DIAGNOSTIC DIAGRAMS®

OPHTHALMOLOGY

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Williams & Wilkins

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Foreword

Why another basic ophthalmology textbook for medical students beginning clinical rotations? There has been an explosive increase in the volume of health science information which medical students must learn. As students rotate through different clinical disciplines they must assimilate pertinent examination skills needed to evaluate disorders of the various organ systems, learn common disease presentations and differential diagnoses, and determine whether a particular medical specialty would be an interesting life work.

Ophthalmology is a unique specialty; it has its own language and many descriptive terms are used only for eye and orbital disease. Esoteric examination techniques are often needed to evaluate ophthalmologic problems. Some common diseases only affect the eye; and some systemic illnesses, ocular fundus changes provide the most lucid data on disease status.

Most of the available ophthalmology textbooks are two encyclopedic for beginning students. There is a need for a concise presentation which gives a broad overview of ophthalmology, describes the use and findings observed with ophthalmoscopy and slit lamp biomicroscopy, and helps the student to develop a coherent approach to common eye problems.

I believe this book serves these needs quite well. It is not meant to be an exhaustive textbook on ophthalmology; a student will receive a basic grounding in clinical approaches to the diagnosis and treatment of common conditions. A short definitive reading list appropriate for medical students is attached; those students who wish to become "real doctors" (i.e., ophthalmologists) will want to delve more deeply into the eye literature.

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San Francisco

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INTRODUCTION

In a medical student's short ophthalmology rotation, the emphasis is primarily on identifying and delineating the ocular diagnosis. *Diagnostic Diagrams* presents a stepwise approach for clinical decision making. Each chapter has a flow chart and supportive text to amplify the diagrammed material. The text provides some insight into how to approach problems related to the eye or adnexa (lids and orbit). It also suggests which conditions may be recognized by the non-ophthalmologist and which require an ophthalmologic consultation.

It is necessary to make an accurate diagnosis before initiating any therapy. This book does not discuss treatment unless it is useful for clarifying a diagnosis.

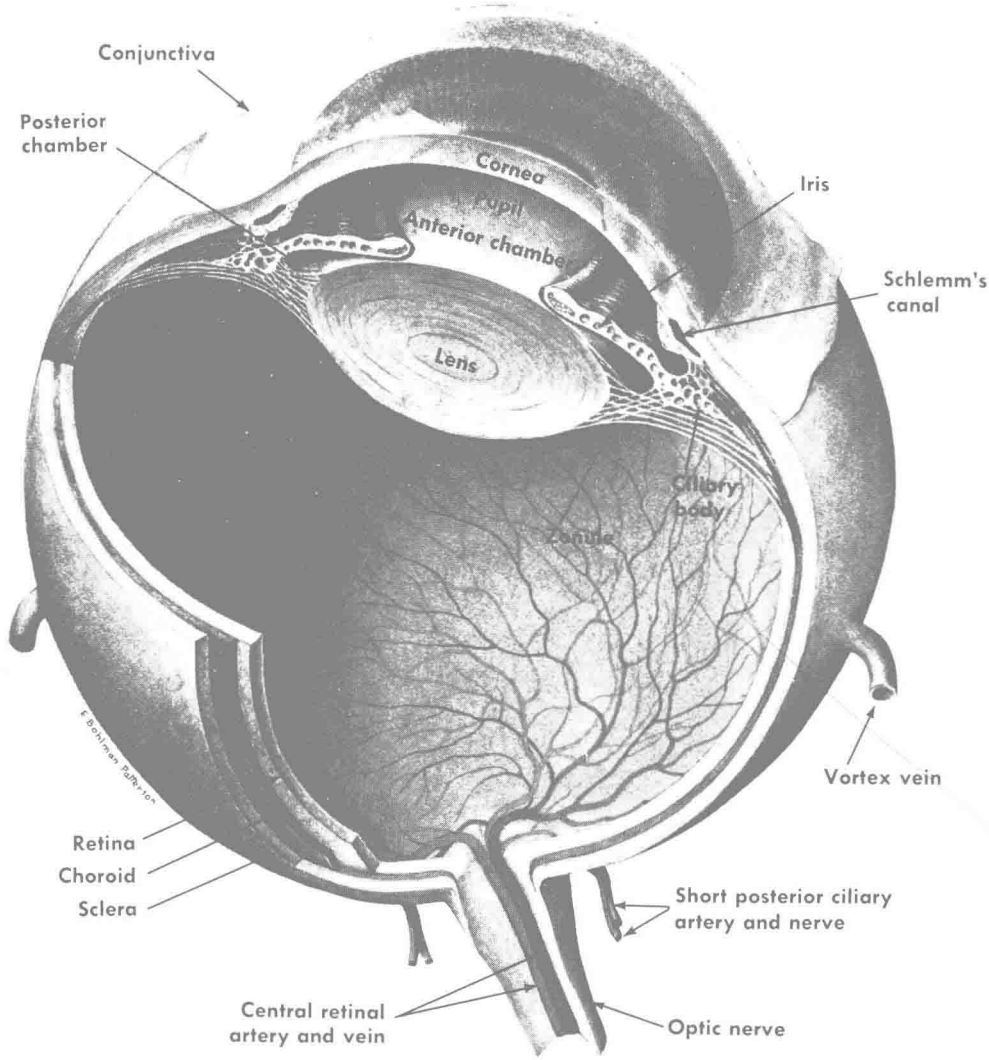
The work stresses the need for a complete eye evaluation, including accurately measuring vision and dilating the pupils. Ophthalmic consultation may be required to finalize the diagnosis; however, a student or practitioner can often diagnose important ophthalmologic problems.

These diagnostic diagrams will not work all the time. They are designed to highlight key steps in the differential diagnosis of eye disorders, not to provide final answers. The text focuses on a limited number of complaints, chosen as common, significant and, for the most part, treatable.

Ophthalmic diagnosis requires a certain amount of pattern recognition. There is no substitute for performing as many eye examinations as possible. A basic understanding of ophthalmology's specialized vocabulary and its diagnostic approach should improve the reader's ability to evaluate patients with eye complaints of local and systemic origin. The book's usefulness will increase as the practitioner performs more eye examinations and becomes familiar with the diagnostic framework.

At the end of the text is a short list of suggested references, including a few major texts as well as some "classic" journal articles. These materials should be available in most medical school and ophthalmic libraries.

INTRODUCTION



The human eye

Figure 1.1 The human eye. (Reprinted with permission from Newell FW, Ernest JT: *Ophthalmology: Principles and Concepts*, ed. 5. St. Louis, C. V. Mosby, Co., 1982.)

Contents

CHAPTER 1
VISUAL LOSS AND THE EYE EXAMINATION

INTRODUCTION

The objective of an ophthalmologic evaluation is either to diagnose and subsequently treat isolated visual system disorders or to use findings obtained from an eye examination to better characterize systemic illness.

HISTORY

The purpose of the history is to delineate the nature of the patient's eye complaint. The reason for his or her visit may be a problem related to visual acuity, lid swelling, double vision or orbital swelling, or may be to complete an evaluation of an endocrine or neurologic condition.

Patients frequently seek ophthalmologic care because of decreased visual acuity. If a patient has one normal eye and one with poor vision, with both eyes open he sees normally (20/20). Very often, people do not realize that they have reduced acuity in one eye until days, weeks, months or even years after the fact. They may discover the loss by randomly covering one eye and suddenly becoming aware of the blurred vision in the other eye.

Of primary importance in the patient history is dating the onset of the visual loss. Has the patient had a previous eye examination? When was that? It is often possible to get some indication of patients' past acuity by asking if their eyes were tested to obtain a driver's license (or get it renewed). Did they have an eye examination in the military? Parents should know if a child passed a school eye examination in the past year.

Other useful questions include the following:

*Has one eye always been weaker?

*Has the visual loss had a gradual onset, or has it been acute? Is it progressive? A sudden loss of vision may be the result of a vascular event for example. Gradual decrease in vision may result from such causes as cataract or metabolic disease.

*Is the visual loss transient? Does it come and go? Transient visual loss may implicate, for example, retinal arteriolar emboli, carotid artery disease, transient obscurations in vision associated with various optic neuropathies, and sometimes, papilledema.

In delineating the patient's medical history, it is important to determine if there is a history of medications which might have ocular effects. Among the known complications are the following:

1) Cycloplegia, induced by such drugs as atropine;

2) Cataractogenesis, a potential complication of systemic or topical steroid use;

3) Retinal toxicity: chloroquine and phenothiazines are two examples that have the potential to reduce vision through retinal damage significantly.

Another important aspect of the medical history is determination of any pre-existing systemic disease that might compromise vision. Examples include diabetes, sickle cell anemia, hypertension and multiple sclerosis. Many systemic diseases cause eye findings, and it is the role of the examiner to discover them through a thorough history interview.

Finally, a complete history includes a record of positive family history for diseases such as glaucoma, sickle cell anemia, and diabetes. These are the primary concerns although family history may also be useful in elucidating certain hereditary eye diseases.

Following the history interview, the physician should perform an abbreviated review of systems, not as thorough as a complete medical work-up but sufficient to document any positive findings in the history.

THE EYE EXAMINATION

The most important component of the eye examination is an accurate assessment of the visual acuity. If the patient has 20/20 vision (with glasses) the

likelihood of a serious problem decreases. If the vision is not 20/20, it is reasonable to request an ophthalmic consultation on that basis alone. Any screening examination should also include assessment of pupillary size and function as well as ocular motility.

Visual Acuity

A complete examination begins with recording the vision in each eye separately, with the most recent available pair of glasses. Hospitals usually have large eye charts or a small pocket-card chart. If neither is available, a book or magazine is useful; vision is recorded as "able to read headlines," "able to read small print," and so on. Near as well as distance acuity should be tested.

Visual acuity is best checked with a Snellen-type distance or projection chart, yielding a ratio measurement such as 20/40 or 20/100. The numerator is the distance at which the patient views the chart. The denominator is the standard distance at which a normally-sighted person reads the line in question. Therefore, someone with 20/100 vision sees at 20 feet letters that a person with normal vision can see 100 feet away. Although "20/20" is the standard for normal vision, about 3% of the population actually see better.

If the patient cannot see the 20/200 letter, he or she moves to 10 feet away from the letter; if the letter is then visible, visual acuity is 10/200. If an individual cannot see any of the letters, the acuity is assessed by testing the ability to count the examiner's fingers at a given distance. Such vision is recorded as, for example, "counts fingers at three feet." One, two or five fingers are used; holding up three or four fingers is often confusing. Anyone who sees 20/200 or less is "legally blind".

Patients unable to count fingers may see "hand motion", judged by the examiner's waving his hand in different areas of the visual field. In the absence of hand motion vision, an examiner asks if light is visible. Such vision may be recorded as "light perception with projection coming from the left and right" or perhaps "questionable light perception".

If vision with glasses is reduced, retesting with a pinhole held in front of the glasses determines whether the visual loss is refractive in nature. Most eye clinics have plastic pinholes. However, a pinhole test can easily be improvised by punching a small hole (1-2mm) in a 3 $\times$ 5 index card. The patient holds the card in front of his eye and reads the letters. Pinhole vision improvement usually suggests the need for a change in lens prescription.

Refraction is routinely done by an ophthalmologist or optometrist; however, there is no reason why a non-specialist cannot do a refraction. The underlying principle is the correction or neutralization of any refractive error. In a nearsighted (myopic) eye, the eye is often too long and the visual image falls in front of the retina; minus ($-$) lenses move the image to its proper position. Similarly, in the farsighted (hyperopic) eye, the eye is usually too short and images focus behind the retina; plus ($+$) lenses correct the positioning. Using a trial lens set or refractometer, the examiner can delineate the correct lens strength to neutralize the refractive error, and the patient has his or her "best corrected vision" (20/20 if refractive error is the only reason for decreased vision).

Correction of corneal irregularity (astigmatism) is somewhat more complex. The examiner must correct not only the position of the image through the media as a whole with plus or minus lenses, but must determine the axis in which additional astigmatic correction is required. Although the process is tedious, with practice the general practitioner can make a reasonably accurate determination.

A gross evaluation of the visual field may be performed using a confrontation technique. The same eye is covered for the patient and the examiner; the patient is then asked to count the examiner's fingers in each quadrant while looking straight ahead. This establishes a relative measure, assuming of course that the examiner's fields are normal.

The Amsler grid provides another measurement of

the visual field. Available in eye clinics, the grid is a grilled piece of paper used to map small central defects in the visual field. With one eye occluded, the patient is asked to look at the center of the grid and outline areas that appear distorted, wavy or otherwise abnormal. Results from this test are useful clues in diagnosing macular disease.

Tangent screens, Goldmann perimeters, and computer-assisted perimeters are available in eye clinics for more reproducible determinations of field defects. Whenever a visual field defect is suspected, ophthalmic advice should be sought.

Color Vision

Although color vision is not routinely tested, measuring color vision is important in patients with visual loss. One of the hallmarks of optic nerve dysfunction is loss of color vision. A gross assessment of color vision in each eye can be obtained by asking the patient what color the examiner's clothing is. Another good way to assess color vision is to hold up a small object (such as the red top of a pen) and ask the color. The patient should cover one eye, and then the other, and describe the relative brightness of the color, whether it appears washed out in one eye. The object should be viewed in various parts of the visual field particularly centrally, and again described. Eye clinics have standardized color plates for more definitive testing.

Pupils

Pupils are normally the same size; if they are not, there is an abnormality termed anisocoria (see Chapter 3). Symmetric pupils do not preclude disease, however, as severe pathology and even blindness is found in eyes with pupils of normal appearance.

After noting the approximate size of the pupils and whether or not they are equal, the examiner should test each pupil's reactivity and the briskness of response to bright light.

One of the most important steps in evaluating a patient with visual loss is the Marcus Gunn test, to detect an "afferent pupillary defect". When one pupil is illuminated, both should contract. There is a relative dilation of both pupils if the light is cast on a blind eye or on an eye with even minimal dysfunction in the afferent visual system.

For this test, the patient looks at an object across the room to eliminate any near response pupillary constriction. The physician shines a flashlight into one of the patient's eyes for a couple seconds, then shifts the light to the second eye, while watching both eyes. If both pupils are normal, when the light is moved from one eye to the other, there is no significant change in the size of *either* pupil. When the light shines on an eye with an afferent defect, there is a gradual dilation of both pupils. Several metronome-like swings of the light may be needed to make the diagnosis. There is a tendency when learning this test to hold the light on the affected pupil for too long a period of time; this bleaches the photoreceptors and may produce a pseudoafferent defect.

If the afferent defect is subtle and difficult to detect, the patient may be asked about the relative brightness of the light when shined in each eye. Though not recorded as an objective afferent pupillary defect (the Marcus Gunn test), such a subjective afferent pupillary defect is worth noting. If there is an objective afferent defect, there should be subjective confirmation.

Extraocular Movements

It is important to determine the position of the patient's eyes in "primary gaze" when the patient is sitting in front of the examiner, looking straight ahead. Any deviation from straight should be recorded.

This is followed by the cover/uncover and alter-

nate cover tests to detect phorias and tropias. (See chapter on strabismus.)

In the primary position, any manifest instability of gaze or nystagmus is recorded. Ocular motility, including degree of excursion, should be assessed, by having the patient look in all directions. During this portion of the exam it is also possible to check for nystagmus in the extremes of gaze. In more thorough examinations (usually neuro-ophthalmologic), an evaluation of the pursuit system (tracking ability) and saccades (rapid eye movements) is done in both the horizontal and vertical pathways.

External Examination

This examination includes a check of lid position, looking for any ptosis (lid droop) or lid retraction. The lashes and lacrimal system are examined for any apparent pathology, such as redness and swelling of the lids, styes, or other skin lesions.

Any obvious proptosis (exophthalmos) should be noted. If subtle proptosis is suspected, an exophthalmometer can confirm the diagnosis.

Finally, the examiner should record the overall appearance of the eyes, whether they are white or red and injected.

Slit Lamp Examination

The slit lamp biomicroscope enables the physician to have a magnified stereoscopic view of the ocular adnexa and the eye. In the emergency room, the general physician may use it primarily to determine whether or not there is an ocular foreign body. In routine eye examinations, the slit lamp is useful to detect any abnormality of the conjunctiva, cornea, anterior chamber, iris or lens. The presence or absence of a Kaiser-Fleischer ring, diagnostic in Wilson's disease, is one example. Cataract formation secondary to long-term systemic corticosteroids is also quickly discerned at slit lamp exam.

Because the slit lamp provides an excellent ocular view, physicians should become familiar with the instrument and the examinations it facilitates. It is appropriate to do a slit lamp evaluation both before and after dilation since it is difficult to comment on the lens and the anterior vitreous cavity without dilation.

Intraocular Pressure

To measure the intraocular pressure, the examiner uses a Schiotz or an applanation tonometer. The beginning examiner cannot estimate the pressure by palpation through the lid unless the globe is rock-hard. Finger tensions are not recommended by these authors.

The Schiotz tonometer is an easily portable instrument. It provides a simple and accurate method of tonometry. The patient is tested in the supine position, following topical anesthesia. The patient should fixate on a spot on the ceiling so that his eyes are in primary position. Since glaucoma is prevalent in the general population, it is important for the general physician to be able to detect it.

Depth of the Anterior Chamber

It is important to determine the anterior chamber depth prior to dilating patients' eyes. Patients with shallow anterior chambers may develop angle closure glaucoma when dilated.

Assessment of the anterior chamber depth is easy to do either at the slit lamp or with a hand light. The light is held laterally to the patient's eye so that it shines across the anterior chamber. If the chamber is shallow, a shadow blocks transmission of the beam; the beam is easily visible across the iris of a normal eye. Once a practitioner has seen a patient with a shallow anterior chamber, he or she should be able to use this test routinely to estimate the anterior chamber depth.

The Lens, Fundus and Vitreous

If the anterior chamber appears shallow and predisposed to narrow angle glaucoma, an ophthalmic consultation is appropriate. (It should be noted that dilation rarely provokes an attack of narrow angle glaucoma.)

If the anterior chamber depth appears normal, it is safe to dilate the eye for a fundus examination. Dilation significantly improves the examiner's ability to view and evaluate ocular structures and should be done whenever possible. The drops that many physicians use are 2½% phenylephrine (Neosynephrine) and/or 1% tropicamide (Mydriacil).

Once the pupils are dilated, the lens and vitreous can be evaluated. A clear view of the fundus implies that the vitreous cavity is clear and that there is no cataract. (See chapter on opacities of the ocular media.) The optic nerve head, the macular area and retinal vessels can also be evaluated at this point.

Whenever a patient is dilated, but especially in the case of inpatients undergoing evaluation, it is of critical importance to note in the chart that the patient has received dilating drops. In the absence of such a notation, the patient may be subjected to acute medical or surgical intervention in the belief that the dilation represents, for example, a ruptured intracranial aneurysm.

THE DIFFERENTIAL DIAGNOSIS

There are four major causes of central visual loss: refractive errors, compromise of the ocular media, optic nerve disease, and macular disease.

Refractive Errors

In normal eyes, the *refractive error* changes over time. Children are relatively hyperopic at birth, and become less so as they get older. Adults, as they age, become presbyopic, needing additional (+) lenses for reading.

Certain diseases cause rapid fluctuations in the refractive error. The most common example is diabetes where elevations in serum glucose cause lens swelling and secondary myopia.

Changes in the *shape of the globe* can change the refraction. For example, a chalazion, or lid tumor, can push on the front of the eye, distorting the cornea, and causing a significant astigmatic alteration of the refraction.

Changes in the position of the lens (lens subluxation or dislocation) can also significantly alter the refractive error. Traumatic or spontaneous lens dislocation with the lens free floating in the vitreous creates a functional blindness in someone previously able to see 20/20. Such aphakia may be corrected with the appropriate refraction although surgery to remove the dislocated lens may become necessary.

Some *retinal diseases* cause refractive errors. Any process which elevates the photoreceptors and moves them anatomically forward (lesions behind the eye, choroidal tumors, or central serous chorioretinopathy) causes hyperopia.

To begin to differentiate among these problems, a refraction is needed. Although the pinhole examination is a sound clue that an alteration in the refractive error is the cause of the problem, to quantify the error and decide whether it is a hyperopic or myopic shift or to determine the axis of the astigmatic error requires refraction.

In summary, a refraction determines the refractive error. Some pathologic causes of refractive error include:

*abnormal corneal topography;

*rapid metabolic shifts, for instance in diabetes, causing myopia (or, sometimes, hyperopia);

*increasing myopia from early incipient nuclear sclerotic lens changes (cataract);

*hyperopia from central serous retinopathy; and,

*orbital or choroidal tumors.

Opacities in the Ocular Media

It is relatively easy to diagnose the presence of opacities in the media; it may be very difficult to determine the cause. For details, see the chapter on opacities in the ocular media.

As part of the eye examination, the examiner grades the media. For example, the "view" of the retina may be anywhere from "20/20" to negligible. At the end of the examination, it should be possible to determine whether the patient's vision is equal to or worse than the media suggests. If someone has approximately "20/100 worth of cataract," he or she cannot see *better* than 20/100; if the patient's actual acuity is only 5/200, this is a clue that visual reduction is not only due to opacification of the media.

Basic Anatomy of the Retina and Major Problems Causing Visual Loss

The *fovea centralis* or macula, some 1,500 microns wide, is approximately as wide as the optic nerve head. The ganglion cell layer in this portion of the retina is greater than one cell thick. The *foveola*, at its center, is about 350 microns wide, contains only cones and is the locus of 20/20 acuity. The visual acuity of peripheral retinal input is approximately 5/200.

The entire retina contains about 7 million cones and 130 million rods. Twenty-twenty acuity can exist in the presence of extensive retinal disease so long as the fovea is not involved. Conversely, a pinpoint lesion in the fovea can reduce vision to legal blindness (20/200 or less).

If retinal pathology is responsible for *reduced acuity*, the foveola (or its axons coursing in the nerve fiber layer to the optic nerve) must be involved; usually this occurs as a result of a compromised vascular supply, distortion of the normal arrangement of photoreceptors, or edema fluid accumulation.

To understand how diseases in the fovea centralis, and not the foveola, can compromise visual acuity, some appreciation of the anatomy of the retinal nerve fiber layers is necessary. Cone photoreceptors send their impulses to horizontal, bipolar and amacrine cells where the initial processing of visual information occurs. The impulses then travel to the ganglion cells, which send their axons through the most superficial layer of the retina (the nerve fiber layer) to the optic nerve.

If these axons in the papillomacular bundle are damaged, the result is similar to directly damaging the photoreceptors in the foveola, and reduced visual acuity may result. To explain reduced acuity, a lesion must be anterior to the optic chiasm. Lesions posterior to the chiasm cause field defects, but do not affect the acuity unless they are bilateral, which is unusual.

Lesions Involving the Macula and Optic Nerve

It may be fairly difficult to differentiate lesions involving the macula and the optic nerve. Visual field exams are helpful to pinpoint certain subclasses of optic neuropathies. Fluorescein angiography is a useful adjunct to diagnose diseases of the macula. This is a relatively simple outpatient test involving intravenous injection of fluorescein, a vegetable dye.

Optic nerve disease may be caused, for example, by anterior ischemic optic neuropathy, demyelinating diseases, compressive disease, toxic optic neuropathies, retrobulbar optic neuropathies, infiltrative optic neuropathies, hereditary optic neuropathies and congenital anomalies. The hallmarks of optic nerve disease are reduced central acuity, reduced color vision and density, and an afferent pupillary defect.

Afferent pupillary defects (Marcus Gunn pupils) are much more common with optice nerve lesions than with macular lesions. They are also seen with relatively gross retinal lesions, for instance, a large retinal detachment. Subtle (0-2+) afferent pupillary

defects may occur with macular degeneration. As a rule, however, 3+ or 4+ afferent defects are only seen with optic nerve defects or large retinal detachments.

In many patients there is no obvious fundus pathology and vision is reduced in both optic nerve and macular disease. A review of the results of the eye exam may assist in differentiating lesions of the macula and the optic nerve:

*Pupillary defects are the rule in optic nerve disease; in general, they are absent or subtle in macular disease.

*On visual field examination, patients with optic neuropathies have areas of decreased vision ("scotomas"), often absolute. Patients with macular disease also have scotomas. They are usually not absolute; patients often complain of distortion (metamorphopsia) of the test object rather than inability to see it.

*In many macular diseases, color vision is often near-normal. Patients with optic nerve disease show abnormal color vision.

In patients with optic nerve disease the fundus lesions, if any, are optic nerve lesions. Similarly, with macular disease any fundus lesions are macular.

If the eye examination is apparently normal in a patient who complains of visual loss, there are four possibilities:

1. Most likely, a pathologic finding has been overlooked.

2. There is subclinical macular disease. This can usually be detected by an ophthalmologist using intravenous fluorescein angiography and various psychophysical tests of macular function, (e.g. electroretinogram, electrooculogram, dark adaptation, etc.).

3. The diagnosis is a retrobulbar optic neuropathy. These patients usually have reduced visual acuity and color vision, visual field defects and an afferent pupillary defect and an abnormal visual evoked response.

4. The patient is malingering, complaining of a problem for which there is no anatomic or pathophysiologic explanation. It is easy to suspect malingering, but difficult to demonstrate it. These cases should be referred to a neuro-ophthalmologist.

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VISUAL LOSS