A DISCUSSION ON GLAUCOMA AND A PROTOCOL OF MANAGEMENT OF GLAUCOMA SUSPECTS

SENIOR PROJECT
BY
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Glaucoma remains a challenging and puzzling ocular disorder. More than 2 million Americans today suffer from this "silent" disease.(1) Despite great advances in the management of glaucoma, it remains a chronic progressive disorder for most patients, and can result in blindness. Thus, the importance of early diagnosis of the disease is evident. The earlier glaucoma is detected, the more effective the management can be to prevent loss of sight. The advent of new instrumentation makes it possible to analyze, interpret, and evaluate glaucoma suspect patients to a greater degree than ever before. This paper discusses the classification of glaucoma, what signs to look for in glaucoma suspect patients, and suggests a protocol of management of glaucoma suspect patients at the Optometry clinic at Ferris State University.

Glaucoma is actually a group of diseases of the eye, having the common feature of an intraocular pressure that the eye cannot tolerate, causing damage to the optic nerve head, and subsequent loss of vision.(2) The glaucomas can be classified into different categories characterized clinically by a variety of symptoms and signs.

The glaucomas can be divided into four broad categories--primary, secondary, congenital, and ocular hypertension. Primary glaucoma is associated with a direct known or unknown disturbance of the aqueous circulation. Secondary glaucoma develops as a consequence of another disease. Congenital glaucoma occurs in the young as a result of a

developmental or birth defect. Ocular hypertension is signified by increased intraocular pressure in the absence of any disc or visual field defect.(3)

The clinical classifications of glaucoma can best be understood by an illustrative table:

Primary Glaucoma
- Open Angle
  - Chronic Simple
  - Low Tension
- Angle Closure Glaucoma
  - With Pupillary Block
  - Without Pupillary Block

Secondary Glaucoma
- Open or Closed Angle
- With or Without Pupillary Block

Congenital Glaucoma

Ocular Hypertension (4)

Primary open angle glaucoma is the most common type of glaucoma and predominantly occurs in people over 50 years of age. It is characterized by elevated intraocular pressure that leads to optic nerve damage and visual field loss. Gonioscopic techniques indicate the anterior chamber angle is open at the time of elevated pressures. The specific etiology is not well understood at the present time. The increase in intraocular pressure is caused by some type of

outflow abnormality.(5) Unfortunately, this type of glaucoma is chronic, slowly progressing, and bilateral, with no symptoms until late in the disease. Most patients are unaware of the loss of vision until a substantial number of nerve fibers have been irreparably damaged.

Low tension glaucoma is illustrated by normal intraocular pressures, but optic nerve signs and visual field defects seen in primary open angle glaucoma. It seems that the optic nerve is susceptible to damage from the low pressure. Some researchers feel that these patients actually have chronic simple glaucoma with an abnormal diurnal variation.(6)

The condition of primary angle closure glaucoma with pupillary block is indeed an ocular emergency. In the normal eye, the aqueous fluid flows from the posterior chamber to the anterior chamber through the pupil. In pupillary block, the aqueous is unable to enter the pupil due to an abnormally tight contact between the lens and the iris. In severe cases, the iris balloons into the trabecular meshwork--iris bombé--causing the anterior chamber angle to close. Since aqueous fluid is continually produced, the intraocular pressure increases to a dangerous level.(7) Fortunately, the patient is well aware there is an "attack" in progress. The signs and symptoms, which are usually unilateral, are increased intraocular pressure,dilation of the limbal vessels, steamy cornea, an oval mid-dilated pupil that is unreactive to light, blurred

vision, significant ocular pain and discomfort, colored rings around point sources of light, nausea, and vomiting. The patient must be treated immediately--loss of vision can occur in 24 hours.(8) The diagnosis is quite simple once a careful case history has been completed, as well as tonometry, gonioscopy, optic nerve evaluation, and a visual field analysis. The patient is usually older, tends to be hyperopic (small anterior globe), has a shallow anterior angle, and a lens that tends to move anteriorly with certain postures. There is often a history of mild "attacks".(9)

A patient with primary angle closure glaucoma without pupillary block also experiences "attacks" with the same signs and symptoms as pupillary block. The main anatomic factor in this type of glaucoma is a plateaued iris, or an obvious elevation to the last iris roll. This last roll of the iris closes the anterior chamber angle when the pupil is dilated.(10) Again, this diagnosis can be made by a case history, gonioscopy, tonometry, slit lamp evaluation, ophthalmoscopy, and visual field testing.

Secondary glaucoma is due to an alteration in the anatomy or physiology of the eye as a result of disease or ocular trauma. It can be present with or without pupillary block, as well as an open or closed angle. This can be due to changes in the uveal tract, such as pigmentary dispersion syndrome, iridocyclitis, rubeosis, iris atrophy, and tumors. An alteration of the lens may also be a factor. Some examples of lens changes are dislocation, pseudoexfoliation,

intumescent cataract, phacotoxic or phacoanaphylactic reaction, or spherophakia. The increase in intraocular pressure may be secondary to trauma--angle recession, iris prolapse into a corneal wound, or massive hemorrhages. The clinician shouldn't forget about iatrogenic factors as well, such as steriod intake.(11)

The etiology of congenital glaucoma depends on the specific congenital abnormality. This type of glaucoma occurs in one of 10,000 births.(12) A thorough case history and examination allows for the detection and diagnosis of congenital glaucoma. The recognition of an abnormal anterior chamber angle with gonioscopy (under general anesthetic) is critical for the diagnosis. Other diagnostic features are photophobia, epiphora, and blepharospasms, leading to eventual corneal edema, increased cupping of the optic nerve, and globe enlargement. If no treatment occurs, total blindness can occur.(13)

A patient is classified as having ocular hypertension when there is an elevation of the intraocular pressure above normal (>21) with no changes in the optic nerve or visual function. One percent per year of these patients will develop glaucoma.(14) These patients must be monitored carefully.

What is the determining factor to decide if a patient is a glaucoma suspect or has glaucoma? This decision is made by collecting and evaluating all of the clinical information needed to make a definitive diagnosis. And it

doesn't simply consist of evaluating the intraocular pressure, the optic disc, and the visual field of a patient. You must begin with a very careful personal/health history considering all of the risk factors associated with glaucoma.

The prevalence of primary glaucoma increases with age. It rarely occurs in patients younger than 40 years of age, but the incidence increases markedly thereafter. By age 80, 14% of all persons have glaucoma. Substantial evidence indicates there is a genetic basis for glaucoma as well. Approximately 20% of patients with glaucoma have a family history of the disease. Racial influences have been found to play a part. Blacks have an increased risk of developing glaucoma compared to whites. Myopic individuals tend to be more susceptible to glaucoma. One theory is that myopic eyes tend to be larger, have a thinner sclera, and thus have a weaker lamina cribrosa. Systemic diseases, such as diabetes and hypertension, also may be associated with increased incidence of glaucoma. Glaucoma is three times more likely to develop in diabetics than in non-diabetic persons.(15) An increase in intraocular pressure can be due to the secondary effects of ocular trauma. This possibility must be investigated.

Tonometry provides valuable information for the diagnosis and management of glaucoma. One evidence from the measurement of intraocular pressure that leads to the diagnosis of glaucoma is a reading of 21 mmHg or above.(16) But one must keep in mind that there is

no clear-cut dividing line between normal and abnormal pressures. What is high for one individual may not result in glaucomatous damage in another. The level of intraocular pressure which causes damage to the optic nerve significantly varies between individuals and even in the same person as he gets older. Several studies have shown that the risk of glaucoma increases as the intraocular pressure increases. Thus, it is important to measure a patient's pressures at as early an age as possible to get a baseline level.

The second factor of the intraocular pressure to consider is the difference of the readings between the two eyes. Primary open angle glaucoma is a bilateral disease, but it tends to appear in one eye before the other. Thus, a difference of 3 mm or more between the two eyes indicates a greater suspicion of glaucoma.(17)

The final factor of intraocular pressure measurements associated with glaucoma is its variability with time. Some patients have a cyclic swing of pressures, upward and/or downward. Glaucoma has a greater chance to develop when a patient is in an upward swing. The practitioner must also be aware of any diurinal fluctuations of intraocular pressures. A diurnal variation in the pressure greater than 7 mm is an indication of possible glaucoma.(18) One must also be familiar with some of the factors that may be associated with high intraocular pressures. These factors include the following: the use of steroids, females

(especially after menopause), smoking, obesity, hypertension, stress, and the use of alcohol.(19)

All glaucoma suspect patients must have thorough biomicroscopy performed. The specific type of glaucoma can not be determined unless the anterior chamber angle is examined. The best way to evaluate the angle is with gonioscopy. In assessing the angle, there are three aspects to consider: shape and position of the insertion of the iris, amount of pigment or debris on the trabecular meshwork, and the magnitude of the angle. The iris normally inserts on the ciliary body. The closer the insertion on the scleral spur, the greater the risk of narrow angle glaucoma. A bowed angle is also a sign that angle closure is a probability. An excess amount of pigment or debris in the trabeculum usually indicates that the outflow of aqueous is stunted, creating increased intraocular pressure. The angle assessment is made by noting the amount of ciliary body and trabeculum that is visible.(20)

The slit lamp is an invaluable tool for the glaucoma suspect besides the use of the gonio lens. The anterior chamber must be observed for signs of trauma or irregularity. The iris must be analyzed carefully noting any atrophy, neovascularization, or iridodialysis. An irregular pupil may indicate synechiae, atrophy, or angle recession. These signs increase the risk of glaucoma. The acute glaucoma pupil won't react to light. Any indication of cells or flare in the anterior chamber should be noted.

The cornea should be evaluated for any endothelial pigment or edema. And finally, the lens should be examined carefully to detect any pseudoexfoliation, exfoliation, or cataract formation. The presence of any of the above mentioned signs suggests previous trauma, inflammation, or ocular systemic disease, and the possibility of secondary glaucoma.(21)

There is a high correlation between optic nerve changes and glaucoma. Thus, information obtained from an evaluation of the posterior segment of the eye is extremely important in the detection, diagnosis, and management of glaucoma. However, similar to intraocular pressure, there is not a fine line between normal and abnormal discs. When evaluating the discs, there are four aspects to consider: (1) Disc topography, (2) Disc hemorrhages, (3) Peri-papillary changes, and (4) nerve fiber layer changes.(22)

When evaluating disc topography, the examiner compares the optic cup to the optic disc. The evidences from the evaluation of the optic disc that lead to the diagnosis of glaucoma are the size of the cup, shape of the optic cup, position of the cup, depth of the cup, difference between the two eyes, and a change over time. The size of the cup, or the cup-to-disc ratio, needs to be observed, since a large ratio definitely increases the probability of glaucoma. And there is a rough relationship between cup-to-disc ratio and intraocular pressure--the higher the pressure, the higher the ratio. The size of the cup should

be determined by its color, as well as its contour, which is accomplished by binocular stereoscopic techniques. Both obervations are necessary, for in glaucoma, the contour cup-to-disc ratio changes before the color ratio.(23) A cup-to-disc ratio of 0.4 or higher should alert the clinician, and sophisticated visual fields should be performed.

The shape of the cup is another useful indication of early glaucomatous change. Though the cup may increase in size in all meridians, studies have shown that 42% of glaucoma patients had in increase in the vertical cup-to-disc ratio.(24)

The optic cup is normally located in the center of the disc. Displacement of the cup may be an indication of glaucoma. The amount of neuroretinal rim tissue in each meridian must be estimated carefully. A large cup is looked at with less suspicion if there is a large neuroretinal rim area 360° around the cup.(25) If the width of the neuroretinal rim is less than 1/8 of the disc diameter, a glaucomatous field loss is likely.(26) The shape and integrity of the rim tissue is vitally important in your diagnosis. Localized pallor or focal obliteration -- notching--of the neuroretinal rim tissue is strongly indicative of glaucoma. Notching usually occurs in the 12 or 6 o'clock position.(27)

The depth of the cup is important to evaluate in glaucoma suspect patients. If there is no cupping, and glaucomatous changes are occurring, the cup will deepen

until it reaches the lamina cribrosa. Then it enlarges at the bottom. The appearance of the lamina is often called the "lamina dot sign". This is an important finding, especially if there is evidence of other clinical signs of optic nerve head damage due to glaucoma.(28)

Evaluation of the asymmetry of the cup-to-disc ratio between the two eyes is an indication of possible glaucoma. The cup-to-disc ratio is generally the same in both eyes. As was mentioned, primary open angle glaucoma is a bilateral disease, but usually occurs asymmetrically. A study has shown that 53% of glaucomatous patients had an asymmetry greater than 0.2 between the two eyes.(29)

The final element of disc topography to consider is a change in the cup-to-disc ratio over time. An increase in the ratio usually occurs prior to visual field loss.(30) The importance of record-keeping, disc drawing, and photographs to document the ratio cannot be stressed enough.

Some glaucoma patients have been noted as having splinter hemorrhages near the disc margins, most commonly in the superior and inferior temporal regions. These hemorrhages are transient, small in size, and simulate normal blood vessels in their configuration. For those reasons, they are easily undetected. It has been shown that visual field defects are more likely to occur in those patients with disc hemorrhages.(31) Splinter hemorrhages

represent an infarction of the blood supply to the optic nerve head.

In healthy eyes, the disc encircling the cup has a healthy, reddish appearance. A peripapillary halo might be a useful diagnostic sign for the detection of glaucoma in the early stages. This halo is often described as a pale yellow ring adjacent to the disc with sharp borders and no pigment changes.(32) The pallor may initially only involve a section of the optic nerve.

Glaucomatous damage to the optic nerve alters the appearance of the retinal nerve fiber layer. Some researchers feel that these changes may be the earliest objective evidence of glaucomatous tissue damage. The earliest signs are localized slitlike defects, usually in the area of the superior or inferior arcades. They give the nerve fiber layer a "raked" appearance.(33) They can be observed with the red-free filter on the ophthalmoscope. But the nerve fiber layer is best observed with red-free, wide-angle photography on a black and white photograph. Unfortunately, the nerve fiber layer defects are not a definitive sign of glaucoma. Further research must be done on this topic.

Visual field testing is an essential part of the diagnostic evaluation of glaucoma. A visual field loss is still the necessary diagnostic evidence for the presence of glaucoma.(34) Every glaucoma suspect patient should therefore have a thorough visual field performed,

peripherally and centrally. The early visual field defects in glaucoma are paracentral scotomas, nasal steps, and temporal wedge-shaped defects.(35) Studies have shown that 91% of glaucomatous visual field losses will occur in a horizontal bow-tie shaped area extending 50° nasally and temporally, and 20° superiorly and inferiorly. And glaucoma tends to respect the horizontal meridian.(36)

The most common early visual field defect found with glaucoma are paracentral scotomas located within the arcuate Bjerrum region 5-15° from fixation. This is most likely caused by vertical elongation of the cup. Later in the disease, these isolated scotomas will coalesce to form arcs.(37) These are most commonly found superior to the horizontal meridian.

The second most common early visual field loss is a nasal depression. A nasal depression is a constriction of the visual field either above or below the horizontal meridian of the nasal field. This requires visual field testing of the periphery. These depressions are caused by temporal unfolding and vertical elongation. A nasal step will later develop, which is a progression of a nasal depression.(38)

A temporal step, or wedge, is sometimes seen in the late stages of glaucoma. This step is a constriction of the visual field either above or below the horizontal meridian in the temporal field. This is a sector-shaped defect. It

often occurs along with a generalized constriction of the entire visual field.(39)

Many researchers feel glaucoma can be detected by using a different approach--psychophysical and electrophysiological testing. These tests evaluate the effectiveness of the optic nerve subjectively and objectively. These tests are utilized to find some reliable clinical evidence of optic nerve damage that precedes glaucomatous visual field loss. Some of the elements of psychophysical and electrophysiological testing for glaucoma are the following: color vision, dark adaptation, contrast sensitivity, visually evoked potential, pattern electroretinogram, and fluorescein angiography.(40) Although no ultimate testing procedures have been found, research is proceeding quite rapidly in this area.

Glaucoma suspect patients should be monitored carefully. The clinician's goal toward the glaucoma suspect is to intervene with some type of treatment before damage occurs or at the earliest possible detection of the ocurring damage. Thus, a standard protocol of management of the glaucoma suspect patients is necessary. This is especially true at the Optometry Clinic at Ferris State University, where there are numerous clinical instructors and students. The Ferris Optometry Clinic serves a very large population of patients. If there is some reason to believe a patient is suspect to glaucoma when completing the primary care exam, the patient is referred to the Ocular Health Clinic

for a more complete glaucoma work-up. Below, I will suggest a protocol of management for the Ferris Optometry Clinic, indicating appropriate instrumentation and their frequency.

The Ferris Optometry Clinic would greatly benefit from the utilization of a glaucoma/glaucoma suspect flowchart. This would aid the clinicians in the timely diagnosis and effective long term management of the glaucoma patient. The flowchart allows one to quickly and accurately document the results of each visit in chronological order, summarized on one page for each eye. This eliminates the need for the clinician to review the patient's records, which are often times quite thick, unless further information is needed. Any changes in intraocular pressure, cup-to-disc ratio, visual fields, etc. can easily be detected by means of this temporal profile.(41)

An example of a glaucoma/glaucoma suspect flowchart is shown in Table 1. The first page has an area on the top for the completion of the following information: name, date of birth, pertinent family history, patient medical history, and any medications (other than those to treat glaucoma) the patient is taking. The remainder of the first page pertains to the right eye. Data collected for the left eye is entered on the second page. The clinical data is entered by visit in a vertical column allowing each category to be monitored with a horizontal scan across the page. Each vertical column is designed for the clinician to enter the date of the exam; glaucoma medications taken; time, method,

and readings of intraocular pressure; nerve head appearance; slit lamp exam and anterior chamber angle estimation; instrumentation and results of visual field testing; gonioscopy results; notation of photographs taken; indication of when a primary care exam was performed; if and to whom a referral was made; and the suggested recall. At the bottom of the page, room for drawings or elaborations on any of the entries is provided. In addition, a brief explanation for the clinician on how to enter the data is provided on the top of the second page.

At the present time, the Goldmann tonometer is considered to be the state of the art instrument.It is relatively inexpensive and has shown to be highly reliable and valid. (42) Because of diurnal variations, glaucoma suspects should have their intraocular pressure measured at various times of the day. The time of each measurement should be recorded in the patient's chart. It has been suggested that intraocular pressures should be measured 2-4+ times yearly for glaucoma suspects, representing a recall of 3 to 6 months.(43)

Gonioscopy should be performed on every glaucoma suspect on the first examination, or very soon after that time. The type of glaucoma suspected can not be stated unless the anterior chamber angle is evaluated. The Goldmann three mirror contact lens is one of the most frequently used instrument because of its versatility.(44) The Zeiss four mirror lens is used often as well. The Shaffer system

is commonly used to grade the anterior chamber angle, interpreting a closed angle as 0 and a wide open angle as 4. Gonioscopy should be performed every 6 to 12 months on glaucoma suspects.(45)

The optic nerve must be evaluated at every visit, since its appearance is such a key factor in the suspicion of glaucoma. The cup-to-disc ratio should be estimated by color and contour. Color ratio can be determined by using the direct ophthalmoscope. The contour ratio is evaluated stereoscopically with a 90 D lens, Hruby lens, or a fundus contact lens. Even with stereoscopic views, there is much variability in the assessment of the cup-to-disc ratio. This is especially true at the Ferris Optometry Clinic, where a patient is very unlikely to work with the same clinician twice. It is thought by some researchers that the intraobserver variations in the interpretations of cup-to-disc ratios is too great to be effectively utilized in the early detection of glaucoma. Thus, written cup-to-disc ratio estimates are often not valuable enough, and other methods must be utilized. Disc drawings at the first visit, and any subsequent visit where change is noted, is very helpful in monitoring the optic nerve head. However, the best method to follow is baseline photographic documentation of the optic nerve at the first visit. At each following examination, the cup-to-disc ratio interpretation should be compared to these baseline photos. These comparisons should

take place with a dilated examination every 6 to 12 months.(46)

In the last few years, clinicians have begun to experiment with stereo disc photographs. Studies have shown that some disc changes observed in stereo disc photographs occurred before visual field loss. (47) Thus, stereo disc photographs should be considered for long term follow-up of glaucoma suspects.

The automated perimeter is the most efficient and effective instrument to aid in the diagnosis of glaucoma. Today's automated devices can determine threshold light sensitivity values throughout the visual field, allowing the clinician to detect glaucomatous defects, monitor progression, and to evaluate the efficacy of treatment. By eliminating the perimetrist from patient interaction, a significant degree of variability and bias has been eliminated. An automated visual field test should be performed as soon as possible once glaucoma is suspected. Once a field has been analyzed, it will be used as one of the parameters for monitoring change. In order to compare, the same test and strategy should be used.

If the initial visual field test shows marked defects or very low overall sensitivity, the field should be repeated in 3 months to confirm the loss. The clinician should not be surprised if an improvement occurs. This is most likely due to the learning curve; i.e. the patient becomes more familiar with the test procedures. A visual

field that shows no changes indicates adequate control. If there are also no changes in intraocular pressure and optic nerves, the visual field can be repeated in a year. If there are optic nerve or intraocualar pressure changes, the field should be repeated in 6 months, or earlier. If there is severe optic nerve damage, fields should be performed every 1-3 months. To say that a visual field has progressed, it should be present on two consecutive fields. Thus, you will have a baseline field, a second field which shows a loss, and a third field which confirms the loss indicated on the second field test.(48)

The last recommendation for any optometric office or clinic is an effective recall system. Currently, the Ferris Optometry Clinic sends reminder postcards through the mail to patients. However, the efficiency of recalls would be greatly improved if a computer was utilized. In addition to providing recall systems, computer software is available that includes a patient database, patient communication modules, accounting systems, patient prescriptions, inventory control, insurance features, referral source analysis systems, service agreements, and much more. The clinician can not rely totally on the patient to arrange for follow-up appointments. Frequent telephone calls by the clinic staff can also be an effective method used jointly with a computer.

I completed a glaucoma flowchart similar to the one presented in Table 1 for 50 glaucoma/glaucoma suspect patients who visited the Ferris Optometry Clinic (chosen at random). I discovered that the clinicians made errors in the care and regularity of tests performed on many of the glaucoma suspect patients when the protocol of management discussed above is considered. It was quite difficult to make comparisons of the data from visit to visit without the flowchart. The following is a statistical analysis of the errors that were made in the care of these 50 patients:

No gonioscopy performed	44 patients
No automated visual fields	39 patients
Lost to recall	13 patients
No photographs taken	11 patients
No primary care exam	6 patients
No stereo view of disc	5 patients
O.N. not evaluated at every visit	5 patients
No Goldmann pressures	2 patients

Two of the flowcharts I completed are demonstrated in Tables 2-3. In Table 2, it is apparent that the patient has not returned for his 6 month recall (this recall date was specified in the patient's record). Upon further observation, you will notice that the time that Goldmann tonometry was performed was not specified in the patient's record at every visit. This is important information to know, since this particular patient had pressures that varied from 14 to

22 mmHg. Clinician variability in cup-to-disc ratio estimation is apparent here. No automated visual fields or gonioscopy was performed on either eye. An attempt was made to schedule this patient for an appointment in the Ocular Health Clinic via postcards and telephone calls with no success.

Table 3 shows another patient that failed to return to the clinic. Again, this demonstrates significant differences in cup-to-disc ratio values from one visit to the other, showing a smaller estimate at the second exam. Besides no automated fields or gonioscopy performed, this patient did not have any photographs taken of the optic nerves.

The optometrist's role and responsibility in the management and treatment of glaucoma/glaucoma suspect patients is greater than ever before and increasing quickly. The management of these patients remains both an art and a science. It requires knowledge and training, as well as experience with a variety of patients over a period of time. The clinician must gather data from many tests and determine the patient's risk for glaucoma. Some information will help us decide for one patient, and other information for another. Each patient has to be considered separately. The clinician should become more familiar with the state-of-the-art instruments and the frequency at which the tests should be performed. My suggested protocol can be reviewed quickly in Table 4. The tests and evaluations of the

glaucoma suspect patient must continue so the clinician has enough information to detect changes at its earliest, and decide upon the appropriate management at that particular point of time. With all of this in mind, the clinician can do his/her best to care for glaucoma and glaucoma suspect patients and prevent the loss of vision.

GLAUCOMA/GLAUCOMA SUSPECT CONTROL SHEET--RIGHT EYE

TABLE 1

NAME: _____ DOB: _____

PER. HX: _____

FAM HX: _____

MEDS/DATE STARTED: _____

(See back for key)

DATE MM DD YY																		
GLC MEDS DATE STARTED																		
IOP METHOD READING TIME																		
NERVE HEAD C/D DEPTH R/T METHOD																		
SLE SIGNS ANGLES																		
VF METHOD RESULT																		
GONIOSCOPY	X	X	X	X	X	X	X	X	X	X	X	X	X	X	X	X	X	X
PHOTOS																		
P/C EXAM																		
REFERRAL																		
RECALL																		

COMMENTS:

GLAUCOMA/GLAUCOMA SUSPECT CONTROL SHEET--LEFT EYE TABLE 1 (Cont.)

NAME:_____ DOB:_____

KEY:

IOP	* METHOD: N=NCT G=GOLDMANN S=SCHIOTZ
NERVE HEAD	* C/D: NOTATE ESTIMATE; DRAW APPEARANCE ON FIRST VISIT AND WHEN ANY CHANGE IS NOTED.
	* R/T: NOTATE WNL OR ABNORMAL. IF ABNORMAL, DETAIL HERE OR IN COMMENTS; INCLUDE DRAWING.
	* METHOD: 90=90 D H=HRUBY LENS F=FUNDUS CONTACT LENS I=INDIRECT OPHTHALMOSCOPE
SLE	* SIGNS: NOTATE WNL IF NORMAL. IF ABNORMAL, DETAIL IN COMMENTS.
	* ANGLES: VON HERRICK GRADING
VF	* METHOD: A=AUTOMATED T=TOPCON A/P =AUTOPLOT TS=TANGENT SCREEN C=CONFRONTATIONS F=FRIEDMANN
	* RESULT: (-) IF NORMAL (+) IF ABNORMAL; DESCRIBE IN COMMENTS
GONIOSCOPY	* SHAFFER METHOD OF GRADING; GRADE EACH QUANDRANT
PHOTO	* CHECK BOX IF DONE
P/C EXAM	* CHECK BOX IF PRIMARY CARE EXAM DONE DURING VISIT
REFERRAL	* NOTATE LAST NAME OF CONSULTANT
RECALL	* RECOMMENDED RECALL

DATE	MM																		
	DD																		
	YY																		
GLC MEDS DATE STARTED																			
IOP	METHOD																		
	READING																		
	TIME																		
NERVE HEAD	C/D																		
	DEPTH																		
	R/T																		
	METHOD																		
SLE	SIGNS																		
	ANGLES																		
VF	METHOD																		
	RESULT																		
GONIOSCOPY		X	X	X	X	X	X	X	X	X	X	X	X	X	X	X	X	X	X
PHOTO																			

COMMENTS:

TABLE 2

TABLE 2

GLAUCOMA SUSPECT FLOW SHEET O.D.

NAME : Don Ellsworth DOB: 6-12-08

FAM Hx: ———

PER Hx: ———

MEDS (GLAUCOMA): ———

MEDS (OTHER): ———

(See back for KEY)

DATE MM DD YY TIME	7 7 1986	7 18 1986 9:00	9 19 1986 9:00	10 24 1986	11 14 1986	3 20 1987	9 23 1987 10:20	10 28 1987 10:45	4 20 1988 10:00	8 24 1988 10:22							
IOP TYPE OD	G 22	G 20	G 19, 20	G 18	G 16	G 16	G 14	G 16	G 14	G 18							
NERVE HEAD	0.6/0.5 2D	0.6/0.5 2D RTN			0.6/0.6 Halo "appearance of RT 2-3D	0.6/0.6 2 D	0.6/0.5 2-3DD RTN	0.6/0.5 2-3DD RTN	0.5/0.5	0.6/0.6 4DD RT 1/8D							
SLE & ANGLES	3 A/P	WNL T					WNL 4 T		WNL 3 T	WNL 2-3 T							
GF	(-)	(-)					(-)		(-)	(-)							
GONIO	|	|	|	|	|	|	|	|	|	|	|	|	|	|	|	|	|
PHOTO																	
P/C EXAM	✓																
REFER																	

(Expand any of the above entries here:)

COMMENTS:

GLAUCOMA SUSPECT FLOW SHEET O.S.

TABLE 2 (Cont.)

NAME: Don Ellsworth DOB: 6-12-08

KEY: MEDS (GLAUCOMA) - DATE AND MED STARTED
IOP - TYPE: N=NCT G=GOLDMANN S=SCHIOTZ ;AND READING
NERVE HEAD - C/D: NOTATE ESTIMATE EACH VISIT; DRAW APPEARANCE IN COMMENTS ON 1st VIS. - ALSO ANY VISIT WHEN CHANGE IS NOTED
- DEPTH: NOTATE ESTIMATE EACH VISIT
- RIM TISSUE: NOTATE WNL OR ABN EACH VISIT - IF ABNORMAL, DETAIL IN COMMENT WITH DRAWING
SLE - NOTATE WNL IF NORMAL, OTHERWISE DETAIL HERE OR IN COMMENTS
ANGLES - VON HERRICK GRADING
VF - METHOD: T=TOPCON, A=AUTOMATED, A/P=AUTOPLOT, F=FRIEDMANN TS=TARGET SCREEN, C=CONFRONTATION - IF WNL, + IF ABNORMAL AND NOTATE IN COMMENTS
GONIO - SCHAFER METHOD OF GRADING (GRADE EACH QUADRIANT) ALSO NOTE IF PIGMENT PRESENT IN TRABECULUM BY P IF PRESENT
PHOTO - CHECK IF DONE (DO PHOTO ON 1st SUSPICIOUS VISIT & ANY TIME CHANGE OCCURS.
P/C EXAM - CHECK IF PRIMARY CARE EXAM DONE DURING VISIT
REFER - NOTATE LAST NAME OF CONSULTANT

DATE MM DD YY TIME	7 7 1986	7 18 1986 9:00	9 19 1986 9:00	10 24 1986	11 14 1986	3 20 1987	9 23 1987 10:20	10 28 1987 10:45	4 00 1988 10:00	8 04 1988 10:02							
IOP TYPE OS	G 22	G 20	G 18, 17	G 17	G 16	G 14	G 15	G 16	G 14	G 18							
NERVE HEAD	0.6/0.5 2D	0.6/0.5 RT N 2D			0.5/0.5 2-3D Halo like RT	0.6/0.6 2D	0.6/0.6 2-3 DD RT N	0.4/0.6 2-3 DD RTN	0.4/0.6 Notch in RT @ 2:00	0.7/0.8 4DD RT NOTCH @ 2:00.							
SLE & ANGLES	3	WNL					WNL 4			WNL 2-3							
VF	A/P —	T —					T Reduced consis fie		T (-)	T (-)							
GONIO																	
PHOTO		✓															

(Expand any of the above entries here:)
COMMENTS:

TABLE 3

GLAUCOMA SUSPECT FLOW SHEET O.D.

NAME : Bill Hardigan DOB: 5-12-32

FAM Hx: ———

PER Hx: ———

MEDS (GLAUCOMA): ———

MEDS (OTHER): ———

(See back for KEY)

DATE MM DD YY TIME	9 27 1985 1:32	10 3 1985 10:40																
IOP TYPE OD	17,14	G 16																
NERVE HEAD	0.6/0.4 RT Notched @ 9:00	0.4/0.3 BTN.																
SLE & ANGLES	WNL 2-3	WNL 3																
UC	F (-)	T (-)																
GONIO	|	|	|	|	|	|	|	|	|	|	|	|	|	|	|	|	|	|	|
PHOTO																		
P/C EXAM	✓																	
REFER																		

(Expand any of the above entries here:)
COMMENTS:

TABLE 3 (Cont.)

GLAUCOMA SUSPECT FLOW SHEET O.S.

NAME : Bill Hardigan DOB: 5-12-32

TABLE 3(Cont.)

KEY: MEDS (GLAUCOMA) - DATE AND MED STARTED
IOP - TYPE: N=NCT G=GOLDMANN S=SCHIOTZ ;AND READING
NERVE HEAD - C/D: NOTATE ESTIMATE EACH VISIT; DRAW APPEARANCE IN COMMENTS ON 1st VIS.
- ALSO ANY VISIT WHEN CHANGE IS NOTED
- DEPTH: NOTATE ESTIMATE EACH VISIT
- RIM TISSUE: NOTATE WNL OR ABN EACH VISIT
- IF ABNORMAL, DETAIL IN COMMENT WITH DRAWING
SLE - NOTATE WNL IF NORMAL, 'OTHERWISE DETAIL HERE OR IN COMMENTS
ANGLES - VON HERRICK GRADING
VF - METHOD: T=TOPCON, A=AUTOMATED, A/P=AUTOPLOT, F=FRIEDMANN
TS=TARGET SCREEN, C=CONFRONTATION
- IF WNL, + IF ABNORMAL AND NOTATE IN COMMENTS
GONIO - SCHAFER METHOD OF GRADING (GRADE EACH QUADRIANT)
ALSO NOTE IF PIGMENT PRESENT IN TRABECULUM BY P IF PRESENT
PHOTO - CHECK IF DONE (DO PHOTO ON 1st SUSPICIOUS VISIT & ANY TIME CHANGE OCCURS
P/C EXAM - CHECK IF PRIMARY CARE EXAM DONE DURING VISIT
REFER - NOTATE LAST NAME OF CONSULTANT

DATE MM / DD / YY / TIME	9 / 27 / 1985 / 1:32	10 / 2 / 1985 / 10:40															
IOP TYPE / OS	13,15	G / 14															
NERVE HEAD	0.4/0.35	0.3/0.3															
SLE & ANGLES	WNL / 2-3	WNL / 3															
VF	F (-)	T (-)															
GONIO	|	|	|	|	|	|	|	|	|	|	|	|	|	|	|	|	|
PHOTO		✓															

(Expand any of the above entries here:)
COMMENTS:

TABLE 4

PROTOCOL OF MANAGEMENT OF GLAUCOMA SUSPECTS

PROCEDURE	METHOD	FREQUENCY/YEAR
INTRAOCULAR PRESSURE	GOLDMANN TONOMETRY	2-4+
GONIOSCOPY	GOLDMANN 3-MIRROR ZEISS 4-MIRROR	1-2
DILATED OPTIC NERVE EVALUATION	DIRECT OPHTHALMOSCOPE (COLOR RATIO) 90 D LENS, RHUBY LENS, FUNDUS CONTACT LENS (CONTOUR RATIO)	1-2
VISUAL FIELDS	AUTOMATED	IF VF AND OTHER SIGNS ARE STABLE--1 IF O.N. OR IOP CHANGES--2 IF SEVERE O.N. DAMAGE--4-12

FOOTNOTES

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5. Johnson, pp. 60-61.
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7. Johnson, p. 61.
8. Bartlett, p. 856.
9. Ibid.
10. Betts, pp. 26-27.
11. Thomas L. Lewis, "Working up a Glaucoma Suspect," Ocular Pathology Lectures, The Ohio State University, 1977.
12. Gerhard W. Cibis, "Congenital Glaucoma," Journal of the American Optometric Association, 58 (1987), pp. 729-30.
13. Betts, PP. 29-30.
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15. Johnson, pp. 59-65.
16. J. Boyd Eskridge, "Ocular Hypertension or Early Undetected Glaucoma?" Journal of the American Optometric Association, 58 (1987), pp. 750-1.
17. Paul Henkind, "Technology: It's Role in our Conception of Glaucoma," American Academy of Ophthalmology, 90 (1983), p. 755.
18. Eskridge, p. 751.
19. Ibid.
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21. Ibid.
22. Eskridge, pp. 752-3.
23. Bartlett, p. 867.
24. RL Weisman, CF Asseff, CD Phelps, et.al., "Vertical Elongation of the Optic Cup in Glaucoma," Trans American Academy of Ophthalmology and Otolaryngology, 77 (1973), pp. 157-61.
25. A. Gordon Balazsi, Stephen Drance, et.al., "Neuroretinal Rim Area in Suspected Glaucoma and Early Chronic Open-Angle Glaucoma," Archives of Ophthalmology, 102 (1984), p. 1014.
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27. V. John Ford, "How to Follow 'Ocular Hypertensives,'" Annals of Ophthalmology, (1982), pp. 309-10.
28. Kevin M. Miller and Harry A Quigley, "The Clinical Appearance of the Lamina Cribrosa as a Function of the Extent of Glaucomatous Optic Nerve Damage," Ophthalmology, 95 (1988), pp. 135-8.
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30. Ibid.
31. Zuhair M. Shihab, Pei-Fei Lee, et.al, "The Significance of Disc Hemorrhage in Open Angle Glaucoma," American Academy of Ophthalmology, 89 (1982), pp. 211-13.
32. Bartlett, p. 812.
33. Alfred Sommer, Harry A. Quigley, et.al, "Evaluation of Nerve Fiber Layer Assessment," Archives of Ophthalmology, 102 (1984), pp. 1766-71.
34. Eskridge, p. 758.
35. Bartlett, p. 872.
36. Betts, p. 9.
37. Bartlett, p. 873.
38. Betts, p. 10.

39. Bartlett, p. 873.
40. Eskridge, pp. 760-65.
41. David J. Kowal and Murray Fingeret, "A Glaucoma Control Chart," Journal of the American Optometric Association, 58 (1987), pp. 734-6.
42. John Carter, "Tonometry in Optometric Practice--the Current Status," Journal of the American Optometric Association, 48 (1977), pp. 191-7.
43. Ford, p. 310.
44. Leonard Thurschwell, "How to Perform Gonioscopy and Peripheral Retinal Examination with a Goldmann Three-Mirror Contact Lens," Southern Journal of Optometry, 1 (1983), p. 18.
45. Paul Ajamian, "Glaucoma Management," North Central Optometric Conference, Minneapolis MN, January 26, 1990.
46. Ford, p. 310.
47. Eskridge, p. 753.
48. Peter Lalle, Murray Fingeret, and S. Barry Eiden, "Automated Perimetry in the Management of Glaucoma," Journal of the American Optometric Association, 60 (1989), pp. 908-9.

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