
The Ophthalmoscope, the Fundus Oculi, and Central and Peripheral Vision

1

The examination of the eye consists of five parts. This chapter deals with the fundus oculi and with central and peripheral vision. The remaining three parts of the examination are described in later chapters.

THE OPHTHALMOSCOPE

There are five mechanical details you need to know about the head of the ophthalmoscope. The following remarks apply to the Welch-Allyn ophthalmoscope (Figure 1–1).

Buy an ophthalmoscope with a halogen bulb and handle that takes D-size batteries. A handle containing a rechargeable cell is almost as big as the D-size battery model and just as good. The ophthalmoscope that takes AA batteries is undesirable. The power does not last long enough, and the scope is difficult to hold.

When you attach the head to the handle, have the on-off button in front, as in Figure 1–1. To turn it on, push in the on-off button and turn the disc that contains it. It turns only one way. As you rotate the lens selector wheel, the numbers change in the lens strength window. This changes the amount of magnification between your eye and the patient's retina when you are looking through the viewing aperture. If the lens selector wheel is turned clockwise in the direction of the heavy arrow, increasingly stronger plus lenses continue to appear in the viewing aperture and increasingly higher black numbers continue to appear in the lens strength window. On the back of the ophthalmoscope there is another adjustable wheel, the aperture selector, which rotates in a horizontal plane. You will find that rotating this wheel produces a green circle, a large white circle, a small white circle, or a grid. Turn it back to the large white circle and leave it there. (If the white circles appear orange, get new batteries, and if still orange, get a new bulb.)

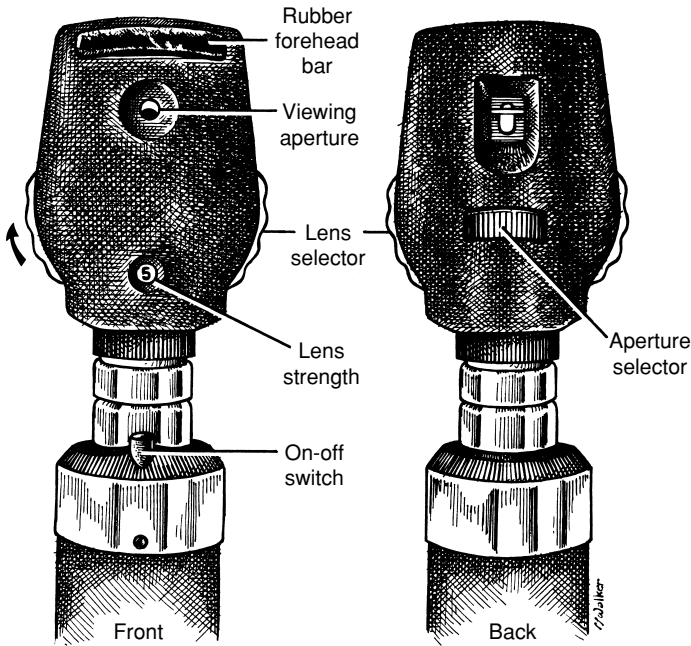


Figure 1-1. Details of the ophthalmoscope head (Welch-Allyn).

THE FUNDUS OCULI

There is a wealth of information in the fundus of the eye for any physician with an ophthalmoscope. Practice looking at the fundus of young persons, such as your classmates. Young people have bigger pupils, more patience, and usually no opacities of the cornea, lens, or vitreous. The retina in a young person is glistening and shiny with beautiful sharp details.

The first time you examine a retina, it is probably best if you and the subject are standing. You have more mobility and will be less awkward. However, as soon as you know how to do it, and as a regular practice, always sit and have the patient sit. Do not sit directly in front of the patient so that your knees hit his; sit facing him with your chair to one side of his. When you get to be an expert, you can examine supine patients, but do not do it until you have to.

- Ask the patient at the start not to hold his breath. This has a practical application on the size and pulsations of his retinal veins.

- With the patient sitting with his back to the window, pull the blinds down and ask the patient to remove his glasses. You do not need a blacked-out room, but remove any incidental light.

There are two things working against you when you use the ophthalmoscope. First, the eye adds all the light it is exposed to. The sum of the ambient light and the ophthalmoscope light, plus the balance between sympathetic and parasympathetic tone, plus the fact that the pupillary sphincter muscle is stronger than the dilator, determines the pupil size. Second, trying to look through a *reflecting* surface is difficult. On a sunny day you cannot look into a lake to any depth because of the reflection off the surface. However, if you hold a hat close to the water and look into the lake in the shadow of the hat, you can see into the water. Similarly, side light or ceiling light reflected on the patient's cornea or surface of his glasses will hinder you. When you examine the patient's fundus, you will partially eliminate reflections by holding the ophthalmoscope so close to the patient that it is touching her forehead. If you rotate the ophthalmoscope 5 or 10 degrees while holding it vertically, the light from the scope strikes the cornea at different angles and you will find the best "reflection-free" angle.

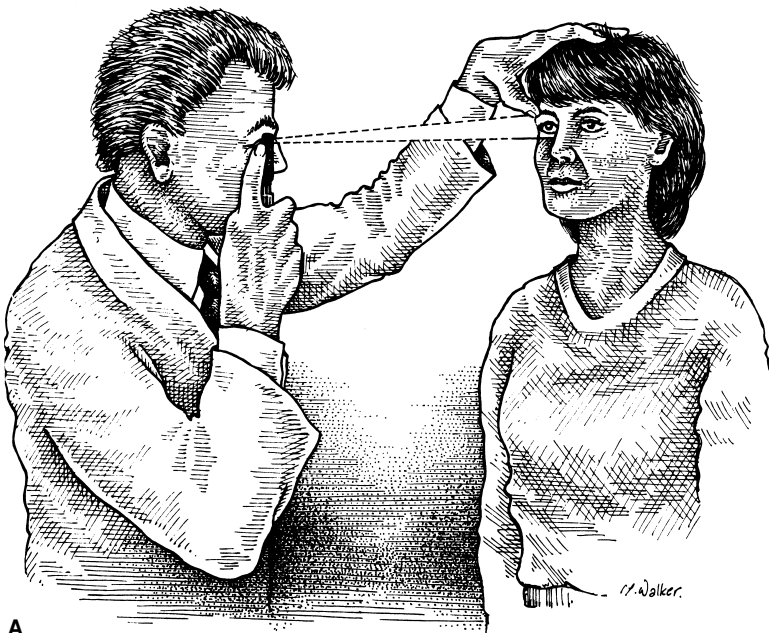
Some doctors keep their glasses on when using the ophthalmoscope; most, however, remove them.

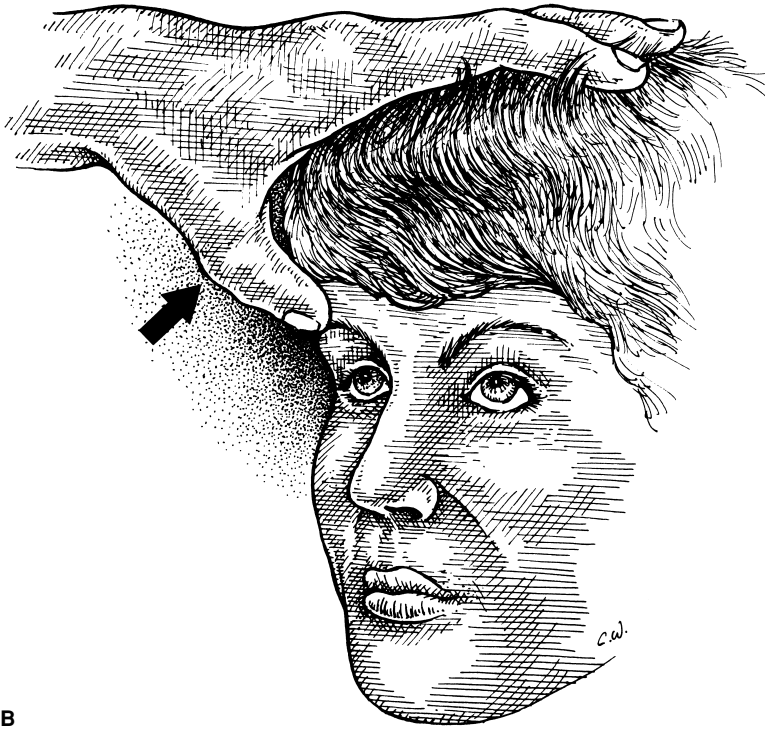
1. **Unless you have marked astigmatism, take your glasses off.** With your glasses off, the head of the ophthalmoscope can be closer to your eye and you will see a larger area of the patient's retina. Try it both ways, beginning with your glasses off. The pinhole effect (see under "Near Vision") of looking through the viewing aperture (Figure 1-1) may take care of your refractive error. If you are astigmatic, your glasses contain a cylinder; hold your glasses at arm's length, look through one lens with one eye and slowly rotate your glasses to the right and then to the left. If the object you are looking at tilts and elongates at one point in the rotation, then your prescription includes a cylinder; you are astigmatic, and you may have to wear your glasses when using the ophthalmoscope.
2. **Hold the ophthalmoscope in your right hand, turn it on, and look through the viewing aperture. You must get the viewing aperture as close as possible to your eye.** The soft rubber bar (Figure 1-1) horizontally placed across the top of the ophthalmoscope head is meant to fit firmly against or just below your eyebrow. There should be some contact between your skin and the rubber bar at all times when you are looking at the fundus oculi. Keep your index finger on the lens selector wheel. If you have the ophthalmoscope halfway down your nose, you

are, in effect, looking through a tube with your pupil at one end and the viewing aperture at the other.

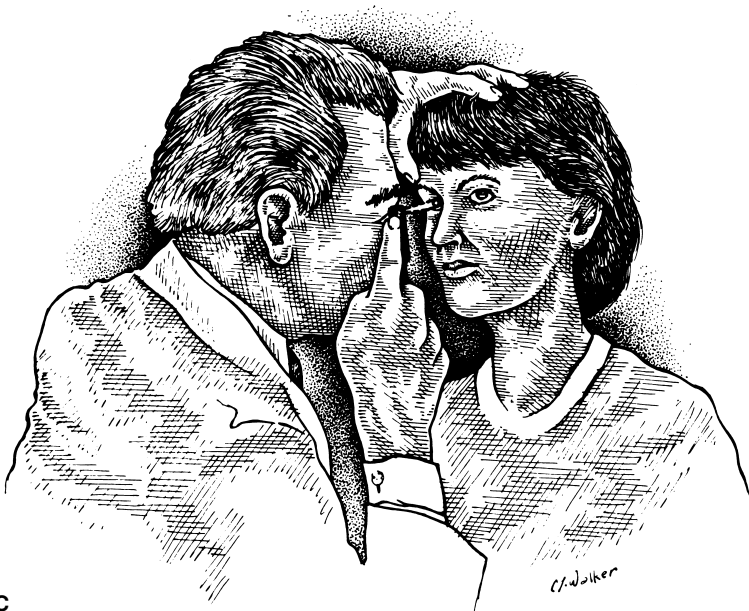
3. **Use your right eye to examine the patient's right eye and vice versa.** However, if you have one "weak" eye, use the other for examining both of the patient's eyes.
4. **Hold the ophthalmoscope in your right hand when examining the patient's right eye and in your left hand when examining the patient's left eye.**
5. **Keep the ophthalmoscope vertical, and keep both your eyes open.**
6. **When examining the patient's right eye, put your left hand on top of the patient's head (Figure 1-2B) and rest your forehead on your flexed thumb and vice versa when examining the left eye.** Ask the patient to stare at something at eye level on the other side of the room and to keep her eyes as still as she can. To examine the patient's right fundus, stand to the patient's right (not directly in front of her) about 60 cm from her. With the ophthalmoscope head up against your eye, shine the light in her right and left eyes by rotating the vertically held ophthalmoscope (Figure 1-2A). You will see two red-orange circles, the red reflexes (much like the reflections of a car's headlights in a cat's eyes at night); these are the patient's retinas.

Follow down onto the right red circle by moving closer to the patient (Figure 1-2C). If you lose the red reflex, move back and start over. Adjust the





B



C

Figure 1–2. **A.** Examining the right retina. Stand in front and to the right of the patient. The red reflex, which is the reflection off the retina, is easily seen although the examiner is 60 cm from the patient. The examiner's forehead will rest on his left thumb. **B.** The examiner's left hand and flexed thumb (arrow) provide a pivot for his forehead. **C.** The final position puts the examiner as close as possible to the patient.

lens wheel if necessary. This will bring any fine artery at the edge of the disc (the head of the optic nerve) into clear, sharp focus. Starting at 0, turn the lens selector wheel one or two clicks in either direction. If the definition of what you see is worse, turn the wheel in the opposite direction, all the time keeping the ophthalmoscope up to your eye.

You need to identify the following and know normal from abnormal:

- Arteries
- Veins
- Optic nerve head or disc
- Physiological cup
- Posterior pole, macula, and fovea

The following comments are helpful when examining each other. Before examining patients, read Chapter 3.

Retinal Arteries

Of the two kinds of vessels in the retina, the arteries are the *smaller*. Orange-red, they reflect the light of the ophthalmoscope so that the center of the artery shows a pale strip along its length. The thicker the wall of the artery, the wider is the shiny white strip down its center. Arteries do *not* pulsate and are somewhat angular. They cross and more commonly are crossed by retinal veins (Figure 1–3).

Retinal Veins

Retinal veins are larger than retinal arteries and are a dusky red. More sinuous and less angular than arteries, they pulsate. Retinal veins do not have as distinctive a clear white strip of reflected light down the center. To see the pulsations, look for a bend or change in direction of the vein. The pulsation is not only an expansion of the caliber of the vein, but a *shunting* of the column of blood up and down within the length of the vessel (Figure 1–4). Veins change direction at the disc or physiological cup edge. One can often see a filling and emptying of the vessel at the bend.

Retinal veins pulsate spontaneously in 80% of people. If you cannot see the pulsations, try the following:

- Watch the biggest vein you can see at the edge of the disc or where it disappears over the edge of the cup.
- Put your finger on the patient's eyelid and press gently against the eye. You will see the vessels empty as the intraocular pressure rises.
- If you let go suddenly, the venous pulsations often become visible.

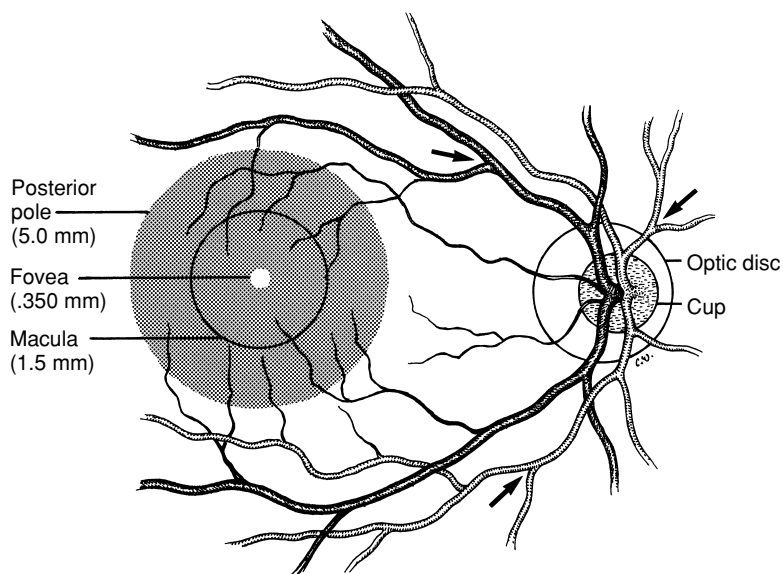


Figure 1–3. Details of the retina. The posterior pole and the macula are not this well-defined in the living retina. The branching vessels (arrows) indicate the direction to the disc.

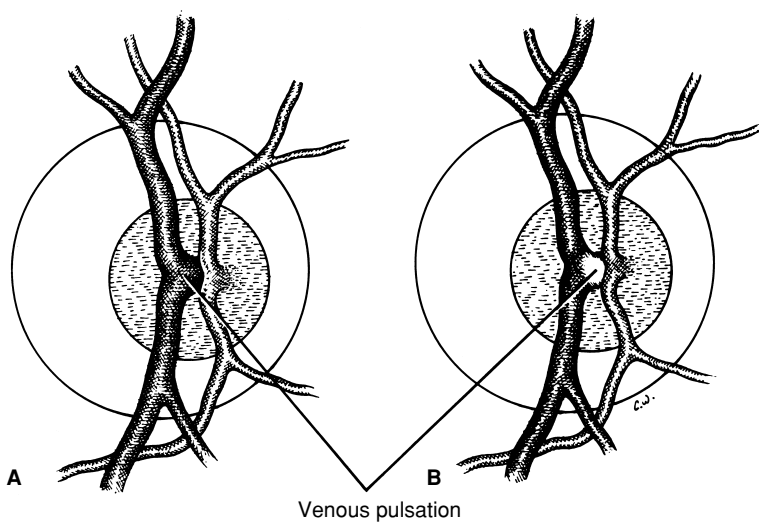


Figure 1–4. **A.** Retinal vein pulsation at the point where the vein disappears into the physiological cup. The vein is filled. **B.** As in A, but with the vein empty.

Alternatively, ask the patient to take in a big breath, hold it (which means closing off the glottis), and then bear down (the Valsalva maneuver). As the intracranial and intraocular venous pressure rises, you can see the retinal veins distend. While still looking at the retinal vessels and after 30 s, ask the patient to slowly breathe out. This should make retinal venous pulsations visible. The pulsation indicates that intracranial pressure is below 200 mm of water *at that moment*.

Follow the vessels as far as you can peripherally. You can help yourself as follows:

When examining her left eye, ask the patient to turn her eyes to her left while you move to her right; you are now looking at the temporal periphery of her left retina.

This is not easy. Instead of looking through the patient's round pupil, you are now looking through a slit pupil and an oblique lens that induces astigmatism. However, if you brace the ophthalmoscope against yourself and the patient and ask her to look left, right, up, and down while you look and move in the opposite directions—and persist at it—you will be able to see the peripheral retina.

The Head of the Optic Nerve or Disc

The disc is located at the nasal side of the center of the retina. The V formed by the bifurcations of veins and arteries is pointing toward the disc (small arrows in Figure 1–3). The disc is pink and white and is much paler than the orange-red retina. It is round or slightly oval, and the veins disappear into it while the arteries arise from it. About 1.5 mm in diameter, the disc usually has a distinct edge around it, but this edge may vanish and the disc will blend into the retina without distinction for a portion of the periphery. This blending into the retina is more evident on the nasal side. A crescent of black pigment around the edge of the disc is common in myopic (shortsighted) persons.

The Physiological Cup

Some part of the disc will appear to be deeper than the rest. This is the cup.

The cup is less than one third of the disc's diameter.

It is usually eccentric and is the point in the disc from which the arteries arise and the veins enter. There may be pearly white fibers, the lamina cribrosa, making a crosshatched appearance on the floor of the cup. It is im-

portant for you to learn the size of the physiological cup relative to the disc and the depth of the cup in those with normal vision (see the section on “Glaucoma” in Chapter 2).

The Posterior Pole

The central region of the retina is divided clinically and anatomically into three areas. The clinical and anatomical terminologies are not the same. In this manual *clinical* terminology is used. The anatomical equivalents are given in parentheses.

We have to consider the following:

- Posterior pole (macula or area centralis)
- Macula (fovea)
- Fovea (foveola)

The **posterior pole** is about 5 mm in diameter. Its outer limits cannot be clearly defined clinically. Histologically, it has more than one layer of ganglion cell nuclei.

The **macula** is at the center of the posterior pole and is a shallow depression about the same size as the disc. It is a darker red than the rest of the fundus, opposite the center of the pupil, and you will not see it well without dilating the pupil. Never give your opinion on its appearance without first dilating the pupil.

- You can see the macula by asking the patient to look into the ophthalmoscope light.
- Look at the retina. You will see several small groups of arteries coming off the temporal side of the disc that rapidly curve toward each other in a vertical direction. They end by surrounding the macula (Figure 1–3).

The **fovea** is the center of the macula. A depression seen as a yellow-white reflecting spot, it lies two disc diameters to the temporal edge of the disc, about 1 mm below its center.

Keep the following points in mind when using the ophthalmoscope:

- Examine the fundus of the eye with the patient sitting.
- Keep the patient’s back to the window, with the overhead lights out.
- Ask the patient not to hold his breath.
- Hold the ophthalmoscope vertically.
- It is easier for the patient if you decrease the intensity of the ophthalmoscope light when looking at the posterior pole.

- Keep your right eye opposite the patient's right eye, and keep both your eyes open.
- Do not breathe into the patient's face.

If the patient wears glasses, ask him to take them off. Look at them. If they are *plus* lenses, as you look through them and move them from side to side, objects move in the *opposite* direction. The reverse is true for the myope who wears minus lenses. For the myope you need a negative (red-numbered) lens in the ophthalmoscope to see retinal details clearly. Start with 0 showing at the lens strength window, and with your finger changing the lens wheel in the opposite direction of the arrow (counterclockwise) (Figure 1–1), you will soon see a clearly defined retina.

You do not need to do anything to allow for a patient with astigmatism, although many astigmatics are also myopic. For a hyperopic patient, reverse the procedure described for a myope. Start with 0 at the lens strength window, but turn the lens selector in the direction of the arrow (clockwise).

When you become proficient at using the ophthalmoscope, you may leave the patient's glasses on and look through them; this is especially helpful in **high myopia**. However, there are disadvantages: (a) both the inside and outside surfaces of his lens reflect incidental light and (b) his glasses prevent you from getting close to his eye and thus reduce the area of his retina that you can see.

Finally, do not spend too long on this part of the examination; you cannot see all there is to see in each patient until you have looked in several hundred eyes. Get to know each feature of the retina individually by looking in every eye that you can, irrespective of the patient's complaints.

VISION

The following terms should be familiar to you:

Amblyopia: Reduced visual acuity

Aphakia: Absence of the lens, such as following cataract removal or dislocation of the lens out of the pupil area

Astigmatism: Impairment of eyesight usually caused by unequal curvature of the cornea

Hypermetropia: Same as hyperopia

Hyperopia: Farsightedness, or focusing of light behind the retina

Myopia: Nearsightedness, or short sight; light focuses in front of the retina

Presbyopia: Impairment of eyesight because of old age

Vision is discussed here in two parts—**central**, or **macular**, **vision** and **peripheral vision**. Central vision can be measured as both near and distance vision.

Central Vision

Distance Vision

Always test and examine the right eye first.

Measure distance vision by using a test card of letters of different sizes. Distance vision is expressed as visual acuity (VA). This is a test of macular function. In the examining room use a Snellen card placed 6 m from the patient. There are also cards for use at 3 m from the patient (Figure 1–5A). The card should be well lit.

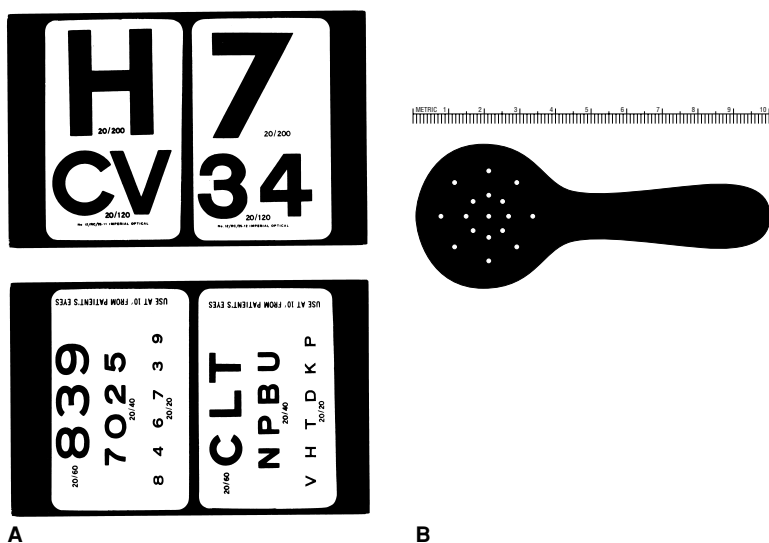


Figure 1–5. **A.** Plastic 6 × 9–cm cards for measuring visual acuity at 3 m. **B.** Plastic pinhole for verifying a refractive error.

Ask the patient to put on her glasses, cover one eye, and read the letters or numbers (or E chart for those who are illiterate) downward, that is, from the bigger to the smaller characters.

The smallest line of readable symbols is the patient's VA, which is expressed as a fraction. The numerator is the distance in feet or meters at which the test is made, and the denominator indicates the distance at which someone with normal vision would be able to read the line. If the patient can read the 20-ft (or 6-m) line when the card is 20 ft (or 6 m) away, her vision is 20/20 (or 6/6) and she has normal macular function. Less than normal ranges from 20/25 to 20/400.

If the patient can read all the letters except one in the 40-ft line, her vision is recorded as 20/40-1. Similarly, if she can read all the letters except two in the 60-ft line, her vision is 20/60-2. If she misses more than two letters in any line, her visual acuity is that of the next line up.

If the patient cannot see the largest letter, hold your hand about 1 m in front of the eye being tested and, with three or four fingers outstretched, ask her, "How many fingers can you see?" If she answers correctly, record this as CF (counting fingers) at 1 m.

Lesser vision than this should be tested and recorded as HM (hand movements) only at 1 m, and even lesser vision should be tested by directing a bright light into one eye from 0.3 m—recorded as LP (light perception) only. A finding of no light perception indicates a sightless eye.

There are Snellen test cards to be used at 6 m, or with the card on the wall above and behind the patient's head, while the patient faces a mirror at 3 m. Distance vision can be recorded from 6/60 to 6/6 (normal) and better than normal as 6/4.

For the patient who has forgotten his glasses, see the remarks on using a pinhole, under the heading "Near Vision."

Test visual acuity in each eye with the patient's other eye covered *and* with both eyes open. Patients with cataracts may have VA 20/60 right and left when the eyes are tested separately and 20/30 to 20/40 when tested with both eyes open. Also, patients with latent nystagmus may have normal acuity with both eyes open. When one eye is covered, the nystagmus occurs in both eyes and the acuity decreases in the uncovered eye.

Remember:

- Visual acuity is a test of macular function.
- Visual acuity is expressed as a fraction.
- The numerator is the distance at which the test is made.
- The denominator is the distance that a person with normal vision can read

the smallest line readable by the patient.

- Measure the best corrected vision of each eye separately, right then left, and both together.

Near Vision The patient with cataracts may have quite good distance vision and poor near vision, and the myope can read newsprint without his glasses but has poor distance vision. Near vision can be tested using the print in a telephone directory or a newspaper. Formal testing requires Jaeger's test type or Birmingham Optical Group test card or the American Medical Association test card. The patient holds the card in his hand at a comfortable distance and reads the smallest typed paragraph that he can. Glasses must be worn; test each eye separately, then together.

If the patient has forgotten his glasses, a pinhole will help (Figure 1-5B). If the patient holds the pinhole up to his eye and reads the test card through one of the holes, refractive errors are eliminated. The light coming through the pinhole is axial and remains unrefracted by the patient's cornea and lens and is sharply focused on the macula.

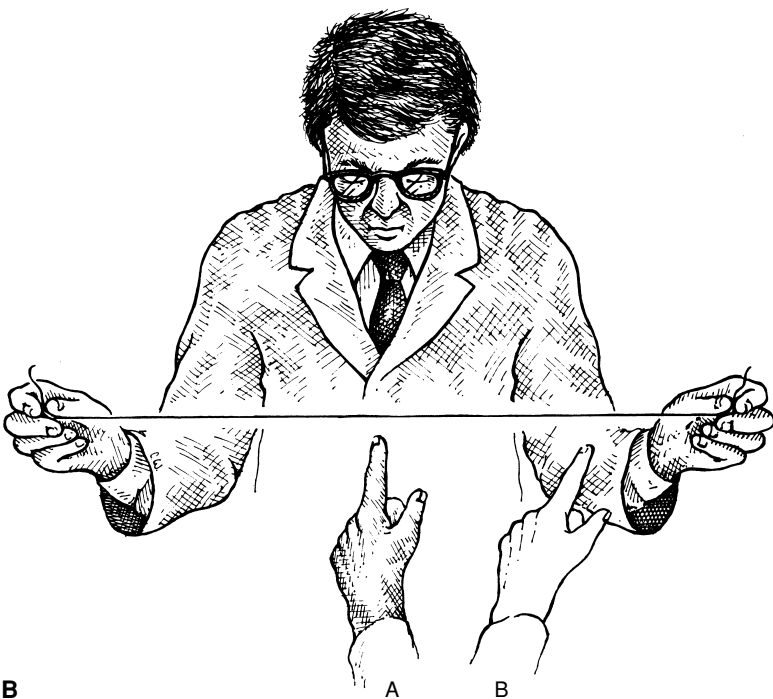
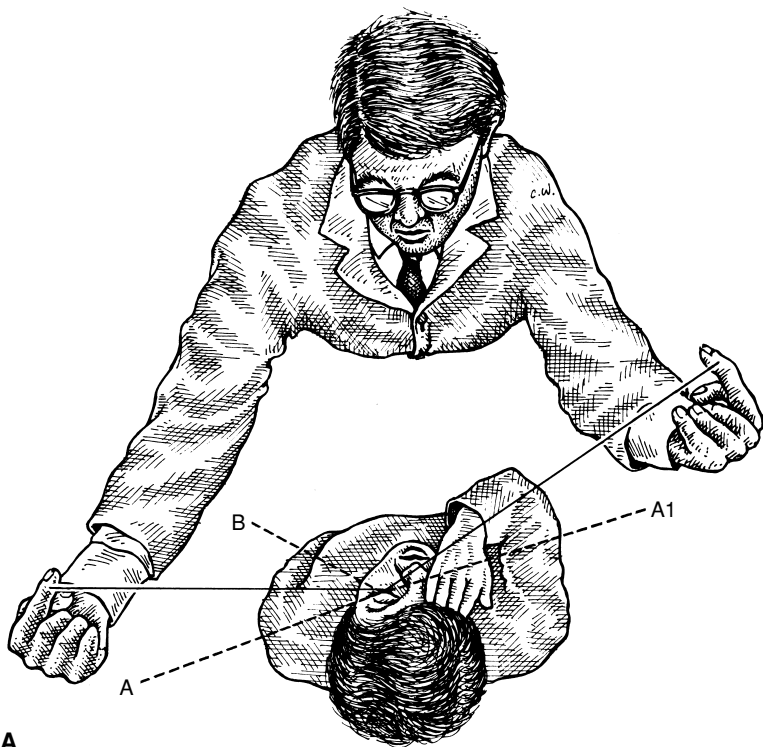
Whether the person is myopic, hyperopic, or presbyopic, testing his vision with the use of a pinhole will bring his vision up to normal. If it does not, his loss of vision is not a result of refractive error.

Figure 1-5A is a reproduction of two pocket cards used to test visual acuity. They are lettered and numbered and are used at 3 m. Similar cards are available at most surgical supply houses or medical college bookstores. Get a set and put them in your ophthalmoscope case with the pinhole.

Peripheral Vision

Peripheral fields of vision reflect the function of the retina (the nonmacular part) and the visual pathways connected to the nonmacular retina. When testing *acuity*, letters and parts of letters and numbers of different size that subtend a portion of an arc are used. When testing *peripheral fields*, the stimulus is movement.

To test visual fields by **confrontation**, seat the patient facing you with his glasses off. Ask him to cover his right eye with his hand and to stare at your right eye with his left eye. Hold your arms out to either side so that your fingers are at the *edges* of his visual fields, as in Figure 1-6A. If the examiner has his right hand at A in Figure 6A, he will get no responses from the patient, as the test object (his moving finger) is behind the patient's field and the patient cannot see it, let alone say whether or not it is moving. If the examiner's right hand is at B, then the test object is inside the field, not on the edge, and a substantial crescent of blindness could be present in the patient's left temporal field and yet be missed because the test is being done incor-



rectly. Keep the examining fingers on the *edges* of the fields. Start with your hands behind the patient's vision at A and A1. Bring your hands forward, one at a time, while wiggling your fingers until the patient can see them. You have thus discovered the edges of the fields and are ready to start testing.

It is important to have the patient continue staring at your right eye with his left. Ask him to tell you on which side he sees fingers moving, that is, right, left, or both. Some patients cannot or will not say "right" or "left." They sometimes mean their right and sometimes your right, and doctor and patient can both become confused. You may ask the patient to *point* to the side that moves and say "both" if both move. Alter the moving side randomly, sometimes right, sometimes left, and sometimes both. Test in both the upper and lower portions of the visual fields.

Do not test with your fingers on the equatorial line of the fields or on the midsagittal meridian.

With a test object as big as your moving finger, you will not know whether you are in the upper or lower half of the field. These halves are anatomically distinct. Test the upper halves well up in the field and the lower halves well down in the field. If movement in any area is being ignored, move the hand toward the midline until it is seen to be moving. Keep the movement small, that is, only a *portion* of one finger, and *slow*. Coarse, fast movements are much easier to see than fine, slow ones.

Always move both right and left fingers simultaneously at least twice in the upper portion of the fields and again in the lower portion.

Simultaneous right and left stimulation is valuable. A patient may be able to see all the movements along the left edge and all the movements along the right edge when the two sides are examined individually. However, when the two sides are stimulated *simultaneously*, the competitive effect of the two *simultaneous stimuli* may bring out a consistent visual field defect. This kind of field defect is not an absolute defect, but is known as an **inattention field defect**.



Figure 1–6. A. Testing the field of vision of the patient's left eye by confrontation. Points A and A1 are behind the edges of the field, point B is well inside the field, and the examiner's fingers are correctly placed on the *edges* of the field. **B.** Visual field assessment of the patient who cannot or will not fix. Ask him to quickly point to the center of the string. If his hand is at A, his lateral fields are approximately full; if at B, he has a left hemianopia (see text for using the string test).

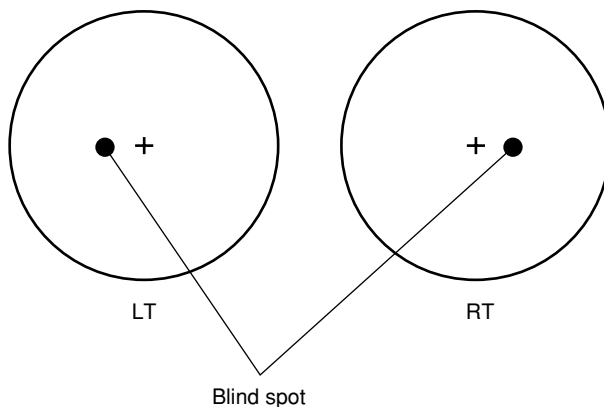


Figure 1–7. Record the patient's visual fields with his right eye field on your right. Mark his right field with *Rt.* and his left field with *Lt.* Indicate central vision with a plus sign. Add the visual acuity (corrected or uncorrected), date, and shade the blind areas.

Sometimes you cannot get the patient's cooperation, and field testing by confrontation will not work. Try the string test as shown in Figure 1–6B. Hold a piece of string with your hands about 24 in apart. Without a lot of instruction or warning, ask the patient to *quickly* point to the center of the string. If his fields are full (from side to side), he will point to the approximate center. If he has a field defect on his left, he will point to the right of center. This can be used to detect altitudinal defects as well if you hold the string vertically.

Finally, in uncooperative patients whose disease is clearly in the hemisphere(s), it is often impossible to test one eye alone. Such patients cannot or will not keep one eye closed. Therefore, you may have to examine the fields with both eyes open, and if the lesion is retrochiasmal, this is good enough.

Visual fields may also be tested by perimeter. This creates a permanent record of visual field loss.

A Bjerrum's screen is used for the formal examination of the central, as opposed to the peripheral, visual fields.

When making notes about visual fields, draw the patterns in the patient's history as though they were your own fields (Figure 1–7), that is, the right eye field on your right and the left eye field on your left. Include the visual acuity and date.

Although visual fields are not really round and they overlap on the nasal side, they are usually drawn this way. The plus sign in the center represents

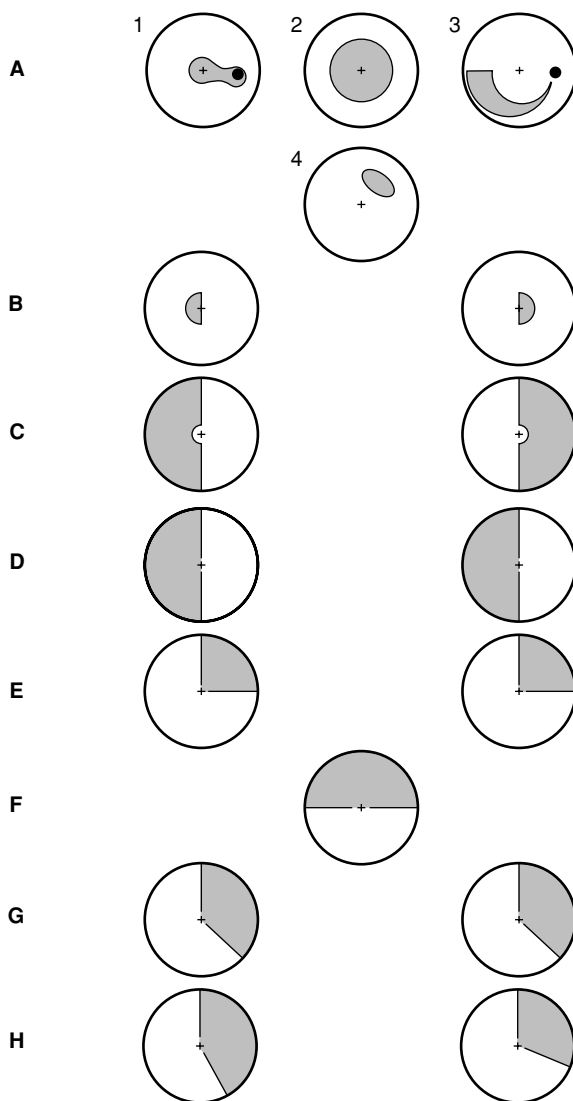


Figure 1-8. Various field defects. **A.** Scotomas: 1—cecocentral, 2—central, 3—arcuate, 4—paracentral. **B.** Bitemporal central scotomas. **C.** Bitemporal complete hemianopia with sparing of central vision. **D.** Left complete homonymous hemianopia with splitting of central vision. **E.** Right upper quadrantanopia. **F.** Altitudinal or horizontal field defect (upper). **G.** Incomplete congruous right homonymous hemianopia. **H.** Incomplete asymmetrical congruous right homonymous hemianopia.

central vision. The shaded oval area, lateral to the center and partially astride the equatorial line, is the normal blind spot. This is an absolute scotoma representing the optic nerve head, or disc, which has no retinal function.

You need to be familiar with the types of field defects shown in Figure 1–8 and their significance. (The blind areas are shaded.)

In testing visual fields by confrontation, remember:

- The patient must fix his gaze on your eye and keep this eye still.
- No one can detect color or definition in his peripheral fields of vision—the stimulus is movement.
- Keep your testing fingers on the *edge* of his fields.
- Test in both the upper and lower quadrants of each eye.
- Always make the right half fields compete against the left half fields by offering simultaneous right and left stimulation.

Loss of Vision

2

This chapter includes a list of definitions you need to know, a few anatomical hints about the visual system, and a listing of some of the kinds of visual problems you can expect to encounter.

Listed with each disease are some common or typical characteristics that will help you with recognition. Some of these diseases do not “belong” in the neurologist’s examining room, but you cannot preselect your patients. Although the neurologist’s opinion is often not the final word on a problem of visual loss, you must know how the visual system works and how diseases of this system present themselves.

DEFINITIONS

Visual acuity: Literally, visual “sharpness” tested by evaluating the recognition of patterns of letters or numbers, graded sizes, brightness discrimination, and color recognition

Macular vision: Same as central vision, the zone in the center of the **field of vision**. It extends as a circle from fixation about 15 degrees. Visual acuity is a measurement of the integrity of macular vision.

Peripheral field of vision: All of the area of space that can be seen, exclusive of central vision, when the eye is stationary. The extent of the peripheral field is (from fixation) about 60 degrees on the nasal side and upward, about 70 degrees inferiorly, and about 90 degrees on the temporal side. The nasal fields of the two eyes overlap.

Scotoma: An area of decreased or absent vision *surrounded* by an area of normal vision. A scotoma may be relative or absolute. A scotoma is *relative* if a small test object cannot be seen in it but a sufficiently large object can be seen. It is *absolute* if nothing can be seen in it.

Central scotoma: A scotoma that includes the fixation point; visual acuity is decreased. The bigger it is, the larger will be the letter on the Snellen test type that may be “hidden” in the

scotoma and the worse will be the visual acuity (type 2 in Figure 1–8A).

Cecocentral scotoma: A horizontal oval scotoma including fixation and extending to and including the blind spot (type 1 in Figure 1–8A)

Arcuate scotoma: As seen in type 3 in Figure 1–8A, corresponds to and represents nerve fiber bundle loss

Blind spot, or physiological scotoma: The area of blindness in the field of vision resulting from the head of the optic nerve. It is oval, 5 degrees wide and 8 degrees high, and on and mostly below the equator on the temporal side of fixation. Its nasal side is about 12 degrees from fixation. It cannot be seen with both eyes open because of the visual field overlap.

Find your own blind spot. Close your left eye. Stare at something at eye level straight ahead of you with your right eye (do not move your eye!). Hold a pencil in your right hand at arm's length. Move the pencil in from the temporal side, on the equator or just below it. As your pencil approaches fixation, you will find you can “lose” the eraser on the end of the pencil in your blind spot. You can slide the eraser around in the blind spot. This is how central field defects are plotted. If you cannot find it, you are not *fixing* your right eye on a distant object.

Hemianopia: Loss of half of the field of vision in both eyes or either eye

Homonymous hemianopia: Loss of half of the field of vision in each eye and on the *same* side of the midsagittal meridian in each eye; that is, a left homonymous hemianopia means the patient has lost vision in his temporal half field of the left eye and nasal half field of the right eye. A bitemporal or binasal field defect may be hemianopic but it is not homonymous. Loss of vision in the upper or lower field respecting the equator and ignoring the midline vertical meridian is called an altitudinal defect (upper or lower) and may be congruous or incongruous (discussed later in this chapter; see also Figures 1–8H and 2–5).

Bitemporal hemianopic central scotoma: The scotomas are in the temporal halves of macular vision. The nasal sides are intact and acuity is normal. The peripheral field is normal (Figure 1–8B). (a) Hemianopia with *splitting* of the macula—the vision in *half* the peripheral field and *half* the mac-

ula is gone, but visual acuity is normal (Figure 1–8D). (b) Hemianopia with *sparing* of the macula—the vision in half the peripheral field is gone, but all of macular or central vision is normal and, of course, acuity is normal (Figures 1–8C and 2–7). (c) Hemianopia with macular loss—hemianopia as above plus all of macular vision and *abnormal* visual acuity. This is not a hemianopia plus a central scotoma. By definition, a scotoma is an area of blindness surrounded by normal vision.

The visual cortex is all of Brodmann’s areas 17–19. Area 17 is the primary visual cortex receiving the optic radiation from the lateral geniculate body.

Area 17 is the striate cortex, area 18 is the parastriate cortex, and area 19 is the peristriate cortex.

The striate cortex is striated by a light strip of myelinated fibers parallel to the surface of the cortex and known as Gennari’s line. You can see it easily with the naked eye in a fresh brain.

Seven important points about the visual pathways are:

1. Optic inversion: Everything you can see from the right eye is divided in two ways. There is a vertical meridian dividing it into the right (or temporal) field and the left (or nasal) field. Everything in the *temporal* field is perceived by the *nasal* retina, and vice versa for the nasal field.

Also, there is an equatorial division. Everything below it is perceived by the upper retina, and vice versa for things above it.

In addition, the nerve fibers in the visual pathways arising from both temporal retinas do *not* cross the midline and they end in the ipsilateral occipital cortices. All the fibers arising from both nasal retinas *do* cross the midline and thus end in the contralateral occipital cortices.

On the medial surface of the occipital lobe you can see the calcarine sulcus, which is roughly horizontal and divides the occipital lobe into upper and lower portions.

All the nerve fibers arising from cells in the retinas above the horizontal division end up in the occipital cortex above the calcarine sulcus and below it for the fibers originating below the horizontal.

The retina is well organized, like a pie divided into four more or less equal pieces, plus the central plum for the posterior pole. In contrast, the visual *fields* are not equal. The temporal field is 60–70% bigger than the nasal field, yet each has about the same amount of retina. Each of the four retinal “pieces” is anatomically distinct with its own connections. The occipital cortex is clearly divided by the calcarine fissure in the horizontal plane. However, the arrangement of fibers in the visual pathways between the retina and

the occiput is not so methodical. For example, macular fibers in the optic nerve immediately behind the eye are in the upper lateral part of the nerve. Immediately in front of the chiasma the same fibers are in the center of the nerve. The chiasma, optic tract, lateral geniculate body (LGB), and optic radiation each have their unique fiber arrangements and proportions of fibers or cells (LGB) devoted to peripheral or central vision. [There is an excellent picture of this on page 916 of the 35th edition of *Gray's Anatomy* (Warwich and Williams, editors), Longman, 1973.]

As an example of proportion, the posterior pole of the retina is about 5 mm in diameter and the macula is about 1.5 mm. The whole retina, when laid out flat, is about 50–55 mm across; thus, over 90% of the retina is devoted to peripheral vision. By contrast, in the occipital cortex the amount of brain representing central vision is huge relative to the amount concerned with the peripheral fields. The occipital cortex representing central vision is the *most posterior* part of the occipital pole, and the peripheral retina is represented by the most anterior part of the occipital cortex.

In general, upper retinal fibers remain upper and lower ones remain lower except in the optic tract and the LGB, where a 90-degree rotation occurs. The rotation becomes undone in the optic radiation.

2. Lesions of the retina and optic nerve are characterized by unilateral, central, altitudinal, ring, and cecocentral scotomas; diminished visual acuity; altered color vision; and afferent pupillary defects. The opposite eye remains normal.
3. Chiasmal lesions are characterized by bitemporal defects. They may be in the peripheral field, the central field, or both (Figure 1–8B and C). Progressive lesions posterior to the chiasma will eventually cause homonymous field defects. The chiasma is about 1 cm above the pituitary gland. This large space can accommodate relatively large suprasellar tumors with no field defect. Although the chiasma is directly above the dorsum sella in most patients, it may be located more anteriorly or posteriorly. This variability will account for atypical field defects. There are unique fiber arrangements in the chiasma.
 - a. **Ventral nasal optic nerve fibers** (serving the superior temporal field) cross in the anterior chiasma and loop into the distal opposite optic nerve and then turn posteriorly to the contralateral optic tract (see Figure 2–1).
 - b. **Macular fibers** make up a large part of the chiasma and are located largely inferiorly.
 - c. **Nasal macular fibers** decussate in the posterior chiasma. (Figures 2–1 to 2–4 are applicable to chiasmal lesions.)

4. Optic tract lesions are characterized by their incongruity. They usually begin as a quadrantic defect with early sparing of macular vision (Figure 2–5).
5. Optic radiation:
 - a. In *temporal* lobe lesions, field defects are principally superior and quadrantic; if hemianopic, they are denser and earlier in the *upper* quadrant.
 - b. In *parietal* lobe lesions, field defects may be inferior and quadrantic but, more commonly, hemianopic. When early and progressive, they will be denser and first evident in the *lower* quadrants (Figure 2–7).
6. Occipital lobe: Monocular temporal crescent, field, or scotomatous defect, in *anterior* occipital lobe lesions (Figures 2–9 and 2–10).
 - a. Central, congruous scotomas, in lesions of the *tip* of the occipital pole (Figure 2–8).
 - b. Hemianopic with macular sparing; bilateral lesions will leave the patient with tubular vision, that is, all blind but for 10 degrees around fixation, or all central and peripheral loss.
7. The more posterior the lesion, the more likely are the areas of field defect in the two eyes to be congruous and macular vision spared. Macular fibers in tract, LGB, and anterior optic radiation are confined to a relatively narrow area. Posteriorly, they form a large part of the radiation and cortex.
8. Visual field defects are congruous when they are the same *shape* in each eye and incongruous when they are not. Unless they are hemianopic, they are seldom the same *size*. Optic radiation lesions are commonly congruous, and tract lesions are usually incongruous.

DIAGNOSIS

The location of the lesion is often obvious in visual system disease because of the *pattern* of the area of visual loss; for example, when visual acuity in the right eye is reduced to 20/200 with a central scotoma but the left eye is normal, the lesion must be between the right cornea and the optic chiasma. When the visual acuity is normal and there is a right upper homonymous quadrantanopia (Figure 1–8E), the lesion must be in the left temporal lobe involving the optic radiations.

As a generalization, the findings of central (type 2 in Figure 1–8A), ring, arcuate (type 3 in Figure 1–8A), or cecocentral (type 1 in Figure 1–8A) scotomas, visual loss in one eye only, abnormal color vision, and afferent pupil defect (see Chapter 5), or an altitudinal field loss (Figure 1–8F) are indicative of disease in the retina or optic nerve.

Normal visual acuity with field defects on the same side of the vertical meridian (ie, homonymous) are caused by lesions in the optic tract, LGB, optic radiation, or occipital cortex. The more posterior the lesion, the more likely the field defect in one eye will be congruous with the defect in the other eye (Figure 1–8G). If the field defect is a *complete* homonymous hemianopia, you cannot tell where the lesion is, except that it must be behind the chiasma.

More diagnoses are missed because of a failure to appreciate the arrangement of the fibers in the optic chiasma than any other part of the visual system.

Histories vary from helpful to misleading. The presbyopic patient complains he cannot get far enough from his newspaper to read it. (The ability to read the fine print in the average newspaper requires 20/30 vision.) The patient with pigmentary retinal degeneration will often forget to mention the progressing night blindness, an extremely important symptom. Another patient with an *inattention* left hemianopia has no complaints and is being examined only at the insistence of, for example, his wife or the motor vehicle licensing authorities. Typically, in the past months he has been driving with the left side of his car over the dividing line into oncoming traffic.

The patient who says he covered one eye with his hand and realized he was virtually blind in the other eye has told you nothing about the tempo of the disease, but has revealed that the lesion is between his cornea and the chiasma.

Also, some people are born with one eye myopic and the other eye normal and they discover the fact incidentally. A man of 40 who wears no glasses and has no visual complaints gets a foreign body in his eye (not in the line of vision). When the foreign body is successfully removed, he discovers that the vision in the previously injured eye is not as good as in the other eye. In his mind the injury and the diminished visual acuity are cause and effect. Examination shows reduced visual acuity but an otherwise normal eye. The pinhole improves his visual acuity to the 20/20 line. Refraction by an ophthalmologist confirms that his best corrected vision is normal. This eye has always been myopic and the trauma has simply brought it to his attention.

When confronted with an apparent recent loss of vision in one eye that makes no sense, *always* get a full refraction before conducting more elaborate investigations.

SOME DISEASES OF THE VISUAL SYSTEM

Retina and Optic Nerve Lesions

Retrobulbar Neuritis Retrobulbar neuritis typically presents in young adults, with onset over 24–48 hr, a large central scotoma, and a painful eye

on palpation and movement. There may be a past history of episodes of multiple sclerosis or this illness may be the first manifestation of it. The fundus is usually normal. Afferent pupillary defect is commonly present.

Optic Neuritis The presentation of optic neuritis is like that of retrobulbar neuritis, except that it may be painless. The terms *retrobulbar neuritis* and *optic neuritis* are used interchangeably. Edema of the optic nerve head may be present. (See the section on papillitis in Chapter 3.)

Central Serous Retinitis Central serous retinitis has subacute onset (days) of slight to moderate vision loss in one eye, usually in a male patient aged 20–40. The disease is caused by fluid exudate lifting the retina, usually at the macula. The patient complains that objects look smaller with the affected eye.

Giant Cell Arteritis of the Central Retinal Artery This disease occurs in those age 60 or older, with a sudden onset of central blindness. Superficial temporal arteries are typically tender, pulseless, and tortuous. There is almost always an elevated erythrocyte sedimentation rate and a low-grade fever. The history will often contain complaints of headache and stiff, aching, weak shoulder and hip muscles. Funduscopy shows total retinal ischemia (see the following section). Diagnosis, including temporal artery biopsy, is an emergency.

Anterior Ischemic Optic Neuropathy In patients over age 50, anterior ischemic optic neuropathy is a manifestation of giant cell arteritis, vasculitis, diabetes mellitus, or Takayasu's disease or is frequently idiopathic. There is a sudden onset of visual loss, sometimes followed by increasing and progressive visual failure over 5–7 days. Altitudinal (inferior) field defects are common. Funduscopy examination shows a swollen disc, hemorrhages near the disc edge, and cotton-wool spots. A macular star is common with vasculopathies. The other eye is commonly involved within weeks or months of the first eye.

Retinitis Pigmentosa The most common symptom of retinitis pigmentosa (RP) is night blindness. The onset and severity of symptoms depend on the pattern of inheritance: the autosomal recessive type presents earliest and is the most severe, X-linked is intermediate, and autosomal dominant is frequently mild. The fundus shows black “bone spicules” of pigment clustered around vessels in the midperiphery. The disc is waxy pale, and there is attenuation of both retinal vessels. Visual fields show ring-shaped arcuate or annular scotomas.

Pseudoretinitis Pigmentosa Pseudoretinitis pigmentosa refers to the fundus findings of a number of disorders that mimic RP. Some pigmentary retinopathies that are *not* RP are those following congenital and acquired syphilis, childhood exanthemas, phenothiazine usage, and trauma.

Atypical RP includes sectoral RP (fundus changes in one sector of the fundus bilaterally, which can mimic chiasmal lesions) and pericentral RP (fundus changes central). Unilateral RP probably does not exist.

Diseases and syndromes associated with RP are Bassen-Kornzweig, Bardet-Biedl, Kearns-Sayre, and Usher's.

Glaucoma Glaucoma exists when elevation of **intraocular pressure** is sufficient to damage optic nerve fibers at the optic disc level. This is the second leading cause of blindness in North America. There are two types: acute angle-closure glaucoma (ACG) and chronic open-angle glaucoma (OAG).

ACG presents with severe pain in the eye and head, blurred vision, and colored halos around lights, plus nausea and vomiting. Pain results from a rapid rise in intraocular pressure. Attacks are precipitated by reduced illumination and may be relieved by sleep, bright light, or miotic agents, all of which constrict the pupil. ACG usually presents unilaterally. The predisposing factors are often bilateral, and the uninvolved eye is therefore at risk. **This is an emergency.**

OAG is the most common form of glaucoma and is **dangerous** because the onset is often gradual and asymptomatic. Central vision is preserved until late, and the field loss is often unnoticed by the patient until the disease is well advanced. It is usually a bilateral disease, although asymmetrical.

Visual field defects in glaucoma respect the *horizontal* division of the visual fields. The arcuate nerve fibers arching above and below the macula from the temporal region are most susceptible to glaucomatous damage. Therefore, the characteristic field defects are paracentral and arcuate nasal scotomas (type 3 in Figure 1–8A). The central area of the visual field is the most resistant, so visual acuity may be normal even in advanced glaucoma.

Optic disc changes are an increase in the *diameter* and *depth* of the physiological cup and increased pallor.

Central Retinal Vein Occlusion Central retinal vein occlusion (CRVO) presents with painless, always unilateral, loss of vision. The patient is commonly a young adult. The extent of visual loss is variable, depending on the degree of venous occlusion, the amount of macular edema, and the presence or absence of complications such as retinal neovascularization and neovascular glaucoma.

The fundus shows dilated, tortuous veins; retinal hemorrhages usually in the peripheral retina; and retinal edema. Cotton-wool exudates are usually

seen only around the disc. The disc edge is indistinct, although the physiological cup remains visible.

Visual fields reveal a relative central scotoma and acuity is usually 20/100, improving to 20/60 with time if there are no complications.

Branch retinal vein occlusion is most common in the superotemporal branch. Fundus changes are confined to the distribution of the branch, and field loss is segmental.

Hypertension often coexists with CRVO, and associated retinal artery disease is a common finding. A picture similar to that of CRVO may be seen in hyperviscosity syndromes, leukemia, and myeloma.

Retinal Artery Occlusion Retinal artery occlusion (RAO) is accompanied by sudden, painless, unilateral loss of vision. It is commonly discovered by the patient on awakening in the morning and occurs in the stroke-prone age group. If the central retinal artery is involved, vision will be completely lost (no light perception). If a cilioretinal artery is present, an island of central vision will be preserved. Visual loss may be confined to a segment of the field if only a branch of the retinal artery is occluded.

The fundus reveals a gray, opaque, edematous retina with a cherry-red fovea. This is normal choroid contrasted against gray retina. Splinter hemorrhages are rarely seen. Branch arteries may show segmented columns of blood (boxcars). There may be a history of transient monocular visual loss lasting minutes, with altitudinal progression as vision fails, that is, the window blind effect.

Causes of RAO include emboli, thrombosis, giant cell arteritis, and collagen-vascular diseases.

Retinal Detachment Retinal detachment (RD) presents with a history of recurrent flashes or floaters in the same area of the visual field. The patient typically complains of a cloud or curtain obscuring part of the field. It usually starts and is most dense peripherally and extends toward central vision. Visual acuity is normal unless or until the macula detaches. An inferior detachment with a superior field defect can be present for a long time before the patient is aware of it. The fundus shows an elevated retina that is gray and wrinkled and undulates.

The elevation will be appreciated as you rack in progressively more plus lenses (black numbers) on the ophthalmoscope to keep the elevated retina in focus.

The most common predisposing factor to RD is degenerative retinal change (as seen in high myopia). Other causes include trauma; a simple "black eye" history may be significant. Malignant melanoma of the choroid can cause a secondary RD, and the retinal neovascularization of diabetes mellitus may result in a tractional RD.

Macular Degeneration Macular degeneration begins at any age but is most common over 60 years. Slow loss of visual acuity occurs bilaterally and is worse in bright light and better in the dark; recovery of vision is slow after exposure to a bright flash of light. The macula may appear normal initially. There are central and paracentral scotomas. Fluorescein retinal angiography and the Amsler grid are helpful in diagnosis.

Optic Neuropathy Optic neuropathy can occur:

1. Without other diseases (eg, Leber's). The typical patient is a young adult male with loss of central vision in one eye, followed by the other eye in days or weeks, with a positive family history. Characteristic fundus changes in the acute stage are followed by optic atrophy. A central scotoma will become cecocentral with upper nasal breakout.
2. With other central nervous system (CNS) diseases, with one or more of the following: congenital deafness, ataxia, spastic quadriplegia, mental deterioration, polyneuropathy, Friedreich's ataxia, Marie's cerebellar ataxia, and Charcot-Marie-Tooth disease
3. With inborn lysosomal disorders: (a) mucopolysaccharidoses or (b) lipidoses

Optic Atrophy Optic atrophy occurs as a consequence of the foregoing and other diseases, such as methyl alcohol or chloroquine ingestion, isoniazid toxicity, and any compressive, ischemic, or toxic disorder of the retinal ganglion cell or fiber from the retina to the LGB. This includes papilledema, which, if severe enough or chronic enough, can result in optic atrophy. Diagnosis of the cause is often impossible. Clinical diagnosis of atrophy is dependent on the color and structure of the disc.

The lesion responsible for optic atrophy may be anywhere from the retina to the LGB inclusive.

Chiasmal Lesions The most common visual field defect is bitemporal, either central or peripheral or both.

Be careful and persistent with patients with visual loss. Lesions in the chiasmal region can be deceptive and extremely chronic. The chronicity and slow progression seem to make the symptoms more acceptable and less demanding. Every neurologist and neurosurgeon has had some bad experience with patients thought to have multiple sclerosis, amblyopia from childhood, low-tension glaucoma, retinitis pigmentosa sine pigmento, or atypical macular degeneration as an explanation for their blindness who eventually turn out to have a chiasmal lesion as the true cause. It does not help the patient to

make the diagnosis after the optic atrophy is marked and the acuity is down to 20/200.

Another reminder about chiasmal lesions is the place of exploratory intracranial surgery. Ordinarily, there is no such operation as an exploratory intracranial procedure. However, if an eye and field examination point to a chiasmal lesion, even if the skull x-ray, carotid angiography, computerized tomography (CT) scan, and nuclear magnetic resonance (NMR) studies are all normal, then the next “investigative” step is **exploration of the chiasmal area**. The neurosurgeon may make the diagnosis of chiasmal arachnoiditis that cannot be treated, but he may find the otherwise undiagnosable 4-mm meningioma, pituitary tumor, or craniopharyngioma.

Finally, lesions of the visual pathways behind the optic chiasma never interfere with color vision (ie, without an accompanying loss of light perception). There are reported examples of defective color appreciation from a parietal cortical lesion, but this is color agnosia, not loss of color vision.

Anterior Chiasmal Syndromes

1. The ipsilateral eye is blind and there is an upper temporal field defect in the contralateral eye. The lesion is shown at **a** in Figure 2-1A.

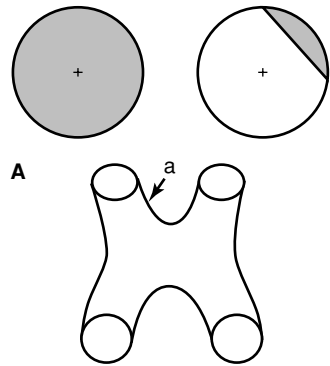


Figure 2-1A

2. The ipsilateral eye is blind and there is a central temporal scotoma in the contralateral eye (The same lesion as in Figure 2-1A).

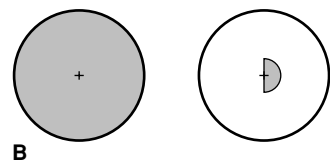


Figure 2-1B

3. The ipsilateral eye is normal and the contralateral eye is as in Figure 2-1A or B (The same lesion as in Figure 2-1A).

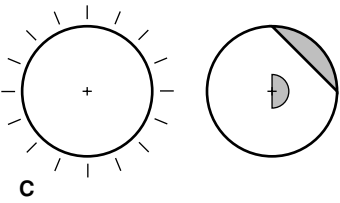


Figure 2-1C

4. The ipsilateral eye has a central scotoma and there is a paracentral scotoma in the temporal field of the contralateral eye (The same lesion as in Figure 2-1A). This can progress to the condition shown in Figure 2-1E.

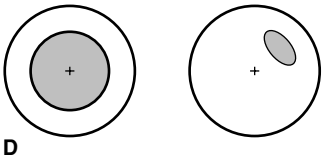


Figure 2-1D

5. The ipsilateral eye is blind and there is a paracentral scotoma in the temporal field and full peripheral field of the contralateral eye (The same lesion as in Figure 2-1A).

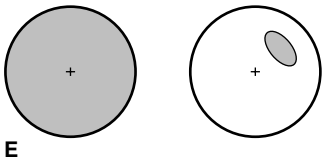
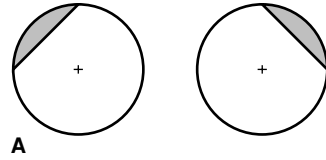


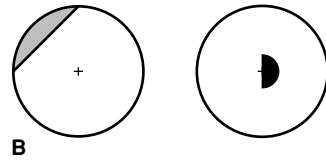
Figure 2-1E

Chiasmal Body Bitemporal peripheral field loss occurs as in Figure 2-2A or central temporal scotomas occur as in Figure 2-2C, or a combination of the two as in Figure 2-2B. Lesions occur as in Figure 2-2D. The lesion is usually below the chiasma (eg, a pituitary tumor).



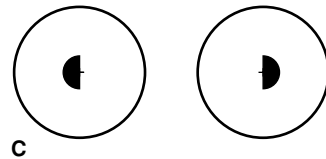
A

Figure 2-2A



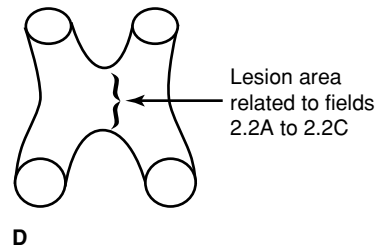
B

Figure 2-2B



C

Figure 2-2C



D

Figure 2-2D

Suprasellar lesions (eg, craniopharyngioma, aneurysm, meningioma, chordoma, or third ventricle distention) can start with a central or peripheral temporal defect that initially may be most marked inferiorly as in Figure 2-2E.

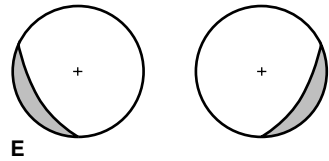


Figure 2-2E

Any of these lesions (suprasellar or infrasellar) may eventually produce the field defect shown in Figure 2-2F.

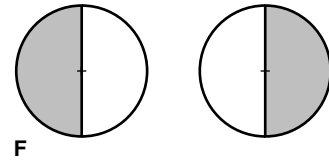


Figure 2-2F

Posterior Chiasma Posterior chiasma presents usually with a bitemporal central scotoma with peripheral defects as well, as in Figure 2-3A. A central scotoma usually occurs first. The lesion is shown at **a** in Figure 2-3B. When big enough to involve the tract at **b**, a homonymous, hemianopic defect is added.

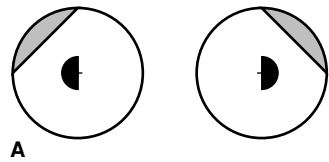


Figure 2-3A

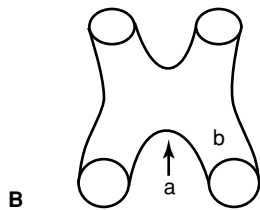


Figure 2-3B

Lateral Chiasma Lateral chiasmal lesions present with nasal field defects. Very rare, they can be unilateral as in Figure 2-4A or bilateral as in Figure 2-4B. The defect rarely comes up to the midline. Unilateral nasal defects have been reported as a result of infarction of the optic nerve, aneurysm, pituitary tumor, and ectatic carotid artery. Bilateral nasal defects have been reported resulting from chiasmal arachnoiditis and secondary to obstructive hydrocephalus and pressure from above from the third ventricle. **Glaucoma** is probably the most common cause of nasal field defects.

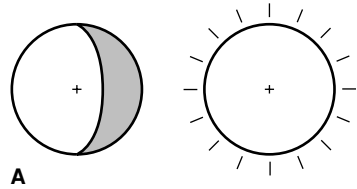


Figure 2-4A

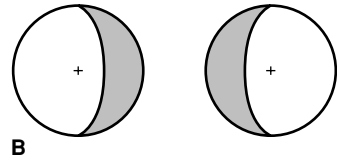


Figure 2-4B

Optic Tract Lesions

Optic tract lesions between the chiasma and the LGB produce incongruous field defects as in Figure 2-5.

Most lesions behind the chiasma, whether tract, LGB, radiation, or cortex, present as homonymous field defects with normal visual acuity. The lesions cause loss of vision in some or all of the two homonymous half fields. When the hemianopia is *complete*, the site of the lesions cannot be located by visual field examination alone.

Blind areas resulting from tract lesions are homonymous and extremely incongruous in shape and size.

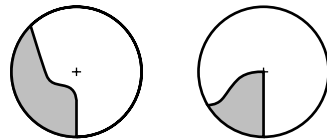


Figure 2-5

1. When a tract lesion produces a complete hemianopia, the eye contralateral to the lesions will show an afferent pupil defect (see Chapter 5). This may be difficult to elicit.
2. When a tract lesion presents with some degree of hemianopic visual loss *plus* decreased visual acuity and color vision, the lesion has involved the chiasma or the optic nerve(s). This area is compact, and distances are small.
3. Some lesions originate in or near the sella and then expand laterally to involve the tract. The patient may present with the confusing combination of a chiasmal (bitemporal) defect plus an optic tract (incongruous hemianopic) defect.
4. Many patients with lesions *anterior* to the LGB (ie, tract, chiasmal, and nerve) are aware of a dimness or loss of vision. Conversely, most patients with lesions *posterior* to the LGB are unaware of their visual defect.

Optic Radiation

Within the Temporal Lobe A homonymous defect, superior and quadrant, is shown in Figure 2–6.

Defects can be congruous or incongruous. When incongruous, the larger defect is usually in the eye on the *same* side as the lesion. It is also denser in this eye. The blind area may cross the horizontal division of the visual fields.

When the defect is incongruous, it is never as incongruous as with optic tract lesions.

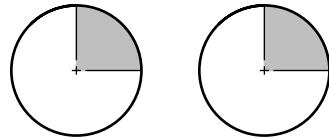


Figure 2–6

Within the Parietal Lobe A hemianopic homonymous defect may initially be *only*, or denser, in the lower fields, as in Figure 2–7.

This is congruous with sparing of the central vision and normal acuity.

Peripheral defects result from medially placed lesions.

There is an abnormal optokinetic response (see Chapter 6).

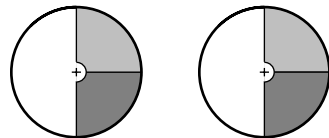


Figure 2–7

Occipital Lobe and Visual Cortex Lesions

The more posterior the homonymous defect, the more likely it is to be completely congruous.

Tip of occipital pole lesions can produce central homonymous scotomas with precise congruity. Figure 2-8 shows a field defect recorded by Sir Gordon Holmes. The tip of the patient's right occipital lobe was injured with the resulting left incomplete homonymous hemianopic central scotomas.

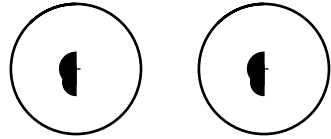


Figure 2-8

Anterior Visual Cortex or Posterior Optic Radiation

There are *unpaired* nasal fibers that correspond to the extreme temporal peripheral field and the most anterior aspect of the area striata. In patients with posterior optic radiation lesions, there may be a unilateral **temporal crescent defect** as in Figure 2-9. It may be above or below the horizontal division and often is the forerunner of a hemianopic defect. The defect may be a temporal crescent scotoma *not* touching the edge of the field (not shown). Thus, a postchiasmal lesion may produce a purely monocular field abnormality.

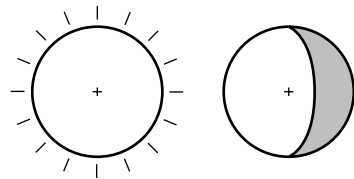


Figure 2-9

The *sparing* of a thin temporal crescent of vision when the rest of the temporal field is hemianopic also occurs as in Figure 2-10.

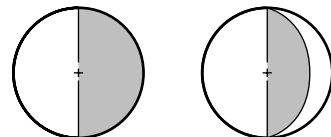


Figure 2-10

Cortical Blindness

The term *cortical blindness* is used synonymously with *bilateral homonymous hemianopia*. The definition includes

- Loss of all vision, including perception of light
- Loss of reflex blinking on being threatened
- Normal pupil response to light and convergence
- Full eye movements

It is common for patients with this disorder not to complain of the vision loss or to deny the blindness when confronted with the fact (Anton's syndrome). Remember that the condition commonly occurs from occlusion of the upper end of the basilar and the two posterior cerebral arteries. As the latter supply the inferior and medial surface of the temporal lobes as well as the primary visual cortex, an acute confusional amnesic syndrome may accompany the acute loss of vision. The latter may account for the denial of the blindness; that is, it is an agnosia.



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