What is Glaucoma?
Visual loss from glaucoma results from characteristic deterioration of the optic nerve leading to progressive loss of the field of vision. At least 3 million Americans suffer from glaucoma. Glaucoma is one of the leading causes of adult blindness, and it is also the leading cause of preventable blindness. Most people who go blind from glaucoma are blind in at least one eye at the time of original detection, which points to the need for better early diagnosis. Because glaucoma usually does not manifest any symptoms until extensive peripheral visual loss becomes apparent in the final stages of the disease, it is often likened to the “sneak thief of sight.” Unlike most eye diseases, most varieties of glaucoma are chronic, virtually lifelong disorders than can be controlled but not cured. Like diabetes, high blood pressure, asthma, or arthritis, glaucoma requires some modification in lifestyle, such as compliance with medical regimens, regular physician visits, and acknowledgment of the disease to achieve successful treatment.

What Parts of the Eye are Involved in Glaucoma?
Three areas of ocular anatomy are key to understanding the group of disorders known as glaucoma. These include the optic nerve (also referred to as the optic nerve head, the optic disk, or the optic papilla), the ciliary body, and
the angle of the anterior chamber. The anatomy of the anterior optic nerve is described in detail below. The ciliary body is the mid-portion of the uveal tract lying just behind the iris and is the site of production of aqueous humor. The angle of the anterior chamber refers to the region between the cornea and the iris that contains the trabecular meshwork, the principal site of outflow of aqueous humor from the eye. Aqueous humor bathes the anterior segment of the eye, providing oxygen and nutrition to the region. The aqueous humor is produced at a relatively constant rate by the ciliary body. The trabecular meshwork acts as a sieve of tissue that connects, via the Canal of Schlemm, to the venous system, where the aqueous humor is resorbed into the bloodstream. Intraocular pressure (eye pressure) is dependent on the production of aqueous humor and resistance of aqueous humor outflow through the trabecular meshwork.

**How is Intraocular Pressure related to Glaucoma?**
The majority of glaucoma cases in North America and Europe are associated with elevation of the intraocular pressure. Elevated intraocular pressure could result from either an excessive production of aqueous humor from the ciliary body or an obstruction of aqueous humor outflow through the chamber angle (trabecular meshwork). In fact, virtually all elevation of intraocular pressure arises from some form of blockade of aqueous humor outflow through the trabecular meshwork. Some patients exhibit the progressive optic nerve damage of glaucoma but seldom or never manifest increased intraocular pressure. Controversy exists about whether these individuals have exquisitely pressure-sensitive optic nerves or whether other damaging factors, such as compromised circulation, cause the optic neuropathy. Glaucomatous optic nerve damage without elevated intraocular pressure is sometimes referred to as "low tension glaucoma" or
"normal pressure glaucoma."

**Are There Different Types of Glaucoma?**
The clinical varieties of glaucoma are classified according to three parameters: 1) Primary (idiopathic) or Secondary (associated with some other ocular or systemic conditions). 2) The state of the anterior chamber angle: open angle (open access of the outflowing aqueous humor to trabecular meshwork) or closed angle (the trabecular meshwork is blocked by apposition of the peripheral iris). 3) Chronicity: acute or chronic. The vast majority of glaucomas are chronic.

Although most glaucoma patients have the primary open-angle type, some 50 different varieties of secondary glaucoma have been described. In general, these are not caused by dysfunction of the trabecular meshwork, but rather by some other ocular or systemic disorders such as inflammation, fragile new blood vessels in the eye, birth defects, ocular trauma or tumors. Although appropriate diagnosis of an unusual secondary glaucoma can make the difference between sight preservation and blindness, most secondary glaucomas are sufficiently uncommon to fall exclusively within the ken of the glaucoma subspecialist.

Glaucoma refers to a group of diseases of the eye, most of which are chronic and, when unrecognized, produce insidious, irreversible visual loss. Blindness from glaucoma can be prevented or greatly reduced by appropriate screening. Glaucoma, like other chronic diseases, requires establishment of a strong physician-patient relationship, ongoing therapeutic regimen, and regular physician office visits. Patients whose glaucoma is recognized in the early stages usually learn to cope with their disease and retain good functional vision throughout their life. Patients whose diagnosis is delayed until advanced visual field loss develops, who cannot cope with
the rigors of chronic disease therapy, or who suffer from non-pressure risk factors have a worse prognosis for retention of useful vision.

**What is the Optic Nerve?**
A characteristic deterioration of the optic nerve associated with cupping and atrophy is the common denominator of all forms of glaucoma (primary or secondary, open or closed angle, chronic or acute). Atrophy of the optic nerve is the primary cause of permanent visual loss in glaucoma. Because of the ready visibility of the anterior optic nerve with any ophthalmoscope, recognition of the early signs of glaucomatous optic neuropathy becomes the single most useful clinical tool for glaucoma screening. With the ophthalmoscope and visualization of the optic nerve head, recognition of the glaucomatous optic nerve is not difficult. The circular optic disk border is visualized as the junction between the nerve head and the surrounding retina.

The optic nerve is primarily composed of the axons (the retinal nerve fibers) from the retinal ganglion cells and acts as the connection between the retina and the brain. The optic nerve head represents the perpendicular transition of the retinal nerve fibers from the surface of the retina to the optic nerve as they exit the eye. The normal, healthy optic nerve is composed of approximately 1.2 to 1.5 million neurons or fibers. With advancing age, there is often some atrophy of the tissue surrounding the optic nerve that gives a pale halo around the disk edge and provides an obvious visual separation of the retina and the optic disk tissue. The neural tissue of the disk has an orange-pinkish hue and has a full, slightly elevated appearance, with a relatively distinct border at the disk edge. Centrally, the orange-pink neural tissue gradually gives way to a yellow-whitish central zone or optic disk cup that is slightly more excavated than the surrounding
neural tissue. This renders a bagel-like or doughnut-like appearance to the nerve head, with the neural tissue or neural "rim" surrounding the central physiologic “cup.” The retinal vessels, arteries and veins, enter and exit the globe through the optic nerve in the area of the optic cup. In glaucoma, as the neural tissue atrophies, the central cup appears to enlarge due to the surrounding tissue loss. The ratio of the diameter of the cup to the total diameter of the disk (cup-disk ratio) is usually 0.3 or less, and greater than 0.5 should raise suspicion of glaucoma.

In addition to diffuse enlargement of the physiologic cup, focal thinning of the optic nerve rim is also very characteristic of glaucoma. Typically, the lower pole of the nerve head shows a focal loss of the neural rim earlier than the superior pole of the optic nerve; this results in a concomitant defect in the upper visual field. (see below). Small, "flame-shaped" hemorrhages at the disk edge are also often harbingers of advancing optic nerve damage and visual field loss and are quite characteristic of glaucomatous optic neuropathy.

As the central cup enlarges and the neural tissue of the optic nerve recedes, support for the retinal vessels traversing the disk disappears, and the vessels get pushed to the nasal, or inside, edge of the disk.

**What is Visual Field Loss?**
Progressive loss of the optic nerve fibers leads eventually to progressive loss of visual field and finally to complete loss or blindness. However, in most forms of glaucoma, a patient will not experience any symptoms until late in the disease. Early peripheral visual field loss is not noticeable to the patient, and its slow progression makes its recognition nearly impossible without special testing. In its normal physiologic state, greater than one million fibers of the optic nerve carry visual information from retinal
ganglion cells through the nerve fiber layer of the retina, along the optic nerve, and to the brain. Fortunately, there is a certain amount of functional reserve in the optic nerve so that a considerable portion, perhaps even half, of the nerve fibers can be lost before significant visual field loss occurs. This offers the opportunity for early diagnosis of disk changes before significant visual loss transpires. The functional status of the optic nerve can be assessed by specialized testing of the peripheral vision, the "visual field."

The special anatomy of the nerve fiber layer in the retina produces visual field defects from glaucoma that follow a characteristic pattern. Visual field loss in glaucoma usually arches from the physiologic blind spot of the optic disk, curves around the central region, and ends abruptly along the horizontal axis nasally. These arch-shaped defects are referred to as “Bjerrum,” or arcuate, scotomas. Loss of the peripheral nasal visual field generally occurs first in glaucoma. Following the progressive change and course of these visual field defects becomes the most critical aspect of managing the glaucoma patient.

**What is Intraocular Pressure?**
The eye is a globe and needs to maintain a level of pressure (intraocular pressure) to maintain proper function. In the past, the level of the intraocular pressure was used to define and diagnose glaucoma. However, in recent decades, it has been recognized that many individuals with glaucomatous optic nerve damage lack elevation of the intraocular pressure. Therefore, intraocular pressure is now considered one of the many risk factors for the development of optic nerve damage. Measurement of intraocular pressure (tonometry) is possible by a variety of techniques, the most accurate of which uses a slit lamp and applanation device.
Measurement of the intraocular pressure is used by the ophthalmologist to monitor the adequacy of intraocular pressure-lowering medications. Intraocular pressure in most individuals ranges between 10 and 20 mmHg, with an average of approximately 16 mmHg. Intraocular pressure above 20 mmHg is considered suspicious and may be a precursor to the development of glaucoma.

**What is Included in the Ophthalmic Examination for Glaucoma?**

In addition to the complete medical and ocular history and examination, particular emphasis on examination of the anterior chamber angle, the optic nerve, and the visual field are essential in the ophthalmic examination for glaucoma. Because the anterior chamber angle structures cannot be seen without special optical prisms or mirrored devices, gonioscopy, or examination of these structures, becomes a crucial aspect of the glaucoma examination. Using one of a variety of gonioscopic lenses, the peripheral iris, cornea and trabecular meshwork can be directly visualized to determine the presence of angle closure, adhesions, inflammatory foci, traumatic injury, masses or other lesions. Gonioscopy is the most important test for the diagnosis of angle closure glaucoma and some of the secondary glaucomas discussed above.

Since the disorder consists primarily of chronic progressive optic nerve deterioration, glaucoma is followed primarily with periodic assessment of optic nerve anatomy (ophthalmoscopy), function (visual field assessment), and assessment of the most common principal causative factor, the intraocular pressure. Glaucomatous optic neuropathy in moderate stages is easily recognized with ophthalmoscopy by the presence of cupping, or enlargement of the central cup on the optic nerve, especially if the cupping is focal within the nerve or asymmetrical between the two eyes. Anatomic changes in
the optic nerve head are readily visualized by ophthalmoscopy, but require sophisticated photographic or digital imaging techniques to record them for documentation of changes over time. Modern visual field testing employs automated computer-generated light detection measurements at multiple locations throughout the field of vision.

**What are the Different Types of Glaucoma?**

**Primary Open Angle Glaucoma**
The vast majority of glaucoma patients have primary open angle glaucoma. These patients manifest a chronic, idiopathic disease associated with progressive degeneration of the anterior optic nerve, known as glaucomatous optic neuropathy. Although elevated intraocular pressure is an important causative risk factor, only half of the 2 to 3 million North Americans with glaucoma will manifest elevated intraocular pressure at a single measurement. Therefore, measurement of intraocular pressure alone is a poor screening technique for glaucoma. Like most biologic parameters, eye pressure fluctuates throughout the day and varies with other influences, including hydration, sleep, blood pressure and body position. With multiple measurements at different testing sessions, most, but not all of these glaucoma subjects, will eventually exhibit elevated intraocular pressure at least part of the time.

The rise in intraocular pressure associated with primary open angle glaucoma derives not from a visible obstruction of the trabecular meshwork, but rather from cellular dysfunction of the trabecular meshwork tissue, which leads to increased aqueous humor outflow resistance. Risk factors for primary open angle glaucoma include family history, corticosteroid sensitivity, myopia, African-American race, systemic high blood pressure, high
intraocular pressure, diabetes, and age. In addition to these risk factors, early age of onset of disease and poor compliance with a medical regimen and physician visits are associated with a worse prognosis. As mentioned above, some patients with progressive optic nerve damage characteristic of glaucoma never manifest intraocular pressures above the statistically normal range. These patients are commonly diagnosed with "low pressure glaucoma," "low tension glaucoma," or normal pressure glaucoma." While recognizing that non-pressure risk factors may play a stronger role in these than in their high-pressure counterparts, these patients are managed similarly to those with conventional primary open angle glaucoma.

**Closed Angle Glaucoma**

All physicians need be cognizant of another form of glaucoma, closed angle or angle-closure glaucoma, which may present acutely or may be silent and chronic. This disorder, quite unrelated to open angle glaucoma, derives entirely from blockade of the trabecular meshwork by the peripheral iris, either by simple and reversible anatomical apposition, or pressing together, of the two tissues or by generally irreversible scarring and adhesion. These irreversible fibrotic adhesions may occur after unrecognized long-standing appositional angle closure (chronic angle closure glaucoma) or from other ocular conditions, such as uveitis or neovascularization (secondary angle closure glaucomas).

Classically, angle closure glaucoma is the well known, less common variety of glaucoma that presents acutely with severe eye pain, blurring of vision, colored halos around lights, nausea and vomiting. Angle closure usually occurs in the hyperopic (farsighted) eye, which is smaller than the average eye and thus crowds the iris, cornea, lens and anterior chamber angle into a smaller than average
Eventually, usually in the fifth to sixth decade of life as the lens gradually increases in size with aging, the lens becomes more firmly applied to the pupillary opening through which aqueous humor from the ciliary body must pass. This obstruction of aqueous humor flow at the pupil, known as relative pupillary block, eventually becomes clinically significant and traps the aqueous behind the pupil, raising the pressure in the posterior chamber above that in the anterior chamber and driving the iris anteriorly to lie against and block the trabecular meshwork. This trabecular meshwork blockade, or angle closure, leads to a sudden and dramatic rise of the intraocular pressure from its baseline normal level in the 10-20 mm Hg range to 60 mm Hg or more. This sudden change in pressure leads to swelling of the cornea with blurring, haloes, and severe ocular pain from iris ischemia and corneal edema. The pupillary margin of the iris becomes most tightly applied to the lens surface when the pupil is in the mid-dilated position; hence, it is often dilation of the pupil by exposure to stress, darkness, or drugs that precipitates an acute attack.

The immediate treatment of acute angle closure is directed toward reversal of the pupillary block, usually by moving the pupil with constriction. Ultimately, however, the pupillary block can be reversed and prevented by creating a new aqueous channel with peripheral iridectomy.

**How is Glaucoma Associated with Ocular Trauma?**

Glaucoma may develop after ocular trauma. Penetrating injuries to the globe disrupt, or even destroy, intraocular contents and may lead to sustained elevation of intraocular pressure and glaucoma. (see Ocular Trauma) A more subtle, insidious glaucoma may arise from blunt ocular injury or ocular contusion, as occurs when the globe is struck with a fist, ball or other object. Blunt injury
temporarily deforms the globe, causing shearing between its internal tissue layers. These shearing forces may tear the insertion of the iris (iridodialysis) or ciliary body (cyclodialysis) from its attachment to the sclera. Most commonly, the fibers of the ciliary muscle that both controls accommodation and modulates aqueous humor outflow become detached, leading to collapse of the trabecular meshwork (known as angle recession) and subsequent secondary glaucoma.

Acutely, the contused eye typically presents with intraocular bleeding (hyphema), and the intraocular pressure may be low, normal, or elevated. Angle recession glaucoma may not manifest for months or even years after the original injury. Treatment of glaucoma from blunt ocular trauma follows a similar protocol to more common open angle glaucomas, except that these eyes do not respond well to pupil-constricting drops such as pilocarpine because of the damage of the ciliary muscle. Because of the damage to the trabecular meshwork, laser trabeculoplasty is similarly ineffective. Thus, when regular glaucoma drops are ineffective, filtration surgery usually becomes necessary.

**What is Pigmentary Glaucoma?**

Pigmentary glaucoma is relatively common secondary glaucoma in the young adult and appears to be exclusively an ocular disorder. This disease is relevant because of the potentially severe consequences in young people. Pigmentary glaucoma occurs primarily in young myopic (nearsighted) adults and is more common in males, usually manifesting between the ages of 20 and 40 years. Melanin pigment granules from the iris circulate freely in the aqueous humor, become deposited or entrapped in the surrounding tissues of the cornea, iris, lens, and particularly within the trabecular meshwork. This leads to obstruction of the meshwork, elevation of intraocular pressure.
pressure, and glaucoma. This condition may manifest with intermittent visual blurring or dull ocular pain, but like other glaucomas may go unnoticed until severe visual loss occurs. Vigorous physical activity or pupillary dilation may induce a shower of pigment granules to be released from the iris in these patients, resulting in a transient, acute rise in eye pressure, corneal edema, blurred vision and ocular pain. Treatment of pigmentary glaucoma is similar to that for primary open angle glaucoma.

**What is Neovascular Glaucoma?**

Glaucoma is typically a disease of the middle aged and the elderly. Thus, its occurrence in children or young adults always raises the question of some associated condition, such as an intraocular tumor. Diabetes mellitus and other vascular disorders are frequently associated with neovascular glaucoma. The devastating consequences of diabetes upon the retina are well known. In addition to retina problems, the diabetic patient may also develop glaucoma as a result of retinal ischemia, known as neovascular glaucoma. Neovascular glaucoma is one of the most devastating varieties of glaucoma. Just as damage to the small blood vessels of the retina results in the formation of fragile new vessels and bleeding, retinal ischemia may also cause new vessels and scarring in the front part of the eye. It is believed that an angiogenic factor is produced by the ischemic retina, leading to new vessel formation inside the eye. Unfortunately, these vessels are abnormal and may cause a variety of vision-threatening problems. Neovascular glaucoma derives from proliferation of fragile new vessels on the iris (rubeosis irides) and into the anterior chamber angle. The trabecular meshwork provides a fertile template for neovascular growth, which ultimately leads to complete blockade of aqueous humor outflow, marked elevation of intraocular pressure and severe, often painful, blinding glaucoma. This process can also follow other vascular
conditions associated with retinal ischemia, including occlusion of the central retinal vein, occlusion of the central retinal artery, and even carotid artery disease without known damage to the eye. Treatment of neovascular glaucoma is multifaceted and involves diligent systemic management of related vascular disease, treatment of retinal ischemia, and lowering of the intraocular pressure with medical and often surgical therapy.

**What is Congenital Glaucoma?**

Although glaucoma is commonly associated with adult, even elderly, patients, childhood or infantile glaucomas also exist. The most common, primary congenital open angle glaucoma occurs in children without other identifiable abnormalities. Most cases become evident in the first year of life and often are discovered in the newborn nursery or during the first few weeks of life. The exact cause of infantile glaucoma is unknown but appears related to a delay in development of the aqueous humor outflow channels.

Infants with congenital glaucoma present with photophobia (shyness to light), epiphora (tearing) and blepharospasm (blinking or squeezing the eyelids). The principal clinical sign is an enlarged cornea, often bilaterally. As the disorder advances, the cornea becomes edematous and appears cloudy. The appearance of an enlarged cloudy cornea in an infant essentially makes the diagnosis of congenital glaucoma and is obvious to casual penlight examination. Prompt referral to an ophthalmologist for intervention can make the difference between sight and permanent blindness.

Various forms of developmental glaucoma may also be associated with congenital malformations of the anterior chamber angle. Common is the Axenfeld-Rieger syndrome
associated with dental, facial and other midline developmental abnormalities, adhesions between the cornea and iris, and glaucoma. Aniridia is a bilateral congenital absence of iris that may be inherited by autosomal dominant transmission or may occur spontaneously. These latter, spontaneous cases, may be associated with kidney tumors or other anomalies of the genito-urinary system. Glaucoma with aniridia usually occurs in early to mid childhood and is not typically associated with enlarged corneas.

**What is the Treatment for Glaucoma?**

Although it is recognized that not all glaucoma patients demonstrate elevated intraocular pressure and not all glaucomatous optic nerve damage is attributable to pressure damage per se, current standard glaucoma care is devoted almost exclusively to reduction of intraocular pressure. Bringing pressure down into the normal or low normal range (17 mm Hg or less) can be expected to arrest progression or dramatically slow its course in the vast majority of cases. At the same time, it must be recognized that some unfortunate individuals, diagnosed late with end-stage nerve damage, with an unusually sensitive optic nerve, or who are primarily sensitive to non-pressure factors, will continue to show unabated visual field loss despite maximal pressure lowering. These are the individuals to whom future research regarding other non-pressure causative factors and their treatment must be directed.

Three methods for glaucoma pressure-lowering treatment are available: medical (usually eyedrops), laser, and surgical. Since the threshold of pressure damage varies among patients, the only reliable indicators of glaucoma stabilization are stability of the visual field and prevention of optic nerve damage. In the usual glaucoma therapy, intraocular pressure may be lowered by any or all of the
three methods (drops, laser and surgery). In a normal, non-glaucomatous population, intraocular pressure averages approximately 16 mmHg and most (95%) will fall between 10 mmHg and 24 mmHg. In the glaucomatous population, the mean intraocular pressure is somewhat higher and the range much broader, even as high as 70 mmHg where arterial circulation to the eye begins to be compromised. Typically, however, the early untreated open angle glaucoma patient will manifest an eye pressure in the mid 20s. Measurement of intraocular pressure at different times of the day establishes the degree of pressure variability before glaucoma therapy is started. The experienced ophthalmologist typically will determine a "target pressure" as a goal to achieve with pressure-lowering therapy, recognizing that the goal may have to be revised based on future assessment of the visual field.

[Back to Top]

**What Laser Therapy is Available for Open Angle Glaucoma?**

During the early 1970's, attempts were made with a variety of lasers to enhance aqueous humor outflow through the trabecular meshwork in open angle glaucoma by puncturing the trabecular meshwork with the laser energy. Despite the failure of these procedures to create holes in the trabecular meshwork, a subsequent decrease in the intraocular pressure, several days to weeks following some of the procedures, was often observed. In 1979, Wise and Witter published a pilot study describing an argon laser procedure for the control of intraocular pressure. This technique, known as argon laser trabeculoplasty, has changed little since its original description. Many theories attempting to explain the effect of the trabeculoplasty laser burns have emerged. It is now thought that a cascade of biological events that
involves renewal of trabecular meshwork cells and accelerated turnover of the extracellular matrix, or tissues between the trabecular cells, enhances outflow through the trabecular meshwork following laser treatment. Argon laser trabeculoplasty is a relatively uncomplicated office procedure and has gained wide acceptance in the treatment of open angle glaucoma. In approximately 80% of eyes treated with argon laser trabeculoplasty, a significant lowering of the intraocular pressure will be achieved. However, the intraocular pressure lowering effect will diminish over time and approximately 10% of initially successful treatments will fail with each year. In patients in whom the initial laser trabeculoplasty was successful, additional laser therapy may be warranted. A modification of this therapy, Selective Laser Trabeculoplasty, is also available to treat open angle glaucoma.

**What Laser Therapy is available for Angle Closure Glaucoma?**

As described above, angle closure involved the lens coming too close to the iris and blocking the passage of fluid into the front of the eye, where it drains into the circulation. Relief of the relative pupillary block allows the iris to move back and the anterior chamber drainage angle to open, allowing escape of aqueous humor and lowering of the intraocular pressure. This is now commonly and simply done by fabricating a small hole in the iris with laser (laser iridotomy), which provides another route for fluid to enter the front of the eye. This equalizes the pressure between the posterior and anterior chambers and allows the iris to fall back to its normal anatomic position and away from the trabecular surface.

**What Surgical Therapy is Available for Glaucoma?**

**Trabeculectomy or Filtering Surgery**
Trabeculectomy is the most common operation for the control of elevated intraocular pressure in adult glaucoma. Various filtering procedures have been developed to shunt the aqueous humor from the anterior chamber to a reservoir under the conjunctiva on the surface of the eye. These procedures provide an alternative low-resistance pathway for aqueous humor egress from the eye. It is believed that the aqueous humor either filters through the conjunctiva from the reservoir, mixing with the tears, or it is absorbed by the blood vessels on the surface of the eye. Postoperative management includes topical dilating drops and antibiotics for the first one to two weeks following surgery. Topical corticosteroids are also used to suppress inflammation. The corticosteroid therapy is thought to reduce scar formation and failure of the filtering bleb. Youth, skin pigmentation, previous surgery, and secondary glaucoma greatly increase the risk of failure. The majority of surgical patients receive some form of additional chemical antimetabolite therapy, either during surgery by sponge application or post operatively as subconjunctival injections. 5-Fluorouracil or Mitomycin-C are the most commonly used antimetabolite adjuncts to trabeculectomy surgery. In addition, a variety of artificial drainage devices are available that employ a plastic shunt tube to divert the aqueous humor from the anterior chamber into the space behind the eye, where it is resorbed. These glaucoma tube shunts are generally reserved for eyes in which trabeculectomy surgery has failed or in which failure is likely due to extensive scar tissue formation, such as neovascular glaucoma.

How Often does a Person with Glaucoma Need to see an Ophthalmologist?
After the initial examination and diagnosis, glaucoma patients are managed much like patients with other chronic disease, requiring regular visits to assess disease
severity and response to therapy. The primary criterion for disease status is the visual field, since it is the most accurate measure of visual function in this disorder. Most patients will need periodic medical examinations, diagnostic testing for progression or new findings, and individualized management with drugs or procedures. Once the diagnosis and treatment regimen are established, the average patient needs to be seen 3-4 times yearly. Frequency of visits and testing depends upon risks for progressive damage and severity of illness, as tabulated below. Because performance on visual field testing shows some variability, visual field tests may have to be repeated if they show any changes.

Ripkin Vision And Laser Center

9424 State Route 14

Streetsboro, Ohio 44241

330-422-1111