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The glaucomas: A review of the literature

L. Akintunde O. Adegite  
*Pacific University*

Alfred Dib  
*Pacific University*

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The glaucomas: A review of the literature

Abstract
Although there are many different types of glaucomas, they can conveniently be broken down into four basic categories, namely: (1) open-angle glaucoma, (2) closed-angle glaucoma, (3) congenital/juvenile glaucoma and (4) secondary glaucoma, the first three being either of primary or secondary origin. In this paper, we review the literature on the four basic categories separately, subdividing each one into its etiology, incidence and prevalence, examination and diagnosis, prognosis, treatment and management.

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THE GLAUCOMAS:
A REVIEW OF THE LITERATURE

by

L. Akintunde O. Adegite

and

Alfred Dib

As partial requirement for the Doctor of Optometry Degree at Pacific University College of Optometry, Forest Grove, Oregon

Advisor: Dr. Alfred Furie

October, 1984
ACKNOWLEDGEMENTS

We take this opportunity to express our sincere gratitude to Dr. Alfred Furie, for his invaluable assistance in guiding us to prepare this paper.
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GENERAL INTRODUCTION AND OVERVIEW

Glaucomas are considered the family of ocular diseases in which increased intra-ocular pressure could lead to optic atrophy accompanied with excavation of the optic disc and with a visual field loss as its characteristic. The degree of increased pressure that causes organic change is not the same in every eye, and some individuals may tolerate for long periods a pressure that would rapidly blind another.

Roughly close to 15% of blindness in adult individuals in the United States is caused by glaucoma. Recent surveys established the fact that one in every forty individuals over age 40 years has this disease. With the present day advancement in medicine and technology, the blindness caused by glaucoma can be prevented.

It is the responsibility of every eye doctor and general practitioner to be aware of the present-day concept of glaucoma and its management.
The intraocular pressure is elevated to a dangerous level in the glaucomatous eye (although there is glaucoma caused by low-pressure, this type is called low tension glaucoma, but the most common is the one due to elevated pressure.) "The glaucomatous eye might be compared to an overly inflated basketball in which pressure is transmitted equally to all parts:" due to the optic nerve high susceptibility to increased pressure, loss of vision results.

The abnormal increase in intraocular pressure could result from obstruction (increased resistance) to outflow from the eye, increased rate of formation of aqueous or increased osmolality of the aqueous with retention of fluid within the eye. The first reason might be the major cause of primary glaucoma, but hypersecretion of aqueous is sometimes another cause.

Glaucoma Classification. (A) Primary glaucoma: (1) open-angle glaucoma/simple glaucoma/wide-angle glaucoma/chronic simple glaucoma. (2) angle-closure glaucoma/narrow angle glaucoma/closed-angle glaucoma/acute congestive glaucoma.

Types: (i) Acute
(ii) Subacute or chronic

(B) Congenital glaucoma: (1) primary congenital or infantile glaucoma/Buphthalmos/hydrophthalmos, (2) glaucoma associated with congenital anomalies.

(C) Secondary glaucoma\textsuperscript{7,8}:- due to:

(1) changes of the lens
(2) changes of the uveal tract
(3) trauma
(4) surgical procedures or complications
(5) rubeosis (diabetes mellitus and central retinal vessel occlusion)

(6) topical corticosteroids

(D) Absolute glaucoma: The end result of any uncontrolled glaucoma is a hard, sightless, and often painful eye.

**Genetics of Glaucoma:** Multifactorial or polygenic inheritance is associated with primary open angle glaucoma.$$^{9,10}$$ Autosomal dominant, autosomal recessive, and x-chromosome-linked inheritance of open angle glaucoma is described below.$^1$ The children and siblings of glaucomatous patients tend to have a higher intraocular pressure, lower facility of outflow, cup/disc ratio of more than 0.3, and increased IOP when topical corticosteroids are administered.

Diabetes mellitus and myopia are also risk factors that increase the likelihood of developing optic nerve disease with increased pressure.$^{11}$

The majority of patients with OAG (open angle glaucoma) develop increased intraocular pressure and a decreased outflow facility following the topical administration of 0.1% dexamethasone four times a day for six weeks. Continued increase in the IOP leads to optic nerve atrophy with excavation and a typical visual field defect; and the dexamethasone must be discontinued.

Based on the studies done by Broughton$^{12}$ and others, using four family members, it was postulated that the response to topical corticosteroids is genetically determined, so that those who respond with a marked increase in pressure (4%) have a genotype with a similar allele pair for high pressure (pH or gg).$^{11}$
Those who do not respond (64%) have a similar allele pair for low pressure (pL or nn), whereas those with an intermediate response (32%) are heterozygous for high and low pressure. Those who are homozygous for a high pressure tend to have positive glucose tolerance tests and low serum protein-bound iodine and tend to be nontasters of phenylthiourea.

Angle-closure glaucoma has a multifactorial inheritance related to the size of the anterior ocular segment.

Congenital, or infantile, glaucoma is transmitted as an autosomal recessive characteristic. Boys are affected twice as often as girls; cardiac, auditory, and cerebral defects may also be present. 12

**OPEN-ANGLE GLAUCOMA**

Open angle glaucoma is one of the most devastating ocular diseases, because once retinal damage is done, there can be no regression. It is characterized by the triad of findings consisting of raised intraocular pressure, optic disc changes and visual field loss. In open-angle glaucoma, the anterior chamber angle is wide open but there is an increased resistance to the outflow of aqueous humor through the trabecular meshwork. The exact mechanism of this increased resistance is not known.

Of all the glaucomas, the primary open-angle type is the most common, accounting for over 90% of the glaucomas. It is clearly established through studies of large sample populations that the distribution of intraocular pressure is not Gaussian but skewed towards the higher values of pressure. 13,14

**Incidence and Prevalence:** Bengtsson 15 found a prevalence of open-angle glaucoma of 0.86% in the age group 56-69 years but different authors have
found slightly differing prevalence rates. However, it is generally accepted that the prevalence rate in persons over 40 years old is between 0.41% to 0.86%, with an incidence rate of 0.1% per year.\(^{16}\)

According to the 1970 registry data\(^{16}\), glaucoma blindness was responsible for 11.1% of all blindness registration. Not only were the glaucomas the second single cause of blindness prevalence, but they were also the second cause of blindness incidence. Large differences exist between white and non-white registrants, with the glaucomas being the first cause of blindness prevalence and incidence in non-whites, whereas they were the fifth cause among whites.

Age, race, sex, socio-economic factors, genetic factors and systemic factors are all variables which seem to influence the risk of developing open-angle glaucoma. In persons over 60 years, there is an almost sevenfold higher incidence than in those under 40 years.\(^{16}\) The applanation pressures increase with age.

There is a difference between the intraocular pressures of men and women. In the study by Hollows and Graham,\(^{17}\) they found that except for the 40-45 year age group, women had higher applanation pressures than men. Although the difference is only of the order of 1 mmHg, it is statistically significant and is present in both right and left eye readings. Since physiological changes occur during the menstrual cycle in women, it would be expected to produce similar changes in intraocular pressure. Bankes and others\(^{18}\) have found that the lowest mean intraocular pressure (IOP) coincided with the 21st to 24th days of the menstrual cycle and the highest mean IOP occurred from the 9th to the 12th day. There is also another peak during the 25th to 28th day. The mean tension in post-menopausal women was higher than that in women who were
still menstruating, even when only those women in the age group 40-49 were compared.

Blacks seem to be at more risk for developing open-angle glaucoma with its associated visual field loss, and blindness from open-angle glaucoma (OAG) is much greater in blacks than in whites and occurs at a younger age. In their glaucoma screening program in Pennsylvania, Coulchan and co-workers found that black participants had higher mean intraocular pressures, more frequent pathological disc changes and more new cases of glaucoma than a sample of white participants matched for sex and age. In fact, the rates for glaucoma in blindness registries in the U.S.A. are 72 and 8.6 per 100,000 for blacks and whites respectively. Working in the Caribbean island of Jamaica, Wallace and Lovell also found that there is a higher prevalence of glaucoma in blacks and people of African origin in Jamaica than in the white population.

Several probable explanations can be offered for the greater end-stage chronic open-angle glaucoma seen in blacks as compared to whites. They are: (a) a greater amount of blacks have elevated intraocular pressures and therefore more develop glaucoma; (b) blacks may be more susceptible to developing visual field defects; (c) glaucoma begins at an earlier age among blacks, hence a greater time span for progression; (d) blacks have a higher blood pressure and this may be a factor responsible for the high IOP; (e) therapy may be less effective in blacks than in whites. The first four reasons would cause a higher incidence of OAG among blacks, while the last reason may account for the more destructive course.

Chronic simple glaucoma has a polygenic inheritance, with autosomal dominant, autosomal recessive and x-chromosome-linked inheritance being
described in the literature. Perkins describes it as a homozygous recessive condition, and found that the mean pressures in the right eyes of both men and women is 0.33 mmHg higher than that in the left eye, with the right eye being measured first. OAG is estimated to be hereditary in approximately one-fifth to one-quarter of the cases.

Socioeconomic and systemic factors seem to play a role in OAG too. Some authors find an increased risk of glaucoma with outdoor exposure, while others find a decreased risk. However, a case-control study by Morgan and Drance (pg. 180 of reference #16) found that persons with predominantly indoor occupations had a risk of 2.5 relative to persons who worked outdoors. Elevated systolic blood pressure, diabetes mellitus and obesity are a few of the systemic factors which have a significant association with open-angle glaucoma. It is generally believed that vascular factors are involved in the pathogenesis of field defects and in susceptible individuals, an elevated IOP would lead to ischemia of the optic nerve fibers and finally to field defects. The vascular changes associated with hypertension could directly increase the risk of field loss by reducing the blood flow to the optic nerve. A ratio of the systolic blood pressure to the IOP of less than 5.75 is suspicious of glaucoma.

**EXAMINATION AND DIAGNOSIS:** Open-angle glaucoma is an insidious disease, with the patient being unaware of the condition. Therefore, great responsibility is placed on the eye care practitioner in properly examining the patient and diagnosing the condition. Although there are other methods that could possibly aid in the diagnosis of OAG, the three most common methods used in examination and diagnosis are: (1) examination of the optic disc by
ophthalmoscopy and serial photography; (2) visual field testing; and (3) measurement of the intraocular pressure.

**Visual Fields:** The characteristic visual field defects in chronic simple glaucoma occur as a result of damage to the nerve fiber bundles. Although it is the most frequent cause, OAG is not the sole cause of such nerve fiber bundle defects. In chronic simple glaucoma, the inferotemporal and superotemporal areas of the nerve head seem to be the most vulnerable to damage, and the nerve fiber bundle defects most frequently involve the arcuate fibers which arch above and below the fovea. These fibers end along the horizontal meridian which extends from fixation to the nasal periphery of the field. Hence, one should always look for peripheral field defects along the nasal horizontal meridian. The visual field defects characteristic of OAG are the Seidel, Bjerrum, Roenne’s nasal step and tubular defects, but the shape, size and location of the nerve fiber bundle scotoma will depend on the extent and site of damage to the nerve fiber bundles at the optic nerve head.

Paracentral circumscribed defects can occur either in the temporal or nasal part of the Bjerrum area and tend to be elongated circumferentially along the course of the nerve fibers. On the temporal side of the central field, they classically occur in the Bjerrum region between 10 and 20 degrees from fixation in the area which constitutes the upward or downward arcuate projection of the appropriate pole of the blind spot. On the nasal side, the scotomata can come almost to fixation or alternately, be as much as 20 or even 30 degrees away from fixation. The Bjerrum region on the nasal side is very wide in
accordance with the course of the arcuate fibers. When first detected, the defects are often absolute or show deep relative nuclei surrounded by areas of less dense involvement. Dense nuclei are often multiple and lie along the course of nerve fibers and paracentral scotomata are often delineated by the nasal horizontal meridian.

The shape and width of the nasal steps depend on many factors, often being wedge-shaped in the periphery of the visual field. In the mid-periphery they tend to be more like a right angle, and closer to fixation, the nasal steps assume the characteristics of an obtuse angle, which is consistent with the shape of the nerve fiber bundles reaching the horizontal nasal meridian at that point. The width of a nasal step in degrees is variable and a nasal step may be present with one isopter and not with another. Usually, nasal steps are found together with paracentral and arcuate scotomata, the latter, bounded by its well-defined nasal horizontal border, often extending to the blind spot.

Static and kinetic perimetry remain the standard tools for assessing visual function in the diagnosis and management of glaucoma. Static testing with a threshold stimulus is the most sensitive way of detecting visual field defects for glaucoma but because it is time-consuming, it is not the most practical or efficient method. Using light sense perimetry, Hart and associates described three phases in which glaucomatous damage involving the distribution of nerve fiber bundles in a given Bjerrum region (superior or inferior) seem to occur. In the initial stage, no defect is found, even though occult damage is occurring. This phase may be marked by prolonged periods during which the visual field appears normal in spite of marked elevations in
intraocular pressure. The second phase is a threshold period during which shallow defects are often transient and are just barely detectable with the most sensitive perimetric tests. The third phase is a critical period during which manifest perimetric defects progress at an uneven pace to become very dense. This progression usually appears within a decade and may occur in spite of only marginally increased intraocular pressure.

Elevated IOP alone may not necessarily cause a field defect. In fact, it has been determined from epidemiological studies that one-third to one-half of persons with glaucomatous field defects have intraocular pressures of less than 21 mm Hg when first detected. Even before visual field defects are detected, ophthalmoscopic changes involving small splinter hemorrhages of the optic disc and defects of the retinal nerve fiber layer may be seen. These have been reported to be the first signs of glaucomatous development. The bleeding recurs at a specific location, probably for as long as there is tissue left, and then shifts to another location which often seems to be more temporal, closer to the macular fibers, or at the opposite pole of the disc. The association between retinal nerve fiber layer defects and disc hemorrhages suggest that the development of the former is somehow connected to localized vascular disturbance in the disc.

In cases where visual field anomalies can be detected, crowding of the peripheral nasal isopters serve as important clues in the early detection of glaucoma. Crowding of the peripheral nasal isopters is said to be present when the nasal isopter, outlined with the peripheral threshold target, is less than 55 degrees from fixation and within 5
degrees of the nasal isopter plotted with the V4e target (largest, most intense) of the Goldmann perimeter.  

The characteristic nasal step of glaucoma is due to a disparate depression of the nasal isopters above and below the median raphe but in some cases the depression is symmetrical on either side of the horizontal midline, thus creating a steep nasal margin with crowding of the peripheral nasal isopters. In such a case, the defect can be easily overlooked.

**Contrast Sensitivity and Electrodiagnostics:** Although contrast sensitivity measurements are rarely used in the diagnosis of glaucoma, there is evidence that in patients with open-angle glaucoma, such measurements are abnormal. Atkin and co-workers found that the contrast sensitivity either to diffuse flicker or to the counterphase flickering grating of low spatial frequency was lower in glaucoma patients than in control subjects. They used the term dynamic response coefficient (DRC) to describe the average of the contrast sensitivities to diffuse flicker and to counterphase flicker for each of their subjects and found that for the glaucoma patients, the DRC's were consistently below the control values, whether or not IOP was high at the time of testing and irrespective of the therapy used. For glaucoma patients who retain nearly normal Snellen acuity, the visual field outside of the central 5 to 10 degrees has been the area in which abnormalities of either spatial or temporal thresholds are most commonly seen.

Hitchings and others also found an abnormal contrast sensitivity in glaucoma patients, which had a positive correlation to the cup/disc
ratio, field loss and age. However, they found no such correlation between contrast sensitivity and IOP. Progressive worsening of the contrast sensitivity in glaucoma suspects may possibly be a precursor of overt glaucomatous damage and is potentially another clinically useful sign of such damage, in addition to nerve head cupping and field loss.

The electroretinogram (ERG) and visually evoked potential (VEP) in open-angle glaucoma patients were also investigated by Bobak and co-workers. They found an abnormal pattern ERG, based on the absence of a significant second harmonic component in three of the four glaucomatous eyes, although the homogeneous field ERG was normal.

**Miscellaneous Findings:** Using fluorescein angiography, it has been determined that the flow of blood through the glaucomatous eye is qualitatively different from normal. Spaeth has described the following differences: (a) A prolonged time for the dye to move from the median antecubital vein, where it is injected, to the eye. (b) Dye entered and left the globe more slowly in patients with glaucoma. (c) The glaucomatous eye did not light up as brightly as the non-glaucomatous eye. (d) There was greater embarrassment of the choroided vessel system than of the central retinal artery system of blood vessels. (e) Some glaucomatous eyes showed abnormal peripapillary flush that was not anticipated on the basis of the ophthalmoscopic appearance of the fundus.

Increased IOP is not solely responsible for the abnormal vascular supply to the choroid and consequently to the laminar and pre-laminar areas of the optic nerve, but it makes the vascular abnormality even more apparent.
It has also been observed that myopic eyes are more vulnerable to the effects of raised intraocular pressure than non-myopic eyes and that myopes with ocular hypertension have a particularly high risk of development of field defects, being worse for high myopes. The results of the study by Perkins showed that myopia occurs more frequently in patients with ocular hypertension, low tension glaucoma and primary open-angle glaucoma than in a normal population of similar age, the biggest difference being found in the primary OAG group and the smallest in the ocular hypertensive group.

Daubs also supported the increase in field loss at higher levels of myopia but showed that IOP has little or no effect on refractive error or vice versa. Diagnosing glaucoma in a high myope is not always easy though. Supertraction of the choroidal and scleral tissue may mask glaucomatous nerve fiber atrophy so that cupping and vessel deviation are not seen, and the myopic conus may make evaluation of the optic disc confusing and could produce an enlargement of the blind spot. Therefore, the visual field may be distorted only because of the high refractive error.

**TREATMENT:** Treatment of open-angle glaucoma primarily involves the use of medical drugs and, in a few instances, of laser or conventional surgery. In many instances, patient non-compliance with the medical regimen is the major hindrance in properly controlling the disease. Such a non-compliance can result from sheer negligence by the patient or from a lack of understanding of the serious ocular consequences of not taking treatment as directed by the eye-care practitioner. The cost of
the medication and the inconvenience, side-effects and discomfort experienced by the patient are also factors worthy of consideration. There is a substantial drop in compliance when three or more daily doses of medication are prescribed as compared to once or twice daily regimens. For the thrice-daily regimen, Novell\textsuperscript{33} found that the noon dose was missed more than twice as often as the morning or evening doses. Hence, the risks and benefits of any medication should be carefully assessed before initiating treatment.

The anti-glaucoma agents are categorized as:
(a) direct acting parasympathomimetics, such as pilocarpine (b) anticholinesterases, such as echothiophate iodide (c) Beta-adrenergic antagonists, such as timolol (d) Adrenergic receptor stimulants, such as epinephrine (e) Alpha-receptor blockers, such as thymoxamine (f) the carbonic anhydrase inhibitors, such as acetazolamide (g) Hyperosmotic agents, such as glycerol

The following table summarizes the dosages of some of the commonly used drugs for open-angle glaucoma:

<table>
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<tr>
<th>Drug</th>
<th>Concentration</th>
<th>Dosage</th>
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<tr>
<td>Timolol (Timoptic, Timoptol)</td>
<td>0.25%, 0.5%</td>
<td>1 gtt. b.i.d.</td>
</tr>
<tr>
<td>Pilocarpine (Pilo-2)</td>
<td>1%, 2%</td>
<td>1 gtt. q6h</td>
</tr>
<tr>
<td>Epinephrine (Eppy)</td>
<td>1%, 2%</td>
<td>1 gtt t.i.d.</td>
</tr>
<tr>
<td>(Propine-prodrug)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Acetazolamide (Diamox)</td>
<td>250 mg</td>
<td>q6h</td>
</tr>
</tbody>
</table>
Further details of the different drugs can be obtained from the books of Pavan-Langston, Scheie and Albert and Newell.

Some patients receive a combination of the anti-glaucoma agents. While they individually lower IOP, combinations are usually only partially additive. Such is the case with timolol maleate and epinephrine, which, when used concurrently, produce a greater reduction in IOP than either agent alone. However, the total percentage reduction in IOP is less than the sum of each individual agent's ocular hypotensive effect. Therefore, when one of the drugs is added to the one being taken, a dramatic long term reduction in IOP is unlikely.

New drugs are constantly being evaluated and Berrospi and others have described favorable responses with little or no side effects, from the topical instillation of 0.25% betaxolol hydrochloride. New surgical techniques are also being used, the argon laser being the most recent. Multiple circumferential low energy treatments, without attempting to penetrate into the canal of Schlemm, appear to produce a persistent reduction in outflow resistance, without excessive inflammation or scarring. The success rate is relatively uniform beyond the age of 40 years but eyes with pressures greater than 35 mm Hg in spite of intensive medical therapy, seem to respond poorly to the argon laser treatment. The anterior chamber must be sufficiently wide (grade 3 or greater) in order to do laser treatment, otherwise peripheral anterior synechiae can occur.

Using conventional surgery, twice as many patients receiving a trabeculectomy required further medical therapy to keep the IOP within
satisfactory boundaries, as compared to those who received a peripheral iridectomy with thermal sclerostomy.\textsuperscript{37}

All in all, the patient with open-angle glaucoma must be monitored carefully and the best possible treatment given with the aim of preventing and controlling any progression of ocular damage.

**CLOSED ANGLE GLAUCOMA**

**Etiology of Closed Angle Glaucoma.**

Glaucoma is caused by a lot of abnormalities in the eye, such as:

(a) Sturge Weber syndrome/Encephalofacial angiomatosis

(b) Plateau iris\textsuperscript{38}

(c) Ghost cell glaucoma following trauma

(d) Buphthalmos

(e) Lens induced glaucoma\textsuperscript{39}, such as lens swelling or lens dislocation

(f) Neurovascular glaucoma\textsuperscript{40}

**Angle Closure Glaucoma**

Types: (a) primary type

(b) secondary type

**Primary Angle Closure Glaucoma.**

Narrow Angle glaucoma (iris block, angle closure, angle congestive glaucoma)

In this type of glaucoma, the peripheral iris covers the trabecular meshwork preventing access of aqueous to the trabecular meshwork. The major predisposing factor is a narrow anterior chamber, or shallow anterior chamber.
As a result of this, at times the iris contact with the trabecular may obstruct the outflow of aqueous and blockage occurs. Due to this outflow blockage, the intra-ocular pressure increases, and this causes the acute congestive attacks, which characterize the disease.

Narrow angled eyes behave normally until the angle is closed or blocked. A narrow anterior chamber angle is a combination of anatomical and functional factors. The anatomical factor is attributed to an anterior position of the lens-iris-diaphragm, probably hereditary. The functional factor is in the form of a physiological iris bombe which occurs from resistance to aqueous flow from the posterior to the anterior chamber over the area of contact between the posterior surface of the lens. The cornea is often small in diameter, and the eye is hyperopic, the lens is closer to the cornea than usual, and with the normal increase in size of the lens with aging, it becomes ever closer. The iris appears to bow forward, so that it seems to closely parallel the posterior convexity of the cornea. (See Figure A).

By shining a penlight into the anterior chamber from the temporal side of the eye, this may be observed. (See Figure B). A shadow which is not present in the normal eye is cast by the nasal portion of the relatively convex iris. When there is anterior displacement of the peripheral iris, increased IOP occurs. This anterior displacement causes the trabecular meshwork to be isolated from the anterior chamber, preventing the exit of aqueous humor. The displacement could be caused by two conditions operating singly or together.

The outflow of aqueous humor through the pupil is impaired, causing aqueous to accumulate in the posterior chamber. This pupillary block (iris bombe) causes increased pressure in the posterior chamber so that the
Mechanical crowding of the angle by the iris isolates the trabecular drainage apparatus from the anterior chamber.

Dilation of the pupil causes the iris to become thicker and crowd into the drainage apparatus, preventing aqueous outflow, and consequentially the IOP increases.

**Incidence of Narrow Angle and Grade:** The size of the angle could be graded into four categories.

- Grade 1 (<1/4) incidence - 0.64%
- Grade 2 (=1/4) incidence - 1%
- Grade 3 (>1/4 <1/2) incidence - 60%
- Grade 4 (>1/2) incidence - 38%

Angle size could be affected by age, refractive error, genetics and race. Overall, the incidence of narrow angle glaucoma is 0.09%.

Narrow angle could be classified into three major groups:

1. Pre-glaucoma and interval phases
2. Acute congestive (iris block, angle closure) phase
3. Chronic narrow angle (iris block, angle closure) phase

Preglaucoma phase occurs in patients with narrow angles but no symptoms of glaucoma. In this individual the IOP is normal but this could be elevated by provoking pupillary dilation. Both preglaucoma and interval phases are very much identical. The interval phase is found in patients with a known history of acute attack of glaucoma, which is not the case with preglaucoma phase.

Classical symptoms of interval phase include: headaches, blur vision and halos around light; discomfort may be marked or only a dull headache localized
around the eye.\textsuperscript{3} The edema of the corneal epithelium resulted in halos. This usually occurs when the pupils tend to dilate, as in semi-darkness or in the movie theatre. In a well lighted room, all these signs and symptoms subside after a few minutes, when the pupils constrict.

An emotional factor also can be a predisposing factor.\textsuperscript{6}

In both preglaucoma and interval phases, the optic nerve is normal; because the eyes have not been subjected to a very long period of elevated pressure.

When the individuals in both preglaucoma and interval phase come for routine eye examinations, they are free of any symptoms. Routine tonometry usually does not or is not helpful in the diagnosis of either preglaucoma or interval glaucoma, because the IOP is normal, visual fields also are normal; reflecting the healthy state of the optic nerve.\textsuperscript{41}

**Acute Congestive Phase (Iris Block, Angle Closure):** Due to the contact of the iris root and the trabecular meshwork, the angle between the iris and trabecular meshwork is completely closed. (Figure C). At this point, the pressure is completely elevated. However, with proper medical therapy, angle closure (iris block)\textsuperscript{38} is very reversible. If the pressure cannot be relieved within 48 to 72 hours, adhesions that develop between the iris root and the trabecular will not be relieved and chronic narrow angle closure ensues.

This type of angle closure is very easy to diagnose. There is always a severe pain in the eye, regional headaches and blurred vision dominate the picture. Sometimes gastro-intestinal symptoms mask the acute glaucoma. Severe nausea and vomiting caused by the vagus reflex may obscure the ocular symptoms and result in a false diagnosis of acute abdominal condition such as gall bladder disease.\textsuperscript{2,6}
In patients with abdominal surgery, acute glaucoma could be overlooked. Pre- or post-operative medication could be a factor. Ocular pain and inflammation in a patient after general surgery are most commonly due to acute congestive glaucoma or corneal abrasion resulting from mechanical injury or from an irritative anesthetic agent.

In acute glaucoma, blur-vision results from edema of the corneal epithelium, as well as from depressed function of the retina and optic nerve. Shallow anterior chamber, an edematous cornea, and a red, inflamed eye, often with edematous conjunctiva, are revealed at external examination.

Ophthalmoscopic, slit lamp biomicroscopic and gonioscopic examinations are often difficult due to corneal haze.

Sub-Acute/Chronic Narrow Angle (Angle Closure) Phase: This is caused by the same etiology as acute-angle closure glaucoma. The difference is that there is no sudden complete block to aqueous outflow by the iris being pushed against the trabeculum. The iris extends its arc of contact with the trabeculum gradually until an adequate area of angle is no longer available for aqueous outflow. Mild or severe symptoms are associated with chronic narrow angle glaucoma. The diagnosis is obvious when it follows an unrelieved attack of acute congestive glaucoma, for the classic signs of acute congestive glaucoma (red eye, hazy cornea, usually marked iris atrophy, shallow anterior and closed angle) are still present.

Chronic narrow angle may result due to pressure gradually rising because of constant increase of synechiae from repeated mild attacks of acute congestive glaucoma.
Progressive cupping and field loss are predominant signs and symptoms of chronic narrow angle glaucoma. Occasional mild attacks of increased intraocular pressure cause transient blurring of vision, halos around lights and possibly slight pain in or about the eyes. On examination, one finds a shallow anterior chamber, high intraocular pressure (25-50 mm Hg), and a closed chamber angle, as seen on gonioscopic examination.

**Diagnosis/Provocative Tests:** Diagnosis - Various factors need to be taken into consideration when trying to diagnose whether an eye is glaucomatous due to anatomical structure or functional orientation of the iris or lens. These factors are: visual acuity, functional symptoms, external examination of the eye, posterior ophthalmoscopic examination of the eye, slit lamp examination, intraocular pressure, gonioscopic examination, visual fields and provocative tests (dark room, mydriatic agents) and tonographic studies.\(^6,43\)

**Visual Acuity:** The acuity is badly affected in both acute congestive and chronic angle closure glaucoma.

**Functional symptoms:** As earlier said, there may be repeated attacks of ocular pain and blurred vision occurring after a prolonged time in darkness (see provocative test under darkroom test); after emotional upset, or after similar situations that cause pupillary dilation. The rapid increase in intraocular pressure causes an epithelial edema of the cornea with blurred vision and rainbows surrounding street lights (iridescent vision).

**External Examination of the Eye:** Shallow anterior chamber is noticed by placing a penlight on the temporal side of the globe. A shadow is created which is absent in an open-angle eye. (See Figure B). Iris atrophy is present.\(^6\)
Posterior Ophthalmoscopic Examination: Posterior examination reveals normal to severe atrophy or cupping. The optic disc is evaluated with respect to the ratio between the horizontal diameter of the physiologic cup and the horizontal diameter of the entire disc; and to the uniformity of the rim of optic nerve surrounding the physiologic cup. The physiologic cup usually has a white, gray, or reddish white color distinctly lighter than the surrounding optic nerve. In glaucoma, the ratio of cup to disc usually exceeds 0.5, and a difference of more than 0.2 between the two eyes should suggest the possibility of glaucoma. The nasal slope is much steeper than the temporal slope. The nerve fiber layer of the retina is reduced, particularly in regions corresponding to notched defects in the nerve tissue of the disc.

Slit Lamp Examination: Possible sector iris atrophy and narrow angle, also epithelial edema and aqueous flare.

Intraocular Pressure: The intraocular pressure is measured by two distinct methods; applanation and indentation. The instruments used are Schiotz—which is an indentation tonometer, Goldmann—which is an applanation tonometer, Mackay-Marg—which is an applanation tonometer, that records the pressure electronically, pneumatic tonometer and the non-contact tonometer. 41

Although normal IOP is presumed to be 20 mm Hg or less, as with all physiologic measurements, variations occur from individual to individual and from day to day. The ability of a given eye to withstand a given level of intraocular pressure may not be the same. A borderline elevation in pressure, therefore, may be normal for one individual whereas for another the eye may not tolerate pressures well within the normal range.

However, the IOP in narrow angle ranges from normal to markedly high. Normal individuals have a mean intraocular pressure of about 15 mmHg +3 mm Hg.
Untreated glaucomatous eyes with field loss have a mean intraocular pressure of 24 mm Hg ± 5 mm Hg. Intraocular pressure tends to increase with aging and to be higher in women than in men.  

**Gonioscopic Examination:** The angle of the anterior chamber is difficult to inspect due to the opaque scleral and corneoscleral limbus. However, it is possible to see this area by means of a contact lens and mirror or a contact lens combined with a prism. The angle varies from closed to synechiae formation, as revealed by the gonioscopy.  

**Visual Fields:** The visual field defects range from normal to slight constriction to extreme glaucomatous damage. According to Harrington's scheme of progressive field loss, the following occur.  

(a) General depression of all isopters  
(b) Greater depression of peripheral isopters  
(c) Particular depression of superior nasal isopters  
(d) Abnormal enlargement of blind spot  
(e) Widening of angioscotoma  
(f) Nerve fiber bundle defects  

**Provocative Tests:** This is done to determine whether or not the angle can be closed and a rise in pressure induced.  

(a) Dark room test: Doing this test, the patient is allowed to stay in the dark room for about one hour. The IOP should be taken immediately, since the pressure normally falls rapidly when the patient is brought into the light and the pupil constricts. If it happens that there is discomfort during the test, the pressure should be taken before an hour. A significant rise in pressure should be treated with miotic medication to reduce the pressure.
(b) **Chemical mydriasis tests:** Various mydriatic agents may be used, such as eucatropine, 5 percent; mydriacyl, 1 percent; and homatropine, 2 percent. Usually drops are instilled twice in each eye, five minutes apart, and the IOP is recorded at 20 minute intervals, for one and a half hours. The advantage of chemical testing is that gonioscopy can be done to confirm angle closure should the pressure rise.

In a situation that the angle remains open by gonioscopy after a rise in pressure, chronic simple glaucoma should be suspected, because pupillary dilation can have the reverse effect of miotics on the trabecula, increasing resistance to aqueous outflow with a resultant rise in pressure. Usually a pressure rise of 8 mm or more is considered positive, however, much higher elevations are characteristic of narrow angle glaucoma.

To neutralize a chemical mydriatic 0.5% physostigmine is used. This is better than pilocarpine because prompt miosis is achieved with 0.5% physostigmine. In cases where the pupil does not constrict and the IOP continues to rise, Diamox should be given as well as urea or glycerol.

After the IOP has been controlled, pilocarpine should be prescribed for use four times daily for the following two days.

Whenever a patient is exposed to the hazards of a mydriasis test, whether or not the test is positive, he should be kept under observation until the pupil constricts.

It is very important to know that a positive mydriasis test is highly significant, but a negative test can be misleading. Most patients whose mydriasis tests are negative often suffer attacks of acute congestive glaucoma in the near or far future. These patients should be informed about this and mydriasis tests should be repeated from time to time.
Treatments: Closed or narrow angle glaucoma is an ophthalmic surgery emergency, and it is very important to make diagnosis before synechiae occur. Although surgery is the most preferrable choice of treatment, it is highly important to exhaust all the drugs or medical therapies before resorting to surgery.\(^1\,\)\(^6\)

Medical or Drug Therapy for Acute Congestive or Angle Closure Glaucoma: An attack of this type of glaucoma, as earlier suggested, should be primarily treated with drugs, followed by surgery whenever possible. The following drugs are of possible use.

**Systemic osmotics:** Oral glycerol. Dosage: 1.0 - 1.5 gm/kg or intravenous mannitol - dosage 1.0 - 2.0 gm/kg. Intravenous acetazolamide, dosage 250 - 500 mg. (A cetazolamide is a carbonic anhydrase inhibitor)

Any of the above mentioned systemic osmotic drugs should be given initially to lower the IOP below 50-60 mm Hg.

**Miotic agents:** (1) Pilocarpine concentration 1% - 4%.

Dosage - One drop in each eye every 15 minutes.

Side effects: (a) myopia-fluctuation

(b) Miosis

(c) Browache

(d) Sweating

(e) Gastro intestinal over-activity

(2) Eserine or any anticholinesterase such as echothiophate, demecarium, diisopropyl-fluorophosphate -- concentration 1/2%.

Dosage - One drop in each eye every 15 minutes. Eserine is more effective than pilocarpine because of its ability to inactivate cholinesterase,
permitting acetylcholine to accumulate in the tissues. Aqueous outflow is increased by ciliary muscle contraction pulling on the trabecular meshwork.

**Side effects:**
(a) painful accommodative spasms  
(b) cataracts  
(c) retinal detachment  
(e) increased G.I. activity

It is very important not to use pilocarpine initially because of its ability to occupy the end plate and partially block the effect of acetylcholine. Strong miotic agents should be avoided; such as floropryl, Humorsol and phospholine iodide; these are dangerous and should be avoided at all cost. It is highly recommended to instill the miotic always in the fellow eye to avoid the danger of an acute attack.

**Carbonic Anhydrase Inhibitors:** This decreases the rate of aqueous formation and allows the pressure to fall.

**Side effects:**
(a) malaise  
(b) fatigue  
(c) depression  
(d) G.I. upsets  
(e) renal stones  
(f) numbness, tingling

(1) Diamox: This is widely used. This should be given by mouth.

**Dosage** - 500 mg, repeated with 250 mg every four hours. In case the patient is nauseated, the same amount can be given intra-venously.

**Surgery:** As earlier said, surgery is the end therapy. The drug therapy is only instituted to lower the IOP until surgery is done.
Peripheral iridectomy is the operation of choice. This is carried out in the superior temporal quadrant through a small scratch incision. A small opening is made in the iris, and the corneoscleral wound is closed with sutures to be watertight. The eye tolerates the procedure well, provided the anterior chamber does not remain flat post operatively, a complication avoided by careful wound closure.  

An iridotomy is another type of procedure which may be done with a laser, instead of surgical incision. Topical local anesthetic is applied, a mild laser beam is used to burn a localized area of the mid peripheral iris, and this is caused to bulge forward. The central portion of this area is then perforated by a laser beam of a much higher energy level. Sometimes many perforations need to be made, or at times this could be impossible to do, if, for instance, the iris is highly pigmented or non-pigmented.

Therapy for:

**Acute chronic closed angle glaucoma:** This type of glaucoma responds poorly to drug therapy. Because the angle is closed due to synechiae, a filtering operation, usually an iridencleisis or an iridectomy with scleral cautery, is necessary to avoid further deterioration of the eye.

**Congenital Glaucoma**

Congenital glaucoma can be divided into two basic groups: (a) primary and (b) secondary glaucoma.

Primary congenital glaucoma is called infantile glaucoma, and this is caused by an increase in intraocular pressure secondary to impairment of the outflow of aqueous humor occurring because of an embryonic defect in the trabecular meshwork.\(^9,\)\(^12\)

Secondary congenital or juvenile glaucoma occurs in systemic disorders. Heredity also plays a part in the cause of congenital glaucoma.
Juvenile Glaucoma. This type of glaucoma occurs between the ages of 3 to 20 years. Some authors would say this occurs up to age thirty but generally the limits of ages set for the juvenile glaucoma is arbitrary. It is possible that glaucoma occurring at age 25 can have the same origin and run the same course as glaucoma at 35 to 40 years.\textsuperscript{3,11}

Juvenile glaucoma runs the insidious course of chronic simple glaucoma and presents the same diagnostic problems; the diagnosis can be more challenging because the presence of glaucoma often is not suspected in younger age groups.

In many instances the disease is discovered only after marked glaucoma cupping and severe visual loss have occurred. (Figure D).

Most often, the glaucoma cupping may be atypical in young persons, especially in the presence of myopia. The cup tends to be shallow and broad in this instance.

Generally speaking, glaucoma is more severe in the juvenile age group, and the intraocular pressure tends to be more labile with greater day to day variance. Dramatic rises in pressure accompanied by halos are more frequent than in chronic simple glaucoma of older persons. All children and young adults with rapidly progressive myopia should be suspected of having glaucoma. The effect of elevated pressure aggravates myopia just as it will affect a distensible sclera and cornea of young people.\textsuperscript{7}

Diagnosis: Methods used in diagnosing juvenile glaucoma and chronic simple glaucoma are identical. The following congenital systemic and ocular disorders are very much predisposing factors to juvenile glaucoma. -- Neurofibromatosis, Sturge-Weber-Dimitri syndrome, or Lowe syndrome, posterior embryotoxon (Axenfield's syndrome), aniridia, spherophakia, congenital
dislocation of the lens, signs of pigment dispersion in the form of Krukenberg's spindle. Heredity is also a compounding factor. Hereditary congenital glaucoma has a genetic heterogenicity, and there are more sporadic than familial cases. Occasional pedigrees are seen with the parent autosomal dominant transmission, while others occur as an autosomal recessive.\(^\text{11}\)

The major pathological-anatomical situation is that the drainage of the aqueous humor is interfered with by the defects, causing an increase in intraocular pressure, which in turn causes stretching of the elastic coats of the eye with marked enlargement of the globe (total staphyloma, Figure F, Buphthalmos) and optic atrophy with excavation.

**Treatment:** Response to drug/medical therapy is very poor in juvenile glaucoma. Surgery is the best method of therapy. Even those people who do very well on drug/medical therapy, hardly can use it to preserve their vision for a lifetime. In situations where miotic drugs are used, they tend to cause spasms of accommodation with discomfort and blurred vision due to artificial myopia, so the youthful and often irresponsible patient tends to use them unfaithfully. In spite of all these, an adequate drug therapy should be given a thorough trial.

A drug such as weak solutions of phospholine iodide, 0.06%, has a prolonged effect that results in a more or less constant refractive change throughout the day. This can be corrected by spectacles. Phospholine iodide is tolerated better than pilocarpine, which must be used three to four times a day and causes episodic spasms of accommodation and artificial myopia after each instillation. The above symptoms disappear in one to two hours but recur with each instillation to the greater inconvenience of the patient.
Epinephrine is another drug that is useful in controlling the pressure and this could be used when the disease is mild or as a supplement to miotic therapy.

Carbonic anhydrase inhibitors have little value because it is not likely that they could be used continuously for a long period of time.

As earlier said, surgery is the best therapy. Reasons for surgery are similar to those for chronic simple glaucoma and include situations where there is progressive cupping of the optic nerve and loss of visual fields.

Filtering procedures such as goniopuncture usually are done. Goniopuncture consists of making a small perforation in the angle wall. It is simple and safe to perform and is accompanied by few operative or post-operative complications.

The eye retains its normal appearance and its function is not disturbed.

If all things fail, more conventional but more hazardous procedures, such as iridencleisis, trephination, iridectomy with scleral cautery, and various types of sclerectomy are used.
Infantile Glaucoma

Infantile glaucoma could be primary or secondary to other ocular congenital anomalies and diseases, such as ocular inflammation and tumors. Infantile glaucoma accounts for approximately 5 percent of blindness among persons in schools for the blind in the U.S.A., a lot of which could have been prevented very early by early diagnosis and proper management. Infantile glaucoma is transmitted as an autosomal recessive characteristic. Boys are affected more often than girls, and the disease is bilateral in 75% of the cases; present also may be cardiac, auditory and cerebral defects. Signs of infantile glaucoma may be present and even far advanced at birth or become apparent before the child has reached 3 months of age.

Earliest symptoms are lacrimation, blepharospasm, and photophobia. Corneal edema with a ground glass appearance is revealed during the examination, which obscures the pattern of the iris. Increase in corneal diameter is noticed from 10.5 to 12 mm or more; breaks in the descemet membrane as glassy lines on the back surface of the cornea is also noticed. The anterior chamber is deeper than normal. The best thing to do when taking the IOP is to avoid the use of general anesthesia, as this might increase the IOP due to ketamine or decrease the IOP due to halothine anesthesia; the use of pneumatographic or air tonometer without general anesthesia has been suggested.

Diagnosis: Early diagnosis (as earlier said) of the infantile glaucoma is very essential. With the present-day surgical techniques, at least 80% of the eyes involved could be saved. However, it is very important to know that, if the surgery is not properly performed, or if the patient is not successfully
operated upon, the eyes would enlarge progressively until profound visual loss and eventual blindness occur.

As in glaucoma of adults, damage is permanent. In one-third of the patients, the disease occurs in utero and typical signs are present at birth. The obvious diagnosis is enlarged eyes (Buphthalmos) and hazy cornea. (Figure F).

Since damage to the eye and impaired development have occurred in utero, the visual prognosis, even with control of pressure, is much poorer than in the other two-thirds of infants in whom the disease develops after birth. If the disease is asymmetrical, insurmountable amblyopia is common, particularly in the eye that shows more severe impairment.

It is worth noting that early diagnosis of infantile glaucoma that develops after birth can be as difficult as diagnosis of chronic simple glaucoma in the adult.

The following are the diagnostic signs stemming from the vulnerability of the cornea and sclera to elevated intraocular pressure: photophobia, Blepharospasm, tearing, corneal haze, enlargement of the cornea, ruptures in Descemet's membrane, cupping of the optic nerve, and elevated intraocular pressure.

**Differential Diagnosis:** Many conditions simulate infantile glaucoma and must always be excluded. The most common are megaloo cornea, high myopia, congenital idiopathic edema of the cornea, rubella keratitis, corneal injury during birth with ruptures of Descemet's membrane and corneal edema, cornea Lipoidosis, cystinosis and the mucopolysaccharidoses (MPS) that are associated with corneal opacity. The latter include Hurler's syndrome (MPS₁), Hunter's
syndrome (MPS$_2$), Sanfilippo's syndrome (MPS$_3$), Morquio's syndrome (MPS$_4$), Scheie's syndrome (MPS$_5$) and Maroteaux-Lamy's syndrome (MPS$_6$).$^{1,2,3,6}$

Differentiation of the above may require examination and measurement of corneal diameter and intraocular pressure.

**Treatment:** Drug or medical therapy is of no value; surgery should be prompt. Drug or medical therapy only permits the eye to deteriorate. Goniotomy, alone or combined with Goniopuncture, is the procedure of choice.

This operation will control IOP in 80 to 85 percent of uncomplicated eyes, and where goniotomy fails, iridectomy with scleral cautery can control the pressure in about half of the eyes.

Good visual prognosis is best when the disease develops in infants from three to six months of age who at the time of surgery show little ocular enlargement or damage. In children born with obvious signs of infantile glaucoma, especially if other anomalies such as aniridia are present, the prognosis is much poorer.

**THE SECONDARY GLAUCOMAS**

The secondary glaucomas can be broken down into the open-angle and closed-angle types.

**Secondary Open-Angle Glaucoma:** Secondary open-angle glaucoma is a glaucoma that results from any associated ocular abnormality or disease but in which the angle remains open. Among the causes of such a glaucoma are:

(a) iritis and uveitis
(b) cataract surgery
(c) corticosteroid use
(d) high episcleral venous pressure
(e) intraocular tumors
(f) neovascularization
(g) pigment dispersion
(h) posterior polymorphous dystrophy
(i) exfoliation and pseudoexfoliation
(j) ocular trauma
(k) systemic diseases

It is not uncommon for glaucoma to develop after surgery, cataract surgery and retinal detachment surgery being prime examples. The routine use of alpha-chymotrypsin with watertight wound closure in intracapsular cataract extraction is a frequent cause of post-operative glaucoma. The presence of a retinal detachment frequently results in a decrease in aqueous secretion and also in the coefficient of aqueous outflow. In most patients, the former is greater than the latter, frequently resulting in hypotony. In retinal detachment, glaucoma can therefore occur pre- or post-operatively. When it does, medical therapy must be started, and glaucoma surgery done for recalcitrant cases, otherwise the persistently elevated IOP could cause Schlemm's canal to collapse, with resultant damage to the outflow passages.

Some drugs, particularly the steroids, predispose to glaucoma development. Glaucoma and cataract are the most important potentially dangerous ocular consequences of the long term use of topical or systemic steroids. Therefore, close follow-up with careful study of the IOP changes is essential for any patient on long term steroid treatment plans. The changes in IOP and the decreased outflow facility occur only after prolonged therapy (3-8 weeks) but when the steroid therapy is stopped, the glaucoma can be controlled.
Central retinal vein occlusion and diabetic retinopathy are the most common causes of neovascularization of the anterior ocular segment. Systemic vascular disease is a particularly unusual cause of neovascular glaucoma but is frequently overlooked. Rice and workers found that the incidence of neovascular glaucoma increased by more than four times when a lensectomy was done during vitrectomy for diabetic retinopathy as compared to eyes in which the lens was not removed. The incidence of iris neovascularization also rose by a factor of more than three. The iris neovascularization obstructs the trabecular outflow channels, resulting in neovascular glaucoma.

The risk of developing neovascular glaucoma (NVG) is approximately 60% in those eyes with extensive retinal ischemia and it seems that eyes treated with penretinal photocoagulation (PRP) prior to iris neovascularization do not develop NVG.

Pigmentary glaucoma is a rare glaucoma that most often occurs in young myopic men. Its incidence decreases with age and in some cases, the disease regresses or improves with time. The pigment is liberated from the posterior iris epithelium and is deposited throughout the anterior segment on structures that are in contact with aqueous currents, such as the posterior peripheral lens surface, the anterior iris surface, the corneal endothelium, and the trabecular meshwork.

The pigmentary obstruction of the trabecular meshwork causes the glaucoma. The pigment from the iris is lost in a peripheral radial slit-like fashion and such slit-like transillumination defects, together with a Krukenberg's spindle and dense pigmentation of the trabecular meshwork, are considered pathognomonic for pigmentary glaucoma. The cause of the loss of iris pigment is unclear, but has usually been attributed to congenital atrophy
or degeneration of the iris. Campbell\textsuperscript{51} states that the loss is due to mechanical rubbing between the peripheral, often concave iris and the anterior zonular packets in pre-disposed individuals. Therapy with thymoxamine may be beneficial.

Glaucoma has also been observed in patients with posterior polymorphous dystrophy but the exact mechanism of this secondary open-angle glaucoma is unclear.\textsuperscript{53} It is possible that the trabecular endothelium may be affected by the same dystrophic process as the cornea.

The exfoliation syndrome and pseudoexfoliation have also been associated with the development of glaucoma. Kozart and associates\textsuperscript{54} reported a 1\% incidence of glaucoma in eyes with exfoliation syndrome, with an additional 15\% of these eyes having ocular hypertension. The sex distribution of exfoliation syndrome patients seems to depend on the coexistence of glaucoma. Women form the majority in those patients without glaucoma but in those with coexisting glaucoma, no sex predilection exists. More patients with bilateral exfoliation syndrome have glaucoma, as compared to those with unilateral exfoliation syndrome.\textsuperscript{54} The cause of the glaucoma is similar to that of pigmentary glaucoma, namely obstruction of the trabecular meshwork.

Ocular trauma is another cause of secondary glaucoma. This traumatic glaucoma may occur either early or late after blunt injury to the eye. As a consequence of the ocular trauma, angle recession and hyphema usually occur. The size of the initial hyphema shows a prognostic bearing to both the early rise in intraocular pressure and the final visual acuity.\textsuperscript{55} Glaucoma is more likely to occur in eyes that have a severe recession of the angle of more than 180 degrees and possibly most often in eyes that have more than 270 degrees of angle recession.
The intraocular pressure may change greatly after an ocular contusion and both increased and decreased pressure may be found. However, most often a period of hypertension is followed by a period of hypotension. Tonjum\textsuperscript{56} described three phases of the course of the IOP. First, there is a hypertensive phase which is observed during the first day after the trauma. Second, there is a steady hypotensive phase lasting 3-4 days. In the third phase, there is a steadily increasing IOP up to normal mate-eye level. Factors cited for the early transient hypertensive phase are an accumulation of blood elements in the trabecular meshwork, edema of the trabecular meshwork region and changes of the endothelial cells lining the trabecular meshwork.\textsuperscript{56}

Therapy for the secondary open-angle glaucomas is basically the same as for primary open-angle glaucoma, in addition to eliminating the cause of such a secondary glaucoma.

**Secondary Angle-Closure Glaucomas:** These occur in a variety of situations in which the aqueous humor's free access to the trabecular meshwork is prevented. The following conditions are very common causes of secondary angle-closure glaucoma.\textsuperscript{3,5}

(a) **Plateau Iris:** Acute glaucoma can recur following peripheral iridectomy for angle closure glaucoma. One type is caused by the iris folding into and occluding the angle following pupil dilation. Affected eyes have a characteristic type of angle recess.\textsuperscript{38}

(b) **Ghost Cell Glaucoma following trauma:** Ghost cell glaucoma occurs following vitrectomy and cataract extraction. Glaucoma of this nature also occurred after trauma.\textsuperscript{43}
The vitrectomy, cataract extraction and trauma have the same characteristics, as explained below.

1) Severe trauma to the eye, either blunt or penetrating, with hyphema and vitreous hemorrhage.

2) Gradual clearing of the anterior chamber hemorrhage and conversion of fresh red blood cells to ghost cells in the vitreous cavity.

3) Forward passage of ghost cells into the anterior chamber through a traumatic opening in the anterior hyaloid face.

4) Elevation of intraocular pressure caused by these cells, approximately two weeks to three months following the trauma.

The trabecular meshwork obstruction is caused by red blood cell debris and laden macrophages; also the degeneration of red blood cells could lead to the trabecular meshwork blockade and outflow facility is impaired and the IOP is elevated; this is called ghost cell glaucoma.\(^5^7\)

Treatment for this type of glaucoma is both medical and surgical. The surgical procedure of choice is anterior chamber irrigation, which is often effective and is advised before proceeding to vitrectomy.

(c) Lens protein induced glaucoma: Abnormalities in the crystalline lens can cause secondarily either angle-closure or open angle glaucoma. The former is invariably a result of pupillary block, whereas the latter has been ascribed to the leakage of lens proteins and particles from the lens or to the cellular reaction to this.\(^3^9\)

(d) Lens-dislocation induced glaucoma: Dislocation of the crystalline lens may cause pupillary block by the filling of the pupil with the lens, vitreous or both.
Treatment for this type or form of glaucoma can be cured by peripheral iridotomy or iridectomy. Laser iridotomy is the treatment of first choice, since it avoids surgical entry of the anterior chamber and the attendant problems of vitreous loss, etc.

(e) **Lens swelling**: Swelling of a cataractous lens may occur and cause secondary angle-closure glaucoma because of pupillary block. Cataract surgery is definitive in restoring vision and curing the glaucoma, unless permanent peripheral anterior synechiae have formed. In eyes with no visual potential, iridotomy or iridectomy may be employed.39

(f) **Neovascular glaucoma**: Neovascular glaucoma (NVG) following central retinal vein obstruction (CRVO) is a devastating complication that occurs in approximately 20% of cases.

Recent clinical and experimental studies have suggested a correlation between the extent of retinal ischemia and neovascularization of the retina (NVE); optic disc (NVD), and iris (NVI). The liberation of a diffusible vaso-proliferative substance from ischemic retinal tissue has been postulated, but has not been proven.40,58,59,60

Non-specific iris vessel leakage is not an uncommon finding on iris angiography, especially in older patients. However, most eyes with definitive NVI following an ischemic CRVO tend to progress rapidly to intractable secondary angle closure glaucoma within a few weeks.

Most eyes that develop NVG following CRVO become painful and blind and many of them ultimately become phthisical. Because of this poor prognosis in eyes that develop NVG, recognition of risk factors for the development of NVG following CRVO is of great potential benefit to affected patients if an effective prophylactic therapy can be found. Pan-retinal photo-coagulation
CONCLUSION

One of the leading causes of blindness in the U.S.A. is glaucoma, and the rise in the incidence of this disease in other parts of the world is being looked into by experts. There are various types of glaucoma, open-angle type, primary and secondary, narrow angle/closed angle type, also with primary and secondary types, congenital type with its sub-groups of juvenile and infantile.

Early diagnosis and treatment of any of these glaucomas are very essential. Open angle glaucoma is most responsive to medical therapy. Number one choice of drug is timoptic. Narrow angle/closed angle glaucomas are primarily treated with medical therapy to get the pressure down before surgical therapy is applied. Preferable surgical therapy is iridectomy or iridotom; other surgical therapy as listed in this paper could be applied. Congenital glaucoma is only treated by surgical methods. Follow-up when treating any of the above mentioned glaucomas is absolutely essential. The majority of the drugs used in treating the glaucomas have a lot of side effects. To avoid the systemic adverse reaction, the patient should be told to close their eyelids for at least two or three minutes. Pressure should then be exerted on the duct (at the medial canthi) for two to three minutes to immobilize the lacrimal pump and decrease tear flow into the nose and blood stream.
All facts about the glaucomas have not been exhausted in this paper. Further information could be obtained in any standard ophthalmology book or any ophthalmological journal. The ocular and systemic anomalies associated with all the types of glaucoma could be found in *The Principles and Concepts of Ophthalmology* by Frank W. Newell and *Textbook of Ophthalmology* by Albert and Scheie.
Figure A - Narrow angle glaucoma. Interval or preglaucoma phase with narrow angle but with normal aqueous outflow and no synechiae.

Figure B - Estimation of depth of anterior chamber by oblique illumination (diagram). (Courtesy of R. Shaffer).

Figure C - Chronic narrow angle phase; iris root adherent to trabecula. Result of fulminating or repeated acute attacks.
Figure D - Juvenile glaucoma. Marked cupping of both left and right eye, also loss of visual field on both left and right eye.
Figure E - Marked enlargement of the right eye of a 3 year old girl with infantile glaucoma.

Figure F - Enlargement of corneas with corneal haze.
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