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AN OVERVIEW ON GLAUCOMA

Sathibabu Talluri[1*], Siva girishbabu Guttula[2]

[1]Department of Pharmaceutics, Aditya college of Pharmaceutical Sciences and Research, Surampalem, East Godavari District, Andhra Pradesh, India.
[2]Department of Pharmaceutics, SRM College of Pharmacy, Potheri, Kanchipuram District, Tamilnadu, India.

ABSTRACT
*Glaucoma is an eye disease in which the **optic nerve** is damaged in a characteristic pattern. This can permanently damage the vision in the affected eye(s) and leads to blindness if left untreated. The word "glaucoma" comes from the Greek γλαύκωμα, "opacity of the **crystalline lens**". Glaucoma has been called the "silent thief of sight" because the loss of vision often occurs gradually over a long period of time and the symptoms only occur when the disease is quite advanced. Once lost, vision cannot normally be recovered, so treatment is aimed at preventing further loss. It is normally associated with increased fluid pressure in the **aqueous humor** of the eye which is called as **intraocular pressure**. The term **ocular hypertension** is used for people with consistently raised intraocular pressure without any associated optic nerve damage. Worldwide, glaucoma is the second-leading cause of blindness after **cataracts**. This article includes a detailed review on the causes, risk factors, types, signs, symptoms, diagnosis and treatment of Glaucoma. It also gives the preventive measures for Glaucoma and deals with the combinations of the drugs used for the treatment of Glaucoma.*

KEYWORDS : *Optic nerve, Crystalline lens, Aqueous humor, Intraocular pressure, Ocular hypertension, Cataracts.*

Correspondence to Author

SATHIBABU TALLURI

D.No.6-3-36, TalluriSuryanarayana sons, Saravaristreet, Peddapuram. Andhra Pradesh, India.

Email: sathibabu.talluri@gmail.com

INTRODUCTION

GLAUCOMA

Glaucoma is an eye disease in which the optic nerve is damaged in a characteristic pattern. This can permanently damage vision in the affected eye(s) and lead to blindness if left untreated. Glaucoma is a disease of the major nerve of vision, called the optic nerve. The optic nerve receives light-generated nerve impulses from the retina and transmits these to the brain, where we recognize those electrical signals as vision. Glaucoma is characterized by a particular pattern of progressive damage to the optic nerve that generally begins with a subtle loss of side vision (peripheral vision).

It is normally associated with increased fluid pressure in the eye aqueous humor.[1] The term "ocular hypertension" is used for people with consistently raised Intra Ocular Pressure (IOP)

without any associated optic nerve damage. Conversely, the term 'normal tension' or 'low tension' glaucoma is used for those with optic nerve damage and associated visual field loss, but normal or low IOP. In some cases, glaucoma may occur in the presence of normal eye pressure. This form of glaucoma is believed to be caused by poor regulation of blood flow to the optic nerve.

The nerve damage involves loss of retinal ganglion cells in a characteristic pattern. The many different subtypes of glaucoma can all be considered to be a type of optic neuropathy. Raised intraocular pressure (above 21 mmHg or 2.8 kPa) is the most important and only modifiable risk factor for glaucoma. However, some may have high eye pressure for years and never develop damage, while others can develop nerve damage at a relatively low pressure. Untreated glaucoma can lead to permanent damage of the optic nerve and resultant visual field loss, which over time can progress to blindness.

Glaucoma has been called the "silent thief of sight" because the loss of vision often occurs gradually over a long period of time, and symptoms only occur when the disease is quite advanced. Once lost, vision cannot normally be recovered, so treatment is aimed at preventing further loss. Worldwide, glaucoma is the second-leading cause of blindness after cataracts.[2][3] It is also the leading cause of blindness among African Americans.[4] Glaucoma affects one in 200 people aged 50 and younger, and one in 10 over the age of eighty. If the condition is detected early enough, it is possible to arrest the development or slow the progression with medical and surgical means. Screening for glaucoma in the general population is however unsupported by the evidence.

The word "glaucoma" comes from the Greek γλαύκωμα, "opacity of the crystalline lens". (Cataracts and glaucoma were not distinguished until *circa* 1705).[5]

OPTIC NERVE

It is the nerve that arises from the back of the eyes and it is directed backward to the brain. It consists of 1.2million axons or bundles in which their cells present in the retina. Those axons that will be damaged when there is high intraocular pressure.

With more axons damaged there will be black spots formed in the visual field especially peripherally and only central vision is clear leading to tunnel vision. When the disease is advanced the central vision will also be affected.

HOW COMMON IS GLAUCOMA….?

Worldwide, glaucoma is the leading cause of irreversible blindness. In fact, as many as 6 million individuals are blind in both eyes from this disease. In the United States alone, according to one estimate, over 3 million people have glaucoma. As many as half of the individuals with glaucoma, however, may not know that they have the disease. The reason they are unaware is that glaucoma initially causes no symptoms, and the subsequent loss of side vision (peripheral vision) is usually not recognized.

CAUSES OF GLAUCOMA

Elevated pressure in the eye is the main factor leading to glaucomatous damage to the eye (optic) nerve. Glaucoma with normal intraocular pressure is discussed below in the section on the different types of glaucoma. The optic nerve, which is located in back of the eye, is the main visual nerve for the eye. This nerve transmits the images we see back to the brain for interpretation. The eye is firm and round, like a basketball. Its tone and shape are maintained by a pressure within the eye (the intraocular pressure), which normally ranges between 8 mm and 22 mm (millimeters) of mercury. When the pressure is too low, the eye becomes softer, while an elevated pressure causes the eye to become harder. The optic nerve is the most susceptible part of the eye to high pressure because the delicate fibers in this nerve are easily damaged.

GLAUCOMA RISK FACTORS

Glaucoma is often called "the sneak thief of sight." This is because, as already mentioned, in most cases, the intraocular pressure can build up and destroy sight without causing obvious symptoms. Thus, awareness and early detection of glaucoma are extremely important because this disease can be successfully treated when

diagnosed early. While everyone is at risk for glaucoma, certain people are at a much higher risk and need to be checked more frequently by their eye doctor. The major risk factors for glaucoma include the following:

- Age over 45 years
- Family history of glaucoma
- Black racial ancestry
- Diabetes
- History of elevated intraocular pressure
- Nearsightedness (high degree ofmyopia), which is the inability to see distant objects clearly
- History of injury to the eye
- Use of cortisone (steroids), either in the eye or systemically (orally or injected)
- Farsightedness (hyperopia), which is seeing distant objects better than close ones (Farsighted people may have narrow drainage angles, which predispose them to acute [sudden] attacks of angle-closure glaucoma.)

CLASSIFICATION

Glaucoma has been classified into specific types:[6]

PRIMARY GLAUCOMA AND ITS VARIANTS

- Primary angle closure glaucoma, also known as primary closed-angle glaucoma, narrow-angle glaucoma, pupil-block glaucoma, acute congestive glaucoma
- Acute angle closure glaucoma
- Chronic angle closure glaucoma
- Intermittent angle closure glaucoma
- Superimposed on chronic open-angle closure glaucoma ("combined mechanism" - uncommon)
- Primary open-angle glaucoma, also known as chronic open-angle glaucoma, chronic simple glaucoma, glaucoma simplex
- High-tension glaucoma
- Low-tension glaucoma

VARIANTS OF PRIMARY GLAUCOMA

- Pigmentary glaucoma.
- Exfoliation glaucoma, also known as pseudoexfoliative glaucoma or glaucoma capsulare.

Primary open-angle glaucoma is when optic nerve damage results in a progressive loss of the visual field.[7] This is associated with increased pressure in the eye. Not all people with primary open-angle glaucoma have eye pressure that is elevated beyond normal, but decreasing the eye pressure further has been shown to stop progression even in these cases.

The increased pressure is caused by trabecular blockage. Because the microscopic passageways are blocked, the pressure builds up in the eye and causes imperceptible very gradual vision loss. Peripheral vision is affected first, but eventually the entire vision will be lost if not treated.

Diagnosis is made by looking for cupping of the optic nerve. Prostaglandin agonists work by opening uveoscleral passageways. Beta blockers, such as timolol, work by decreasing aqueous formation. Carbonic anhydrase inhibitors decrease bicarbonate formation from ciliary processes in the eye, thus decreasing formation of Aqueous humor. Parasympathetic analogs are drugs that work on the trabecular outflow by opening up the passageway and constricting the pupil. Alpha 2 agonists (brimonidine, apraclonidine) both decrease fluid production (via. inhibition of AC) and increase drainage.

Primary angle closure glaucoma is caused by contact between the iris and trabecular meshwork, which in turn obstructs outflow of the aqueous humor from the eye. This contact between iris and Trabecular Meshwork (TM) may gradually damage the function of the meshwork until it fails to keep pace with aqueous production, and the pressure rises. In over half of all cases, prolonged contact between iris and TM causes the formation of synechiae (effectively "scars").

These cause permanent obstruction of aqueous outflow. In some cases, pressure may rapidly build up in the eye, causing pain and redness (symptomatic, or so called "acute" angle closure). In this situation, the vision may become blurred, and halos may be seen around bright lights. Accompanying symptoms may include headache and vomiting.

Diagnosis is made from physical signs and symptoms: pupils mid-dilated and unresponsive to light, cornea edematous (cloudy), reduced vision, redness, and pain. However, the majority of cases are asymptomatic. Prior to very severe loss of vision, these cases can only be identified by examination, generally by an eye care professional.

DEVELOPMENTAL GLAUCOMA

- Primary congenital glaucoma
- Infantile glaucoma
- Glaucoma associated with hereditary of familial diseases

SECONDARY GLAUCOMA

- Inflammatory glaucoma
- Uveitis of all types
- Fuchs heterochromiciridocyclitis
- Phacogenic glaucoma
- Angle-closure glaucoma with mature cataract
- Phacoanaphylactic glaucoma secondary to rupture of lens capsule
- Phacolytic glaucoma due to phacotoxic meshwork blockage
- Subluxation of lens
- Glaucoma secondary to intraocular hemorrhage
- Hyphema
- Hemolytic glaucoma, also known as erythroclastic glaucoma
- Traumatic glaucoma
- Angle recession glaucoma: Traumatic recession on anterior chamber angle
- Postsurgical glaucoma
- Aphakic pupillary block
- Ciliary block glaucoma
- Neovascular glaucoma (see below for more details)
- Drug-induced glaucoma
- Corticosteroid induced glaucoma
- Alpha-chymotrypsin glaucoma. Postoperative ocular hypertension from use of alpha chymotrypsin.
- Glaucoma of miscellaneous origin
- Associated with intraocular tumors
- Associated with retinal detachments
- Secondary to severe chemical burns of the eye
- Associated with essential iris atrophy
- Toxic glaucoma

Neovascular glaucoma, an uncommon type of glaucoma, is difficult or nearly impossible to treat, and is often caused by Proliferative Diabetic Retinopathy (PDR) or Central Retinal Vein Occlusion (CRVO). It may also be triggered by other conditions that result in ischemia of the retina or ciliary body. Individuals with poor blood flow to the eye are highly at risk for this condition.

Neovascular glaucoma results when new, abnormal vessels begin developing in the angle of the eye that begins blocking the drainage. Patients with such condition begin to rapidly lose their eyesight. Sometimes, the disease appears very rapidly, especially after cataract surgery procedures. A new treatment for this disease, as first reported by Kahook and colleagues, involves use of a novel group of medications known as anti-VEGF agents. These injectable medications can lead to a dramatic decrease in new vessel formation and, if injected early enough in the disease process, may lead to normalization of intraocular pressure.

Toxic glaucoma is open angle glaucoma with an unexplained significant rise of intraocular pressure following unknown pathogenesis. Intraocular pressure can sometimes reach 80 mmHg (11 kPa). It characteristically manifests as ciliary body inflammation and massive trabecular oedema that sometimes extends to Schlemm's canal. This condition is differentiated from malignant glaucoma by the presence of a deep and clear anterior chamber and a lack of aqueous misdirection. Also, the corneal appearance is not as hazy. A reduction in visual acuity can occur followed neuroretinal breakdown.[8]

ABSOLUTE GLAUCOMA

Absolute glaucoma is the end stage of all types of glaucoma. The eye has no vision, absence of pupillary light reflex and pupillary response, and has a stony appearance. Severe pain is present in the eye. The treatment of absolute glaucoma is a

destructive procedure like cyclocryoapplication, cyclophotocoagulation, or injection of 99% alcohol.

SIGNS AND SYMPTOMS

The two main types of glaucoma are open-angle glaucoma and closed-angle glaucoma (also called angle closure glaucoma). Open-angle glaucoma accounts for 90% of glaucoma cases in the United States. It is painless and does not have acute attacks. The only signs are gradually progressive visual field loss, and optic nerve changes (increased cup-to-disc ratio on fundoscopic examination).

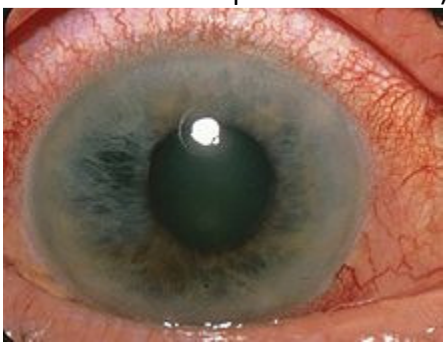

Photo showing conjunctival vessels dilated at the corneal edge (ciliary flush, circumcorneal flush) and hazy cornea characteristic of acute angle closure glaucoma.

Closed-angle glaucoma accounts for less than 10% of glaucoma cases in the United States, but as many as half of glaucoma cases in other nations (particularly Asian countries). About 10% of patients with closed angles present with acute angle closure crises characterized by sudden ocular pain, seeing halos around lights, red eye, very high intraocular pressure (>30 mmHg), nausea and vomiting, suddenly decreased vision, and a fixed, mid-dilated pupil. It is also associated with an oval pupil in some cases. Acute angle closure is an emergency.

A normal range of vision

The same view with advanced vision loss from glaucoma

Of the several causes for glaucoma, ocular hypertension (increased pressure within the eye) is the most important risk factor in most types of glaucoma but in some populations, only 50% of people with primary open-angle glaucoma actually have elevated ocular pressure.[9]

DIETARY

No clear evidence indicates vitamin deficiencies cause glaucoma in humans. It follows, then, that oral vitamin supplementation is not a recommended treatment for glaucoma.[10] Caffeine increases intraocular pressure in those with glaucoma, but do not appear to affect normal individuals.[11]

ETHNICITY AND GENDER

Many people of East Asian descent are prone to developing angle closure glaucoma due to shallower anterior chamber depths, with the majority of cases of glaucoma in this population consisting of some form of angle closure.[12] Inuit also have a 20 to 40 times higher risk of developing primary angle closure glaucoma. Women are three times more likely than men to develop acute angle closure glaucoma due to their shallower anterior chambers. People of African descent are three times more likely to develop primary open angle glaucoma.

GENETICS

Positive family history is a risk factor for glaucoma. The relative risk of having primary open angle glaucoma (POAG) is increased approximately 2-4 fold for individuals who have a sibling with glaucoma.[13] Glaucoma, particularly primary open angle glaucoma, is associated with mutations in several different genes,[14] although most cases of

glaucoma do not involve these genetic mutations. Normal tension glaucoma, which comprises one-third of POAG, is also associated with genetic mutations.[15]

Various rare congenital/genetic eye malformations are associated with glaucoma. Occasionally, failure of the normal third trimester gestational atrophy of the hyaloid canal and the tunica vasculosalentis is associated with other anomalies. Angle closure-induced ocular hypertension and glaucomatous optic neuropathy may also occur with these anomalies,[16][17][18] and modeled in mice.[19]

OTHER

Other factors can cause glaucoma, known as "secondary glaucomas", including prolonged use of steroids (steroid-induced glaucoma); conditions that severely restrict blood flow to the eye, such as severe diabetic retinopathy and central retinal vein occlusion (neovascular glaucoma); ocular trauma(angle recession glaucoma); and uveitis (uveitic glaucoma). In a large study in the UK, glaucoma patients had a 29% increased incidence of systemic hypertension compared to age- and sex-matched controls.[20]

DIAGNOSIS OF GLAUCOMA

An eye doctor (ophthalmologist) can usually detect those individuals who are at risk for glaucoma (because of, for example, a narrow drainage angle or increased intraocular pressure) before nerve damage occurs. The doctor also can diagnose patients who already have glaucoma by observing their nerve damage or visual field loss. The following tests, all of which are painless, may be part of this evaluation.

Glaucoma Tests[21][22][23]	**What Test Examines**	**How Examination is Accomplished**
Tonometry	Inner eye pressure	The eye is numbed via eye drops. The examiner then uses a tonometer to measure the inner pressure of the eye through pressure applied by a puff of warm air or a tiny tool.
Ophthalmoscopy (dilated eye exam)	Shape and color of the optic nerve	The pupil is dilated via the application of eye drops. Using a small magnification device with a light on the end, the examiner can examine the magnified optic nerve.
Perimetry (visual field test)	Complete field of vision	The patient looks straight ahead and is asked to indicate when light passes the patients peripheral field of vision. This allows the examiner to map the patient's field of vision.
Gonioscopy	Angle in the eye where the iris meets the cornea	Eye drops are used to numb the eye. A hand-held contact lens with a mirror is placed gently on the eye to allow the examiner to see the angle between the cornea and the iris.
Pachymetry	Thickness of the cornea	The examiner places a pachymeter gently on the front of the eye to measure its thickness.
Nerve fiber analysis	Thickness of the nerve fiber layer	Using one of several techniques, the nerve fibers are examined.

HOW OFTEN SHOULD SOMEONE BE CHECKED (SCREENED) FOR GLAUCOMA…?[24][25]

The following are the **American Academy of Ophthalmology**'s recommended intervals for eye exams:

- Age 20-29: At least once during this period.
- Age 30-39: At least twice during this period.
- Age 40-64: Every two to four years.
- Age 65 or older: Every one to two years.

TREATMENT FOR GLAUCOMA

GENERAL APPROACH

Although nerve damage and visual loss from glaucoma cannot usually be reversed, glaucoma is a disease that can generally be controlled. That is, treatment can make the intraocular pressure normal and, therefore, prevent or retard further nerve damage and visual loss. Treatment may involve the use of eyedrops, pills (rarely), laser or surgery.

In the United States, eyedrops are usually used first in treating most types of open-angle glaucoma. In contrast, in Europe, laser or surgery is sometimes the first choice of treatment. One or more types of eyedrops may have to be taken up to several times a day to lower intraocular pressure. These drops work either by reducing the production of the aqueous fluid (shutting the faucet) or by increasing the drainage of the fluid out of the eye. Each type of therapy has its benefits and potential complications.

GLAUCOMA MEDICATIONS (EYEDROPS)

Intraocular pressure can be lowered with medication, usually eye drops. Several different classes of medications are used to treat glaucoma, with several different medications in each class.

Each of these medicines may have local and systemic side effects. Adherence to medication protocol can be confusing and expensive; if side effects occur, the patient must be willing either to tolerate them, or to communicate with the treating physician to improve the drug regimen. Initially, glaucoma drops may reasonably be started in either one or in both eyes.[26]

Poor compliance with medications and follow-up visits is a major reason for vision loss in glaucoma patients. A 2003 study of patients in an HMO found half failed to fill their prescriptions the first time, and one-fourth failed to refill their prescriptions a second time.[27] Patient education and communication must be ongoing to sustain successful treatment plans for this lifelong disease with no early symptoms.

The possible neuroprotective effects of various topical and systemic medications are also being investigated.[28][29][30]

TYPES OF GLAUCOMA EYE DROPS

Medication	Mechanism	Dosage	Side effects	Marketed products
Prostaglandin analogs	By increasing trabecular outflow	One drop Once a day	Changes in eye color and eyelid skin, stinging, blurred vision, eye redness, itching, burning.	Latanoprost (Xalatan) Travoprost (TravatanZ) Bimatoprost (Lumigan)
Beta blockers	By decreasing aqueous humor production by the ciliary body	1 drop once or twice a day	Bradicardia Hypotension Fatigue Impotence	Timolol (Timoptic) Levobunolol (Betagan) Betaxolol(Optipres) Carteolol(Ocupress) Metipranolol (Optipranolol)
Alpha agonists	Work to both decrease and increasing uveoscleral outflow	One drop Three times a day	Burning or stinging, fatigue, headache, drowsiness, dry mouth and nose, relatively higher likelihood of allergic reaction.	Brimonidine (Alphagan P) Apraclonidine (Iopidine) Dipivefrin (Propine) Epinephrine
Parasympatho mimetic agents	Contraction of the ciliary muscle,	Three to four	A small pupil, blurred vision, an	Pilocarpine (Salagen), demecarium (Humorsol),

(Mitotics)	tightening the trabecular meshwork and allowing increased outflow of the aqueous humour	times a day	aching brow, and an increased risk of retinal detachment.	echothiophate (Phospholine Iodide), and isoflurophate (Floropryl).
Carbonic anhydrase inhibitors	By reducing the production of fluid in the aqueous humor by inhibiting carbonic anhydrase in the ciliary body..	Two to three times daily	Stinging, burning, eye discomfort; inpill form: tingling hands and feet, stomach upset, memory problems, depression, frequent urination.	(Trusopt®) Brinzolamide (Azopt™).
Oral forms			Systemic side effects including reduction of body potassium, kidney stones, numbness or tingling sensations in the arms and legs, fatigue, and nausea	Acetazolamide (Diamox) Methazolamide (Neptazane)

- **Combined medications** can offer an alternative for patients who need more than one type of medication. In addition to the convenience of using one eyedrop bottle instead of two, there may also be a financial advantage, depending on your insurance plan. Cosopt® is a combination of a beta blocker (timolol) and a carbonic anhydrase inhibitor (Trusopt). Combigan™ is new and combines an alpha agonist (brimonidine) with a beta blocker (timolol).
- Physostigmine is also used to treat glaucoma.
- Marijuana was found, in the early 1970s, to reduce pressure in the eyes, though how the cannabinoids in marijuana produce this effect remains unknown.

Oral forms of these medications used for glaucoma include **acetazolamide (Diamox)** and **methazolamide (Neptazane).** Their use in this condition, however, is limited due to their systemic (throughout the body) side effects, including reduction of body potassium, kidney stones, numbness or tingling sensations in the arms and legs, fatigue, and nausea.

Osmotic agents are an additional class of medications used to treat sudden (acute) forms of glaucoma where the eye pressure remains extremely high despite other treatments. These medications include isosorbide (Ismotic, given by mouth) and mannitol (Osmitrol, given through the veins). These medications must be used cautiously as they have significant side effects, including nausea, fluid accumulation in the heart and/or lungs (congestive heart failure and/or pulmonary edema), bleeding in the brain, and kidney problems. Their use is prohibited in patients with uncontrolled diabetes, heart, kidney, or liver problems.

Ophthalmologists often prescribe an eye drop containing more than one class of drug to patients who require more than one type of drug for control of their glaucoma. This simplifies the taking of drops by the patient. The most common

example of this is the combination of timolol and dorzolamidein the same drop (Cosopt).

Several new classes of glaucoma drops are currently under development or awaiting FDA approval. Although marijuana use has been shown to reduce intraocular pressure, eye drops are available which accomplish the same purpose with greater efficacy and less systemic risk.

GLAUCOMA SURGERY OR LASER

There are several forms of laser therapy for glaucoma.

- Laser iridotomy
- Laser trabeculoplasty
- Laser cyclo-ablation
- Trabeculectomy
- Aqueous shunt devices
- Viscocanalostomy

The surgeon sometimes creates other types of drainage systems. While glaucoma surgery is often effective, complications, such as infection or bleeding, are possible. Accordingly, surgery is usually reserved for cases that cannot otherwise be controlled.

CAN GLAUCOMA BE PREVENTED..?

Primary open-angle glaucoma cannot be prevented, given our current state of knowledge. However the optic-nerve damage and visual loss resulting from glaucoma can be prevented by earlier diagnosis, effective treatment, and compliance with treatment.

Secondary types of glaucoma can often be prevented by avoidance of trauma to the eye and prompt treatment of eye inflammation and other diseases of the eye or body that may cause secondary forms of glaucoma.

Most cases of visual loss from angle-closure glaucomas can be prevented by the appropriate use of laser iridotomy in eyes at risk for the development of acute or chronic angle-closure glaucoma.

WHAT IS IN THE FUTURE FOR GLAUCOMA…?

New eyedrops will continue to become available for the treatment of glaucoma. Some drops will be new classes of agents. Other drops will combine some already existing agents into one bottle to achieve an additive effect and to make it easier and more economical for patients to take their medication.

Many researchers are investigating the therapeutic role of neuro protection of the optic nerve, especially in patients who seem to be having progressive nerve damage and visual field loss despite relatively normal intraocular pressures. Animal models have shown that certain chemical mediators can reduce injury or death of nerve cells. Proving such a benefit for the human optic nerve, however, is more difficult because, for one thing, biopsy or tissue specimens are not readily available. Nevertheless, if any of these mediators in eye drops can be shown to protect the human optic nerve from glaucomatous damage, this would be a wonderful advance in preventing blindness.

In other studies, new surgical methods are being evaluated to lower the intraocular pressure more safely without significant risk of damage to the eye or loss of vision.

Finally, increased efforts to enhance public awareness of glaucoma, national free screenings for those individuals at risk, earlier diagnosis and treatment and better compliance with treatment are our best hopes to reduce vision loss from glaucoma.

RESEARCH

- **The Advanced Glaucoma Intervention Study** is a large American National Eye Institute-sponsored study designed "to assess the long-range outcomes of sequences of interventions involving trabeculectomy and argon laser trabeculoplasty in eyes that have failed initial medical treatment for glaucoma". It recommends different treatments based on race.
- **The Early Manifest Glaucoma Trial** is another NEI study which found immediate treatment of people who have early-stage glaucoma can delay progression of the disease.
- **The Ocular Hypertension Treatment Study**, also an NEI study, found topical ocular hypotensive medication was effective in delaying or preventing onset of primary open-angle glaucoma (POAG) in individuals with elevated intraocular pressure (IOP). Although

this does not imply all patients with borderline or elevated IOP should receive medication, clinicians should consider initiating treatment for individuals with ocular hypertension who are at moderate or high risk for developing POAG.

The Blue Mountains Eye Study was the first large, population-based assessment of visual impairment and common eye diseases of a representative older Australian community sample. Risk factors for glaucoma and other eye disease were determined.

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