Visual Field Pupil Exam

7

Pupillary Examination

The pupil is the window to the inner eye, through which light passes to reach retinal photoreceptors. Because of its potential to reveal serious neurologic or other disease, examination of the pupil is an important element of a thorough ophthalmic evaluation, requiring meticulous attention to detail. Pathologic disorders can alter the size, shape, and location of the pupil, as well as the way the pupil reacts to light and near-focus stimulation. The patient who has a pupillary abnormality generally comes to the attention of the ophthalmologist as a result of either anisocoria (difference in size between the two pupils) or reduced pupillary light reaction.

This chapter provides a brief, basic background about pupillary pathway anatomy and supplies instructions for the principal tests to evaluate pupillary responses. It also gives a brief overview of the pupillary abnormalities that are most commonly encountered through pupillary evaluation and testing.

Anatomy of Pupillary Pathways

Disorders of the pupil generally arise from dysfunction of the afferent or efferent pupillary pathways. The afferent pathway is composed of optic nerve axons emanating from the globe, passing through the chiasm, and exiting the optic tract before the lateral geniculate body to synapse in the dorsal midbrain. The efferent pathway includes the parasympathetic and the sympathetic input to the iris muscles. In addition, lesions affecting the dorsal midbrain often cause light-near dissociation (see below).

The size of the pupil is controlled by the opposed actions of the sympathetic and parasympathetic nervous systems that control the tone of two smooth muscles, the pupillary sphincter muscle and the pupillary dilator muscle. The pupillary sphincter muscle is supplied by cholinergic fibers of the parasympathetic system through the third cranial (oculomotor) nerve; the pupillary dilator muscle is supplied by adrenergic fibers of the sympathetic system. The pupils tend to be smaller in infants and larger in children and young adults, becoming smaller again with advancing age.

Parasympathetic Pathway (Light-Reflex Pathway)

The pupillary parasympathetic pathway subserves the pupillary light reflex (Figure 7.1). The afferent arc begins in the retina and ends in the midbrain tectum. When light stimulates the retinal photoreceptors (rods and cones), impulses are transmitted through retinal ganglion cell axons, which include pupillomotor fibers. The pupillary fibers pass through the optic nerve to the chiasm, where hemidecussation occurs, and then to the optic tract. They bypass the lateral geniculate body to enter the midbrain. The first synapse occurs at the pretectal nuclei, near the superior colliculus. The fibers then project to synapse at both the ipsilateral and contralateral Edinger-Westphal nuclei, the parasympathetic motor center of the oculomotor nerve. Contralateral projections pass through the posterior commissure and the periaqueductal gray matter.

The efferent fibers from the Edinger-Westphal nuclei then travel superficially in the oculomotor nerve as it leaves the brain stem and enter the orbit within the inferior division of the oculomotor nerve to synapse at the ciliary ganglion. Postganglionic fibers then travel within the short posterior ciliary nerves, passing through the suprachoroidal space

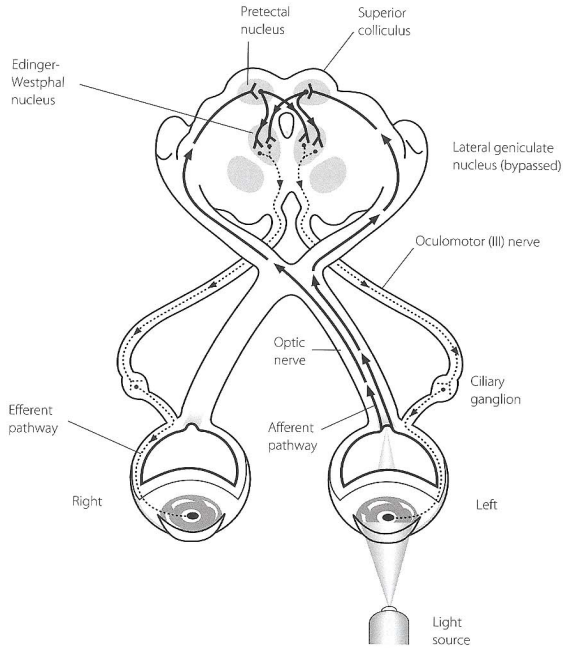


Figure 7.1 Light reflex pathway. In this schematic cross-section, the solid line represents the afferent pathway and the dotted line represents the efferent pathway. A light stimulating the left retina will generate impulses that travel up the left optic nerve and divide at the chiasm. Some impulses continue up the left tract; some cross and travel up the right tract. The impulses arrive at each pretectal nucleus and stimulate cells, which in turn send impulses down the third cranial nerve to each iris sphincter, causing each pupil to constrict. Because of the double decussation, the first in the chiasm and the second between the pretectal nuclei and the Edinger-Westphal nuclei, the direct pupil response in the left eye equals the consensual response in the right eye.

to innervate the pupillary sphincter muscle and the ciliary muscle. This complex anatomic pattern, with partial crossings at the chiasm and in the posterior commissure, results in the symmetry of the direct and consensual pupillary responses.

Near-Reflex Pathway

The near-reflex pathway subserves pupillary constriction when fixating a target at near. It is less well defined than the light-reflex pathway. The final common pathway is mediated through the oculomotor nerve with a synapse in the ciliary ganglion. Its central fibers are located more ventrally in the midbrain than those of the light-reflex pathway; that is, ventral to the Edinger-Westphal nuclei. Unlike the light-reflex pathway, which is entirely subcortical, the near-reflex pathway sends fibers to the cerebral cortex bilaterally.

Sympathetic Pathway

Pain, fear, and certain other psychic stimuli lead to pupillary dilation through the sympathetic innervation of the pupillary dilator muscle. The oculosympathetic pathway consists of a three-neuron arc (Figure 7.2). The first-order neurons of the sympathetic pathway originate in the posterior hypothalamus, descend to the intermediolateral gray column of the spinal cord, and synapse at the ciliospinal center of Budge at spinal levels C8 to T2. Preganglionic second-order neurons arise from the intermediolateral column, leave the spinal cord by the ventral spinal roots, and enter the rami communicans. They join the paravertebral cervical sympathetic chain and ascend through this chain to synapse at the superior cervical ganglion. Postganglionic third-order neurons originate in the superior cervical ganglion, entering the cranium with the internal carotid artery. The fibers join the ophthalmic division of the fifth cranial (trigeminal) nerve within the cavernous sinus, reaching the ciliary muscle and pupillary dilator muscle by means of the nasociliary nerve and the long posterior ciliary nerves. Some sympathetic fibers might transiently join the sixth cranial (abducens) nerve within the cavernous sinus.

Examination of the Pupils

A pupillary examination begins with a general observation of the pupils. The pupillary reflexes are tested via the light-reflex test and the swinging flashlight test (to evaluate the direct and consensual pupillary reflexes) and the near-reflex test (to evaluate the near vision response).

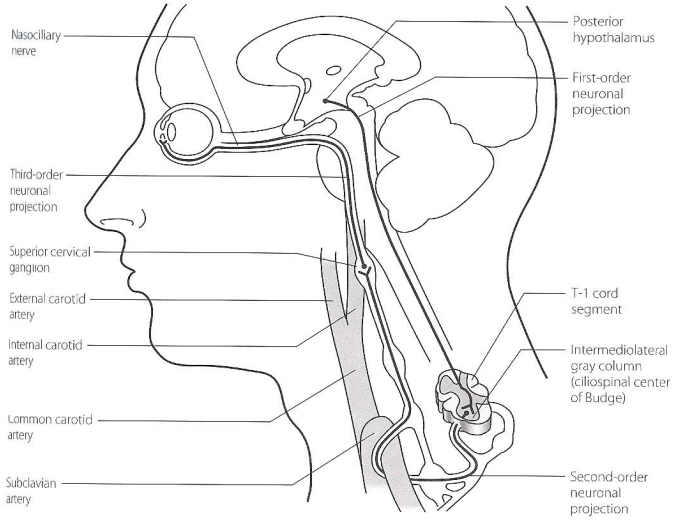


Figure 7.2 Oculosympathetic pathway.

General Pupillary Observation

In a room with standardized ambient light, begin a general observation of the pupils by noting the shape of each pupil and the color of the irides. Have the patient fixate a distance target across the room to minimize accommodation and the accompanying miosis. Sit on one side of the seated patient and diffusely illuminate both pupils from below the nose with a handheld light (eg, muscle light), using the least amount of light necessary to discern pupil size (Figure 7.3). Carefully measure the pupil diameter in both eyes with a millimeter ruler, the pupil gauge usually printed on the near vision chart, or an Iowa pupil gauge. Record the size in millimeters in the patient's chart. Refer to "Size (mm)" in Figure 7.4 for an example.

If the patient has anisocoria, the pupillary diameters should be measured under both dim and bright illumination. A subtle degree of anisocoria (usually less than 1 mm) is normally present in many individuals, and is called *essential*, or *physiologic*, *anisocoria*. In physiologic

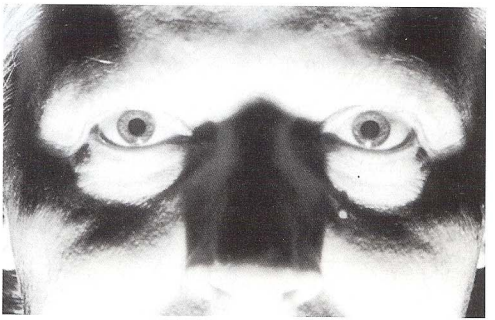


Figure 7.3 Pupils can be seen in dim light by shining a handheld light on the patient's face from below while the patient looks into the darkness. (Reprinted with permission from Thompson HS, Kardon RH. Clinical importance of pupillary inequality. *Focal Points: Clinical Modules for Ophthalmologists,* Vol. 10, Module 10. San Francisco: American Academy of Ophthalmology; 1992.)

	Size (mm)	Briskness of Light Reaction	RAPD	Near Reaction
Pupil OD	4.5	3+		3+
Pupil OS	4.5	2+	2+	3+

Figure 7.4 Example for recording principal pupillary testing measurements.

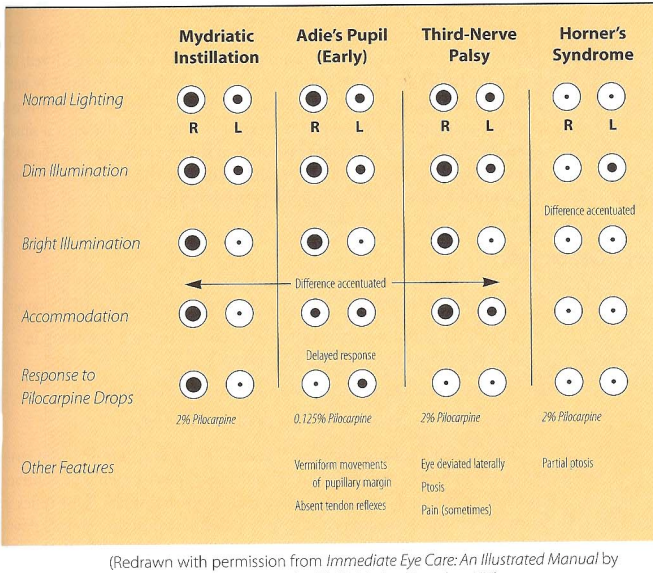
anisocoria, the inequality in pupillary size remains the same under all lighting conditions, and the pupillary light reflexes are equally brisk.

With true anisocoria, the examiner must document the size in millimeters of the palpebral fissure and note the presence and degree of any ptosis. Ptosis found on the side of a larger, sluggish pupil suggests the possibility of oculomotor paresis. Ptosis found on the side of a smaller pupil requires pharmacologic testing to rule out Horner's syndrome, which results from a lesion affecting the sympathetic pathway. Table 7.1 depicts four common causes of anisocoria and their differential diagnoses.

Light-Reflex Test

Shining a light in one eye normally causes both pupils to constrict equally, a result of the hemidecussation of pupillomotor fibers at the chiasm and in the midbrain tectum. The pupillary reaction in the illu-

Table 7.1 Anisocoria: Common Causes and Differential Diagnosis



(Redrawn with permission from *Immediate Eye Care: An Illustrated Manual* by Ragge and Easty, 1991. Mosby-Wolfe Limited, London, UK.)

minated eye is called the *direct reflex*, and the reaction in the nonilluminated eye is called the *consensual reflex*. The basic test used to evaluate these light reflexes as they occur in both eyes simultaneously is described in Clinical Protocol 7.1.

Swinging Flashlight Test

The swinging flashlight test is done after the light-reflex test to compare the direct and consensual responses in each eye individually (Clinical Protocol 7.2). This test is used to detect the presence of a relative afferent pupillary defect (also called the Marcus Gunn pupil), one of the most helpful neuro-ophthalmic signs. During the swinging flashlight test, the examiner alternately and briskly illuminates each

eye several times, noting pupillary response. A normal consensual response is for the pupils to become initially constricted and to remain so as the light is swung from eye to eye.

A relative afferent pupillary defect can be detected even when the pupil on the defective side is bound down by adhesions, paralyzed, or pharmacologically dilated, as long as the contralateral pupil is not. Swinging the light to the eye with optic nerve disease may show no pupillary change in that eye if the pupil is immobile. In such a situation, the degree of consensual response in the normal eye reflects optic nerve activity in the affected eye, and swinging the light to the intact eye should result in further pupillary constriction.

Near-Reflex Test

When a person looks at a near target, three reactions normally occur:

- Accommodation (contraction of the ciliary muscle, resulting in increased lens thickness and curvature)
- Convergence (contraction of the medial rectus muscles)
- Miosis (by contraction of the pupillary sphincter)

This combination of actions is called the *near synkinesis*. The term *synkinesis* indicates concurrent or simultaneous movements or contractions. The pupillary near reflex should be normal if the light reflex is normal, but the opposite is not true (see light-near dissociation below). Clinical Protocol 7.3 describes the near-reflex test.

Abnormal Pupils

Pupillary evaluation can reveal a variety of ophthalmic and neurologic abnormalities, including iris muscle damage, lesions of the sympathetic or parasympathetic pathways, optic nerve or retinal pathology, and dorsal midbrain lesions. This section discusses the primary findings commonly encountered when examining the pupils and the implications of those findings. Table 7.2 summarizes the characteristics of pupils in a variety of clinical situations.

Iris Abnormalities

Trauma, surgery, inflammation, or ischemia can damage the iris and alter its appearance. The pupil can be somewhat dilated, sluggishly reactive, and irregular because of traumatic sphincter rupture. There

Table 7.2 Pupillary Findings in Common Clinical Situations

Clinical Entity	General Features	Neuroanatomic Site of Lesion	Response to Light and Near Stimulation	Anisocoria?	Response to Mydriatics	Response to Miotics	Response to Other Pharmacologic Agents
Essential (physiologic) anisocoria	Round, regular	Benign, normal finding	Both brisk	No change	Dilates	Constricts	Normal
Traumatized iris	Irregular, notched pupil border on slit-lamp examination	Pupillary iris sphincter muscle	Both variable; depend on extent of damage	Greater in light	Dilates	Variable constriction; depends on extent of damage	NA
Relative afferent pupillary defect (Marcus Gunn pupil)	Round, regular; positive swinging flashlight test	Optic nerve or extensive retinal damage	Affected pupil shows better consensual than direct light reaction	No change	Dilates	Constricts	Normal
Midbrain pupils	Mid-dilated bilaterally	Dorsal midbrain	Poor to light, better to near	No change	Dilates	Constricts	NA
Tonic pupil (Adie's syndrome)	Acutely dilated, eventually may become miotic; sector pupil palsy, vermiform movement	Ciliary ganglion	Absent to light, tonic to near, tonic redilation	Greater in light	Dilates	Constricts	Pilocarpine 0.125% constricts; Mecholyl 2.5% constricts
Pharmacologically dilated pupil	Very large, round, unilateral	Iris sphincter	Fixed at 8–9 mm	Greater in light	Already maximally dilated	No constriction	Pilocarpine 0.5% –2% will not constrict
Oculomotor palsy (nonvascular)	Mid-dilated (5–7 mm), mild to severe ptosis, unilateral	Third cranial nerve; suspect aneurysm	+/- fixed	Greater in light	Dilates	Constricts	Pilocarpine 0.5% –2% will constrict
Horner's syndrome	Small, round, mild ptosis, unilateral	Sympathetic pathway	Both brisk	Greater in darkness	Dilates	Constricts	Cocaine 4%–10%: poor or no dilation; Paredrine 1%: poor or no dilation if third-order neuron damage, dilates otherwise
Argyll Robertson pupil	Small, irregular, bilateral	Midbrain	Poor to light, better to near	No change	Poor	Constricts	NA

might be notches in the pupillary margin. Additional sequelae of trauma include iritis and reactive miosis. Inflammation can result in anterior and posterior synechiae, affecting the appearance and reactivity of the pupil. Various disorders may cause iris neovascularization with closure of the chamber angle. Developmental anomalies and genetic disorders can be associated with such iris abnormalities as coloboma, aniridia, polycoria, Brushfield spots, Lisch nodules, or iris transillumination. Iris abnormalities are best observed at the slit lamp.

Relative Afferent Pupillary Defect

A relative afferent pupillary defect (RAPD), or Marcus Gunn pupil, is detected with the swinging flashlight test, described earlier. An RAPD indicates unilateral or asymmetric damage to the anterior visual pathways (eg, optic nerve disease or extensive retinal damage). It is not seen with symmetric damage to the anterior visual pathways and it is not present in patients with cataract or other media opacities, refractive errors, functional visual loss, or cortical lesions. The RAPD is often proportional to the amount of visual loss. It is graded from +1 to +4, with +4 designating an amaurotic pupil, an extreme example in which the eye shows no direct light reaction as a result of profound optic nerve damage.

Light-Near Dissociation

Light-near dissociation refers to a case in which the patient has significantly better pupillary near reflex than light reflex. Light-near dissociation can result from damage to bilateral structures that constitute the afferent limb of the pupillary light reaction (eg, bilateral optic atrophy) or from damage to the fibers that mediate the pupillary light reflex in the dorsal aspect of the midbrain. Mesencephalic fibers for the near reflex are located more ventrally than fibers for the light reflex. Thus, the near-reflex fibers are sometimes spared from the effect of compressive or superficial inflammatory lesions involving the dorsal midbrain.

Light-near dissociation is evident in dorsal midbrain (Parinaud's) syndrome, which arises most commonly from a pineal-region tumor compressing the dorsal midbrain but can be caused by multiple sclerosis, stroke, or hydrocephalus. The patient has mid-dilated pupils with light-near dissociation, up-gaze palsy, eyelid retraction, and convergence-retraction nystagmus. The light-near dissociation arises from compression of the superficially located fibers needed for the light reflex; the more ventral near fibers are spared.

Another syndrome that shows light-near dissociation is Argyll Robertson pupil, a rare but classic sign of neurosyphilis (particularly tabes dorsalis). Both pupils are miotic, asymmetric in size, and irregular in shape. Other causes of light-near dissociation include bilateral optic atrophy and Wernicke's encephalopathy. Certain disorders such as diabetes mellitus (probably the most common cause of light-near dissociation) and amyloidosis cause light-near dissociation as a result of their associated peripheral autonomic neuropathies.

Horner's Syndrome

Horner's syndrome results from damage to ocular sympathetic fibers at any level along the sympathetic pathway (central, preganglionic, or postganglionic neurons). Features of this syndrome include

- Mild ptosis (due to paresis of Müller's muscle)
- Miosis (by paralysis of the pupillary dilator muscle)
- Ipsilateral decrease in facial sweating (anhidrosis)
- Apparent enophthalmos
- Heterochromia iridis (usually in congenital cases)

Other signs include lower eyelid reverse ptosis (the margin of the eyelid is higher than normal), transient decrease in intraocular pressure, and increased tear breakup time. The associated anisocoria is more apparent in dim illumination, and a dilation lag is noted when shifting from bright to dim illumination.

Testing

Pharmacologic testing to confirm the diagnosis of Horner's syndrome consists of the instillation of a drop of 4% to 10% cocaine solution in each eye, which dilates the normal eye only. It works by blocking the reuptake of norepinephrine from sympathetic nerve endings, allowing the norepinephrine to remain in contact with its effector muscle longer. If the sympathetic pathway on one side is interrupted at any level, norepinephrine is not released from the nerve endings; the cocaine will have no effect; and the pupil on that side will remain miotic.

Once the diagnosis of Horner's syndrome is confirmed with the cocaine test, hydroxyamphetamine can be used to differentiate central and preganglionic lesions from postganglionic lesions. The hydroxyamphetamine test cannot be performed on the same day as the cocaine test because cocaine interferes with the action of hydroxyamphetamine. One drop of hydroxyamphetamine (Paredrine 1%) is instilled in

each eye. Because hydroxyamphetamine stimulates the release of norepinephrine from sympathetic postganglionic nerve terminals, it will fail to dilate the pupil in patients who have postganglionic lesions but will dilate the pupils in those with central or preganglionic lesions.

Differentiation of lesions is clinically useful because central and preganglionic lesions are more likely to be harbingers of more serious disease than postganglionic lesions. For example, central lesions arise from central nervous system vascular events and tumors. Preganglionic lesions are caused by apical lung tumors (Pancoast tumors), chest surgery, and thoracic artery aneurysm. The causes of postganglionic lesions include cluster headaches, spontaneous dissection of the carotid artery, and neck trauma.

Do not perform applanation tonometry, test corneal sensation, or otherwise irritate or touch the corneas before pharmacologic testing; any resulting epithelial defect can lead to unequal absorption of the diagnostic drops and perhaps a false negative or a false positive test result.

The Fixed and Dilated Pupil

The differential diagnosis of a fixed, dilated pupil is important to master. This sign can be seen in the following conditions:

- Adie's tonic pupil
- Oculomotor nerve palsy
- Pharmacologic blockade
- Traumatic iris sphincter rupture
- Angle-closure glaucoma

Adie's tonic pupil

Adie's tonic pupil is typically seen as unilateral mydriasis in an otherwise healthy young woman. Acutely the pupil is large, but it diminishes in size over months to years and can become miotic eventually. The pupil shows sluggish or no reaction to light and a slow (tonic) near response. Redilation is also tonic. At the slit lamp, slow, wormlike (vermiform) contractions of the iris help in making the diagnosis. The precise cause of the disorder is unknown. Postganglionic parasympathetic denervation is present, and the lesion is thought to localize to the ciliary ganglion. Many patients with Adie's tonic pupil show impaired knee or ankle jerks and corneal hypesthesia. The condition can be diagnosed by its hypersensitivity to weak miotic drops; an Adie's tonic pupil constricts to 0.05% to 0.1% pilocarpine drops, which affect a normal pupil only minimally.

Oculomotor nerve palsy

Compression of the third cranial (oculomotor) nerve results in the typical features of oculomotor nerve palsy, which include ptosis, motility abnormalities, and a dilated pupil on the affected side. Because the parasympathetic fibers are located in the peripheral (superficial) portion of the oculomotor nerve as it exits the brain stem, they are typically affected by a compressive lesion (eg, tumor, aneurysm) and spared by a vasculopathic lesion (eg, diabetes mellitus). When an acute third-nerve palsy is accompanied by pupillary mydriasis, an aneurysm at the junction of the internal carotid and posterior communicating arteries must be vigorously and urgently investigated with appropriate neuroimaging. Other causes of oculomotor palsy include brain tumor, basal meningitis, or uncal herniation. Diabetic (vasculopathic) oculomotor nerve palsy usually spares the pupil.

Pharmacologic blockade

Pharmacologic blockade is one of the most frequent causes of a dilated and fixed pupil in an otherwise healthy patient. It results from purposeful or inadvertent instillation of atropine-like drugs into the eyes. It can be differentiated from a dilated pupil accompanying a third-nerve palsy or Adie's syndrome by the absence of ptosis and motility abnormalities and by failure of the pupil to constrict upon instillation of pilocarpine 0.5% or 1% drops into the eye. These drops would cause constriction of a mydriatic pupil accompanying an oculomotor palsy.

Other causes

Another cause of pupillary mydriasis is traumatic iris sphincter rupture. Careful slit-lamp examination will reveal irregular pupillary borders at the sites of sphincter rupture. Acute angle-closure glaucoma classically presents with a mid-dilated and poorly reactive pupil; look for a red eye with corneal edema and increased intraocular pressure.

Pitfalls and Pointers

- Unilateral blindness, or a unilateral relative afferent pupillary defect, does not cause anisocoria. Whereas the pupil on the defective side might react only sluggishly or not at all to direct light stimulation, it will constrict consensually when the normal contralateral eye is stimulated.

- A relative afferent pupillary defect indicates that the afferent visual pathway is defective on one side in comparison with the contralateral pathway (the adjective *relative* emphasizes this). There is no such thing as a *bilateral* relative afferent pupillary defect.
- When performing the swinging flashlight test, be careful to spend equal time with the light illuminating each pupil to avoid differential bleaching of photoreceptors and possible artifactitious relative afferent pupillary defect. Anisocoria and severe ptosis can also cause differential photoreceptor bleaching that can confound pupillary testing.
- Recognize that a pupil-involving oculomotor nerve palsy is often a harbinger of cerebral aneurysm. Perform the workup expeditiously, and obtain a neurosurgical consultation emergently.
- When performing pharmacologic pupillary testing, instill drops in both eyes for comparison.
- Media opacities such as cataract or even dense vitreous hemorrhage almost never cause a relative afferent pupillary defect.
- Do not administer hydroxyamphetamine on the same day as cocaine (for diagnosis of Horner's syndrome), because cocaine interferes with the action of hydroxyamphetamine.

Suggested Resources

Burde RM, Savino PJ, Trobe JD. *Clinical Decisions in Neuro-Ophthalmology*, 2nd ed. St Louis: Mosby-Year Book; 1992; pp 321–346.

Thompson HS, Kardon RH. Clinical importance of pupillary inequality. *Focal Points: Clinical Modules for Ophthalmologists*, Vol X, Module 10. San Francisco: American Academy of Ophthalmology; 1992.

Clinical Protocol 7.1

Performing the Light-Reflex Test

1. Under dim room illumination, ask the patient to maintain fixation on a distance target, such as a large letter on the Snellen acuity chart.
2. Shine a bright handheld light directly into the right eye by approaching it from the side or from below. Do not stand in front of the patient or allow the patient to look directly at the light, which would stimulate the near reflex and preclude accurate light-reflex testing.
3. Record the direct pupillary response to light in the right eye in terms of the briskness of the response, graded from 0, indicating no response, to 4+, indicating a brisk response (see "Briskness of Light Reaction" in Figure 7.4).
4. Repeat steps 1–3 for the left eye.
5. Repeat steps 1 and 2 in the right eye, observing the consensual reflex by noting the response to the light of the nonilluminated (left) pupil. The rapidity of the response and change in pupil size should normally be equivalent to that seen in the direct light reaction and is graded on the same numeric scale.
6. Repeat steps 1, 2, and 5 in the left eye.

Clinical Protocol 7.2

Performing the Swinging Flashlight Test

1. Under dim room illumination with the patient fixating a distance target, illuminate the patient's right eye directly with a bright handheld light, in a manner identical to that used when testing the light reflex (Figure 1A). Note the pupillary constriction in both eyes.

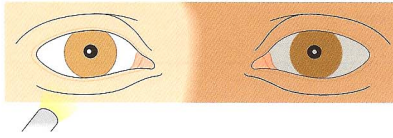


Figure 1A

continued

2. Move the light beam immediately and swiftly over the bridge of the patient's nose to the left eye, noting the pupillary response in that eye. Normally, the pupil will either constrict slightly or remain at its previous size (Figure 1B). If, instead, the pupil dilates when the light illuminates it (ie, the direct light reflex is weaker than the consensual reflex), a relative afferent pupillary defect is present, which usually indicates a disorder of the optic nerve or severe retinal pathology (Figure 1C).

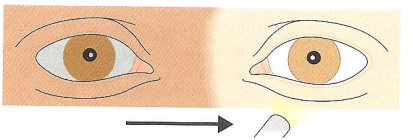


Figure 1B

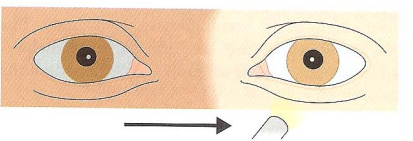


Figure 1C

3. Quickly swing the light back to the right eye and evaluate the response. A normal response is again a mild constriction or no change in size at all. Net pupillary constriction or dilation is an abnormal response.
4. Repeat steps 1–3 rhythmically, spending equal intervals illuminating each pupil, until it is clear whether pupillary responses are normal or whether one pupil consistently dilates.
5. Record a relative afferent pupillary defect (RAPD) as 1+ to 4+, with 1+ indicating a mild afferent defect and 4+ indicating an amaurotic pupil, a severe defect in which the affected eye shows no direct light response (see "RAPD" in Figure 7.4). Neutral density filters may be used for more accurate quantification.

Clinical Protocol 7.3

Performing the Near-Reflex Test

1. Under normal room illumination, ask the patient to fixate a distance target.
2. While seated next to the patient, move a detailed target toward the patient's line of vision at near. (The patient's thumb is sometimes an excellent near target, providing proprioceptive as well as visual near clues to ensure adequate near efforts). A flashlight should not be used for this purpose.
3. Instruct the patient to shift fixation to the near target. If using the patient's thumb as a target, the examiner holds the patient's thumb and moves it, asking the patient to view it intently.
4. Observe the pupillary reflex when the patient shifts fixation to the near target. Normal pupils constrict upon viewing the near target.
5. Repeat steps 1–4 several times.
6. Record the near reaction in terms of the briskness of the response, graded from 0, for no response, to 4+, for brisk response (see "Near Reaction" in Figure 7.4).

8

Visual Field Examination

The field of vision is that portion of a subject's surroundings that is visible at any one time. The visual field properly includes central fixation, conventionally measured by visual acuity tests, and extrafoveal (or peripheral) vision. Central fixation, or visual acuity, and the visual field are tested in different ways and provide information on different aspects of visual function. Visual acuity testing measures resolution, the ability to identify forms. Visual field testing measures sensitivity, the ability to detect light thresholds at different locations. An abnormal visual field can indicate a problem in the retina, optic nerve, or visual pathway.

Visual field testing is part of a thorough ophthalmic examination. The visual field of each eye is tested separately by one or more tests. The visual fields are routinely screened with the confrontation fields test. If macular disease is suspected to be causing a central visual field defect, a device called an Amsler grid is used to test the central area of each eye's visual field. If a visual field defect is detected by screening, further evaluation is conducted by

manual or automated procedures known as perimetry. Perimetry is used to document the presence and severity of a visual field defect and to monitor progression of previously known visual field loss.

This chapter introduces the concept of the visual field and discusses its anatomic correlates. It describes the most common methods of screening and clinical testing, giving step-by-step instructions in basic methods, and discusses general categories of visual field defects.

The Visual Field

The visual field is an inverted and reversed map of corresponding retinal points. Limited by the ora serrata and any intervening obstructions (eg, orbital rim and nose), the normal visual field extends about 50° superiorly, 60° nasally, 70° inferiorly, and 90° temporally from fixation. The visual field can be divided into central, intermediate, and peripheral zones. The central zone includes an area from the fixation point to a circle 30° away (a 5 mm radius from the fovea). The central zone contains the temporal physiologic blind spot, which corresponds to the optic nerve head centered about 15° from the fovea. The intermediate zone extends from 30° to 50° and the peripheral zone is the area beyond 50°.

A *scotoma*, also called a *visual field defect*, is a place in the visual field where an object cannot be seen. A *relative scotoma* is an area in the visual field where test objects of low luminance cannot be seen, but larger or brighter ones can. An *absolute scotoma* is an area where no test object can be seen (eg, the physiologic blind spot or a scotoma in advanced disease). In addition to its density, a scotoma is described by its shape (eg, hemianopia, quadrantanopia, etc) and its location (eg, temporal, superonasal, etc).

Perimetry is the measurement of the visual field during central fixation using either moving objects (kinetic perimetry) or stationary test stimuli (static perimetry). In kinetic perimetry, points along the edge of the visual field are determined by finding the weakest light stimulus (visual threshold) that evokes a visual sensation. The line that joins points having the same threshold is called an *isopter*. In static perimetry, the use of fixed targets that change in brightness gives a threshold map of different points where the patient has an equal chance of either seeing or not seeing the target.

The map of the visual field is a topographic plot of neural sensitivity at different locations. The clinical record of the normal field of vision varies with the size, color, brightness, and movement of the test stimulus, or target; with the background illumination; and with a patient's alertness and familiarity with the test. Visual fields are conventionally recorded on charts that represent the field as the patient sees it (ie, the temporal field of the right eye is to the right), and the field for the right eye is always placed to the right when comparing the visual field maps of both eyes.

The visual field is a three-dimensional concept that is presented in two dimensions (Figure 8.1A). Different types of perimetry give maps with different appearances. A visual field performed by kinetic perimetry (eg, with the Goldmann perimeter) is plotted on polar graph paper (Figure 8.1B) with radial meridians (measured counterclockwise from 0° at the right-hand horizontal) and circles of eccentricity (concentric rings every 10° out from fixation). With such a map obtained by kinetic perimetry, the examiner looks down onto the "hill of vision," with its contours represented by isopters. In older maps of static perimetry, the examiner looked across the visual field landscape and focused on a certain cross-section. Most maps produced by automated static perimetry present an array of sensitivity values; these values can also be pictured by the program in gray tones in a "map" resembling an isoptric plot (Figure 8.1C).

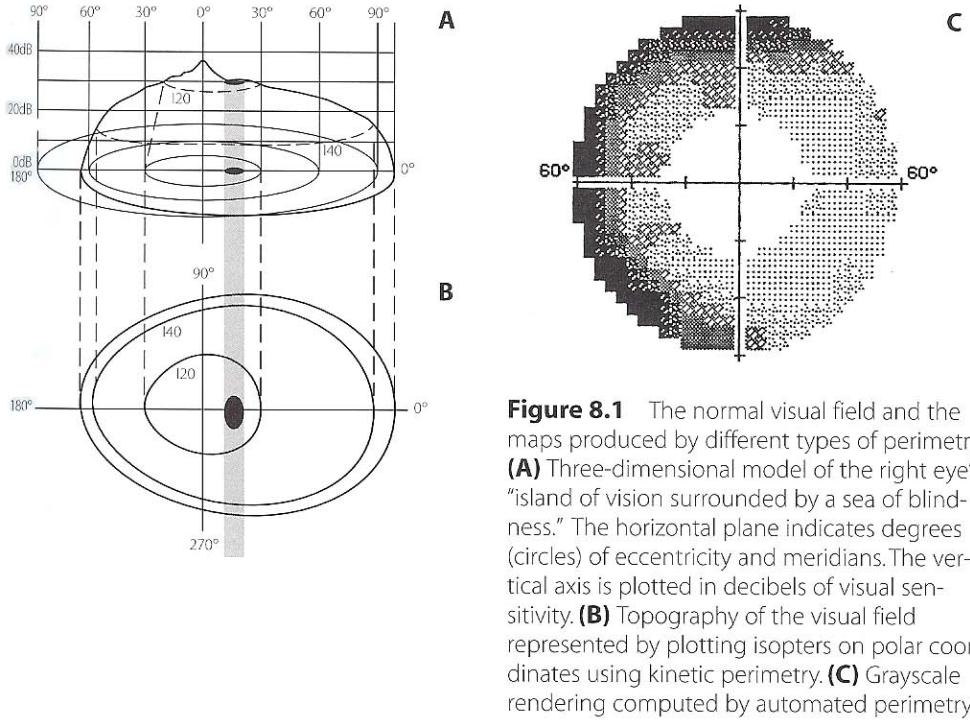


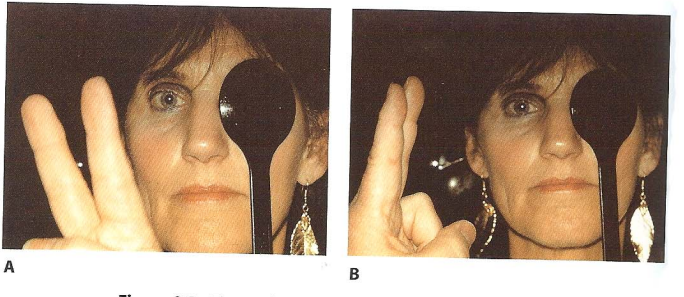
Figure 8.1 The normal visual field and the maps produced by different types of perimetry. **(A)** Three-dimensional model of the right eye's "island of vision surrounded by a sea of blindness." The horizontal plane indicates degrees (circles) of eccentricity and meridians. The vertical axis is plotted in decibels of visual sensitivity. **(B)** Topography of the visual field represented by plotting isopters on polar coordinates using kinetic perimetry. **(C)** Grayscale rendering computed by automated perimetry.

Screening Tests

Visual field screening is routinely done at a patient's initial eye examination. The confrontation fields test can screen for an unsuspected visual field defect caused by a lesion of the central nervous system, but confrontation testing is often unreliable for detecting subtle visual field loss, as in glaucoma. The Amsler grid, used when the patient has symptoms of central distortion or loss, helps evaluate macular function. Both of these screening tests are discussed below.

Confrontation Fields Testing

Confrontation testing of a patient's visual fields is done in a face-to-face position at a distance of about 1 meter (3 feet). By convention, the right eye is tested first, although if there is a marked difference in visual acuity it is advisable to begin with whichever is the better eye. The eye not being tested must be completely occluded, either by using a handheld or press-on occluder, by putting a folded facial tissue under an elastic eye occluder, or by asking the patient to cover the eye with the palm of the hand. When the patient's left eye is covered, the examiner's right eye should be closed, and vice versa, to permit comparison. Then present fingers midway between yourself and the patient, testing all four quadrants (Figure 8.2).



A B

Figure 8.2 The confrontation fields test. **(A)** Correct presentation of fingers, side by side frontally. **(B)** Incorrect presentation, so that one finger hides the other. (Reprinted by permission from Walsh TJ, ed: *Visual Fields: Examination and Interpretation*. 2nd ed. Ophthalmology Monograph 3. San Francisco, American Academy of Ophthalmology; 1996.)

To assess the patient's visual field, compare the patient's responses to your own normal visual field. Testing the edges of the central visual field is more rewarding than testing the extreme periphery, because the hand is often about the size of the 20/400 optotype, which is the visual level 30° from the fovea. (All examiners should know how their hand compares in size to the Snellen chart.) To outline the visual field determined by the confrontation screening method, slowly bring your hand inward from different directions, testing each of the patient's meridians. Instructions for performing the confrontation fields test are provided in Clinical Protocol 8.1.

Special Situations

Confrontation testing to screen for visual field defects may not be possible in infants, obtunded patients, and patients with optic nerve disease. Alternative screening methods for such patients are described below.

Reflex eye movement test for infants

Test the visual field of infants and toddlers by making use of their involuntary fixational reflexes. First get the child's attention in a frontal gaze. While the child is watching your face, silently bring an interesting toy or other object from the periphery to elicit fixational head and eye movements.

Blink reflex test for obtunded patients

Quickly flicking your hand toward a sighted patient's open eye normally elicits a blink reflex. This test can help find a dense hemianopia or quadrantanopia.

Comparison testing for patients with optic nerve disease

The blind spot usually cannot be adequately evaluated by confrontation fields testing. If you suspect optic neuropathy, ask the patient to subjectively compare the brightness of a light between the two eyes. To do this, shine a penlight directly into the patient's open eye, first into one and then into the other. By assigning a 100% score to the brighter eye, have the patient estimate the relative reduction in light intensity perceived by the dimmer eye. For example, ask the patient, "If the right eye is one dollar, how much is the left eye worth?"

The color desaturation test requires the use of a bright red object to compare the two eyes. Neutral density filters can be used to help quantify the difference in brightness.

Common Visual Field Defects

An abnormal visual field test result should be described in the medical record according to which eye is involved, the shape of the field abnormality, its location, and its symmetry. These attributes, and certain typical perimetric patterns, help to localize a lesion along the visual pathway. Table 8.3 lists some common descriptions of visual field

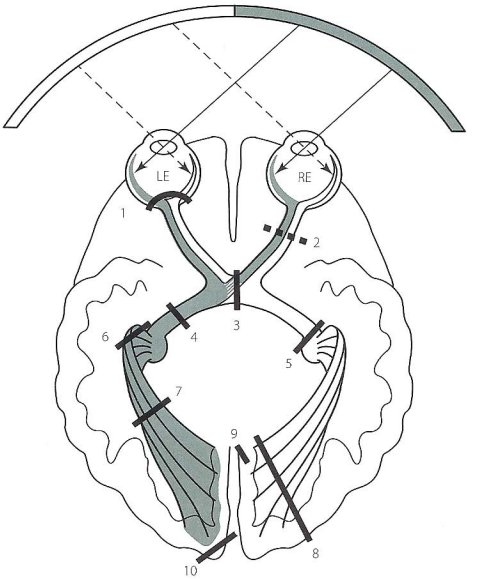
Table 8.3 Terms Used to Describe Visual Field Defects

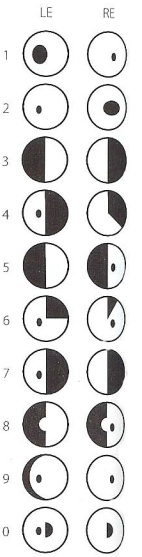
Monocular Field Defects	Binocular Field Defects
Localized Defects	*Homonymous Hemianopias*
■ Wedge-shaped temporal field defect	■ With macular splitting
■ Arcuate nasal field defect	■ With macular sparing
■ Central scotoma	■ Paramidline-sparing vertical hemianopia
■ Enlarged blind spot	■ With unilateral sparing of temporal crescent
■ Cecocentral scotoma	*Bitemporal Hemianopias*
■ Annular scotoma	*Binasal Hemianopias*
Generalized Defects	*Altitudinal Field Defects*
■ Generalized depression (peripheral constriction)	*Quadrantanopias*
	Bilateral Central Field Defects
	Bilateral Peripheral Field Defects

defects, some of which are discussed below. After a field defect has been documented, its progression is followed via multiple testings over time.

Eye and Shape

A visual field abnormality can be classified as monocular (a defect of one eye's visual field) or binocular (a defect of both eyes' visual fields).





Location	Visual Field
1. Optic disc	Enlarged blind spot
2. Optic nerve	Cecocentral scotoma
3. Chiasm	Bitemporal hemianopia
4. Optic tract	Noncongruous homonymous hemianopia
5. Lateral geniculate body	Homonymous hemianopia
6. Temporal lobe	Noncongruous superior quadrantanopia
7. Parietal lobe	Homonymous hemianopia
8. Occipital lobe	Homonymous hemianopia with macular sparing
9. Anterior calcarine fissure	Temporal crescent
10. Tip of occipital lobe	Homonymous hemianopic scotomas

Figure 8.8 Abnormal visual fields produced by lesions of the visual pathway.

Figure 8.8 shows a variety of commonly seen shapes of visual field defects together with a diagram of their anatomic origins. One of the most common shapes is a scotoma, a localized defect surrounded by detectable visual field. Some examples of common scotomas associated with glaucoma are shown in Figure 8.9. These scotomas often extend from the blind spot (eg, arcuate scotoma) or appear to make the visual field smaller (peripheral constriction).

A Bjerrum scotoma, a monocular isolated paracentral defect, is an example of an arcuate scotoma, so called because it yields an arc-like shape when plotted. This crescentic form is caused by the normal course of the retinal ganglion cell nerve fibers. Defects in the arcuate zone may connect with the blind spot (Seidel scotoma), appear as one or more scattered paracentral scotomas, or end at the horizontal raphe (Rønne's nasal step). A nasal step is a scotoma that, when plotted, abuts onto the horizontal meridian and appears as a step-like loss of vision at the outer limit of the nasal field. An altitudinal scotoma is one that causes loss of the upper or lower visual field. There also may be generalized depression (also called peripheral constriction in kinetic perimetry) in which retinal sensitivity is diffusely reduced.

A binocular visual field defect in each eye's hemifield is called a hemianopia. Incomplete hemianopias are referred to as quadrantanopias and sectoral defects. A chiasmal or retrochiasmal lesion produces visual field defects that respect the vertical meridian and that remain in one hemifield of each eye (see Figure 8.8). Retinal and optic nerve lesions produce visual field defects that can cross the vertical meridian (see Figure 8.9).

Location

A hemianopia may be homonymous (ie, impairing visual function on the same side of each eye), bitemporal, or binasal. Quadrantanopias and altitudinal defects are described as being superior, inferior, or checkerboard.

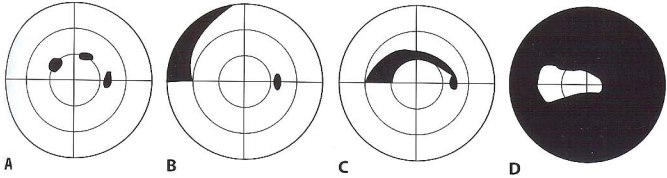


Figure 8.9 Visual field defects produced by glaucomatous optic neuropathy (right eye). **(A)** Paracentral scotoma. **(B)** Superior nasal step. **(C)** Arcuate scotoma. **(D)** Advanced peripheral constriction.

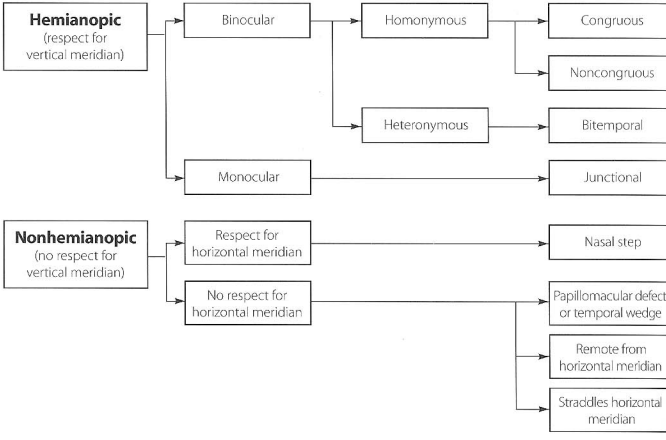


Figure 8.10 Interpretation tree for visual field defects. (From Trobe JD, Glaser JS. *The Visual Fields Manual: A Practical Guide to Testing and Interpretation.* Gainesville, FL: Triad; 1983.)

Symmetry

Field defects that are similar between the two eyes are called congruous, and defects that are asymmetric or differently sized for each eye are noncongruous. Because corresponding fibers from the two retinas lie close together as they near the visual cortex, lesions of the posterior radiations tend to be congruous, while lesions of the anterior visual pathways are more frequently noncongruous.

Localizing Visual Field Defects

The physician needs to know the typical patterns obtained in perimetry to determine the probable location of a lesion. A decision-making approach based on knowledge of neuroanatomy helps the examiner make an accurate medical interpretation. Figure 8.10 depicts a variety of common perimetric defects and their likely anatomic origins.

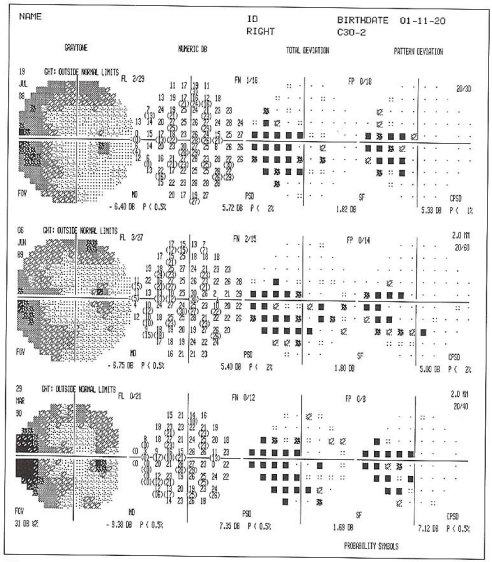


Figure 8.11 Progressive visual field loss as detected by repeat automated perimetry in a patient with chronic glaucoma.

Progression

Visual fields often must be tested on several occasions to get a reliable picture of the patient's status. Chronic diseases such as glaucoma can produce progressive visual field loss that may be detected before optic nerve or other changes are visible (Figure 8.11).

Pitfalls and Pointers

- *Make the patient comfortable with the examination.* The results of a visual field examination will be most accurate if the examiner provides a quiet, nondisturbing environment and ensures that the patient fully understands the testing procedure. Some patients

Suggested Resources

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Clinical Protocol 8.1

Performing the Confrontation Fields Test

Test Setup

1. Seat the patient and make sure the eye not being tested is occluded.
2. Seat yourself facing the patient at a distance of about 1 m. Close your eye that is directly opposite the patient's occluded eye.
3. Ask the patient to fixate on your nose or on your open eye.

Check for Scotoma

4. *Finger counting.* Hold your hands stationary midway between yourself and the patient in opposite quadrants about 30° from central fixation

(60 mm [24 inches] from your mutual based axis). Quickly extend then retract a finger or fingers on one hand in one quadrant of the monocular field, asking the patient to state the number. To avoid confusion, limit the number of fingers shown to 1, 2, and 5, and hold the fingers side by side in the frontal plane. Repeat in all four quadrants, testing at least two times per quadrant.

a. Test patients who have marked visual loss by waving your hand in each quadrant individually and asking if the patient perceives the motion. With patients who can only perceive light, test in each quadrant individually for the ability to correctly determine the direction of light projection by pointing a transilluminator or penlight toward the pupil while keeping the patient's other eye completely shielded.

b. Test young children with a finger-mimicking procedure. First teach the child to hold up the same number of fingers as you do, then conduct the test as usual. Test rapidly, because a child will soon glance directly at your hand (although this involuntary movement can also indicate a normal response).

5. *Simultaneous finger counting.* Present fingers simultaneously in opposite quadrants, asking the patient to state the total number, using the following combinations: 1 and 1, 1 and 2, and 2 and 2. This test can reveal a more subtle field defect than finger counting in each quadrant separately. Sometimes a patient with a relative scotoma can detect fingers presented to the defective hemifield but has problems with simultaneous targets.

6. *Simultaneous comparison.* Hold both palms toward the patient, close to the line of sight, in opposite superior, then inferior, quadrants. Ask the patient to state whether one hand appears darker or less distinct. This test is very subjective and relies on equal illumination but can reveal a subtle defect in a hemifield.

a. A similar test can be done by asking the patient to compare the relative hue or intensity of two identically colored objects, such as the red caps of two eyedropper bottles. Hold the targets in separate quadrants. If there is a hemianopia, the patient may describe one cap as red and the other as faded or colorless. This test can also be done with one colored item by bringing it across from a defective to a normal area, to determine whether there is a sudden change in intensity.

b. Check a central scotoma by comparing central and eccentric locations, using hands or other identical objects.

continued

8.1

Diagram the Confrontation Field

7. If an abnormality is detected, sketch a 360° visual field chart, labeled for right and left eye and temporal and nasal field, and plot the visual field as the patient sees it (Figure 1). Record a failure to detect an abnormality as "no defect to finger confrontation."

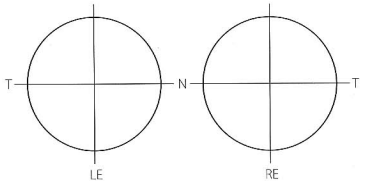

Figure 1