Continuing Medical Education

The Diagnosis and Treatment of Glaucoma

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Summary

Background: Glaucoma is a group of chronically progressive disorders of the optic nerve. In this article, we present the epidemiology of and risk factors for glaucoma, as well as the diagnostic work-up and treatment options.

Methods: This review is based on pertinent publications retrieved by a selective search in Medline and the Cochrane Library, supplemented by further articles chosen by the authors.

Results: In Europe, the prevalence of glaucoma is 2.93% among persons aged 40 to 80 years. The prevalence rises with age, reaching 10% in persons over 90 years old. The available diagnostic methods include ophthalmoscopy, tonometry, perimetry, and imaging techniques. The treatment of glaucoma is focused on lowering the intraocular pressure with topical drugs, laser therapy, and glaucoma surgery. In patients with manifest glaucoma, lowering the intraocular pressure prevents the progression of visual field defects, with a number needed to treat of 7.

Conclusion: The diagnostic evaluation of glaucoma rests on multiple pillars, all of which must be considered for establishing the diagnosis and defining the desired target pressure: these are, among others, the intraocular pressure and ocular function and morphology. Individually tailored pressure-lowering treatment should be evaluated in regularly scheduled follow-up visits for assessment of function and morphology and adjusted as necessary to minimize the risk of progression.

Cite this as:
Schuster AK, Erb C, Hoffmann EM, Dietlein T, Pfeiffer N: The diagnosis and treatment of glaucoma. Dtsch Arztebl Int 2020; 117: 225–34. DOI: 10.3238/arztebl.2020.0225

Glaucoma (from the Greek *glaukós,* a nonspecific term for green or light gray [1]) is a group of disorders that differ in their pathophysiology, risk factors, manifestations, treatments, and prognoses. Their common feature is progressive degeneration of the optic nerve, with loss of retinal ganglion cells, thinning of the retinal nerve fiber layer, and progressive excavation of the optic disc *(Figure 1)* (2, 3).

Learning objectives

After reading this article, the reader should know:

- How the various types of glaucoma differ from one another
- How a targeted diagnostic evaluation should be structured
- What treatment options are available for each of the disease entities

Method

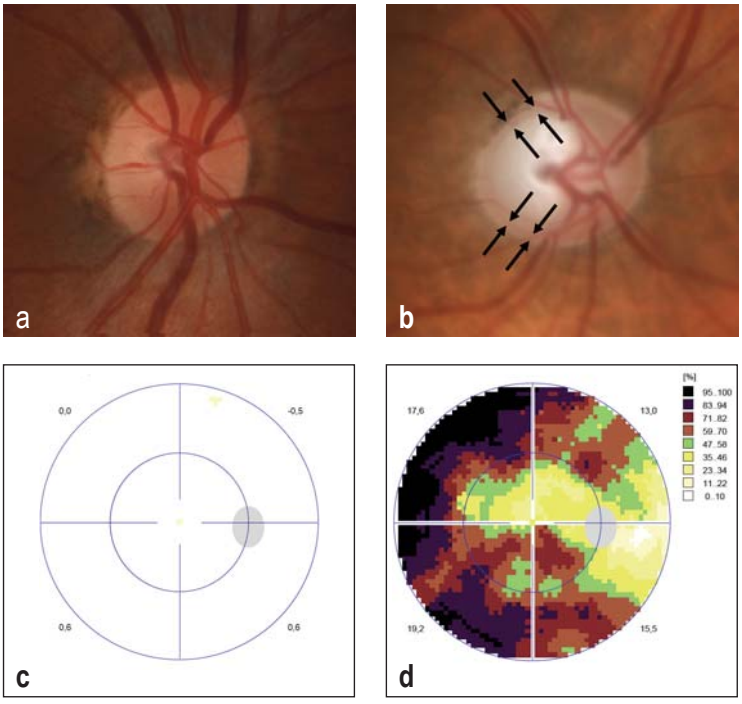
A literature search from January 2014 to December 2018 was carried out in Medline and the Cochrane Library, with the search terms "open-angle glaucoma," "angle-closure glaucoma," "epidemiology," "diagnosis," "tonometry," "perimetry," "optical coherence tomography," "glaucoma therapy," and "glaucoma surgery." From the articles retrieved, those that were relevant to the subject of this review were selected. The reference lists of the chosen articles were scrutinized, and further publications considered important by the authors were added. Only articles on glaucoma in human beings were considered. There was no language restriction.

Definition

Glaucoma is a group of disorders whose common feature is progressive degeneration of the optic nerve, with loss of retinal ganglion cells, thinning of the retinal nerve fiber layer, and increasing excavation of the optic disc.

Pathophysiology

Elevated intraocular pressure and low perfusion pressure increase the gradient across the lamina cribrosa and cause papillary hypoperfusion, leading to structural changes and remodeling of the lamina cribrosa and to impaired axonal transport in the optic nerve fibers.



Images: Department of Ophthalmology, University Medical Center Mainz

Figure 1: Optic disc images: a) normal and b) glaucomatous optic disc; c) and d) the respective visual field measurements. The findings from one eye (the right eye) are shown in all images. The optic disc in b) displays, particularly in the upper and lower temporal quadrants, marked thinning of the neuroretinal edge zone (black arrows), with a large excavation. The corresponding visual field examination d) reveals marked defects with central sparing. Darker shading represents the areas in which light is less well perceived; a normal visual field is shown for comparison (c). Visual field measurements depend on the patient's concentration and cooperation, which can be quantified, for example, with automatic fixation detection and trick questions.

Pathophysiology

The retinal ganglion cells are neurons of the central nervous system that receive signals from the photoreceptors, process them, and transmit them in axons through the optic nerve to further centers in the brain. These axons run from the ganglion cell nuclei in the retina to the optic disc (2), and then together with the retinal vessels through the lamina cribrosa, a sieve-like structure composed of collagen. Behind the lamina cribrosa, the axons, surrounded by a myelin sheath, continue as the optic nerve. Elevated intraocular pressure, low perfusion pressure, and/or low cerebrospinal fluid pressure increase the gradient across the lamina cribrosa and cause papillary hypoperfusion, leading to structural changes and remodeling of the lamina cribrosa and to impaired axonal transport in the optic nerve fibers (4). In particular, the pores in the anterior region of the lamina cribrosa are elongated in open-angle glaucoma (5).

The increasing loss of retinal ganglion cells leads to progressive impairment of the visual field, generally beginning in the mid-periphery and then advancing until only a central or peripheral island of intact vision remains. Further functional disturbances include impaired contrast and color perception and difficulty in reading (6). The mechanisms by which retinal ganglion cells are lost are not yet fully understood.

The different types of glaucoma are classified according to the respective structural changes in the anterior segment of the eye. The aqueous humor is mainly drained in the chamber angle via the trabecular meshwork and the canal of Schlemm, and partly via the uveoscleral outflow (root of the iris, ciliary body). The chamber angle lies between the iris and the peripheral posterior surface of the cornea, and at its end the canal of Schlemm lies under the trabecular meshwork. While in open-angle glaucoma the chamber angle is macroscopically open, in acute angle closure it is occluded by the iris *(Figure 2)*; this suddenly blocks the outflow of aqueous humor via the trabecular meshwork and the canal of Schlemm, causing a marked elevation of intraocular pressure. In secondary open-angle glaucoma, there are changes of the chamber angle that are visible under the microscope (gonioscope), such as pigment deposition (in pigmentary glaucoma) (7) or protein deposition (in pseudoexfoliation glaucoma) (8), that elevate the intraocular pressure. The mechanisms leading to elevated intraocular pressure in primary open-angle glaucoma are not fully understood.

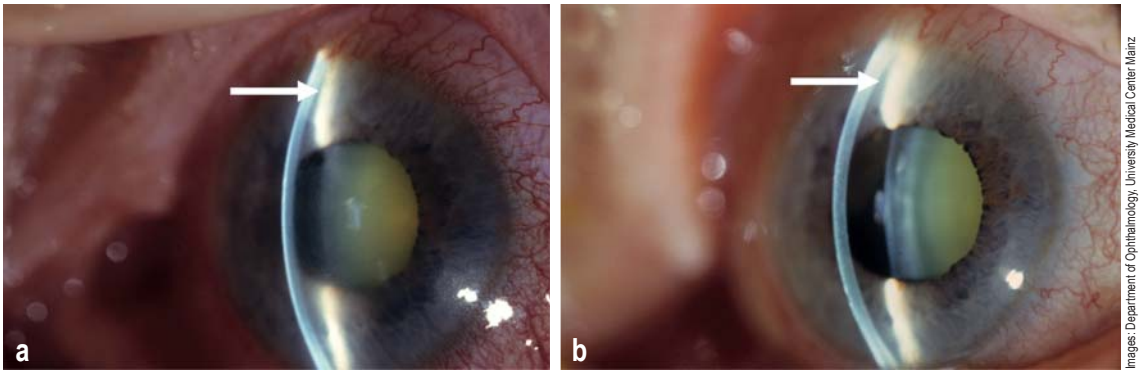
The normal intraocular pressure has an average value of 15.7 mm Hg (9) but displays marked chronobiological and interindividual variation even in healthy persons. It is regulated by the balance between the secretion of aqueous humor by the ciliary body and its outflow. Elevated intraocular pressure may thus be a consequence of increased outflow resistance. This can be due to gonioscopically visible changes in the chamber angle in secondary open-angle glaucoma, as mentioned above, but it can also arise without any such changes, as in primary open-angle glaucoma. Glaucomatous changes in the optic nerve may arise even when the intraocular pressure is within normal limits (normal-pressure glaucoma). Among persons of European ancestry, the intraocular pressure is normal in 30% of all cases of glaucoma (10), with regional variation in prevalence. This disorder is apparently caused by an intraocular pressure

Resulting disturbances

The progressive loss of retinal ganglion cells leads to increasing impairment of the visual field. Further functional disturbances include impaired contrast and color perception and difficulty in reading.

Chamber angle

The chamber angle lies between the iris and the peripheral posterior surface of the cornea, and at its end the canal of Schlemm lies under the trabecular meshwork.



Images: Department of Ophthalmology, University Medical Center Mainz

Figure 2: Slit-lamp examination a) of an occluded chamber angle in acute angle closure and b) after successful treatment (laser iridotomy). There is marked enlargement of the chamber angle (white arrow) and deepening of the anterior chamber from a) to b), as well as corneal clearing and regression of conjunctival hyperemia and scleral vascular distention.

that, although within normal limits, nonetheless exceeds the pressure sensitivity of the optic disc. The importance of the pressure sensitivity of the optic disc is also indicated by the fact that a 25% pressure reduction lowers the risk of glaucoma progression by 50% (11). Moreover, vascular changes seem to play a role in the pathophysiology of open-angle glaucoma, and of normal-pressure glaucoma in particular (12), e.g., an excessive nocturnal drop in blood pressure in otherwise normotensive persons (13).

Epidemiology

In 2010, 2.1 million persons around the world went blind because of glaucoma (14). In western Europe, glaucoma is the second most common cause of irreversible blindness, after age-related macular degeneration (15). The prevalence of glaucoma in Europe among persons aged 40 to 80 years is 2.93% *(Figure 3)* (16). Most suffer from open-angle glaucoma, which has a prevalence of 2.51% from age 40 to age 80 (16). In Germany, too, most persons with glaucoma have open-angle glaucoma (17). The commonly available surgical treatment of cataract in industrialized countries has lowered the risk of a narrow angle and acute angle closure. The thin artificial lens deepens the anterior chamber, with flattening of the iris (18) and widening of the chamber angle.

The prevalence of open-angle glaucoma rises with age (19), from 0.4% at the age of 40–44 years to 2.7% at age 70–74 and 10.0% over age 90 in persons of European ancestry. Men are more commonly affected than women (odds ratio [OR] 1.30) (19). A systematic review revealed marked differences between ethnic groups: in particular, persons of African ancestry have a prevalence of glaucoma 2.8 times higher than Europeans, while angle-closure glaucoma and normal-pressure glaucoma are more common in Asians (16). Congenital glaucoma and juvenile glaucoma are rare in general (20).

Risk factors

The main risk factors for glaucoma are:

- Advanced age (21–23)
- Elevated intraocular pressure (21–23)
- High myopia (24)
- A positive family history of glaucoma (22, 23).

The risk also depends on ethnicity (16). Moreover, excavation of the optic disc is particularly hard to assess in highly myopic eyes. It is thought that enlargement of the optic disc due to myopia, with consequent thinning of the lamina cribrosa, may predispose to glaucoma (25). Increased shear forces in the lamina cribrosa caused by eye movements in persons with highly myopic (long) eyes have been mentioned as another possible pathogenetic factor (26).

Elevated intraocular pressure, or an elevated translaminar pressure gradient (27), is the sole modifiable risk factor for open-angle glaucoma that has been identified so far. The randomized, controlled Ocular Hypertension Treatment Study led to the conclusion that lowering elevated intraocular pressure (21–32 mm Hg) by 22.5% can decrease the 5-year risk of developing open-angle glaucoma from 9.5% to 4.4% (28).

The putative relation between open-angle glaucoma and cardiovascular disease is currently debated.

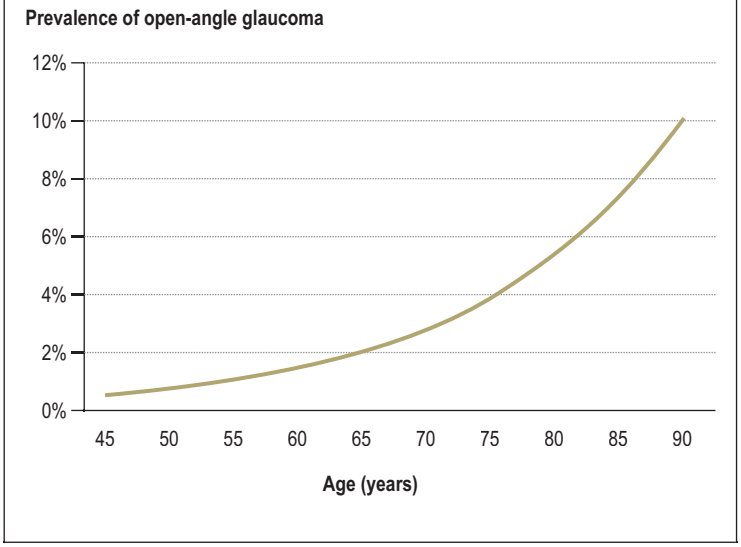
Epidemiology

In 2010, 2.1 million persons around the world went blind because of glaucoma. In western Europe, glaucoma is the second most common cause of irreversible blindness, after age-related macular degeneration.

The main risk factors for glaucoma

- Advanced age
- Elevated intraocular pressure
- High myopia
- A positive family history

FIGURE 3



The increasing prevalence of glaucoma with advancing age in persons of European extraction. Data from Kapetanakis et al. (19)

Systematic reviews have shown small effect sizes for arterial hypertension (29), diabetes mellitus (30), and obstructive sleep apnea (31) with respect to open-angle glaucoma. The corneal configuration is also under discussion as a potential structural risk factor, although no association between a thin cornea and a thin lamina cribrosa has yet been demonstrated (32).

The corneal thickness does, however, influence the measurement of intraocular pressure: a thin cornea can artifactually lower the measured pressure (33). However, this holds only for very large deviations in corneal thickness, as the thickness of the cornea normally fluctuates over a range of approximately 40 µm over the course of the day (34). Formulae to take account of the influence of the corneal thickness on the measured intraocular pressure have not proven useful in practice (35).

Diagnostic evaluation

Symptoms

Acute angle closure can manifest itself with pain radiating from the eye, visual impairment, conjunctival hyperemia, and sometimes nausea and vomiting with a tense, rock-hard globe. This is an ophthalmological emergency that demands immediate treatment to prevent severe ocular damage and blindness.

In contrast, open-angle glaucoma usually does not become symptomatic until it has reached an advanced stage. If visual field defects are present, they usually do not lie in the same part of the fields of the two eyes and are therefore well compensated by binocular vision. Thus, persons with open-angle glaucoma generally report no symptoms (36), and many are completely unaware that they have the condition (37). One-third of patients already have the condition in an advanced or late stage in at least one eye at the time of diagnosis (38). Gramer et al. reported that 10–20% of patients were already unable to drive a vehicle at the time of presentation at the clinic because of binocular visual field defects (39).

Early detection

As this condition only becomes symptomatic when it has reached an advanced stage, the German ophthalmological associations recommend regular screening examinations for early detection from age 40 onward (40). Because of the low prevalence of the disorder (19) and the low sensitivity and specificity of the tests (e1, e2), The rate of false positives is high (> 65%, and even higher in younger patients), and thus any positive finding must be followed up by further testing. Regular examination is especially important in risk groups with elevated incidence and prevalence of the disorder, so that it can be diagnosed and treated early in its course. No randomized, controlled trials on this topic have yet been conducted. The recommended screening examination consists of at least a clinical history, stereoscopic examination of the papilla and peripapillary nerve layer, tonometry, and slit-lamp examination of the eye (40). Screening examinations for glaucoma are not covered by the statutory health insurance providers in Germany, nor is there any population-wide periodic screening for glaucoma in other European countries such as the UK, France, or the Netherlands.

Clinical investigations

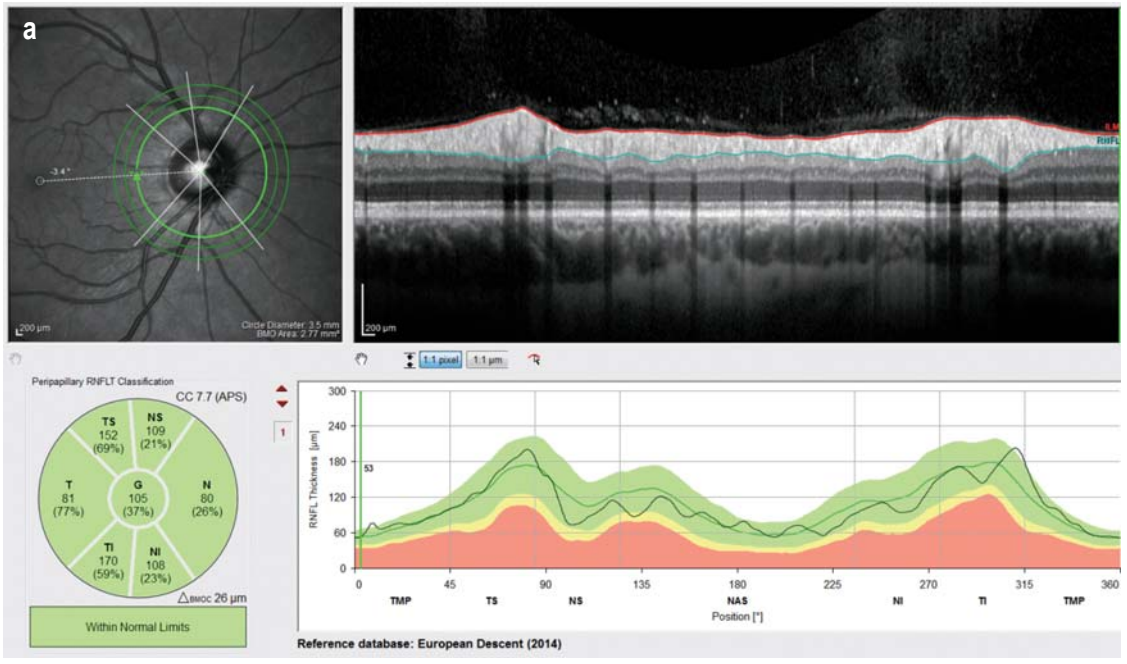
The mainstay of glaucoma diagnosis is funduscopic examination of the optic disc and the retinal nerve fiber layer. Glaucomatous changes are manifested by tissue loss at the neuroretinal rim and enlargement of the optic nerve excavation, a non-physiological discrepancy between the optic nerve excavations in the two eyes, hemorrhages at the edge of the optic disc, thinning of the retinal nerve fiber layer, and parapapillary tissue atrophy (the beta zone) (e3–e5). Morphometric techniques enable quantitative examination of the optic

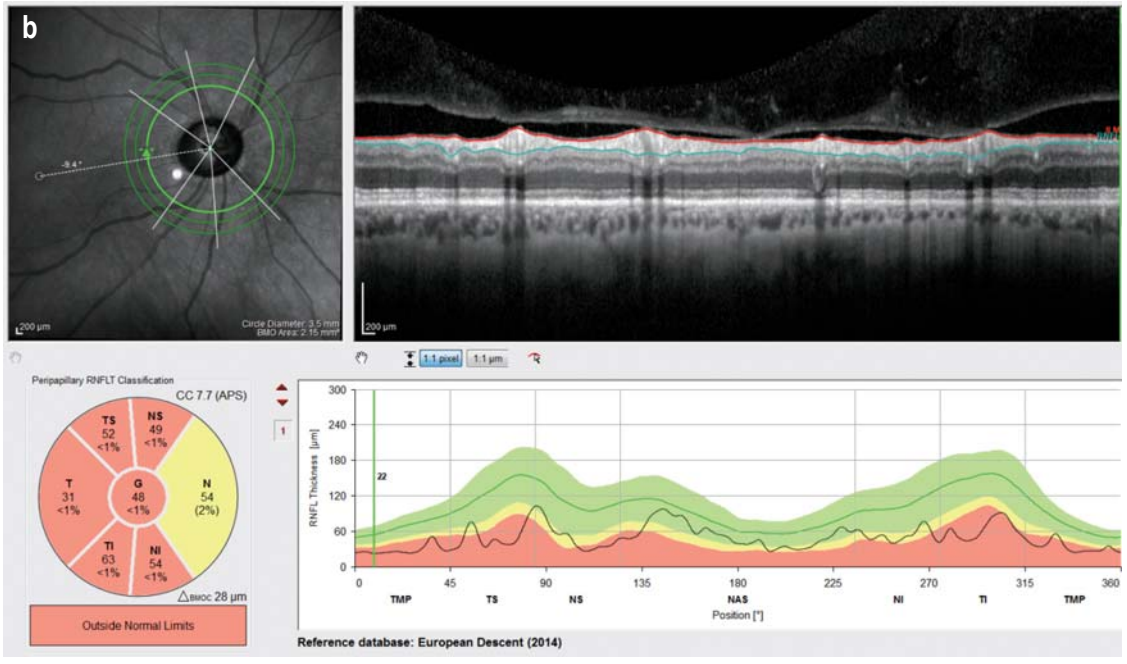
Risk reduction

Lowering an elevated intraocular pressure (21–32 mm Hg) by 22.5% can lower the 5-year risk of developing open-angle glaucoma from 9.5% to 4.4% .

Manifestations of acute angle closure

Acute angle closure can manifest itself with pain radiating from the eye, visual impairment, conjunctival hyperemia, and sometimes nausea and vomiting with a tense, rock-hard globe. This is an emergency demanding the immediate consultation of an ophthalmologist.





Images: Department of Ophthalmology, University Medical Center Mainz

Figure 4: Examination of peripapillary nerve fiber layer thickness a) in a normal eye and b) in a glaucomatous eye. The retinal nerve fiber layer lies between the internal limiting membrane (red line) and the border between the retinal nerve fiber layer and the ganglion cell layer (turquoise line). Beyond this layer, reflections from the vitreous body can be seen.

Imaging for diagnostic evaluation of glaucoma

Morphometric techniques enable quantitative examination of the optic nerve head and measurement of the retinal nerve fiber layer and neuroretinal rim with optical coherence tomography (OCT).

Imaging for follow-up evaluation

Objective examination is especially important for assessing the course of the disease and should be performed at the time of initial diagnosis. Morphometric techniques such as OCT enable quantitative follow-up evaluation.

TABLE

Overview of treatments that lower the intraocular pressure in open-angle glaucoma

Treatment category	Treatment type		Mechanism of action
Drugs	Local application	Prostaglandin analogs Beta-blockers Alpha$_2$-adrenergic agonists Carbonic anhydrase inhibitors Miotic agents	Improved uveoscleral and trabecular outflow Reduced aqueous humor production Reduced aqueous humor production, increased uveoscleral outflow Reduced aqueous humor production Widening of the chamber angle
	Systemic intake	Carbonic anhydrase inhibitors Osmotically active substances (mannitol IV)	Reduced aqueous humor production Osmotic removal of water from the globe
Operative interventions	Laser therapy	Laser trabeculoplasty	Increased outflow of aqueous humor via the canal of Schlemm
		Cyclophotocoagulation	Reduced aqueous humor production
	Surgery	Cyclocryocoagulation	Reduced aqueous humor production
		Minimally invasive procedure	For example, implantation of a stent in the canal of Schlemm to lessen the outflow resistance of the trabecular meshwork
		Non-filtering procedure	For example, deep sclerotomy: widening of the outflow pathways without incising the eye
		Filtering procedure	For example, trabeculectomy: creation of an accessory pathway for the aqueous humor to flow out of the eye under the conjunctiva

IV, Intravenous

disc (e6) and measurement of the retinal nerve fiber layer and neuroretinal rim with optical coherence tomography (OCT) *(Figure 4)* (e7). Objective examination (photography of the optic disc, morphometric measurement of the optic nerve head and of the thickness of the retinal nerve fiber layer, e.g., with OCT [e8]) should be carried out at the initial visit and is especially important for assessing the course of the disease. New imaging procedures enable more precise assessment of the dynamic nature of the disease in the individual case, even if these techniques are not covered by the legally mandated health insurance scheme in Germany.

Measurement of the intraocular pressure (tonometry) on initial diagnosis is mandatory. The intraocular pressure is currently the only modifiable risk factor for the occurrence and progression of glaucoma. The corneal thickness and curvature should be measured at the same time to determine the likelihood of the intraocular pressure measurements being artifactually high or low (33); the size of the artifact may vary with the tonometric method used. Corrective formulae for the intraocular pressure have not been found to be of value and should not be used (35). The intraocular pressure also fluctuates over the course of the day. For this reason, the intraocular pressure is measured at different times of day to produce a daily pressure profile for better titration of the treatment. Gonioscopic examination of the chamber angle at the time of initial diagnosis yields information about the pathogenesis of the disease.

The visual fields should also be examined to evaluate the degree of functional impairment resulting from the loss of optic nerve fibers, and to provide a guide to treatment (e9). Visual field findings may vary depending on the concentration and cooperation of the patient, and progression may therefore be difficult to detect. For this reason, it is recommended that the visual fields should be examined at least three times in the first year after the diagnosis is made (e9).

Treatment

The only form of treatment that has been shown to be efficacious and is generally accepted for the prevention of glaucoma progression is reduction of the intraocular pressure (11, e10), which is effective with a number needed to treat of 7 (e11). In patients with open-angle glaucoma, the intraocular pressure can be reduced with regularly applied eyedrops (11, e10, e12), laser therapy

Glaucomatous changes

These include tissue loss at the neuroretinal rim and enlargement of the optic nerve excavation, a non-physiological discrepancy between the optic nerve excavations in the two eyes, hemorrhages at the edge of the optic nerve head, thinning of the retinal nerve fiber layer, and parapapillary tissue atrophy.

Intraocular pressure

Measurement of the intraocular pressure (tonometry) on initial diagnosis is mandatory. The intraocular pressure is currently the only modifiable risk factor for glaucoma and for the progression of glaucoma.

(e13), and/or surgery (e14, e15) *(Table)*. The goal is to achieve an individually set target pressure at which glaucoma is not expected to progress, and at which the lack of progression can be observed and documented. In each patient, the target pressure is determined on the basis of the existing glaucomatous damage, the current intraocular pressure, the rate of structural and functional progression, other existing risk factors, and the potential side effects of treatment. These considerations determine the initial treatment measures (e16). A pressure reduction rate in percent is no longer recommended as the sole treatment objective, because this takes no account of the absolute level of the initial intraocular pressure, which is an important component. Typical target pressures are < 21 mm Hg for early glaucoma, < 18 mm Hg for moderate glaucoma, and < 15 mm Hg for advanced glaucoma. Whatever target is set, the further course of the intraocular pressure should be regularly checked to determine whether the disease has stabilized as desired or progressed further, in which case the target pressure may need to be revised (e9).

Topical treatment

Various substance classes are available for topical use to reduce the intraocular pressure. They differ in their mechanisms of action, in the degree to which they lower the intraocular pressure (e12), and in their dosing, side effects, and cost. A network meta-analysis on topical first-line drugs showed that the intraocular pressure is lowered to the greatest extent by prostaglandin analogs (bimatoprost by 5.61 mm Hg, latanoprost 4.85 mm Hg, travoprost 4.83 mm Hg, tafluprost 4.37 mm Hg), followed by beta-blockers (levobunolol 4.51 mm Hg, timolol 3.70 mm Hg, carteolol 3.44 mm Hg, levobetaxolol 2.56 mm Hg, betaxolol 2.24 mm Hg), alpha$_2$-adrenergic agonists (brimonidine 3.59 mm Hg, apraclonidine 2.52 mm Hg), and carbonic anhydrase inhibitors (dorzolamide 2.49 mm Hg, brinzolamide 2.42 mm Hg) (e12).

Prostaglandin analogs are usually prescribed for the initial treatment and are applied once daily, in the evening. These drugs improve the uveoscleral and trabecular outflow and thereby lower the intraocular pressure. Their side effects include conjunctival hyperemia, increased growth of the eyelashes, reduction of periorbital fat, and increased pigmentation of the iris and periocular skin *(eFigure)* (e17). Systemic conditions limiting the use of prostaglandin analogs include bronchial asthma, severe cardiovascular conditions, and diseases of the liver or kidneys.

Topically applied beta-blockers are an alternative. These are usually applied twice per day; they lower the intraocular pressure by diminishing the production of the aqueous humor. Their main local side effect is dry eye disease, or exacerbation of existing dry eye disease. Systemic contraindications include bronchial asthma, sinus bradycardia, second- or third-degree AV block, decompensated congestive heart failure, severe allergic rhinitis, cerebral hypoperfusion, and muscle weakness. Beta-blockers can exacerbate hyperglycemia and mask the symptoms of hypoglycemia in diabetic patients.

Alpha$_2$-adrenergic agonists lessen the secretion of aqueous humor and increase the uveoscleral outflow. The local side effects include initial white discoloration of the conjunctiva after the drops are applied, and, over the long term, topical intolerance in more than one-third of patients. Less commonly, there can be lid retraction, dry mouth, bradycardia, and fatigue (e18). Simultaneous treatment with monoamine oxidase inhibitors, sympathomimetic drugs, or tricyclic antidepressants, which can affect noradrenergic transmission, is a systemic contraindication. Topical treatment with alpha$_2$-adrenergic agonists is contraindicated in children under 12 because of extremely severe side effects (ranging up to coma in toddlers). Conditions requiring special caution include bradycardia, hypotension, arteriosclerosis, and impaired hepatic or renal function. Topical carbonic anhydrase inhibitors likewise function by decreasing the production of aqueous humor; local undesired side effects include tearing, burning, and corneal endothelial decompensation.

Miotic drugs can be a further alternative, but are now hardly ever used as the treatment of first resort.

Drugs from these different substance classes may be combined with one another, with due consideration of their side effect profiles and mechanisms of action. It is recommended that the eyes should be kept closed for a few minutes after the local, touch-free application of eyedrops in the lower conjunctival sac; if indicated, the tear ducts should be manually occluded with the index fingers. This lessens outflow of the drug through the tear duct system and resorption through the nasal mucosa, thereby reducing the chance of systemic side effects. Multiple studies have shown that patients tend to display poor compliance with local antiglaucomatous treatment (e19).

In the past, most eyedrops contained benzalkonium chloride as preservative, but in recent years multiple eyedrop preparations have been developed and

Target range for intraocular pressure

The target pressure is set individually and the further course of the intraocular pressure should be regularly checked to determine whether the disease has stabilized as desired.

Prostaglandin analogs

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approved without this ingredient (so-called preservative-free eyedrops). These have lessened the undesired side effects, conjunctival hyperemia in particular, and improved local tolerance (e20). Artificial tears (e.g., hyaluronic acid preparations) are used to treat side effects such as dry eye.

Laser therapy

Laser therapy may be considered as a supplementary measure if local treatment does not adequately lower the intraocular pressure or fails to achieve the target pressure (e.g., because of lacking compliance with treatment). Laser therapy, however, generally results in a moderate lowering of the intraocular pressure, by way of increased aqueous humor outflow after laser trabeculoplasty (e13) or diminished aqueous humor production after cyclophotocoagulation (e21). The latter lowers the intraocular pressure by at least 20% in 47% of the treated eyes (e22); its potential complications include inadequate or excessive pressure reduction, inflammation, and pupillary deformity, which may lead to highly bothersome glare. Micropulse laser techniques can be used for both applications as well, but their efficacy has not yet been fully documented.

Glaucoma surgery

Surgery is indicated if nonsurgical treatment options are insufficient to lower the intraocular pressure to the target pressure, or cause intolerable side effects. Minimally invasive, filtering, and non-filtering types of glaucoma surgery are available. For example, in one type of minimally invasive procedure a stent is placed in the canal of Schlemm (e14) to lower the outflow resistance through the trabecular meshwork. In general, this operation, which can be performed in combination with cataract surgery, does not lower the intraocular pressure enough, unless the glaucoma is only moderate (e23); in recent years, the surgical options have expanded markedly. Minimally invasive glaucoma surgery seems to have fewer side effects than a filtering procedure (e24), but also lowers the intraocular pressure by a lesser amount (e25).

In a filtering operation, an accessory pathway is created for the aqueous humor to flow out of the eye under the conjunctiva. Trabeculectomy is now considered the reference standard for this type of procedure. Various antimetabolites are applied intraoperatively and postoperatively to inhibit local conjunctival scarring (e26). Patients with advanced glaucoma have less worsening of their visual fields if they are treated with trabeculectomy than if they undergo laser trabeculoplasty (hazard ratio [HR] = 3.95 in 10 years for Caucasian patients, HR = 1.62 in 10 years for patients with dark skin) (e27). Other methods include deep sclerectomy and canaloplasty; these seem to have a lower risk of complications (cataract, endophthalmitis, etc.) (e28).

Treatments for acute angle closure, aside from intraocular pressure reduction with topical agents and systemic drugs (carbonic anhydrase inhibitors), include surgical procedures such as lentectomy with intraocular lens implantation or mechanical opening of the occluded angle (iridotomy, iridectomy) (e29), which can be performed as an emergency procedure for persistent acute angle closure. The other eye should also be treated surgically shortly afterwards, as the risk of acute angle closure in the second eye is 51%, but can be reduced by successful treatment to 2% (e30).

Laser therapy

Laser therapy may be considered as supplementary treatment if local treatment does not adequately lower the intraocular pressure or fails to achieve the target pressure (e.g., because of non-compliance).

Conflict of interest statement

Prof. Schuster has an endowed professorship for ophthalmic health care research sponsored by the German Eye Foundation (*Stiftung Auge*) and financed by the German Ophthalmological Society (*Deutsche Ophthalmologische Gesellschaft*) and the Professional Association of German Ophthalmologists (*Berufsverband der Augenärzte Deutschlands e.V.*). He has received third-party research funding and lecture honoraria from Allergan, Bayer Vital, Novartis, and Heidelberg Engineering.

Prof. Erb has served as a paid consultant for Allergan, Alcon, and Santen. He has received payment for preparing continuing medical education events from Allergan, Alcon, Bayer, Santen, Glaukos, Zeiss, Thea, and Visufarma.

Prof. Hoffmann has served as a paid consultant for Santen, Allergan, Novartis, Glaukos, and Heidelberg Engineering. She has received reimbursement of meeting participation fees and of travel and accommodation expenses from Santen, Allergan, Glaukos, and Novartis. She has also received payment from the same companies for preparing continuing medical education events.

Prof. Dietlein owns Johnson and Johnson stock. He has served as a paid consultant for Alcon and Santen. He has received payment for preparing continuing medical education events from Theapharma, Alcon, and Santen.

Prof. Pfeiffer has served as a paid consultant for Aerie, Alcon, Atheneum, Böhringer, Ivantis, InnFocus, Isarna, Novartis, Santen, and Thea. He has received reimbursement of meeting participation fees and of travel and accommodation expenses from Aerie and Santen.

Manuscript submitted on 1 April 2019, revised version accepted on 28 October 2019.

Translated from the original German by Ethan Taub, M.D.

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Cite this as:
Schuster AK, Erb C, Hoffmann EM, Dietlein T, Pfeiffer N: The diagnosis and treatment of glaucoma. Dtsch Arztebl Int 2020; 117: 225–34. DOI: 10.3238/arztebl.2020.0225

▶ **Supplementary material**

For eReferences please refer to:
www.aerzteblatt-international.de/ref1320

eFigure:
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- Participation in the CME certification program is possible only over the Internet: cme.aerzteblatt.de. This unit can be accessed until 21 June 2020. Submissions by letter, e-mail or fax cannot be considered.
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Only one answer is possible per question. Please select the answer that is most appropriate.

Question 1

What is the approximate prevalence of glaucoma in persons aged 40 to 80 years?

a) 1%
b) 3%
c) 7%
d) 10%
e) 25%

Question 2

Which of the following is a risk factor for glaucoma?

a) Macular degeneration
b) Female sex
c) Lagophthalmus
d) Positive family history
e) Keratoconjunctivitis sicca

Question 3

What is the most common type of glaucoma?

a) Open-angle glaucoma
b) Angle-closure glaucoma
c) Congenital glaucoma
d) Normal-pressure glaucoma
e) Juvenile glaucoma

Question 4

What diagnostic technique is mandatory when glaucoma is initially diagnosed?

a) Orthoptic examination for strabismus
b) Intraocular pressure measurement (tonometry)
c) Measurement of blinking frequency
d) Assessment of the pupillary light reflex
e) Inspection of the lid margins

Question 5

Which of the following predisposes to acute angle closure?

a) A narrow pupil (miosis)
b) A deep anterior chamber
c) Prior cataract surgery
d) A narrow chamber angle
e) A thin lens

Question 6

What is the sole treatment that has been shown to be effective for the prevention of glaucoma progression?

a) Intravitreal bevacizumab
b) Cortisone eyedrops
c) Artificial tears
d) Intraocular pressure reduction
e) Photodynamic therapy

Question 7

What type of eyedrops leaves the intraocular pressure unchanged?

a) Prostaglandin analogs
b) Beta-blockers
c) Carbonic anhydrase inhibitors
d) Hyaluronic acid preparations
e) Alpha$_2$-adrenergic agonists

Question 8

What method is used for the follow-up assessment of the optic disc in glaucoma?

a) Magnetic resonance imaging
b) Optical coherence tomography
c) Ultrasonography
d) Electroretinography
e) Visual evoked potentials

Question 9

What is the best description of the mechanism of action of carbonic anhydrase inhibitors in the topical treatment of glaucoma?

a) They promote angiogenesis on the optic disc
b) They enlarge the trabecular meshwork.
c) They improve tear secretion
d) They increase aqueous humor outflow via Schlemm's canal
e) They decrease aqueous humor secretion

Question 10

What is the goal of surgery for open-angle glaucoma?

a) Lowering the intraocular pressure
b) Restoring optic nerve fibers
c) Improving visual acuity

► **Participation is possible only over the Internet: cme.aerzteblatt.de**

Supplementary material to:

The Diagnosis and Treatment of Glaucoma

by Alexander K. Schuster, Carl Erb, Esther M. Hoffmann, Thomas Dietlein, and Norbert Pfeiffer

Dtsch Arztebl Int 2020; 117: 225–34. DOI: 10.3238/arztebl.2020.0225

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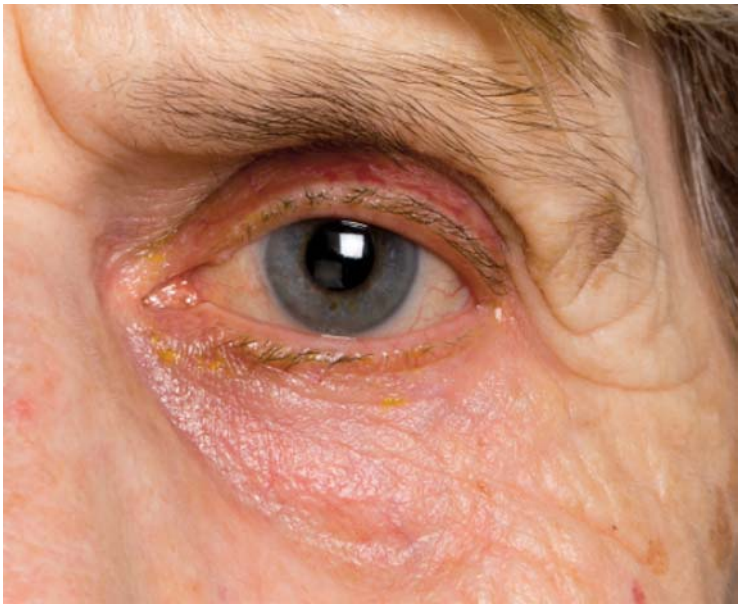

Images: Department of Ophthalmology, University Medical Center Mainz

eFigure: Side effects of local antiglaucomatous treatment
There is redness of the periocular skin, as well as a reduction of periorbital fat and mild conjunctival hyperemia.