

Eye and Eye Problems

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Overview The visual system provides a rapid and efficient means for the rapid assimilation of information from the surrounding. The act of seeing begins with the capture of images; transmit it to an area in the back of the brain called occipital. The image is processed using other related information such as time element, coordinate and other attribute to recognize and react to it. The brain needs all these elements to recognize an image, for example if time attribute is missed the person experience day je vou. The eyes must be rotated constantly within their orbits to place and maintain targets of visual interest upon the fovea. This activity looking, is governed by an elaborate efferent motor system. Each eye is moved by six extraocular muscles, supplied by cranial nerves from the oculomotor (III), trochlear (IV), and abducens (VI) nuclei. Activity in these ocular motor nuclei is coordinated by pontine and midbrain mechanisms for smooth pursuit, saccades, and gaze stabilization during head and body movements. Visual function can be disturbed in myriad ways. The eyes are mounted in a prominent position on the head, where they are vulnerable to trauma, exposure, and infection. Vision can be damaged by diseases intrinsic to the eye, such as glaucoma, cataract, or retinal detachment. Many neurologic diseases produce ocular symptoms, because extensive areas of the cortex, thalamus, cerebellum, and brainstem are devoted to visual perception or to the execution of eye movements. In genetic disorders, eye manifestations are common and often help the clinician to recognize a rare syndrome such as Leber's hereditary. Finally, the eyes are affected frequently by acquired systemic diseases. [\[TOP\]](#)

CLINICAL ASSESSMENT OF VISUAL FUNCTION In approaching the patient with reduced vision, the first step is to decide whether refractive error is responsible. In emmetropia, parallel rays from infinity are focused perfectly upon the retina. Sadly, this condition is enjoyed by only a minority of the population. In myopia, the globe is too long, and light rays come to a focal point in front of the retina. Near objects can be seen clearly, but distant objects require a diverging lens in front of the eye. In hyperopia, the globe is too short, and hence a converging lens is used to supplement the refractive power of the eye. In astigmatism, the corneal surface is not perfectly spherical, necessitating a cylindrical corrective lens. In recent years it has become possible to correct refractive error with the excimer laser by performing either LASIK (laser in situ keratomileusis) or PRK (photorefractive keratectomy) to alter the curvature of the cornea. With the onset of middle age, presbyopia develops as the lens within the eye becomes unable to increase its refractive power to accommodate upon near objects. To compensate for presbyopia, the emmetropic patient must use reading glasses. The patient already wearing glasses for distance correction usually

switches to bifocals. The only exception is the myopic patient, who may achieve clear vision at near simply by removing glasses containing the distance prescription. Refractive errors usually develop slowly and remain stable after adolescence, except in unusual circumstances. For example, the acute onset of diabetes mellitus can produce sudden myopia because of fluid imbibition and swelling of the lens induced by hyperglycemia. Testing vision through a pinhole aperture is a useful way to screen quickly for refractive error. If the visual acuity is better through a pinhole than with the unaided eye, the patient needs a refraction to obtain best corrected visual acuity. **[TOP] VISUAL ACUITY** The Snellen chart is used to test acuity at a distance of 6 m (20 ft). For convenience, a scale version of the Snellen chart, called the Rosenbaum card, is held at 36 cm (14 in) from the patient. All subjects should be able to read the 6/6 m (20/20 ft) line with each eye using their refractive correction, if any. Patients who need reading glasses because of presbyopia must wear them for accurate testing with the Rosenbaum card. If 6/6 (20/20) acuity is not present in each eye, the deficiency in vision must be explained. For acuity worse than 6/240 (20/800), the ability to count fingers, see hand motions, or perceive a bright light should be recorded. Legal blindness is defined by the Internal Revenue Service as a best corrected acuity of 6/60 (20/200) or less in the better eye, or a binocular visual field subtending 20° or less. For driving the laws vary by state, but most require a corrected acuity of 6/12 (20/40) in at least one eye. Patients with a homonymous hemianopia should not drive. **[TOP] PUPILS** The pupils should be tested individually in dim light with the patient fixating upon a distant target. If they respond briskly to light, there is no need to check the near response, because isolated loss of constriction (miosis) to accommodation does not occur. For this reason, the ubiquitous abbreviation PERRLA (pupils equal, round, and reactive to light and accommodation) implies a wasted effort with the last step. However, it is important to test the near response if the light response is poor or absent. Light-near dissociation occurs with neurosyphilis (Argyll Robertson pupil), lesions of the dorsal midbrain (obstructive hydrocephalus, pineal region tumors), and after aberrant regeneration (oculomotor nerve palsy, Adie's tonic pupil). An eye with no light perception has no pupillary response to direct light stimulation. If the retina or optic nerve is only partially injured, the direct pupillary response will be weaker than the consensual pupillary response evoked by shining a light into the other eye. This relative afferent pupillary defect (Marcus Gunn pupil) can be elicited with the swinging flashlight test. It is an extremely useful sign in retrobulbar optic neuritis and other optic nerve diseases, where it may be the sole objective evidence for disease. Subtle inequality in pupil size, up to 0.5 mm, is a fairly common finding in normal persons. The diagnosis of essential or physiologic anisocoria is secure as long as the relative pupil asymmetry remains constant as ambient lighting varies. Anisocoria that increases in dim light indicates a sympathetic paresis of the iris dilator muscle. The triad of miosis with ipsilateral ptosis and anhidrosis constitutes Horner's syndrome, although anhidrosis is an inconstant feature. Brainstem stroke, carotid dissection, or neoplasm impinging upon the sympathetic chain are occasionally identified as the cause of Horner's syndrome, but most of cases are idiopathic. Anisocoria that increases in bright light suggests a parasympathetic palsy. The first concern is an oculomotor nerve paresis. This possibility is excluded if the eye movements are full and the patient has no ptosis or diplopia. Acute pupillary dilation (mydriasis) can occur from damage to the ciliary ganglion in the orbit. Common mechanisms are infection (herpes zoster, influenza), trauma (blunt, penetrating, surgical), or ischemia (diabetes, temporal arteritis). After denervation of the iris sphincter the pupil does not respond well to light, but the response to near is often relatively intact. When the near stimulus is removed, the pupil redilates very slowly compared with the normal pupil, hence the term tonic pupil. In Adie's syndrome, a tonic pupil occurs in conjunction with weak or absent tendon reflexes in the lower extremities. This benign disorder, which occurs predominantly in healthy young women, is assumed to represent a mild dysautonomia. Tonic pupils are also associated with Shy-Drager syndrome, segmental hypohidrosis, diabetes, and amyloidosis. Occasionally, a tonic pupil is discovered incidentally in an otherwise completely normal, asymptomatic individual. The diagnosis is confirmed by placing a drop of dilute (0.125%) pilocarpine into each eye. Denervation hypersensitivity produces pupillary constriction in a tonic pupil, whereas the normal pupil shows no response. Pharmacologic dilation from accidental or deliberate instillation of anticholinergic agents (atropine, scopolamine drops) into the eye can also produce pupillary mydriasis. In this situation, normal strength (1%) pilocarpine causes no constriction. Both pupils are affected equally by systemic medications. They are small with narcotic use (morphine, heroin) and large with anticholinergics (scopolamine). Parasympathetic agents (pilocarpine, demecarium bromide) used to treat glaucoma produce miosis. In any patient with an unexplained

pupillary abnormality, a slit-lamp examination is helpful to exclude surgical trauma to the iris, an occult foreign body, perforating injury, intraocular inflammation, adhesions (synechia), angle-closure glaucoma, and iris sphincter rupture from blunt trauma. **[TOP] EYE MOVEMENTS AND ALIGNMENT** Eye movements are tested by asking the patient with both eyes open to pursue a small target such as a penlight into the cardinal fields of gaze. Normal ocular versions are smooth, symmetric, full, and maintained in all directions without nystagmus. Saccades, or quick refixation eye movements, are assessed by having the patient look back and forth between two stationary targets. The eyes should move rapidly and accurately in a single jump to their target. Ocular alignment can be judged by holding a penlight directly in front of the patient at about 1 m. If the eyes are straight, the corneal light reflex will be centered in the middle of each pupil. To test eye alignment more precisely, the cover test is useful. The patient is instructed to gaze upon a small fixation target in the distance. One eye is covered suddenly while observing the second eye. If the second eye shifts to fixate upon the target, it was misaligned. If it does not move, the first eye is uncovered and the test is repeated on the second eye. If neither eye moves, the eyes are aligned orthotropically. If the eyes are orthotropic in primary gaze but the patient complains of diplopia, the cover test should be performed with the head tilted or turned in whatever direction elicits the patient's diplopia. With practice the examiner can detect an ocular deviation (heterotropia) as small as 1 to 2° with the cover test. Deviations can be measured by placing prisms in front of the misaligned eye to determine the power required to neutralize the fixation shift evoked by covering the other eye. **STEREOPSIS** Stereoacuity is determined by presenting targets with retinal disparity separately to each eye using polarized images. The most popular office tests measure a range of thresholds from 800 to 40 seconds of arc. Normal stereoacuity is 40 seconds of arc. If a patient achieves this level of stereoacuity, one is assured that the eyes are aligned orthotropically and that vision is intact in each eye. Random dot stereograms have no monocular depth cues and provide an excellent screening test for strabismus and amblyopia in children. **[TOP] COLOR VISION** The retina contains three classes of cones, with visual pigments of differing peak spectral sensitivity: red (560 nm), green (530 nm), and blue (430 nm). The red and green cone pigments are encoded on the X chromosome; the blue cone pigment on chromosome 7. Mutations of the blue cone pigment are exceedingly rare. Mutations of the red and green pigments cause congenital X-linked color blindness in 8% of males. Affected individuals are not truly color blind; rather, they differ from normal subjects in how they perceive color and how they combine primary monochromatic lights to match a given color. Anomalous trichromats have three cone types, but a mutation in one cone pigment (usually red or green) causes a shift in peak spectral sensitivity, altering the proportion of primary colors required to achieve a color match. Dichromats have only two cone types and will therefore accept a color match based upon only two primary colors. Anomalous trichromats and dichromats have 6/6 (20/20) visual acuity, but their hue discrimination is impaired. Ishihara color plates can be used to detect red-green color blindness. The test plates contain a hidden number, visible only to subjects with color confusion from red-green color blindness. Because color blindness is almost exclusively X-linked, it is worth screening only male children. The Ishihara plates are often used to detect acquired defects in color vision, although they are intended as a screening test for congenital color blindness. Acquired defects in color vision frequently result from disease of the macula or optic nerve. For example, patients with a history of optic neuritis often complain of color desaturation long after their visual acuity has returned to normal. Color blindness can also occur from bilateral strokes involving the ventral portion of the occipital lobe (cerebral achromatopsia). Such patients can perceive only shades of gray and may also have difficulty recognizing faces (prosopagnosia). Infarcts of the dominant occipital lobe sometimes give rise to color anomia. Affected patients can discriminate colors, but they cannot name them. **[TOP] VISUAL FIELDS** Vision can be impaired by damage to the visual system anywhere from the eyes to the occipital lobes. One can localize the site of the lesion with considerable accuracy by mapping the visual field deficit by finger confrontation and then correlating it with the topographic anatomy of the visual pathway. More quantitative data can be obtained by formal perimetric examination of the visual fields. In kinetic perimetry, the patient faces a tangent screen or a hemispheric bowl (Goldmann perimeter) while the examiner moves a small light target from the periphery towards the center. Such manual techniques have largely been supplanted by computer-driven perimeters (Humphrey, Octopus) that present a target of variable intensity at fixed positions in the visual field. By generating an automated printout of light thresholds, these static perimeters provide a sensitive means of detecting scotomas in the visual field. They are also useful for serial assessment of visual

function in chronic diseases such as glaucoma or pseudotumor cerebri. The crux of visual field analysis is to decide whether a lesion is before, at, or behind the optic chiasm. If a scotoma is confined to one eye, it must be due to a lesion anterior to the chiasm, involving either the optic nerve or retina. Retinal lesions produce scotomas that correspond optically to their location in the fundus. For example, a superior-nasal retinal detachment results in an inferior-temporal field cut. Damage to the macula causes a central scotoma. Optic nerve disease produces characteristic patterns of visual field loss. Glaucoma selectively destroys axons that enter the superotemporal or inferotemporal poles of the optic disc, resulting in arcuate scotomas shaped like a Turkish scimitar, which emanate from the blind spot and curve around fixation to end flat against the horizontal meridian. This type of field defect mirrors the arrangement of the nerve fiber layer in the temporal retina. The superb acuity of humans is achieved by thrusting aside all retinal elements at the fovea except photoreceptors, to minimize absorption and scattering of light. To avoid passing over the fovea, axons from cells in the temporal retina must follow an indirect course arching around the fovea to reach the optic disc. Arcuate or nerve fiber layer scotomas also occur from optic neuritis, ischemic optic neuropathy, optic disc drusen, and branch retinal artery or vein occlusion. Damage to the entire upper or lower pole of the optic disc causes an altitudinal field cut that follows the horizontal meridian. This pattern of visual field loss is typical of ischemic optic neuropathy but also occurs from retinal vascular occlusion, advanced glaucoma, and optic neuritis. About half the fibers in the optic nerve originate from ganglion cells serving the macula. Damage to papillomacular fibers causes a cecocentral scotoma encompassing the blind spot and macula. If the damage is irreversible, pallor eventually appears in the temporal portion of the optic disc. Temporal pallor from a cecocentral scotoma may develop in optic neuritis, nutritional optic neuropathy, toxic optic neuropathy, Leber's hereditary optic neuropathy, and compressive optic neuropathy. It is worth mentioning that the temporal side of the optic disc is slightly more pale than the nasal side in most normal individuals. Therefore, it can sometimes be difficult to decide whether the temporal pallor visible on fundus examination represents a pathologic change. Pallor of the nasal rim of the optic disc is a less equivocal sign of optic atrophy. At the optic chiasm, fibers from nasal ganglion cells decussate into the contralateral optic tract. Crossed fibers are damaged more by compression than uncrossed fibers. As a result, mass lesions of the sellar region cause a temporal hemianopia in each eye. Tumors anterior to the optic chiasm, such as meningiomas of the tuberculum sellae, produce a junctional scotoma characterized by an optic neuropathy in one eye and a superior temporal field cut in the other eye. More symmetric compression of the optic chiasm by a pituitary adenoma, meningioma, craniopharyngioma, glioma, or aneurysm results in a bitemporal hemianopia. The insidious development of a bitemporal hemianopia often goes unnoticed by the patient and will escape detection by the physician unless each eye is tested separately. It is difficult to localize a postchiasmal lesion accurately, because injury anywhere in the optic tract, lateral geniculate body, optic radiations, or visual cortex can produce a homonymous hemianopia, i.e., a temporal hemifield defect in the contralateral eye and a matching nasal hemifield defect in the ipsilateral eye. A unilateral postchiasmal lesion leaves the visual acuity in each eye unaffected, although the patient may read the letters on only the left or right half of the eye chart. Lesions of the optic radiations tend to cause poorly matched or incongruous field defects in each eye. Damage to the optic radiations in the temporal lobe (Meyer's loop) produces a superior quadrantic homonymous hemianopia, whereas injury to the optic radiations in the parietal lobe results in an inferior quadrantic homonymous hemianopia. Lesions of the primary visual cortex give rise to dense, congruous hemianopic field defects. Occlusion of the posterior cerebral artery supplying the occipital lobe is a frequent cause of total homonymous hemianopia. Some patients with hemianopia after occipital stroke have macular sparing, because the macular representation at the tip of the occipital lobe is supplied by collaterals from the middle cerebral artery. Destruction of both occipital lobes produces cortical blindness. This condition can be distinguished from bilateral prechiasmal visual loss by noting that the pupil responses and optic fundi remain normal. **[TOP] PAINFUL EYE** Corneal Abrasions: These are seen best by placing a drop of fluorescein in the eye and looking with the slit lamp using a cobalt-blue light. A penlight with a blue filter will suffice if no slit lamp is available. Damage to the corneal epithelium is revealed by yellow fluorescence of the exposed basement membrane underlying the epithelium. It is important to check for foreign bodies. To search the conjunctival fornices, the lower lid should be pulled down and the upper lid everted. A foreign body can be removed with a moistened cotton-tipped applicator after placing a drop of topical anesthetic, such as proparacaine, in the eye. Alternatively, it may be possible to flush the foreign body from

the eye by irrigating copiously with saline or artificial tears. If the corneal epithelium has been abraded, antibiotic ointment and a patch should be applied to the eye. A drop of an intermediate-acting cycloplegic, such as cyclopentolate hydrochloride 1%, helps to reduce pain by relaxing the ciliary body. The eye should be reexamined the next day. Minor abrasions may not require patching and cycloplegia. **Subconjunctival Hemorrhage:** This results from rupture of small vessels bridging the potential space between the episclera and conjunctiva. Blood dissecting into this space can produce a spectacular red eye, but vision is not affected and the hemorrhage resolves without treatment. Subconjunctival hemorrhage is usually spontaneous but can occur from blunt trauma, eye rubbing, or vigorous coughing. Occasionally it is a clue to an underlying bleeding disorder. **Pinguecula:** This is a small, raised conjunctival nodule at the temporal or nasal limbus. In adults such lesions are extremely common and have little significance, unless they become inflamed (pingueculitis). A pterygium resembles a pinguecula but has crossed the limbus to encroach upon the corneal surface. Removal is justified when symptoms of irritation or blurring develop, but recurrence is a common problem. **Blepharitis:** This refers to inflammation of the eyelids. The most common form occurs in association with acne rosacea or seborrheic dermatitis. The eyelid margins are usually colonized heavily by staphylococcus. Upon close inspection, they appear greasy, ulcerated, and crusted with scaling debris that clings to the lashes. Treatment consists of warm compresses, strict eyelid hygiene, and topical antibiotics such as erythromycin. An external hordeolum (sty) is caused by staphylococcal infection of the superficial accessory glands of Zeis or Moll located in the eyelid margins. An internal hordeolum occurs after suppurative infection of the oil-secreting meibomian glands within the tarsal plate of the eyelid. Systemic antibiotics, usually tetracyclines, are sometimes necessary for treatment of meibomian gland inflammation (meibomitis) or chronic, severe blepharitis. A chalazion is a painless, granulomatous inflammation of a meibomian gland that produces a pealike nodule within the eyelid. It can be incised and drained, or injected with glucocorticoids. Basal cell, squamous cell, or meibomian gland carcinoma should be suspected for any nonhealing, ulcerative lesion of the eyelids. **Dacrocystitis:** An inflammation of the lacrimal drainage system, this can produce epiphora (tearing) and ocular injection. Gentle pressure over the lacrimal sac evokes pain and reflux of mucus or pus from the tear puncta. Dacrocystitis usually occurs after obstruction of the lacrimal system. It is treated with topical and systemic antibiotics, followed by probing or surgery to reestablish patency. Entropion (inversion of the eyelid) or ectropion (sagging or eversion of the eyelid) can also lead to epiphora and ocular irritation. **Conjunctivitis:** This is the most common cause of a red, irritated eye. Pain is minimal, and the visual acuity is reduced only slightly. The most common viral etiology is adenovirus infection. It causes a watery discharge, mild foreign-body sensation, and photophobia. Bacterial infection tends to produce a more mucopurulent exudate. Mild cases of infectious conjunctivitis are usually treated empirically with broad-spectrum topical ocular antibiotics, such as sulfacetamide 10%, polymixin-bacitracin-neomycin, or trimethoprim-polymixin combination. Smears and cultures are usually reserved for severe, resistant, or recurrent cases of conjunctivitis. To prevent contagion, patients should be admonished to wash their hands frequently, not to touch their eyes, and to avoid direct contact with others. **Allergic Conjunctivitis:** This condition is extremely common and often mistaken for infectious conjunctivitis. Three forms of allergic conjunctivitis are recognized, with closely overlapping manifestations. Hay fever conjunctivitis has a seasonal incidence, related to the release of airborne antigens into the air by plants. IgE-mediated activation of mast cells in the conjunctiva causes itching, redness, and edema. Vernal conjunctivitis is also seasonal, becoming worse during warm months. It affects exclusively children or adolescents and is more common in boys. The cause is unknown, but airborne antigens are thought to trigger symptoms. Itching, photophobia, epiphora, and mucous discharge are typical. The palpebral conjunctiva may become hypertrophic with giant excrescences called cobblestone papillae. Irritation from contact lenses or any chronic foreign body can also induce formation of cobblestone papillae. Atopic conjunctivitis occurs in subjects with atopic dermatitis or asthma. Symptoms caused by allergic conjunctivitis can be alleviated with cold compresses, topical vasoconstrictors, antihistamines, and mast-cell stabilizers such as cromolyn sodium. Topical glucocorticoid solutions provide dramatic relief of immune-mediated forms of conjunctivitis, but their long-term use is ill-advised because of the complications of glaucoma, cataract, and secondary infection. Topical nonsteroidal anti-inflammatory agents (NSAIDs) such as ketorolac tromethamine are a better alternative. **Keratoconjunctivitis Sicca:** Also known as dry eye, it produces a burning, foreign-body sensation, injection, and photophobia. In mild cases the eye appears surprisingly normal, but

tear production measured by wetting of a filter paper (Schirmer strip) is deficient. A variety of systemic drugs, including antihistaminic, anticholinergic, and psychotropic medications, result in dry eye by reducing lacrimal secretion. Disorders that involve the lacrimal gland directly, such as sarcoidosis or Sjogren's syndrome, also cause dry eye. Patients may develop dry eye after radiation therapy if the treatment field includes the orbits. Problems with ocular drying are also common after lesions affecting cranial nerves V or VII. Corneal anesthesia is particularly dangerous, because the absence of a normal blink reflex exposes the cornea to injury without pain to warn the patient. Dry eye is managed by frequent and liberal application of artificial tears and ocular lubricants. In severe cases the tear puncta can be plugged or cauterized to reduce lacrimal outflow. **Keratitis:** This is a threat to vision because of the risk of corneal clouding, scarring, and perforation. Worldwide, the two leading causes of blindness from keratitis are trachoma from chlamydial infection and vitamin A deficiency related to malnutrition. In the United States, contact lenses play a major role in corneal infection and ulceration. They should not be worn by anyone with an active eye infection. In evaluating the cornea, it is important to differentiate between a superficial infection (keratoconjunctivitis) and a deeper, more serious ulcerative process. The latter is accompanied by greater visual loss, pain, photophobia, redness, and discharge. Slit-lamp examination shows disruption of the corneal epithelium, a cloudy infiltrate or abscess in the stroma, and an inflammatory cellular reaction in the anterior chamber. In severe cases, pus settles at the bottom of the anterior chamber, giving rise to a hypopyon. Immediate empirical antibiotic therapy should be initiated after corneal scrapings are obtained for Gram's stain, Giemsa stain, and cultures. Fortified topical antibiotics are most effective, supplemented with subconjunctival antibiotics as required. The most frequent bacterial pathogens are Staphylococcus, Streptococcus (particularly *S. pneumoniae*), Pseudomonas, Enterobacteriaceae, Haemophilus, and Neisseria. For Neisseria, systemic antibiotics should be given in addition to topical antibiotics to eliminate systemic infection. A fungal etiology should always be considered in the patient with keratitis. Fungal infection is common in warm humid climates, especially after penetration of the cornea by plant or vegetable material. **Herpes Simplex:** The herpes viruses are a major cause of blindness from keratitis. Most adults in the United States have serum antibodies to herpes simplex, indicating prior viral infection. Primary ocular infection is generally caused by herpes simplex type 1, rather than type 2. It manifests as a unilateral follicular blepharoconjunctivitis, easily confused with adenoviral conjunctivitis unless telltale vesicles appear on the periocular skin or conjunctiva. A dendritic pattern of corneal epithelial ulceration revealed by fluorescein staining is pathognomonic for herpes infection but is seen in only a minority of primary infections. Recurrent ocular infection arises from reactivation of the latent herpes virus. Viral eruption in the corneal epithelium may result in the characteristic herpes dendrite. Involvement of the corneal stroma produces edema, vascularization, and iridocyclitis. Herpes keratitis is treated with topical antiviral agents, cycloplegics, and oral acyclovir. Topical glucocorticoids are effective in mitigating corneal scarring but must be used with extreme caution because of the danger of corneal melting and perforation. Topical glucocorticoids also carry the risk of prolonging infection and inducing glaucoma. **Herpes Zoster** Herpes zoster from reactivation of latent varicella (chickenpox) virus causes a dermatomal pattern of painful vesicular dermatitis. Ocular symptoms can occur after zoster eruption in any branch of the trigeminal nerve but are particularly common when vesicles form on the nose, reflecting nasociliary (V1) nerve involvement (Hutchinson's sign). Herpes zoster ophthalmicus produces corneal dendrites, which can be difficult to distinguish from those seen in herpes simplex. Stromal keratitis, anterior uveitis, raised intraocular pressure, ocular motor nerve palsies, acute retinal necrosis, and postherpetic scarring and neuralgia are other common sequelae. Herpes zoster ophthalmicus is treated with antiviral agents and cycloplegics. In severe cases, glucocorticoids may be added to prevent permanent visual loss from corneal scarring. **Episcleritis** This is an inflammation of the episclera, a thin layer of connective tissue between the conjunctiva and sclera. Episcleritis resembles conjunctivitis but is a more localized process and discharge is absent. Most cases of episcleritis are idiopathic, but some occur in the setting of an autoimmune disease. Scleritis refers to a deeper, more severe inflammatory process, frequently associated with a connective tissue disease such as rheumatoid arthritis, lupus erythematosus, polyarteritis nodosa, Wegener's granulomatosis, or relapsing polychondritis. The inflammation and thickening of the sclera can be diffuse or nodular. In anterior forms of scleritis, the globe assumes a violet hue and the patient complains of severe ocular tenderness and pain. With posterior scleritis the pain and redness may be less marked, but there is often proptosis, choroidal effusion, reduced motility, and visual loss. Episcleritis and scleritis

should be treated with NSAIDs. If these agents fail, topical or even systemic glucocorticoid therapy may be necessary, especially if an underlying autoimmune process is active. **Uveitis** Involving the anterior structures of the eye, this is called iritis or iridocyclitis. The diagnosis requires slit-lamp examination to identify inflammatory cells floating in the aqueous humor or deposited upon the corneal endothelium (keratic precipitates). Anterior uveitis develops in sarcoidosis, ankylosing spondylitis, juvenile rheumatoid arthritis, inflammatory bowel disease, psoriasis, Reiter's syndrome, and Behcet's disease. It is also associated with herpes infections, syphilis, Lyme disease, onchocerciasis, tuberculosis, and leprosy. Although anterior uveitis can occur in conjunction with many diseases, no cause is found to explain the majority of cases. For this reason, laboratory evaluation is usually reserved for patients with recurrent or severe anterior uveitis. Treatment is aimed at reducing inflammation and scarring by judicious use of topical glucocorticoids. Dilation of the pupil reduces pain and prevents the formation of synechiae. **Posterior Uveitis** This is diagnosed by observing inflammation of the vitreous, retina, or choroid on fundus examination. It is more likely than anterior uveitis to be associated with an identifiable systemic disease. Some patients have panuveitis, or inflammation of both the anterior and posterior segments of the eye. Posterior uveitis is a manifestation of autoimmune diseases such as sarcoidosis, Behcet's disease, Vogt-Koyanagi-Harada syndrome, and inflammatory bowel disease. It also accompanies diseases such as toxoplasmosis, onchocerciasis, cysticercosis, coccidioidomycosis, toxocariasis, and histoplasmosis; infections caused by organisms such as *Candida*, *Pneumocystis carinii*, *Cryptococcus*, *Aspergillus*, herpes, and cytomegalovirus; and other diseases such as syphilis, Lyme disease, tuberculosis, cat-scratch disease, Whipple's disease, and brucellosis. In multiple sclerosis, chronic inflammatory changes can develop in the extreme periphery of the retina (pars planitis or intermediate uveitis). **Acute Angle-Closure Glaucoma** This is a rare and frequently misdiagnosed cause of a red, painful eye. Susceptible eyes have a shallow anterior chamber, either because the eye has a short axial length (hyperopia) or a lens enlarged by the gradual development of cataract. When the pupil becomes mid-dilated, the peripheral iris blocks aqueous outflow via the anterior chamber angle and the intraocular pressure rises abruptly, producing pain, injection, corneal edema, obscurations, and blurred vision. In some patients, ocular symptoms are overshadowed by nausea, vomiting, or headache, prompting a fruitless workup for abdominal or neurologic disease. The diagnosis is made by measuring the intraocular pressure during an acute attack or by performing gonioscopy to reveal the narrowed chamber angle by means of a specially mirrored contact lens. Acute angle closure is treated with oral or intravenous acetazolamide, topical beta blockers, apraclonidine, and pilocarpine to induce miosis. If these measures fail, a laser can be used to create a hole in the peripheral iris to relieve pupillary block. Many physicians are reluctant to dilate patients routinely for fundus examination because they fear precipitating an angle-closure glaucoma. The risk is actually remote and more than outweighed by the potential benefit to patients of discovering a hidden fundus lesion visible only through a fully dilated pupil. Moreover, a single attack of angle closure after pharmacologic dilation rarely causes any permanent damage to the eye and serves as an inadvertent provocative test to identify patients with narrow angles who would benefit from prophylactic laser iridectomy. **Endophthalmitis** This occurs from bacterial, viral, fungal, or parasitic infection of the internal structures of the eye. It is usually acquired by hematogenous seeding from a remote site. Chronically ill, diabetic, or immunosuppressed patients, especially those with a history of indwelling intravenous catheters or positive blood cultures, are at greatest risk for endogenous endophthalmitis. Although most patients have ocular pain and injection, visual loss is sometimes the only symptom. Septic emboli, from a diseased heart valve or a dental abscess, that lodge in the retinal circulation can give rise to endophthalmitis. White-centered retinal hemorrhages (Roth's spots) are considered pathognomonic for subacute bacterial endocarditis, but they also appear in leukemia, diabetes, and many other conditions. Endophthalmitis also occurs as a complication of ocular surgery, occasionally months or even years after the operation. An occult penetrating foreign body or unrecognized trauma to the globe should be considered in any patient with unexplained intraocular infection or inflammation. [\[TOP\]](#) **TRANSIENT OR SUDDEN VISUAL LOSS** Amaurosis Fugax This term refers to a transient ischemic attack of the retina. Because neural tissue has a high rate of metabolism, interruption of blood flow to the retina for more than a few seconds results in transient monocular blindness, a term used interchangeably with amaurosis fugax. Patients describe a rapid fading of vision like a curtain descending, sometimes affecting only a portion of the visual field. Amaurosis fugax usually occurs from an embolus that becomes stuck within a retinal arteriole. If the embolus breaks

up or passes, flow is restored and vision returns quickly to normal without permanent damage. With prolonged interruption of blood flow, the inner retina suffers infarction. Ophthalmoscopy reveals zones of whitened, edematous retina following the distribution of branch retinal arterioles. Complete occlusion of the central retinal artery produces arrest of blood flow and a milky retina with a cherry-red fovea. Emboli are composed of either cholesterol (Hollenhorst plaque), calcium, or platelet-fibrin debris. The most common source is an atherosclerotic plaque in the carotid artery or aorta, although emboli can also arise from the heart, especially in patients with diseased valves, atrial fibrillation, or wall motion abnormalities. In rare instances, amaurosis fugax occurs from low central retinal artery perfusion pressure in a patient with a critical stenosis of the ipsilateral carotid artery and poor collateral flow via the circle of Willis. In this situation, amaurosis fugax develops when there is a dip in systemic blood pressure or a slight worsening of the carotid stenosis. Sometimes there is contralateral motor or sensory loss, indicating concomitant hemispheric cerebral ischemia. Retinal arterial occlusion also occurs rarely in association with retinal migraine, lupus erythematosus, anticardiolipin antibodies, anticoagulant deficiency states (protein S, protein C, and antithrombin III deficiency), pregnancy, intravenous drug abuse, blood dyscrasias, dysproteinemias, and temporal arteritis. Amaurosis fugax warns of a patient at high risk for stroke. The carotid arteries should be studied by ultrasound. Endarterectomy for a stenosis of $\geq 60\%$, even in asymptomatic patients, has been shown to reduce the subsequent rate of ipsilateral stroke. Therapy with aspirin, warfarin, or other anticoagulants is appropriate in selected patients. If no carotid lesion is found, cardiac ultrasound should be performed. Ambulatory electrocardiographic monitoring may reveal that intermittent atrial fibrillation is giving rise to emboli. Marked systemic hypertension causes sclerosis of retinal arterioles, splinter hemorrhages, focal infarcts of the nerve fiber layer (cotton-wool spots), and leakage of lipid and fluid (hard exudate) into the macula. In hypertensive crisis, sudden visual loss can result from vasospasm of retinal arterioles and consequent retinal ischemia. In addition, acute hypertension may produce visual loss from ischemic swelling of the optic disc. Patients with acute hypertensive retinopathy should be treated by lowering the blood pressure. However, the blood pressure should not be reduced precipitously, because there is a danger of optic disc infarction from sudden hypoperfusion. Impending branch or central retinal vein occlusion can produce prolonged visual obscurations that resemble those described by patients with amaurosis fugax. The veins appear engorged and phlebotic, with numerous retinal hemorrhages. In some patients, venous blood flow recovers spontaneously, while others evolve a frank obstruction with extensive retinal bleeding (blood and thunder appearance), infarction, and visual loss. Venous occlusion of the retina is often idiopathic, but hypertension, diabetes, and glaucoma are prominent risk factors. The benefit of treatment with anticoagulants is unproven and carries the risk of hemorrhage into the vitreous. Polycythemia, thrombocythemia, or other factors leading to an underlying hypercoagulable state should be corrected.

Anterior Ischemic Optic Neuropathy (AION) This is caused by insufficient blood flow through the posterior ciliary arteries supplying the optic disc. It produces sudden, painless, monocular visual loss, although patients occasionally report premonitory obscurations. The optic disc appears swollen and surrounded by nerve fiber layer splinter hemorrhages. AION is divided into two forms: arteritic and nonarteritic. The nonarteritic form of AION is most common. No specific cause can be identified, although diabetes and hypertension are frequent risk factors. No treatment is available. About 5% of patients, especially those over age 60, develop the arteritic form of AION in conjunction with giant cell (temporal) arteritis. It is urgent to recognize arteritic AION so that high doses of glucocorticoids can be instituted immediately to prevent blindness in the second eye. Symptoms of polymyalgia rheumatica may be present, and the sedimentation rate is usually elevated. In a patient with visual loss from suspected arteritic AION, temporal artery biopsy is helpful to confirm the diagnosis, but glucocorticoids should be started without waiting for the biopsy to be completed. The diagnosis of arteritic AION is difficult to sustain in the face of a normal sedimentation rate and a negative temporal artery biopsy, but such cases do occur rarely.

Posterior Ischemic Optic Neuropathy This is an infrequent cause of acute visual loss. It is induced by the combination of severe anemia and hypotension, causing infarction of the retrobulbar optic nerve. Cases have been reported after major blood loss during surgery, exsanguinating trauma, gastrointestinal bleeding, and renal dialysis. The fundus usually appears normal, although optic disc swelling develops if the process extends far enough anteriorly. Vision can be salvaged in some patients by prompt blood transfusion and reversal of hypotension.

Optic Neuritis This is a common inflammatory disease of the optic nerve. In the Optic Neuritis Treatment Trial (ONTT), the mean age of patients was 32 years, 77% were female, 92%

had ocular pain (especially with eye movements), and 35% had optic disc swelling. In most patients, the demyelinating event was retrobulbar and the ocular fundus appeared normal on initial examination, although optic disc pallor slowly developed over subsequent months. Virtually all patients experience a gradual recovery of vision after a single episode of optic neuritis, even without treatment. This rule is so reliable that failure of vision to improve considerably after a first attack of optic neuritis casts doubt upon the original diagnosis. Treatment of optic neuritis is controversial because the favorable prognosis for visual recovery has made it difficult to demonstrate any benefit from glucocorticoids. The ONTT showed that patients treated with a conventional dose of oral glucocorticoids (prednisone, 1 mg/kg per day for 14 days) did no better than patients treated with a placebo. A recent Danish trial of oral high-dose methylprednisolone (500 mg daily for 5 days, followed by a 10-day taper) reported a slight response at 1 and 3 weeks but none at 8 weeks. From these studies, it is apparent that oral glucocorticoids have little to offer in the treatment of optic neuritis. According to the ONTT, even high-dose intravenous methylprednisolone (250 mg every 6 h for 3 days) followed by oral prednisone (1 mg/kg per day for 11 days) makes no difference in final acuity (measured 6 months after the attack), although the recovery of visual function occurs more rapidly. For some patients, optic neuritis remains an isolated event. However, the ONTT showed that the 5-year cumulative probability of developing clinically definite multiple sclerosis following optic neuritis is 30%. Remarkably, intravenous glucocorticoids were associated with a reduced rate of development of multiple sclerosis over a 2-year follow-up period, especially in the subgroup of patients with multiple foci of demyelination on their magnetic resonance (MR) scan. However, by the end of a 3-year follow-up period, patients treated with intravenous glucocorticoids versus placebo showed no difference in the rate of multiple sclerosis. Moreover, intravenous glucocorticoids did not reduce the likelihood of subsequent attacks of optic neuritis. To summarize, the organizers of the ONTT recommend an MR scan in patients with optic neuritis. If two or more foci of demyelination are found or visual loss is severe, they suggest treatment with intravenous glucocorticoids.

The potential benefits of intravenous glucocorticoids are: (1) a slightly faster recovery of visual function, and (2) a potential reduction in the risk of subsequent neurologic events that would signify multiple sclerosis. Critics of the ONTT have questioned these recommendations, pointing out that: (1) visual outcome is the same in the long run, (2) evidence indicating a reduced risk of eventual multiple sclerosis with intravenous glucocorticoid treatment is based upon follow-up data in a rather small number of patients, and (3) the protection against multiple sclerosis is transient, and no longer apparent beyond 2 years of follow-up. In cases of unilateral optic neuritis, the decision whether to obtain an MR scan or to treat with intravenous glucocorticoids should be based upon clinical judgment and careful discussion with the patient. In cases of bilateral, simultaneous optic neuritis, the rationale for intravenous glucocorticoids is stronger.

Leber's Hereditary Optic Neuropathy This is a disease of young men, characterized by onset over a few weeks of painless, severe, central visual loss in one eye, followed weeks or months later by the same process in the other eye. Acutely, the optic disc appears mildly plethoric with surface capillary telangiectases, but no vascular leakage on fluorescein angiography. Eventually optic atrophy ensues. There is no treatment. Leber's optic neuropathy is caused by a point mutation at codon 11778 in the mitochondrial gene encoding nicotinamide adenine dinucleotide dehydrogenase (NADH) subunit 4. Subsequently, additional mutations responsible for the disease have been identified, most in mitochondrial genes encoding proteins involved in electron transport. Mitochondrial mutations causing Leber's neuropathy are inherited from the mother by all her children, but usually only sons develop symptoms. This curious male predilection is a mystery.

Toxic Optic Neuropathy This can result in acute visual loss with bilateral optic disc swelling and central or cecocentral scotomas. Such cases have been reported to result from exposure to ethambutol, methyl alcohol (moonshine), ethylene glycol (antifreeze), or carbon monoxide. In toxic optic neuropathy, visual loss can also develop gradually and produce optic atrophy without a phase of acute optic disc edema. Many agents have been implicated as a cause of toxic optic neuropathy, but the evidence supporting the association for many is weak. The following is a partial list of potential offending drugs or toxins: disulfiram, ethchlorvynol, chloramphenicol, amiodarone, monoclonal anti-CD3 antibody, ciprofloxacin, digitalis, streptomycin, lead, arsenic, thallium, D-penicillamine, isoniazid, emetine, and sulfonamides. Deficiency states, induced either by starvation, malabsorption, or alcoholism, can lead to insidious visual loss. Thiamine, vitamin B12, and folate levels should be checked in any patient with unexplained, bilateral central scotomas and optic pallor.

Papilledema This connotes bilateral optic disc swelling from raised intracranial pressure. Headache is a frequent, but not invariable,

accompaniment. All other forms of optic disc swelling, e.g., from optic neuritis or ischemic optic neuropathy, should be called optic disc edema. This convention is arbitrary but serves to avoid confusion. Often it is difficult to differentiate papilledema from other forms of optic disc edema by fundus examination alone. Transient visual obscurations are a classic symptom of papilledema. They can occur in only one eye or simultaneously in both eyes. They usually last seconds but can persist for minutes if the papilledema is fulminant. Obscurations follow abrupt shifts in posture or happen spontaneously. When obscurations are prolonged or spontaneous, the papilledema is more threatening. Visual acuity is not affected by papilledema unless the papilledema is severe, long-standing, or accompanied by macular edema and hemorrhage. Visual field testing shows enlarged blind spots and peripheral constriction. With unremitting papilledema, peripheral visual field loss progresses in an insidious fashion while the optic nerve develops atrophy. In this setting, reduction of optic disc swelling is an ominous sign of a dying nerve rather than an encouraging indication of resolving papilledema. Evaluation of papilledema requires computed tomography (CT) or MR imaging to exclude an intracranial lesion. MR angiography is appropriate in selected cases to search for a dural venous sinus occlusion or an arteriovenous shunt. If neuroradiologic studies are negative, the subarachnoid opening pressure should be measured by lumbar puncture. An elevated pressure, with normal cerebrospinal fluid, points by exclusion to the diagnosis of pseudotumor cerebri (idiopathic intracranial hypertension). The majority of patients are young, female, and obese. Treatment with a carbonic anhydrase inhibitor such as acetazolamide lowers intracranial pressure by reducing the production of cerebrospinal fluid. Weight reduction is vital but often unsuccessful. If acetazolamide and weight loss fail, and visual field loss is progressive, lumboperitoneal shunting or optic nerve sheath fenestration should be undertaken without delay to prevent blindness. Occasionally, emergency surgery is required for sudden blindness caused by fulminant papilledema.

Optic Disc Drusen These are refractile deposits within the substance of the optic nerve head. They are unrelated to drusen of the retina, which occur in age-related macular degeneration. Optic disc drusen are most common in people of northern European descent, with an incidence of 0.3 to 0.4%. Their diagnosis is obvious when they are visible as glittering particles upon the surface of the optic disc. However, in many patients they are hidden beneath the surface, producing an elevated optic disc with blurred margins that is easily mistaken for papilledema. It is important to recognize pseudo-papilledema due to optic disc drusen to avoid an unnecessary evaluation for papilledema. Ultrasound or CT scanning are sensitive for detection of buried optic disc drusen because they contain calcium. In most patients, optic disc drusen are an incidental, innocuous finding, but they can produce visual obscurations. On perimetry they give rise to enlarged blind spots and arcuate scotomas from damage to the optic disc. With increasing age, drusen tend to become more exposed on the disc surface as optic atrophy develops. Hemorrhage, choroidal neovascular membrane, and AION are more likely to occur in patients with optic disc drusen. No treatment for drusen is available.

Vitreous Degeneration This occurs in all individuals with advancing age, leading to chronic and acute visual symptoms. Opacities develop in the vitreous, casting annoying shadows upon the retina. As the eye moves, these distracting floaters move synchronously, with a slight lag caused by inertia of the vitreous gel. Vitreous traction upon the retina causes mechanical stimulation, resulting in perception of flashing lights. This photopsia is brief and confined to one eye, in contrast to the bilateral, prolonged scintillations of cortical migraine. Contraction of the vitreous can result in sudden separation from the retina, heralded by an alarming shower of floaters and photopsia. This process, known as vitreous detachment, is a frequent involitional event in the elderly. It is not harmful unless it damages the retina. A careful examination of the dilated fundus is mandatory in any patient complaining of floaters or photopsia to search for peripheral tears or holes. If such a lesion is found, laser application or cryotherapy can forestall a retinal detachment. Occasionally a tear ruptures a retinal blood vessel, causing vitreous hemorrhage and sudden loss of vision. On attempted ophthalmoscopy the fundus is hidden by a dark red haze of blood. Ultrasound is required to examine the interior of the eye for a retinal tear or detachment. If the hemorrhage does not resolve spontaneously, the vitreous can be removed surgically. Vitreous hemorrhage also occurs from the fragile neovascular vessels that proliferate on the surface of the retina in diabetes, sickle cell anemia, and other ischemic ocular diseases.

Retinal Detachment This produces symptoms of floaters, flashing lights, and a scotoma in the peripheral visual field corresponding to the detachment. If the detachment includes the fovea, there is an afferent pupil defect and the visual acuity is reduced. In most eyes, retinal detachment starts with a hole, flap, or tear in the peripheral retina

(rhegmatogenous retinal detachment). Patients with peripheral retinal thinning (lattice degeneration) are particularly vulnerable to this process. Once a break has developed in the retina, liquified vitreous is free to enter the subretinal space, separating the retina from the pigment epithelium. The combination of vitreous traction upon the retinal surface and passage of fluid behind the retina leads inexorably to detachment. Patients with a history of myopia, trauma, or prior cataract extraction are at greatest risk for retinal detachment. The diagnosis is confirmed by ophthalmoscopic examination of the dilated eye. **Classic Migraine** This usually occurs with a visual aura lasting about 20 min. In a typical attack, a small central disturbance in the field of vision marches toward the periphery, leaving a transient scotoma in its wake. The expanding border of migraine scotoma has a scintillating, dancing, or zig-zag edge, resembling the bastions of a fortified city, hence the term fortification spectra. Patients' descriptions of fortification spectra vary widely and can be confused with amaurosis fugax. Migraine patterns usually last longer and are perceived in both eyes, whereas amaurosis fugax is briefer and occurs in only one eye. Migraine phenomena also remain visible in the dark or with the eyes closed. Generally they are confined to either the right or left visual hemifield, but sometimes both fields are involved simultaneously. Patients often have a long history of stereotypic attacks. After the visual symptoms recede, headache develops in most patients. **Transient ischemic attacks** from vertebrobasilar insufficiency result in acute homonymous visual symptoms. Many patients mistakenly describe symptoms in their left or right eye, when in fact they are occurring in the left or right hemifield of both eyes. Interruption of blood supply to the visual cortex causes a sudden fogging or graying of vision, occasionally with flashing lights or other positive phenomena that mimic migraine. Cortical ischemic attacks are briefer in duration than migraine, occur in older patients, and are not followed by headache. There may be associated signs of brainstem ischemia, such as diplopia, vertigo, numbness, weakness, or dysarthria. **Stroke** This occurs when interruption of blood supply from the posterior cerebral artery to the visual cortex is prolonged. The only finding on examination is a homonymous visual field defect that stops abruptly at the vertical meridian. Occipital lobe stroke is usually due to thrombotic occlusion of the vertebrobasilar system, embolus, or dissection. Lobar hemorrhage, tumor, abscess, and arteriovenous malformation are other common causes of hemianopic cortical visual loss. **Factitious (Functional, Nonorganic) Visual Loss** This is claimed by hysterics or malingers. The latter comprise the vast majority, seeking sympathy, special treatment, or financial gain by feigning loss of sight. The diagnosis is suspected when the history is atypical, physical findings are lacking or contradictory, inconsistencies emerge on testing, and a secondary motive can be identified. In our litigious society, the fraudulent pursuit of recompense has spawned an epidemic of factitious visual loss. [\[TOP\]](#) **CHRONIC VISUAL LOSS** **Cataract** This is a clouding of the lens sufficient to reduce vision. Most cataracts develop slowly as a result of aging, leading to gradual impairment of vision. The formation of cataract occurs more rapidly in patients with a history of ocular trauma, uveitis, or diabetes mellitus. Cataracts are acquired in a variety of genetic diseases, such as myotonic dystrophy, neurofibromatosis type 2, and galactosemia. Radiation therapy and glucocorticoid treatment can induce cataract as a side effect. The cataracts associated with radiation or glucocorticoids have a typical posterior subcapsular location. Cataract can be detected by noting an impaired red reflex when viewing light reflected from the fundus with an ophthalmoscope or by examining the dilated eye using the slit lamp. The only treatment for cataract is surgical extraction of the opacified lens. Over a million cataract operations are performed each year in the United States. The operation is generally done under local anesthesia on an outpatient basis. Remarkable technical innovations have made it possible to aspirate the cataract while leaving the lens capsule intact (extracapsular cataract extraction), rather than removing the entire lens with its capsule (intracapsular cataract extraction). A plastic or silicone intraocular lens is then placed within the empty lens capsule in the posterior chamber, substituting for the natural lens, and leading to rapid recovery of sight. More than 95% of patients who undergo cataract extraction can expect an improvement in vision. In many patients, the lens capsule remaining in the eye after cataract extraction eventually turns cloudy, causing a secondary loss of vision. A small opening is made in the lens capsule with a laser to restore clarity. **Glaucoma** This is a slowly progressive, insidious optic neuropathy, usually associated with chronic elevation of intraocular pressure. In Americans of African descent it is the leading cause of blindness. The mechanism whereby raised intraocular pressure injures the optic nerve is not understood. Axons entering the inferotemporal and superotemporal aspects of the optic disc are damaged first, producing typical nerve fiber bundle or arcuate scotomas on perimetric testing. As fibers are destroyed, the neural rim of the optic disc shrinks and the physiologic cup within

the optic disc enlarges. This process is referred to colloquially as pathologic cupping. The cup-to-disc diameter is expressed as a ratio, e.g., 0.2/1. The cup-to-disc ratio ranges widely in normal individuals, making it difficult to diagnose glaucoma reliably simply by observing an unusually large or deep optic cup. Careful documentation of serial prospective examinations is helpful. In the patient with physiologic cupping, the large cup remains stable, whereas in the patient with glaucoma it expands relentlessly over the years. Detection of visual field loss on formal perimetry also contributes to the diagnosis of glaucoma. Finally, most patients with glaucoma have raised intraocular pressure. However, a surprising number of patients with typical glaucomatous cupping and visual field loss have intraocular pressures that apparently never exceed the normal limit of 20 mmHg (so-called low-tension glaucoma). In acute angle-closure glaucoma, the eye is red and painful due to abrupt, severe elevation of intraocular pressure. Such cases account for only a handful of patients with glaucoma. Most patients with glaucoma have open, nonoccludable anterior chamber angles. The cause of raised intraocular pressure in these patients is uncertain. Recent studies have implicated mutations in a gene encoding a glycoprotein expressed in the trabecular meshwork. This structure serves as a filter to drain aqueous from the eye. Because the elevation of intraocular pressure develops gradually and is less marked than in angle-closure glaucoma, there is no pain or ocular injection. The central visual field and foveal acuity are spared until end-stage disease is reached. For these reasons, severe and irreversible damage can occur before either the patient or physician recognizes the diagnosis. Screening of patients for glaucoma by noting the cup-to-disc ratio on ophthalmoscopy and by measuring intraocular pressure (using a Schiotz, To