle of the anterior chamber according to Wurgaft. Teach students how to diagnose glaucoma.

To give students an idea of the place of eye injuries in general injuries, the frequency of domestic, school and occupational injuries. To familiarize students with the features of the clinic, the course and treatment of burns. Emergency care for burns. Outcomes. Prevention Features of childhood injuries.

Objectives of the lesson:

Student should know:

1. Anatomy of the angle of the anterior chamber.
2. Ways of the outflow of intraocular fluid.
3. The mechanism of production of intraocular fluid.
4. Glaucoma classification.
5. Methods for diagnosing glaucoma.
6. Clinic, diagnosis of open-angle, angle-angle glaucoma, acute attack of glaucoma.
7. Medication for glaucoma.
8. Principles of pathogenetic surgery for glaucoma.
9. Clinical examination of patients with glaucoma.
10. Classification of eye damage according to etiology, localization, severity, presence and properties of foreign bodies.
11. Diagnostic methods. The main types of primary health care.
12. Methods for the determination and localization of foreign bodies. X-ray diagnosis of foreign bodies in the eye.
13. Complications of penetrating wounds: traumatic non-purulent iridocyclitis, purulent iridocyclitis, endophthalmitis, panophthalmitis. Clinic, course, treatment, outcomes.
14. Sympathetic ophthalmia. The frequency and timing of occurrence, etiology and pathogenesis. Indications for the removal of the wounded eye and the timing of the operation.
15. Burns of the organ of vision: chemical, thermal, radiation. Classification of burns according to their severity and prevalence. Features of the clinic, course and treatment of burns caused by acids, alkalis, manganese crystals. Emergency assistance. Burn treatment: conservative and surgical.
16. Radiation injuries of the organ of vision.
17. Ophthalmic symptoms in cardiovascular, neurological, infectious diseases, HIV infection, blood diseases and endocrine pathology.

***Student must be able to*:**

1. IOP measurements by palpation and tonometry according to Maklakov and Goldman.
2. Teach you how to write prescriptions for drugs used to treat glaucoma.
3. Provide first aid for injuries, blunt injuries, burns and eye microtraumas.
4. Master the technique of removing foreign bodies from the conjunctiva and cornea.
5. Master the technique of washing the conjunctival cavity.
6. Master the technique of applying monocular and binocular dressings.
7. Removal of superficial foreign bodies from the conjunctiva and cornea.
8. Lavage of the conjunctival cavity.
9. The application of monocular and binocular dressings.
10. Examination of the fundus using direct and reverse ophthalmoscopy.
11. Biomicroscopy. Principle of operation. Opportunities.

The student must perform the following practical skills:

1. IOP measurement by palpation.
2. IOP measurement with a Maklakov tonometer.

3. Determining the width of the angle of the anterior chamber according to Wurgaft.
4. Biomicroscopy. The principle of work, opportunities.

CHECKLIST OF QUESTIONS FROM RELATED SUBJECTS

1. Physiology of intraocular fluid production.
2. Anatomy and physiology of ciliary body and trabecular meshwork.
3. X-ray examination of the orbit.
4. Indications and contraindications for MRI, CT.

CHECKLIST OF CONTROL QUESTIONS FOR THE LESSON

1. Definition of glaucoma. The frequency and prevalence of the disease. The social significance of glaucoma as one of the main causes of blindness. Intraocular pressure. Circulation of aqueous humor. Regulation of intraocular pressure. The main types of glaucoma.
2. Primary glaucoma. Modern views on the etiology and pathogenesis. Factors predisposing to the development of glaucoma. Hereditary factors in glaucoma.
3. Classification of primary glaucoma. The clinical course of open-angle and angle-angle glaucoma. Absolute glaucoma.
4. Methods for the diagnosis of glaucoma: tonometry, electrotonometry, biomicroscopy, ophthalmoscopy, gonioscopy, perimetry.
5. Methods for early diagnosis of glaucoma. Subjective and objective symptoms depending on the stage of the disease.
6. The clinical course of an acute attack of glaucoma, general and local symptoms. Pathogenesis of an acute attack. Differential diagnosis with acute iridocyclitis; with a number of common diseases. Emergency treatment for an acute attack of glaucoma. Indications for surgical treatment. Fulminant glaucoma.
7. Principles of conservative treatment of open-angle and angle-angle glaucoma, principles of prescribing drugs depending on the form of glaucoma. The use of general exposure. Regime, diet, employment.
8. Indications for surgical treatment. The principles of pathogenetically oriented operations. The use of physical factors in the treatment of glaucoma (lasers, high and low temperatures). Clinical examination of patients with glaucoma. Prevention of blindness from glaucoma.
9. Secondary glaucoma: uveal, phacogenic, vascular, traumatic, degenerative, neoplastic. Features of the course and treatment. Outcomes.
10. Congenital glaucoma. Frequency. Etiology and pathogenesis. The influence of various pathological conditions of pregnant women on the development of the anterior chamber angle. The role of heredity. The earliest signs of the disease. Classification, forms. Clinic, stages, course.
11. Hydrophthalmus, butphthalmos. Principles, terms and methods of surgical treatment of congenital glaucoma. Outcomes.

12. Place of eye damage in general injuries. Frequency of domestic, school and occupational injuries. Classification of eye damage according to etiology, localization, severity, presence and properties of foreign bodies.

13. Diagnostic methods. The main types of primary health care. Outcomes. Treatment of complications. Prevention of eye injuries.

14. Injuries of the eyelids, conjunctiva and lacrimal organs. Primary care with them.

15. Eye injuries. Classification: non-penetrating, penetrating; simple, complex, with complications. Symptoms of perforated wounds of the cornea and sclera. Signs of through wounds. Primary care Primary surgical treatment.

16. Methods for the determination and localization of foreign bodies. X-ray diagnosis of foreign bodies in the eye. Principles of removal of magnetic and magnetic foreign bodies. Metallosis and the timing of its manifestation. The mechanism of development of various symptoms in metalloses.

17. Complications of penetrating wounds: traumatic non-purulent iridocyclitis, purulent iridocyclitis, endophthalmitis, panophthalmitis. Clinic, course, treatment, outcomes.

18. Sympathetic ophthalmia. The frequency and timing of occurrence, etiology and pathogenesis. Clinical forms. General and local treatment, prognosis, prevention. Indications for the removal of the wounded eye and the timing of the operation.

19. Dull eyeball damage. Their frequency, clinic, course, outcomes. Classification by severity, treatment principles.

20. Features combat damage. Features of industrial injuries, microtraumas, methods of individual and social prevention.

21. Burns of the organ of vision: chemical, thermal, radiation. Classification of burns according to their severity and prevalence. Features of the clinic, course and treatment of burns caused by acids, alkalis, manganese crystals. Emergency assistance. Burn treatment: conservative and surgical.

22. Radiation injuries of the organ of vision. Ultraviolet radiation, infrared radiation, x-ray and ionizing radiation, laser radiation in various parts of the spectrum, radio waves, UHF, microwave, ultrasound, the visible part of the spectrum of high brightness.

23. Ophthalmic symptoms in cardiovascular, neurological, infectious diseases, HIV infection, blood diseases and endocrine pathology.

PRACTICAL PART OF THE LESSON

Work of students is carried out in the ophthalmology department in the presence of the teacher of the department in order to develop and consolidate practical skills. The acquired skills are consolidated in the training room when examining patients or at a seminar. In the classroom, students independently study modern clinical protocols for examination and treatment, methodological recommendations of the ministry of health of the Republic of Belarus.

The control of the final level of knowledge is carried out at a seminar or in the clinical analysis of a patient, an outpatient card or a medical card.

1. Analysis of the thematic patient.

2. Clinical analysis of outpatient cards, medical records.

3. Answers to questions of a computer test program in ophthalmology on the topic.

QUESTIONS FOR SELF STUDY AND ADDITIONAL RESEARCH TASKS

1. Primary glaucoma. Classification of primary glaucoma.
2. Secondary glaucoma: Classification
3. Conservative treatment and surgical treatment of glaucoma.
4. Damage to the organ of vision. Frequency, classification. Treatment. Prevention
5. Injuries to the eyeball. Classification. Diagnostics. Urgent Care. Treatment.
6. Local and general complications of perforated wounds of the eyeball. Their prevention. Treatment.
7. Features of child, industrial, agricultural eye injuries. Preventive measures.
8. Removal of surface foreign bodies.
9. Sympathetic inflammation. Clinical forms. Diagnosis, treatment.
10. Damage to the orbit. Classification. Diagnostics. Treatment.
11. Dull eyeball damage. Severity. Urgent Care. Treatment.
12. Burns to the organ of vision. Classification. Urgent Care. Treatment. Prevention
13. Electrophthalmia. Diagnostics. Urgent Care. Prevention
14. Injuries of the eyelids, conjunctiva of the lacrimal organs. Diagnosis, treatment.

Glaucoma has been recognized as a clinical disease for 150years. Until recently, intraocular pressure (IOP) was considered to be the only pathogenetic factor, but its significance has now been qualified to some extent. The glaucomas are a diverse group of disorders that damage the optic nerve, resulting in characteristic optic nerve head cupping and visual field loss. The intraocular pressure (IOP) in such persons is presumed to be inconsistent with normal optic nerve function and is usually elevated above normal values. Nevertheless, IOP elevation need not be present to make the diagnosis of glaucoma. Patients with characteristic optic nerve cupping and visual field loss in the presence of normal IOP are diagnosed as having normal-pressure (low-pressure) glaucoma. Conversely, patients with elevated IOP in the absence of detectable secondary abnormalities in ocular structure and function are classified as ocular hypertensives.

Three steps in the development of glaucoma need to be taken into account: risk factors, the pathogenetic mechanism, and injury.

Three risk factors need to be taken into account:

- Intraocular pressure
- Vascular dysregulation
- Systemic blood pressure

In many other disease conditions, there is also another aspect that is important: cure. As is well known, however, glaucoma is not curable. Definition: The term “glaucoma” covers several diseases with differing etiologies that share the common finding of optic neuropathy with characteristic pathologic findings in the optic nerve head and a specific pattern of visual field defects. The disease is often, but not always, associated with increased intraocular pressure. The final stage of glaucoma is blindness.

- Primary glaucoma refers to glaucoma that is not caused by other ocular disorders.

- Secondary glaucoma may occur as the result of another ocular disorder or as an undesired side effect of medication or other treatment.

Epidemiology. Throughout the world, there are about 70million people suffering from glaucoma and 7million who have been blinded by the disease. Glaucoma is the second most frequent cause of blindness in developing countries after diabetes mellitus. Some 15–20% of blind persons have lost their eyesight as a result of glaucoma. In Germany, for example, approximately10% of the population over the age of 40 suffer from increased intraocular pressure. Approximately 10% of patients seen by ophthalmologists have glaucomas [5,6].

Physiology and pathophysiology of aqueous humor circulation. The average normal intraocular pressure of 15mmHg in adults is significantly higher than the average tissue pressure in almost every other organ in the body. This high pressure is important for optical imaging and helps to ensure:

- Uniformly smooth curvature of the surface of the cornea.
- Constant distance between the cornea, lens, and retina.
- Uniform alignment of the photoreceptors of the retina and the pigmented epithelium on Bruch's membrane, which is normally taut and smooth.

The aqueous humor is formed by the ciliary processes and secreted into the posterior chamber of the eye. At a rate of about 2–6μL/min and a total anterior and posterior chamber volume of about 0.2–0.4mL, about1–2% of the aqueous humor is replaced each minute.

The aqueous humor passes through the pupil into the anterior chamber. As their is lies flat along the anterior surface of the lens, the aqueous humor cannot overcome the pupillary resistance until sufficient pressure has built up to lift their is off the surface of the lens. The flow of the aqueous humor from the posterior chamber into the anterior chamber is therefore not continuous, but pulsatile.

Any increase in the resistance to papillary outflow (papillary block) leads to an increase in the pressure in the posterior chamber; the iris inflates anteriorly on its root like a sail and presses against the trabecular meshwork. This is the pathogenesis of angle closure glaucoma. Various factors can increase the resistance to pupillary outflow. The aqueous humor flows out of the angle of the anterior chamber through two channels:

- The trabecular meshwork receives about 85% of the outflow, which then drains into the canal of Schlemm. From here it is conducted by 20–30 radial collecting channels into the episcleral venous plexus.
- An uveoscleral vascular system receives about 15%of the outflow, which joins the venous blood.

The trabecular meshwork is the second source of physiologic resistance. The trabecular meshwork is a body of loose sponge-like avascular tissue between the scleral spur and Schwalbe's line. Increased resistance in present in open angle glaucoma.

Classification

The following is a useful classification of glaucoma, constructed with reference to the anatomy of the anterior chamber angle as well as to specific pathophysiologic and etiologic factors:

- Normal outflow
- Hypersecretion glaucoma

- Impaired outflow
- Congenital glaucoma (developmental)
- Primary glaucoma
- Angle-closure
- With pupillary block
- Without pupillary block
- Open-angle
- Secondary glaucoma
- Angle-closure
- With pupillary block
- Without pupillary block
- Open-angle
- Variable outflow
- Normal-pressure (low-pressure) glaucoma

Secondary angle-closure glaucoma

Secondary angle-closure glaucoma (SACG) can be subdivided in a manner similar to PACG as follows:

- SACG with pupillary block
- Untreated PACG
- Phacogenic
- Phacomorphic
- Secondary to lens subluxation or dislocation
- Posterior synechiae induced
- Inflammatory
- Aphakic or pseudophakic pupillary block
- Ciliary block (malignant glaucoma)
- SACG without pupillary block
- Secondary to sheetlike cellular or vascular proliferations in the anterior segment
- Neovascular glaucoma (rubeosis iridis) [8]
- ICE syndrome
- Epithelial downgrowth
- Stromal ingrowth
- Endothelialization of the anterior chamber angle
- Secondary to anterior displacement of anterior segment structures
- Postoperative failure of formation of the anterior chamber
- Tumor or cyst related
- Retinopathy of prematurity
- Persistent hyperplastic primary vitreous
- Miscellaneous
- Iridoschisis

SECONDARY OPEN-ANGLE GLAUCOMA

SOAG caused by cells or debris in the angle

- Hyphema
- Uveitis
- Pigmentary glaucoma
- Pseudoexfoliation
- Hemolytic and вЂњghost cellвЂќ glaucoma
- Phacolytic glaucoma
- Nondenatured lens material-induced glaucoma
- Melanomalytic or melanocytomalytic glaucoma
- Tumor seeding of the trabecular meshwork
- Schwartz-Matsuo syndrome
- SOAG caused by damaged outflow channels
- Previous uveitis
- Blunt trauma [9]
- Repeated hyphema
- Siderosis and hemosiderosis oculi [10]
- Repeated attacks of acute angle-closure glaucoma
- Early rubeosis or other anterior segment cellular proliferative disorder
- SOAG caused by corneoscleral and extraocular disease
- Interstitial keratitis
- Orbital venous thrombosis
- Encircling element after retinal disease
- Retrobulbar mass
- Leukemia
- Mediastinal mass
- SOAG secondary to miscellaneous causes
- Steroid-induced glaucoma
- Alpha-chymotrypsin glaucoma
- Glaucomatocyclitic crisis (Posner-Schlossman syndrome)
- Fuchs' heterochromic iridocyclitis

Examination Methods of glaukoma

Oblique Illumination of the Anterior Chamber

The anterior chamber is illuminated by abeam of light tangential to the plane of the iris. In eyes with an anterior chamber of normal depth, the iris is uniformly illuminated. This is a sign of a deep anterior chamber with an open angle.

In eyes with a shallow anterior chamber and an angle that is partially or completely closed, the iris protrudes anteriorly and is not uniformly illuminated.

Slit Lamp Examination

The central and peripheral depth of the anterior chamber should be evaluated on the basis of the thickness of the cornea. An anterior chamber that is less than three times as deep as the thickness of the cornea in the center with a peripheral depth less than the thickness of the cornea suggests a narrow angle. Other diagnostic signs during slit lamp examination may be noted for example, the presence of inflammatory cell deposits (keratic precipitates [KPs]) on the corneal epithelium, anterior chamber cells and flare.

Gonioscopy is essential for further evaluation.

To evaluate the depth of the anterior chamber with a slit lamp biomicroscope, a narrow setting for the light beam should be selected. The beam should strike the eye at a slight angle to the examiner's line of sight.

Anterior segment imaging devices that have recently become available (Visante OCT, Zeiss) provide a tomographic overview of the entire anterior chamber and its size.

Gonioscopy

The angle of the anterior chamber is evaluated with a gonioscope placed directly on the cornea.

Gonioscopy can differentiate the following conditions:

- Open angle: open angle glaucoma.
- Occluded angle: angle closure glaucoma.
- Angle access is narrowed: configuration with imminent risk angle of an acute closure glaucoma.
- Angle is occluded: secondary angle closure glaucoma, for example due to neovascularization in rubeosis iridis.
- Angle open but with inflammatory cellular deposits, erythrocytes, or pigment in the trabecular meshwork: secondary open angle glaucoma.

Gonioscopy is the examination of choice for identifying the respective presenting form of glaucoma.

Measuring Intraocular Pressure

Palpation: Comparative palpation of both eyeballs is a preliminary examination that can detect increased intraocular pressure.

- If the examiner can indent the eyeball, which fluctuates under palpation, pressure is less than20mmHg.
- An eyeball that is not resilient but rock hard is a sign of about 30–40mmHg of pressure (acute angle closure glaucoma).

Applanation tonometry. This method is the most common method of measuring intraocular pressure. It permits the examiner to obtain a measurement on a patient within a few seconds (Maclacovs method)

Pneumatic noncontact tonometry. The electronic tonometer directs a 3ms blast of air against the cornea. The tonometer records the deflection of the cornea and calculates the intraocular pressure on the basis of this deformation.

Advantages.

- Does not require the use of a topical anesthetic.
- Non contact measurement eliminates the risk of infection (can be used to measure intraocular pressure in the presence of conjunctivitis).

Disadvantages.

- Calibration is difficult.
- Precise measurements are possible only within low to middle range pressures.
- Cannot be used in the presence of corneal scarring.
- Examination is unpleasant for the patient.
- Air flow is loud.

Measuring the 24-hour pressure curve.

This examination is performed to analyze fluctuations of the pressure level over a 24-hour period in patients with suspected glaucoma. A single measurement may not be representative. Only a 24-hour curve provides reliable information about the pressure level.

Intraocular pressure fluctuates in a rhythmic pattern. The highest values frequently occur at night or in the early morning hours. In normal patients, these fluctuations in intraocular pressure rarely exceed 4–6mmHg. Pressure is measured on the ward at 6:00 a.m., noon, 6:00 p.m., 9:00p.m., and midnight. Out patient 24-hour pressure curves without nighttime and early morning measurements are less reliable

Sources of error in tonometry. Corneal thickness needs to be taken into account in applanation tonometry. Tonometric measurements are too low in patients with thin corneas and too high in those with thick corneas. The correction factor is about 2–3mmHg/50μm corneal thickness (given a regular corneal thickness of 539μm). This means that in an eye with a corneal thickness of 590μm, the real intraocular pressure is about 2–3mmHg lower than the measured value.

Primary Open Angle Glaucoma

Primary open-angle glaucoma (POAG) is a bilateral condition that may be asymmetric both in time of onset and in severity when each eye is compared. By definition, the filtration angle is open to gonioscopic examination. The diagnosis is to some extent one of exclusion because secondary causes must be ruled out before the diagnosis is made. The incidence of the disorder shows wide racial and ethnic variation. Its overall incidence is probably 1% to 3% and increases with increasing patient age: by age 75 years, 5% of the population is affected. It is documented to be a more severe problem among American blacks, of whom more than 11% are affected by age 80 years. Conversely, it is found less frequently than PACG in Inuits and Asians.

POAG is considered a genetically determined disorder. A positive family history for POAG is found in 50% of patients with POAG. The exact mode of inheritance is not definitely established for most glaucomas; however, some juvenile and adult open-angle glaucomas have been mapped to the long arm of chromosome 1.

Etiology. The nature of the obstruction to aqueous outflow in POAG is not known, although it is commonly believed to be located in the area of the juxtacanalicular connective tissue adjacent to Schlemm's canal. Unfortunately, few early cases of POAG have been examined histologically. One must be cautious in interpreting specimens from more advanced cases because changes may reflect secondary effects resulting from elevated IOP or from the medication used to treat it.

The relation of changes in the trabecular extracellular matrix (including acid mucopolysaccharides, glycoproteins, glycosaminoglycans, and collagen fibrils) to the pathophysiology of POAG remains to be determined. A significant decrease in the number of trabecular endothelial cells, when compared to age-matched controls, has been reported in patients with POAG. This finding can be viewed as an acceleration of the usual age-related decline in the number of trabecular endothelial cells.In the normal person, an age-related decrease in aqueous facility of outflow is accompanied by a parallel decrease in aqueous production. The net result is a quantitative balance between aqueous production and drainage. POAG can be viewed as an imbalance in the usual parallel relation of these aging changes. From this perspective, it is easy to speculate that POAG itself may really

represent a multifactoral problem with a final common pathway of elevated IOP in most patients.

Symptoms. The majority of patients with primary open angle glaucoma do not experience any subjective symptoms for years. However, a small number of patients experience occasional unspecific symptoms such as headache, a burning sensation in the eyes, or blur red or decreased vision that the patient may attribute to lack of eyeglasses or in sufficient correction. The patient may also perceive rings of color around light sources at night, which has traditionally been regarded as a symptom of angle closure glaucoma.

Primary open angle glaucoma often does not exhibit typical symptoms for years. Regular examination by an ophthalmologist is crucial for early diagnosis.

Primary open angle glaucoma can be far advanced before the patient notices an extensive visual field defect in one or both eyes.

It is crucial to diagnose the disorder as early as possible because the prognosis for glaucoma detected in its early stages is far better than for advanced glaucoma. Where increased intraocular pressure remains undiagnosed or untreated for years, glaucomatous optic nerve damage and the associated visual field defect will increase to the point of blindness.

Diagnostic considerations. Measurement of intraocular pressure. Elevated intraocular pressure in a routine ophthalmic examination is an alarming sign.

Twenty-four-hour pressure curve. Fluctuations in intraocular pressure of over 5–6 mm Hg may occur over a24-hour period.

Gonioscopy.The angle of the anterior chamber is open and appears as normal as the angle in patients without glaucoma.

Ophthalmoscopy. Examination of the optic nerve reveals whether glaucomatous cupping has already occurred and how far advanced the glaucoma is. Where the optic disc and visual field are normal, ophthalmoscopic examination of the posterior pole under green light may reveal fascicular nerve fiber defects as early abnormal findings.

Perimetry. Noise field perimetry is suitable as a screening test as it makes the patient aware of scotomas and makes it possible to detect and describe them.

The patient will not see the flickering points in the region of the scotoma. After this test, the defect should be quantified by more specific methods. Automatic grid perimetry is suitable for the early stages of glaucoma. Special programs(suchastheG1program on the Octopus perimeter and the 30–2 program on the Humphrey perimeter devices) reveal the earliest glaucomatous changes. In advanced glaucoma, kinetic hand perimetry with the Goldmann perimeter device is a useful preliminary examination to evaluate the remaining field of vision.

Differential diagnosis.

Two disorders are important in this context.

Ocular hypertension. Patients with ocular hypertension have significantly increased intraocular pressure over a period of years without signs of glaucomatous optic nerve damage or visual field defects. Some patients in this group will continue to have elevated intraocular pressure but will not develop glaucomatous lesions; the others will develop primary open angle glaucoma.

The probability that a patient will develop definitive glaucoma increases the higher the intraocular pressure, the younger the patient, and the more compelling the evidence of a history of glaucoma in the family.

Low-tension glaucoma. Patients with low-tension glaucoma exhibit typical progressive glaucomatous changes in the optic disc and visual field without elevated intraocular pressure. These patients are very difficult to treat because management cannot focus on the control of intraocular pressure. Often these patients will have a history of hemodynamic crises such as gastrointestinal or uterine bleeding with significant loss of blood, low blood pressure, and peripheral vascular spasms (cold hands and feet).A continuous 24-hour blood pressure profile often shows phases of low blood pressure during the night that would other wise be undetected. These time intervals of low perfusion lead to optic neuropathy. Patients with glaucoma may also experience further worsening of the visual field due to a drop in blood pressure. Caution should be exercised when using cardiovascular and antihypertensive drugs in patients with glaucoma [7].

Principles of Therapy

In all cases of glaucoma it is essential to establish whether the glaucoma is open or closed angle. This is accomplished by placing a gonioscopic contact lens on the eye and actually viewing the angle structures.

Indications for initiating treatment.

- Glaucomatous changes in the optic cup. Medical treatment should be initiated where there are signs of glaucomatous changes in the optic cup or when there is a difference of more than 20% between the optic cups of the two eyes.

- Any intraocular pressure exceeding 25mmHg should be treated.

- Increasing glaucomatous changes in the optic cup or increasing visual field defects inspite of antiglaucomatous medication. Regard less of the pressure measured, these changes show that the current pressure level is too high for the optic nerve and that additional medical therapy is indicated. A target IOP should be defined about 30%under the initial level. In addition to reducing the pressure, minimization of the daily pressure fluctuations is attempted. This also applies to patients with advanced glaucomatous damage and threshold pressure levels (around 22mmHg). The strongest possible medications are indicated in these cases, to lower pressure as much as possible (10–12mmHg).

- Early stages. It is often difficult to determine whether therapy is indicated in the early stages, especially where intraocular pressure is elevated slightly above threshold values. Patients with low-tension glaucoma exhibit increasing cupping of the optical disc even at normal pressures (less than 22mmHg), where as patients with elevated intraocular pressure (25–33 mm Hg) may exhibit an unchanged optic nerve for years.

Patients with suspected glaucoma and risk factors such as a family history of the disorder, middle myopia, glaucoma in the other eye, or differences between the optic cup in the two eyes should be monitored closely. Follow-up examinations should be performed three to four times a year, especially for patients not undergoing treatment.

Therapeutic goals. The aimis to reduce the IOP to the level of the target pressure. The target pressure needs to be determined individually for each patient as a specific IOP level low enough to prevent the progression of visual field loss and glaucomatous optic nerve

damage. The definition of the target pressure may be a continuing process that takes several office visits.

Medical therapy. Available options in medical treatment of glaucoma

- Inhibit aqueous humor production
- Increase trabecular outflow
- Increase uveoscleral outflow

Hypotensive drugs

A .Beta-adrenergic blockers (timolol, betaxolol, carteolol, levobunolol, metipranolol). Mechanism of action. Timolol, levobunolol, carteolol, and metipranolol are nonselective $beta_1$- (cardiac) and $beta_2$- (smooth muscle, pulmonary) receptor blocking agents. Betaxolol has 100 times more affinity for $beta_1$- than $beta_2$-receptors.

Physiologic effects. The nonselective drugs decrease IOP by blockade of $beta_2$-receptors in the ciliary processes, resulting in decreased aqueous production. The mechanism for betaxolol is unknown because there are so few $beta_1$-receptors in the eye, but there may be вЂњspill overвЂќ to bind $beta_2$-receptors as well. There is no effect on facility of outflow. The drug molecule timolol (and probably betaxolol and levobunolol) releases from the beta-receptor site as early as 3 hours after topical administration, yet clinical effect may last up to 2 weeks. This prolonged effect may result from re-release of beta-blocker from depots in the iris pigment epithelial melanin. Carteolol, unlike the other beta-blockers, has intrinsic sympathomimetic activity, possibly resulting in fewer side effects. It also lacks timolol's tendency to increase serum cholesterol and decrease high-density lipoproteins, a factor to consider in cardiovascular patients.

Indications are primary and secondary open-angle glaucomas, including inflammatory glaucomas, acute and chronic primary and secondary angle-closure glaucomas, ocular hypertension, and childhood glaucomas.

Precautions and contraindications include known drug allergy. These drugs should be used with caution or not at all, depending on severity of disease, in patients with asthma, emphysema, chronic obstructive pulmonary disease, bronchitis, heart block, congestive heart failure, cardiovascular disease, or cardiomyopathy. Although betaxolol is the blocker of choice in patients at risk for pulmonary reaction because of its greater $beta_1$ (cardiac) selectivity, the drug may induce bronchospasm in some patients.

Available preparations. Timolol (Timoptic, Betimol), 0.25% to 0.50%; betaxolol (Betoptic-S) and levobunolol (Betagan), 0.25% to 0.50%; metipranolol (OptiPranolol), 0.3%; carteolol (Ocupress), 1% eye drops. Once-a-day dosing with drops or a gel formulation of timolol is also available as Timoptic XE 0.25% or 0.50% and Istalol 0.50%.

Recommended dosage is qd or q12h topically. All beta-blockers may be used with significant additive effect in combination with miotic agents, $alpha_2$-agonists, prostaglandin analogs, or carbonic anhydrase inhibitors (CAIs).

Side effects Bradycardia, cardiac arrest, acute asthma, and pulmonary edema have all been reported in susceptible individuals and result from systemic absorption of topical drug. Lacrimal canalicular compression should be practiced by patients at any risk, and the drug used with caution or not at all in those patients with moderate to severe cardiac or pulmonary disease.

Full adult dosage should be avoided in children because apnea may result; 0.25% qd to bid with canalicular compression is the advisable dosage.

B. Prostaglandin analogs (latanoprost, bimatoprost, travoprost, and unoprostone)

Mechanism of action. Lowering of IOP by increasing uveoscleral outflow through a prostaglandin F_2 O±-mediated mechanism.

Indications. First-line or additive therapy for lowering of high IOP. Preparation and dosage. Latanoprost (Xalatan) 0.005%, bimatoprost (Lumigan) 0.03%, and travoprost (Travatan) 0.004% are each given once daily, usually at bedtime. Unoprostone (Rescula) 0.15% is given twice daily. Latanoprost is temperature sensitive and needs to be refrigerated prior to and after opening.

Side effects include increased iris pigmentation, darkening of the eyelid skin, increased thickness and number of eyelashes, ocular irritation (redness, itching, etc.), uveitis, cystoid macular edema, and probable reactivation of herpes simplex virus.

C. Carbonic anhydrase inhibitors (CAIs). Acetazolamide and methazolamide are oral agents. Acetazolamide is also available as an intravenous (i.v.) agent. Topical CAIs are dorzolamide and brinzolamide.

Mechanism of action. CAIs inhibit the enzyme carbonic anhydrase.

Physiologic effects. The ciliary body enzyme, carbonic anhydrase, is related to the process of aqueous humor formation, most likely via active secretion of bicarbonate. CAIs decrease the rate of aqueous humor formation.

Indications. CAIs are additive therapy in the management of various acute glaucomas as well as in the chronic management of primary and secondary open-angle and angle-closure glaucomas not adequately controlled by topical medication.

Contraindications. Because of the metabolic and possible respiratory acidosis effects, patients with significant respiratory disease should be given oral CAIs cautiously and in lower dosages. Patients with a history of calcium phosphate kidney stone formation should be given the oral medication cautiously and only after consultation with their primary care provider. Known allergy is a contraindication. Patients with allergies to other sulfonamides should be given these agents with caution.

Available preparations include acetazolamide (Diamox, generic) 125 mg and 250 mg tablets, 500 mg sequels, 500 mg per 5 mL i.v.; methazolamide tablets (Neptazane, GlaucTabs, MZM, generic) 25 mg and 50 mg tablets; dorzolamide (Trusopt) 2% drops; and brinzolamide (Azopt) 1% drops.

Recommended dosage. Established dosages for near-maximum effect are acetazolamide tablets 250 mg q6h; methazolamide tablets 50 to 100 mg bid to tid; acetazolamide sustained-release capsules 500 mg q12h. Because acetazolamide is excreted unchanged by the kidneys, patients with renal disease such as diabetic nephropathy should be started on lower than standard dosages. Methazolamide may be used more safely in this situation. Dorzolamide 2% drops and brinzolamide 1% tid decrease IOP by about 20%.

Side effects. Unfortunately, 40% to 50% of glaucoma patients are unable to tolerate systemic CAIs long term because of various disabling side effects. A symptom complex of malaise, fatigue, depression, anorexia, and weight loss is the most frequent side effect. Loss of libido, especially in young males, may also occur. These symptoms show some correlation with the degree of systemic metabolic acidosis on therapy. They may have a gradual, insidious onset over several months. Often neither the patient nor the physician relates these symptoms to the systemic CAI therapy. Frequently, patients erroneously undergo extensive medical evaluations searching for occult malignancies.

D.Miotics Pilocarpine. Mechanism of action. Pilocarpine is a direct-acting parasympathomimetic (muscarinic) cholinergic drug.

Physiologic effects. The drug is used in chronic open-angle glaucoma to increase the facility of aqueous outflow. The mechanism of action is probably exclusively mechanical, via ciliary muscle contraction and pull on the scleral spur and TM. It is used in acute angle-closure glaucoma to move the iris away from the angle. Miosis is a side effect and is of no therapeutic benefit.

Indications are chronic open-angle glaucoma, acute angle-closure glaucoma, chronic synechial angle-closure glaucoma (following peripheral iridectomy), and following cyclodialysis surgery.

Contraindications are inflammatory glaucoma, malignant glaucoma, or known allergy.

Available preparations are pilocarpine 0.5% to 8% eye drops (Isopto Carpine, Pilocar, Piloptic), and 4% gel (Pilopine-HS gel).

Recommended dosage

Eye drops. Except in very darkly pigmented irides, maximum effect is probably obtained with a 4% solution. In milder open-angle glaucoma, therapy is usually initiated with a 1% concentration. Duration of effect is 4 to 6 hours. It is usually prescribed for use every 6 hours.

Combination. Pilocarpine can be used in conjunction with other glaucoma medications and, in most instances, confers additional pressure-lowering effects.

E.Hyperosmotic agents (mannitol, glycerin)

Mechanism of action. Reduction of IOP by increasing plasma tonicity sufficiently to draw water out of the eye.

Indications. Additive therapy for rapid reduction of high IOP. Onset of action is 30 minutes and lasts 4 to 6 hours. Side effects may include severe systemic hypertension aggravation, nausea, vomiting, confusion, congestive heart failure, pulmonary edema, or diabetic hyperglycemia (mannitol). The drugs are contraindicated in oliguria or anuria.

Principles of medical treatment of primary open angle glaucoma:

Medical therapy is the treatment of choice for primary open angle glaucoma.

Surgery is indicated only where medical therapy fails.

- There is no single generally applicable therapy plan. The choice of medication depends on the efficacy, side effects and contraindications of different drugs. At first, a single drug treatment should be targeted with an IOP-reducing effect of at least 20%of the untreated level.

Surgical treatment of primary open angle glaucoma.

Indications:

- Medical therapy is inadequate.

- The patient does not tolerate medical therapy. Reactions include allergy, reduced vision due to narrowing of the pupil, pain, and ciliary spasms, and ptosis.

- The patient is not a suitable candidate for medical therapy due to a lack of compliance or a lack of dexterity in applying eye drops.

1. Argon laser trabeculoplasty:

- Principle. Laser burns in the trabecular meshwork cause tissue contraction that widens the intervening spaces and improves outflow through the trabecular meshwork.

- Technique. focal laser burns are placed in the anterior trabecular meshwork.

- Comment. Laser surgery in the angle of the anterior chamber is possible only if the angle is open. The surgery itself is largely painless, can be performed as an outpatient procedure, and involves few possible complications. These may include bleeding from vascular structures near the angle and synechiae between the iris and individual laser burns. Argon laser trabeculoplasty can bring improvement with intraocular pressures up to30mmHg. It decreases intraocular pressure by about 6–8 mm Hg for about 2years.Argonlaser trabeculoplasty is only effective in about every second patient. The full effect occurs about 4–6weeks postoperatively.

2.Filtration surgery:

Principle. The aqueous humor is drained through the anterior chamber through a subconjunctival scleral opening, circumventing the trabecular meshwork. Formation of at hinwalled filtration bleb is a sign of sufficient drainage of aqueous humor.

Technique. First, a conjunctival flap is raised, which may be either fornix-basedor limbal-based.Then a partial-thicknesss cleral flap is raised. The scleral flap is then loosely closed and covered with conjunctiva. The short time application of an antiproliferative substance (mitomycinC) inhibits wound healing postoperatively and keeps the filtration way open. Comment. A permanent reduction in intraocular pressure is achieved in 80–85%of these operations.

3.Cyclodialysis:

Principle. The aqueous humor is drained through an opening into the suprachoroidal space.

Technique. A full-thickness scleral incision is made down to the ciliary body 4 mm posterior to the limbus. The sclera is then separated from the ciliary body with a retractor and retracted anteriorly into the anterior chamber. The ciliary body atrophies in the area of the incision, which also helps to decrease the production of aqueous humor.

Comment. This procedure is less common today than it was in the1980s. One reason for this is that it is difficult to gauge accurately the atrophy to the ciliary body. Occasionally severe hypotonia of the globe results, which then requires surgical intervention to close the dialysis opening.

4.Cycloablation (cyclodestructiveprocedures):

Principle. Atrophy is induced in portions of the ciliary body through the intact sclera to reduce intraocular pressure by decreasing the amount of tissue producing aqueous humor.

Technique

– Cyclocryotherapy. A cryoprobe is used to freeze the ciliary body at several points through the sclera. This procedure can be repeated if necessary; the interventions have a cumulative effect.

– Cyclodiathermy. This method is similar to cyclocryotherapy, except that a diathermy needle is advanced through the sclera into the ciliary body to cauterize it with heat. The procedure can be performed with or without prior dissection of apartial-thickness scleral flap.

– Lasercycloablation induces atrophy in the ciliary body usinga YAG laser orhigh-energy diode laser pulses.

– Ultrasound disruption induces atrophy in the ciliary body with high-frequency ultrasound waves. These last two forms of therapy have been developed to induce atrophy

more effectively, more accurately, and in more controlled doses, which is less traumatic for the eye.

Comment. All these forms of cycloablation are irreversible and cause permanent hypotonia. They therefore represent the last line of treatment options.

Primary Angle Closure Glaucoma

Definition: Acute episodic increase in intraocular pressure to several times the normal value (10–20mmHg) due to sudden blockage of drainage. Production of aqueous humor and trabecular resistance are normal.

Epidemiology. The incidence among persons over the age of 60 is one per thousand. Women are three times as likely to be affected as men. Inuit are more frequently affected than other ethnic groups, whereas the disorder is rare in blacks.

Etiology

Anatomically predisposed eyes with shallow anterior chambers pose a relative impediment to the flow of aqueous humor through the pupil. This papillary block increases the pressure in the posterior chamber. The pressure displaces the iris anteriorly toward the trabecular meshwork, suddenly blocking the outflow of aqueous humor (angle closure).A typical glaucoma attack occurs unilaterally due to widening of the pupil either in dark surroundings and/or under emotional stress (dismay or fear). A typical situation is the evening mystery movie on television.

Iatrogenic pharmacologic mydriasis and systemic psychotropic drugs can also trigger aglaucoma attack. It should be borne in mind that mydriatic agents entail a risk of triggering a glaucoma attack by widening the pupil. It is therefore important to evaluate the depth of the anterior chamber in every patient even before a routine fundus examination.

Symptoms. Acute onset of intense pain. The elevated intraocular pressure acts on the corneal nerves (the ophthalmic nerve or first branchof the trigeminal nerve) to cause dull pain. This pain may be referred to the temples, back of the head, and jaws via the three branches of the trigeminal nerve, which can mask its ocular origin. Nausea and vomiting occur due to irritation of the vagus nerve and can simulate abdominal disorders. The generalized symptoms such as headache, vomiting, and nausea may dominate to the extent that the patient fails to notice local symptoms.

Diminished visual acuity. Patients notice obscured vision and colored halos around lights in the affected eye. These symptoms are caused by the corneal epithelial edema precipitated by the enormous increase in pressure.

Prodromal symptoms. Patients report transitory episodes of blurred vision or the appearance of colored halos around lights prior to the attack. These prodromal symptoms may goun noticed or may be dismissed as unimportant by the patient in mild episodes where the eye returns to normal. Early identification of high-risk patients with shallow anterior chambers and gonioscopic findings is important, as damage to the structures of the angle may be well advanced before clinical symptoms appear.

The full clinical syndrome of acute glaucoma is not always present. The diminished visual acuity may goun noticed if the other eye has normal vision.

Patients' subjective perception of pain intensity can vary greatly.

Diagnostic considerations:

The diagnosis is made on the basis of a triad of symptoms:

- Unilateral red eye with conjunctival or ciliary injection.
- Fixed and dilated pupil.
- Hard eyeball on palpation.

Other findings.

- The cornea is dull and steamy with epithelial edema.
- The anterior chamber is shallow or completely collapsed. This will be apparent when the eye is illuminated by a focused lateral light source and on slit lamp examination. Inspection of the shallow anterior chamber will be difficult. Details of the surface of the iris will be visible, and the iris will appear faded.
- The fundus is generally obscured due to opacification of the corneal epithelium. When the fundus can be visualized as symptoms subside and the cornea clears, the spectrum of changes to the optic disc will range from a normal vital optic disc to anill-defined hyperemic optic nerve. In the latter case, venous congestion will be present. The central artery of the retina will be seen to pulsate on the optic disc as blood can only enter the eye during the systolic phase due to the high intraocular pressure.
- Visual acuity is reduced to perception of hand motions.

Differential diagnosis. Misdiagnosis is possible, as the wide variety of symptoms can simulate other disorders.

- General symptoms such as headache, vomiting, and nausea often predominate and can easily be mistaken for appendicitis or a brain tumor.
- In iritis and iridocyclitis, the eye is also red and the iris appears faded. However, intraocular pressure tends to be decreased rather than elevated.

Treatment.

An acute glaucoma attack is an emergency, and the patient requires immediate treatment by an ophthalmologist. The underlying causes of the disorder require surgical treatment, although initial therapy is conservative.

Medical therapy. The goals of conservative therapy are:

- Decrease intraocular pressure.
- Allow the cornea to clear (important for subsequent surgery).
- Relieve pain.

Principles of medical therapy in primary angle closure glaucoma.

- Osmotic reduction in the volume of the vitreous body is achieved via systemic hyperosmotic solutions (oral glycerin, 1.0–1.5g/kg of body weight, or intravenous mannitol, 1.0–2.0g/kg of body weight).
- Production of aqueous humor is decreased by inhibiting carbonic anhydrase (intravenous acetazolamide, 250–500mg). Both steps are taken initially to reduce intraocular pressure to below 50–60 mm Hg. The iris is with drawn from the angle of the anterior chamber by administering topical miotic agents. Pilocarpine 1% eye drops should be applied every 15minutes. If this is not effective, pilocarpine can be applied more often, every 5minutes, and in concentrations up to4%.Miotic agents are not the medications of first choice because the sphincter pupillae muscle is ischemic at pressures exceeding 40–50mmHg and will not respond to miotic agents. Miotic agents also relax the zonule fibers, which causes anterior displacement of the lens that further compresses the anterior

chamber. This makes it important to first initiate therapy with hyperosmotic agents to reduce the volume of the vitreous body.

- Symptomatic therapy with analgesic agents, antiemetic agents, and sedatives can be initiated where necessary.

Surgical management (shunt between the posterior and anterior chambers).

Once the cornea is clear, the underlying causes of the disorder are treated surgically by creating a shunt between the posterior and anterior chambers.

Neodymium: yttrium–aluminum–garnet laser iridotomy (nonincisional procedure): The Nd: YAG laser can be used to create an opening in the peripheral iris (iridotomy) by tissue lysiswithout having to open the globe.

The operation can be carried out with topical anesthesia.

Peripheral iridectomy (incisional procedure).Where the cornea is still swollen with edema or the iris is very thick, an open procedure may be required to create a shunt. A limbal incision is made at the 12-o'clock position with the patient under topical anesthesia or general anesthesia, through which a basal iridectomy is performed.

Secondary Open Angle Glaucoma

Definition: The anatomic relationships between the root of the iris, the trabecular meshwork, and peripheral cornea are not disturbed. However, the trabecular meshwork is congested and the resistance to drainage is increased.

The most important forms.

Pseudoexfoliative glaucoma. Deposits of amorphous acellular material form through out the anterior chamber and congest the trabecular meshwork.

Pigmentary glaucoma. Young myopic men typically are affected. The disorder is characterized by release of pigment granules from the pigmentary epithelium of the iris that congest the trabecular meshwork.

Cortisone glaucoma. Some35–40%of the population react to3-weektopical or systemic steroid therapy with elevated intraocular pressure. Increased deposits of mucopolysaccharides in the trabecular meshwork presumably increase resistance to outflow; this is reversible when the steroids are discontinued.

Inflammatory glaucoma. Two mechanisms contribute to the increase in intraocular pressure:

1.The viscosity of the aqueous humor increases as a result of the influx of protein from inflamed iris vessels.

2.The trabecular meshwork becomes congested with inflammatory cells and cellular debris.

Phacolytic glaucoma.This is acute glaucoma in eyes with mature or hypermature cataracts. Denatured lens protein passes through the intact lens capsule into the anterior chamber and is phagocytized. The trabecular meshwork becomes congested with protein binding macrophages and the protein itself.

SecondaryAngleClosureGlaucoma

Definition: In secondary angle closure glaucoma as in primary angle closure glaucoma, the increase in intraocular pressure is due to blockage of the trabecular meshwork. However, the primary configuration of the anterior chamber is not the decisive factor.

The most important causes.

Rubeosis iridis. Neovascularization draws the angle of the anterior chamber together like a zipper (neovascular glaucoma). Ischemic retinal disorders such as diabetic retinopathy and retinal vein occlusion can lead to rubeosis iridis with progressive closure of the angle of the anterior chamber. Other forms of retinopathy or intraocular tumors can also cause rubeosis iridis. The prognosis for eyes with neovascular glaucoma is poor.

Trauma.Post-traumatic presence of blood or exudates in the angle of the anterior chamber and prolonged contact between the iris and trabecular meshwork in a collapsed anterior chamber (following injury, surgery, or insufficient treatment of primary angle closure) can lead to anterior synechiae and angle closure without rubeosis iridis.

Treatment of secondary glaucomas.

Medical therapy of secondary glaucomas is usually identical to the treatment of primary chronic open angle glaucoma.

Ocular Trauma

Eye injuries occurmore often in combination with other injuries (in cases of polytrauma) than in isolation. Life-threatening injuries should always be treated before ophthalmological treatment is started [5].

Definition: Life takes priority over vision. Eye injuries should be treated in patients who have been fully examined and stabilized.

Examination Methods

The incidence of ocular injuries is still high, despite improved safety regulations in recent years, such as the mandatory use of seat belts and protective eyewear for persons operating high-speed rotary machinery. It is therefore important that every general practitioner and member of healthcare staff should be able to recognize an ocular injury and provide initial treatment. The patient should then be referred to an ophthalmologist, who should be solely responsible for evaluation of the injury and definitive treatment. The following diagnostic options are available to determine the nature of the injury more precisely:

Patient history. Obtaining a thorough history will provide important information about the cause of the injury.

- Work with a hammer and chisel nearly always suggests an intraocular foreign body.
- Cutting and grinding work suggests corneal foreign bodies.
- Welding and flame-cutting work suggests ultraviolet keratoconjunctivitis.

The examiner should always ascertain whether the patient has adequate tetanus immunization.

Inspection (gross morphologic examination).Ocular injuries frequently cause pain, photophobia, and blepharospasm. A few drops of topical anesthetic are recommended to allow the injured eye to be examined at rest with minimal pain to the patient. The cornea and conjunctiva are then examined for signs of trauma using a focused light, preferably one combined with a magnifying loupe. The eyelids can be everted to inspect the tarsal surface and conjunctival fornix. A foreign body can then be removed immediately.

Ophthalmoscopy. Examination with a focused light or ophthalmoscope will allow gross evaluation of deeper intraocular structures, such as whether a vitreous or retinal hemorrhage is present. Vitreous hemorrhage can be identified by the lack of red reflex on retroillumination. Care should be taken to avoid unnecessary manipulation of the eye in an obviously severe open-globe injury (characterized by a soft globe, pupil displaced toward

the penetration site, prolapsed iris, and intraocular bleeding in the anterior chamber and vitreous body). Such manipulation might otherwise cause further damage, such as extrusion of intraocular contents.

To properly estimate the urgency of treating palpebral and ocular trauma, it is particularly important to differentiate between open-globe injuries and closed-globe injuries. Open-globe injuries have highest priority due to the risk of losing the eye.

Classification of Ocular Injuries by Mechanism of Injury

- Mechanical injuries.

– Eyelid injuries.
– Injuries to the lacrimal system.
– Conjunctival laceration.
– Foreign body in the cornea and conjunctiva.
– Corneal erosion.
– Non penetrating injury (blunt trauma to the globe).
– Injury to the floor of the orbit (blow-out fracture).
– Penetrating injury (open-globe injury).
– Impalement injury to the orbit.

- Chemical injuries [6].
- Injuries due to physical agents.

– Burns [7].
– Radiation injuries (ionizing radiation).
– Ultraviolet keratoconjunctivitis.

- Indirect ocular trauma. Transient traumatic retinal angiopathy (Purtscher retinopathy).

Mechanical Injuries

Eyelid Injury

Etiology. Eyelid injuries can occur in practically every facial injury. The following types warrant special mention:

- Eyelid lacerations with involvement of the eyelid margin.

- Avulsions of the eyelid in the medial canthus with avulsion of the lacrimal canaliculus.

Clinical picture. The highly vascularized and loosely textured tissue of the eyelids causes them to bleed profusely when injured. Hematoma and swelling will be severe. Abrasions usually involve only the superficial layers of the skin, whereas punctures, cuts, and all eyelid avulsions due to blunt trauma (such as a fist) frequently involve all layers. Bite wounds (such as dog bites) are often accompanied by injuries to the lacrimal system.

Treatment. Surgical repair of eyelid injuries, especially lacerations with involvement of the eyelid margin, should be performed with care. The wound should be closed in layers and the edges properly approximated to ensure a smooth margin without tension to avoid later complications, such as cicatricial ectropion. Lid swellings are best treated by wool pads or ice compresses.

Injuries to the Lacrimal System

Etiology. Lacerations and tears in the medial canthus (such as dog bites or glass splinters) can divide the lacrimal duct. Obliteration of the punctum and lacrimal

canaliculus is usually the result of a burn or chemical injury. Injury to the lacrimal sac or lacrimal gland usually occurs in conjunction with severe craniofacial trauma (such as a kick from a horse or a trafficac cident). Dacryocystitis is a common sequela, which often can only be treated by surgery (dacryocystorhinostomy).

Treatment. Lacrimal system injuries are repaired under an operating microscope. A ring-shaped silicone stent is advanced into the canaliculus using a special probe. The silicone stent remains in situ for 3–4 months and is then removed.

Surgical repair of eyelid and lacrimal system injuries must be performed by an ophthalmologist.

Conjunctival Laceration

Epidemiology. Due to its exposed position, thinness, and mobility, the conjunctiva is susceptible to lacerations, which are usually associated with subconjunctival hemorrhage.

Etiology. Conjunctival lacerations most commonly occur as a result of penetrating wounds (such as from bending over a spiked-leaf palm tree or from a branch that snaps back onto the eye).

Symptoms and diagnostic considerations. The patient experiences a foreign body sensation. Usually this will be rather mild. Examination will reveal circumscribed conjunctival reddening or subconjunctival hemorrhage in the injured area. Occasionally only application of fluorescein dye to the injury will reveal the size of the conjunctival gap.

Treatment. Minor conjunctival injuries do not require treatment as the conjunctiva heals quickly. Larger lacerations with mobile edges are approximated with absorbable sutures.

Corneal and Conjunctival Foreign Bodies

Epidemiology. Foreign bodies on the cornea and conjunctiva are the commonest ocular emergency encountered by general practitioners and ophthalmologists.

Etiology. Airborne foreign bodies and metal splinters from grinding or cutting discs in particular often become lodged in the conjunctiva or cornea or burn their way into the tissue.

Symptoms and diagnostic considerations. The patient experiences a foreign-body sensation with every blink of the eye. This is accompanied by epiphora (tearing) and blepharospasm. Depending on the time elapsed since the injury—i.e., after a few hours or several days—conjunctival or ciliary injection will be present. The foreign bodies on the conjunctiva or cornea are themselves often so small that they are visible only under loupe magnification. There may be visible in filtration or a ring of rust. Where there is no visible foreign body but fluorescein dye reveals vertical corneal striations, the foreign body will be beneath the tarsus.

A foreign-body sensation with every blink of the eye accompanied by epiphora, blepharospasm, and vertical striations on the surface of the cornea are typical signs of a subtarsal foreign body.

Treatment. Corneal and conjunctival foreign bodies. The foreign body is prised out of its bed with a fine needle or cannula. The defect created by the foreign body will often be contaminated with rust or infiltrated with leukocytes. This defect is carefully reamed out with a drill and treated with an antibiotic eye ointment and bandaged if necessary.

Subtarsal foreign bodies. Everting the upper and lower eyelids will usually reveal the foreign body, which may then be removed with a moist cotton swab. An antibiotic eye bandage is placed until the patient is completely free of symptoms.

Corneal Erosion

Etiology. This disorder follows initial trauma to the surface cornea, such as the fingernail of a child carried in the parent's arms, a spiked-leaf palm tree, or a branch that snaps back onto the eye. Properly treated, this epithelial defect usually heals within a short time—i.e., 24–48 hours depending on the size of the defect. However, occasionally the epithelial cells do not properly adhere to Bowman's layer so that the epithelium repeatedly ruptures at the site of the initial injury. This characteristically occurs in the morning when the patient wakes up and suddenly opens his or her eyes. This recurring erosion often creates severee motional stress for the patient.

Symptoms and diagnostic considerations. Immediately after the injury, the patient experiences a severe foreign-body sensation associated with tearing. Because there is actually a defect in the surface of the cornea, the patient has the subjective sensation of a foreign body within the eye. The epithelial defect causes severe pain, which immediately elicits a blepharospasm. Additional symptoms associated with corneal erosion include immediate eyelid swelling and conjunctival injection. Fluorescein sodium dye will readily reveal the corneal defect when the eye is examined through a blue light.

Treatment. An antibiotic ointment eye bandage is used. Treatment of recurrent corneal erosion often requires hospitalization. Bilateral bandages are placed to ensure that the eyes are completely immobilized.

Blunt Ocular Trauma (Ocular Contusion)

Epidemiology and etiology. Ocular contusions resulting from blunt trauma such as a fist, ball, champagne cork, stone, falling on the eye, or a cow's horn are very common. Significant deformation of the globe can result where the diameter of the blunt object is less than that of the bony structures of the orbit.

Clinical picture and diagnostic considerations. Deformation exerts significant traction on intraocular structures and can cause them to tear. Often there will be blood in the anterior chamber, which will initially prevent the examiner from evaluating the more posterior intraocular structures.

Do not administer medications that act on the pupilas there is a risk of irreversible mydriasis from a sphincter tear, and papillary movements increase the risk of subsequent bleeding. The posterior intraocular structures should only be thoroughly examined in mydriasis to determine the extent of injury after a week to 10 days.

Late sequelae of blunt ocular trauma include:

- Secondary glaucoma [13].
- Retinal detachment.
- Cataract.

Late sequelae of blunt ocular trauma may occur years after the injury.

Treatment. This involves immobilizing the eye initially, to allow intraocular blood to settle.Subsequent bleeding 3–4 days after the injury is common.

Blow-Out Fracture

Etiology. Blow-out fractures of the orbit result from blunt trauma. Blunt objects of small diameter, such as a fist, tennis ball, or baseball, can compress the contents of the

orbit so severely that the orbital wall fractures. This fracture usually occurs where the bone is thinnest, along the paper-thin floor of the orbit over the maxillary sinus. The ring-shaped bony orbital rim usually remains intact. The fracture can result in protrusion and impingement of orbital fat and the inferior rectus and its sheaths in the fracture gap. Where the medial ethmoid wall fractures instead of the orbital floor, emphysema in the eyelids will result.

Symptoms and diagnostic considerations. The more severe the contusion, the more severe the intraocular injuries and resulting visual impairment will be. Impingement of the inferior rectus can result in diplopia, especially in upward gaze. Initially, the diplopia may go unnoticed when the eye is still swollen shut. A large bone defect may result in displacement of larger portions of the contents of the orbital cavity. The eye may recede into the orbit (enophthalmos) and the palpebral fissure may narrow. Injury to the infraorbital nerve, which runs along the floor of the orbit, may result. This can cause hypesthesia of the facial skin. Crepitus upon palpation during examination of the eyelids welling is a sign of emphysema due to collapse of the ethmoidal air cells. The crepitus is caused by air entering the orbit from the paranasal sinuses. The patient should refrain from blowing his or her nose for the next 4–5days to avoid forcing air or germs into the orbit. Radiographs should be obtained and a near, nose, and throat specialist consulted to help determine the exact location of the fracture. CT studies are more precise and may be indicated to evaluate difficult cases. Tissue displaced into the maxillary sinus will resemble a hanging drop of water in the CT image.

Treatment. Surgery to restore normal anatomy and the integrity of the orbit should be performed within 10days. This minimizes the risk of irreversible damage from scarring of the impinged inferior rectus. Where treatment is prompt, the prognosis is good. Tetanus prophylaxis and treatment with antibiotics are crucial.

Open-Globe Injuries

Etiology. Together with severe chemical injuries, open-globe injuries are the most devastating forms of ocular trauma. They are caused by sharp objects that penetrate the cornea and sclera. A distinction is made between penetration with and without an intraocular foreign body. However, even blunt trauma can cause an open-globe injury in an eye weakened by previous surgery or injury where extremely high-energy forces are involved (such as falling on a cane or a blow from a cow’s horn) [14].

Clinical picture and diagnostic considerations. Penetrating injuries cover the entire spectrum of clinical syndromes. Symptoms can range from massive penetration of the cornea and sclera with loss of the anterior chamber to tiny, nearly invisible injuries that close spontaneously. The latter may include a fine penetrating wound or the entry wound of a foreign body.

Depending on the severity of the injury, the patient’s visual acuity may be severely compromised or not influenced at all. One of the most common sequelae is a traumatic cataract. The rupture in the lens capsule allows aqueous humor to penetrate, causing the lens to swell. This results in lens opacification of varying severity. Large defects will lead to total opacification of the lens within hours or a few days. Smaller defects that close spontaneously often cause a circumscribed opacity. Typically, penetration results in a rosette-shaped anterior or posterior subcapsular opacity.

Depending on the severity of the injury, the following diagnostic signs will be present in an open-globe injury:

- The anterior chamber will be shallow or absent.
- The pupil will be displaced toward the penetration site.
- Swelling of the lens will be present (traumatic cataract).
- There will be bleeding in the anterior chamber and vitreous body.
- Hypotonia of the globe will be present.

The rupture of the lens capsule and vitreous hemorrhage often rend erexamination difficult as they prevent direct inspection. These cases, and any patient whose history suggests an intraocular foreign body, require one or both of the following diagnostic imaging studies:

- Radiographs in two planes to determine whether there is a foreign body in the eye.
- CT studies, which allow precise localization of the foreign body and can also image radiolucent foreign bodies.

An injury sustained while working with a hammer and chisel suggests an intraocular foreign body. The diagnosis can be confirmed by examining the fundus in mydriasis and obtaining radiographic studies.

Treatment. First aid. Where penetrating trauma is suspected, a sterile bandage should be applied and the patient referred to an eye clinic for treatment.

Tetanus immunization or prophylaxis and prophylactic antibiotic treatment are indicated as a matter of course.

Surgery. Surgical treatment of penetrating injuries must include suturing the globe and reconstructing the anterior chamber. Any extruded intraocular tissue (such as the iris) must be removed. Intraocular foreign bodies should be removed when the wound is repaired (i.e., by vitrectomy and extraction of the foreign body).

Late sequelae:

- Improper reconstruction of the anterior chamber may lead to adhesions between the iris and the angle of the anterior chamber, resulting in secondary angle closure glaucoma.
- A retinal injury (for example at the site of the impact of the foreign body) can lead to retinal detachment.
- Failure to remove iron foreign bodies can lead to ocular siderosis, which causes irreparable damage to the receptors and may manifest itself years later.
- Copper foreign bodies cause severe inflammatory reactions in the eye (ocular chalcosis) within a few hours. Symptoms range from uveitis and hypopyon to phthisis bulbi (shrink age and hypotonia of the eye ball).
- Organic foreign bodies (such as wood) in the eye lead to fulminant endophthalmitis.

Impalement Injuries in the Orbit

Etiology. Impalement injuries occur most frequently in situations such as these:

- Children may fall on pencils held in their hands.
- Injuries may result from the actions of other persons (such as arrows or darts).
- A knife may slip while a butcher is removing a bone from a cut of meat.

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