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Eye disorders

Introduction

Vision, the most complex sense, has been the focus of significant medical and surgical innovations. Disorders that affect the eye generally lead to vision loss or impairment; routine ophthalmic examinations and early treatment can help prevent it.

Review of anatomy

The visual system consists mainly of the bony orbit, which houses the eye; the contents of the orbit, including the eyeball, optic nerves, extraocular muscles, cranial nerves, blood vessels, orbital fat, and lacrimal system; and the eyelid, which covers the eye, moistens it, and protects it from injury.

The orbit (also called the *socket*) encloses the eye in a protective recess in the skull. Its seven bones—frontal, sphenoid, zygomatic, maxillary, palatine, ethmoid, and lacrimal—form a cone. The apex of this cone points toward the brain, and the cone's base forms the orbital rim. The periorbita covers the bones of the orbit.

Extraocular muscles hold the eyes in place and control their movement as described below:

- *superior rectus:* elevates the eye upward; adducts and rotates the eye inward
- *inferior rectus:* depresses the eye downward; adducts and rotates the eye outward

- *lateral rectus:* abducts or turns the eye outward (laterally)
- *medial rectus:* adducts or turns the eye inward (medially)
- *superior oblique:* rotates the eye inward; abducts and depresses the eye
- *inferior oblique:* rotates the eye outward; abducts and elevates the eye.

The actions of these muscles are mutually antagonistic: As one contracts, its opposing muscle relaxes.

ELDER TIP

Eye structure and activity change with age. The eyes set deeper in their sockets or have laid down more fat, and the eyelids lose their elasticity and become saggy and wrinkled.

Ocular layers

The eye has three structural layers: the sclera and cornea, the uveal tract, and the retina. (See *Cross section of the eye*.)

The sclera is the dense, white, fibrous outer protective coat of the eye. It meets the cornea at the limbus (corneoscleral junction) anteriorly, and the dural sheath of the optic nerve posteriorly. The lamina cribrosa is a sievelike structure composed of a few strands of scleral tissue through which the optic nerve bundles pass. The sclera is covered by the episclera, a thin layer of fine elastic tissue.

ELDER TIP

In older adults, lens changes occur typically with formation of a cataract. The vitreous body liquefies and pulls away from the retina, generating floating vitreous debris and peripheral vitreous detachments.

The cornea is the transparent, avascular, curved layer of the eye that's continuous with the sclera. The cornea consists of five layers: the epithelium, which contains sensory nerves; Bowman's membrane, the basement membrane for the epithelial cells; the stroma, or supporting

tissue (90% of the corneal structure); Descemet's membrane, containing many elastic fibers; and the endothelium, a single layer of cells that acts as a pump to maintain proper dehydration or detumescence of the cornea. Aqueous humor bathes the posterior surface of the cornea, maintaining intraocular pressure (IOP) by volume and rate of outflow. The anterior cornea is kept moist by the tear film. The cornea's sole function is to refract light rays.

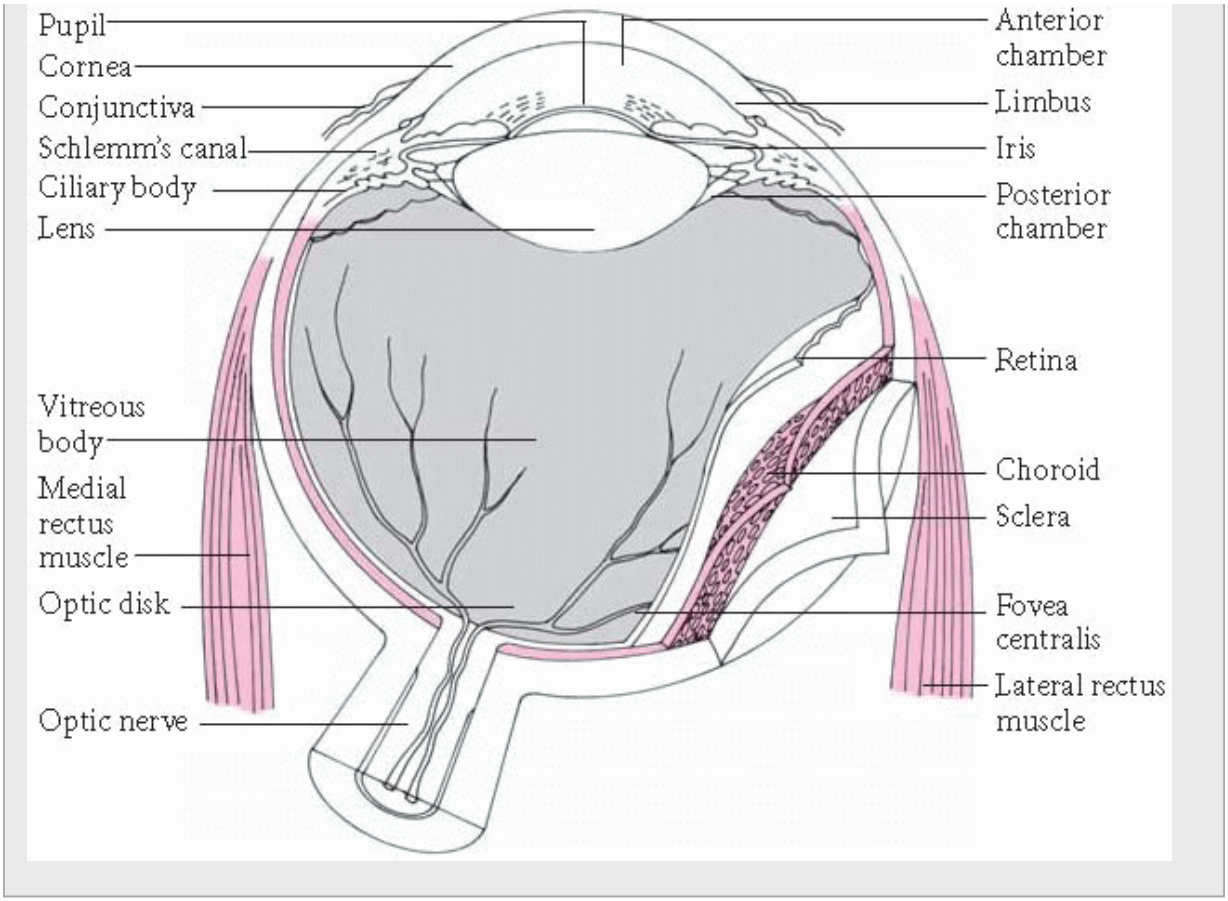
ELDER TIP

Corneal sensitivity to touch decreases with age, and corneal curvature probably changes slightly also. It's believed that atrophy of the dilator muscle fibers and increased rigidity of the blood vessels of the iris reduce pupil size, decreasing the amount of light that reaches the retina. Consequently, higher levels of illumination may be needed to improve uncorrected visual acuity in the older adult.

The middle layer of the eye, the uveal tract, is pigmented and vascular. It consists of the iris and the ciliary body in the anterior portion, and the choroid in the posterior

portion. In the center of the iris is the pupil. The sphincter and dilator muscles control the amount of light that enters the eye through the pupil, and the pupil itself allows aqueous humor to flow from the posterior chamber into the anterior chamber.

CROSS SECTION OF THE EYE



The angle formed by the anterior iris and the posterior corneal structures contains many minute collecting channels of the trabecular meshwork. Aqueous humor drains through these channels into an encircling venous system called the canal of Schlemm.

The ciliary body, which extends from the root of the iris to the ora serrata, produces aqueous humor and controls lens accommodation through its action on the zonular fibers. The choroid, the largest part of the uveal tract, is made up of blood vessels bound externally by the suprachoroid and internally by Bruch's membrane.

The retina is the neural coat of the eye. It receives visual images and transmits them to the brain for interpretation. It extends from the ora serrata to the optic nerve; the retinal pigment epithelium (RPE) adheres lightly to the choroid. Located next to the RPE are rods and cones. Although both rods and cones are light receptors, they respond to light differently. Rods, scattered throughout the retina, respond to low levels of light and detect moving objects; cones, located in the fovea centralis, function best in brighter light and perceive finer details.

Three types of cones contain different visual pigments and react to specific light wavelengths: one type reacts to red light, one to green, and one to blue-violet. The eye mixes these colors into various shades; the cones can detect 150 shades.

ELDER TIP

Many elderly patients lose their ability to discriminate blue-greens, and white objects appear yellowish; these patients may also have difficulty discriminating among pastels, violets, and yellow-greens.

The lens and accommodation

The lens of the eye is biconvex, avascular, and transparent; the lens capsule is a semi-permeable membrane that can admit water and electrolytes. The lens changes shape (accommodation) for near and far vision. For near vision, the ciliary body contracts and relaxes the zonules, the lens becomes spherical, the pupil constricts, and the eyes converge; for far vision, the ciliary body relaxes, the zonules tighten, the lens becomes flatter, the eyes straighten, and the pupils dilate. The lens refines the refraction necessary to focus a clear image on the retina.

The vitreous body, which is 99% water and a small amount of insoluble protein, constitutes two-thirds of the eye's volume. This transparent, gelatinous body gives the eye its shape and contributes to the refraction of light rays. The vitreous is firmly attached to the ora serrata of the ciliary body (anteriorly) and to the optic disk (posteriorly). The vitreous face contacts the lens; the vitreous gel rests against the retina.

Lacrimal apparatus and eyelids

The lacrimal apparatus consists of the lacrimal glands, upper and lower canaliculi, lacrimal sac, and nasolacrimal duct. The main gland, located in a shallow fossa beneath the superior temporal orbital rim, secretes reflex tears, which keep the cornea and conjunctiva moist. These tears flow through 12 to 14 excretory ducts and contain lysozyme, an enzyme that protects the conjunctiva from bacterial invasion. Multiple

sebaceous glands in the eyelids produce an oily secretion that prevents tears from evaporating. With every blink, the eyelids direct the flow to the inner canthus, where the tears pool and then drain through a tiny opening called the punctum. The tears then pass through the canaliculi and lacrimal sac and down the nasolacrimal duct, which opens into the nasal cavity.

The eyelids (palpebrae) consist of tarsal plates that are composed of dense connective tissue. The orbital septum—the fascia behind the orbicularis oculi muscle—acts as a barrier between the lids and the orbit. The levator palpebrae muscle elevates the upper lid. The eyelids contain three types of glands:

- glands of Zeis—modified sebaceous glands connected to the follicles of the eyelashes
- meibomian glands—sebaceous glands in the tarsal plates that secrete an oily substance as a tear film component (About 25 of these glands are found in the upper lid and about 20 in the lower lid.)
- Moll's glands—ordinary sweat glands.

The conjunctiva is the thin mucous membrane that lines the eyelids (palpebral conjunctiva), folds over at the fornix, and covers the surface of the eyeball (bulbar conjunctiva). The conjunctiva produces mucin, another component of the tear film. The ophthalmic, lacrimal, and multiple anastomoses of facial arteries supply blood to the lids. The space between the open lids is the palpebral fissure; the juncture of the upper and lower lids is the canthus. The junction near the nose is called the nasal, medial, or inner canthus; the junction on the temporal side, the lateral or external canthus.

ELDER TIP

Age-related vision changes are usually first noticed during the fifth decade of life and may include the inability to focus, narrowing of the visual field, reduced peripheral vision, and loss of iris elasticity producing decreased response to light and dark. In addition, as people age, production of any of the three tear film components may decrease, causing dry eyes.

Depth perception

In normal binocular vision, a perceived image is projected onto the two foveae. Impulses then travel along the optic pathways to the occipital cortex, which perceives a single image. However, the cortex receives two images—each from a slightly different angle—giving the images perspective and providing depth perception.

Vision testing

Several tests assess visual acuity and identify visual defects:

- Ishihara's test determines color blindness by using a series of plates composed of a colored background, with a letter, number, or pattern of a contrasting color located in the center of each plate. The patient with deficient color perception can't perceive

  the differences in color or, consequently, the designs formed by the color contrasts.
- The Snellen chart or other eye charts evaluate visual acuity. Such charts use progressively smaller letters or symbols to determine central vision on a numerical scale. A person with normal acuity should be able to read the letters or recognize the symbols on the 20/20 line of the eye chart at a distance of 20 feet.

Subjective testing

Several tests accomplish subjective testing of the eyes.

- B-mode ultrasonography delineates retinal tumors, detachments, and vitreous hemorrhages—even in the presence of opacities of the cornea and lens. A hand-held B-scanner has simplified ultrasonic examination of the eye, making it possible to perform such studies in the ophthalmologist's office.
- The convergence test locates the breaking point of fusion (before double vision occurs). For this test, the examiner holds a small object in front of the patient's nose and slowly brings it closer to the patient. The point at which the eyes “break” is termed the near point of convergence and is measured in prism diopters.

- The cover-uncover test assesses eye muscle misalignment or tendency toward misalignment. In this test, the patient stares at a small, fixed object—first from a distance of 20′(6.1 m) and then from 1′ (0.3 m). The examiner covers the patient's eyes one at a time, noting any movement of the uncovered eye, the direction of any deviation, and the rate at which the eyes recover normal binocular vision when latent heterophoria is present.
- Duction test checks eye movement in all directions of gaze. While one eye is covered, the other eye follows a moving light. This test detects weakness of rotation due to muscle paralysis or structural dysfunction.
- Fluorescein angiography evaluates the blood vessels in the choroid and retina after I.V. injection of fluorescein dye; images of the dye-enhanced vasculature are recorded by rapid-sequence photographs of the fundus.
- Goldmann's applanation, Tonopen tonometry, and Schiøtz tonometry all measure IOP. After instilling a local anesthetic in the patient's eye, the examiner places the Schiøtz tonometer lightly on the corneal surface and measures the indentation of the cornea produced by a given weight. The Schiøtz tonometer has been largely replaced by an electronic Tonopen tonometer for bedside use. Applanation tonometry gauges the force required to flatten a small area of central cornea, and is the most accurate method of measuring IOP. For this test, a patient must be seated at a slit lamp and the cornea stained with fluorescein dye before the prism of the applanation tonometer touches the cornea and the examiner adjusts the controls until the two lines form an “S.”
- Gonioscopy allows for direct visualization of the anterior chamber angle.
- The Maddox rod test assesses muscle dysfunction; it's especially useful in disclosing and measuring heterophoria (the tendency of the eyes to deviate). It can reveal horizontal, vertical and, especially, torsional deviations.
- Ophthalmodynamometry measures the relative central retinal artery pressures and indirectly assesses carotid artery flow on each side. This

test has been largely supplanted by the color Doppler imaging test, which can assess blood flow velocities in ophthalmic vessels.

- Ophthalmoscopy—direct ophthalmoscopy or binocular indirect ophthalmoscopy allows examination of the interior of the eye after the pupil has been dilated with a mydriatic.
- Refraction tests may be performed with or without cycloplegics. In cycloplegic refraction, eyedrops weaken the accommodative power of the ciliary muscle. Lenses placed in front of the eye direct light rays onto the retina, thus focusing the image so that it can be transmitted along the visual pathway. A retinoscope may be used in the same way by directing a beam of light through the pupil onto the retina; the light's shadow is neutralized by placing the appropriate lens in front of the eye.
- Slit-lamp biomicroscopic examination allows a well-illuminated examination of

the eyelids and the anterior segment of the eyeball.

- Visual field tests assess the function of the retina, the optic nerve, and the optic pathways when both central and peripheral visual fields are examined.

EYELID AND LACRIMAL DUCTS

Blepharitis

A common inflammation, blepharitis produces a red-rimmed appearance of the margins of the eyelids. It's frequently chronic and bilateral and can affect both upper and lower lids. Seborrheic blepharitis is characterized by formation of waxy scales and symptoms of burning and foreign-body sensation. Staphylococcal (ulcerative) blepharitis is characterized by formation of dry scales along the inflamed lid margins, which also have ulcerated areas and may be associated with keratoconjunctivitis sicca (KCS), a dry-eye syndrome. Both types may coexist. Blepharitis tends to recur and become chronic. It can be controlled if treatment begins before onset of ocular involvement.

Causes and incidence

Seborrheic blepharitis may be seen in conjunction with seborrhea of the scalp, eyebrows, and ears. It's common in elderly people and in people with red hair. Staphylococcal blepharitis is associated with *Staphylococcus aureus* infection and is more common in females than in males. Allergies and eyelash infestations with lice are less-common causes of blepharitis. Blepharitis may also be associated with repeated styes and chalazion.

Complications

- Ocular involvement
- Keratitis
- Conjunctivitis

Signs and symptoms

Clinical features of blepharitis include itching, burning, foreign-body sensation, and sticky, crusted eyelids on waking. This constant irritation results in unconscious rubbing of the eyes (causing reddened rims) or continual blinking. Other signs include waxy scales in seborrheic blepharitis; and flaky scales on lashes, loss of lashes, and ulcerated areas on lid margins in ulcerative blepharitis. In association with KCS, dry eyes may also be a problem.

Diagnosis

Diagnosis depends on patient history and characteristic symptoms. In staphylococcal blepharitis, culture of ulcerated lid margin shows *S. aureus*.

Treatment

The goals of therapy are to control the disease and its underlying causes, maintain vision, and avoid secondary complications. Treatment depends on the type of blepharitis:

- blepharitis resulting from pediculosis— removal of nits (with forceps) or application of ophthalmic physostigmine or other ointment as an insecticide (This may cause pupil constriction and, possibly, headache, conjunctival irritation, and blurred vision from the film of ointment on the cornea.)
- seborrheic blepharitis—daily lid hygiene (using a mild shampoo on a damp applicator stick or a washcloth) and hot compresses to remove scales from the lid margins; also, frequent shampooing of the scalp and eyebrows
- staphylococcal blepharitis—warm compresses and an antibiotic, such as tetracycline or erythromycin eye ointment, may be used. For some patients, systemic antibiotics are indicated. Patients with severe staphylococcal blepharitis may require antibiotic sensitivity studies.

Special considerations

- Instruct the patient to gently remove scales from the lid margins daily, with an applicator stick or a clean washcloth.
- Teach the patient the following method for applying warm compresses: First, run warm water into a clean bowl. Then, immerse a clean cloth in the water and wring it out. Place the warm cloth against the closed eyelid (be careful not to burn the skin). Hold the compress in place until it cools. Continue this procedure for 15 minutes.
- Antibiotic ophthalmic ointment should be applied after 15-minute application of warm compresses.
- Treatment for seborrheic blepharitis also requires attention to the face and scalp.

RECOGNIZING EXOPHTHALMOS

This photo shows the characteristic forward protrusion of the eyes from the orbit associated with exophthalmos.

Exophthalmos

Exophthalmos (also called *proptosis*) is the unilateral or bilateral bulging or protrusion of the eyeballs or their apparent forward displacement (with lid retraction). The prognosis depends on the underlying cause.

Causes and incidence

Exophthalmos commonly results from hyperthyroidism, particularly ophthalmic Graves' disease in which the eyeballs are displaced forward and the lids retract. Unilateral exophthalmos may also result from trauma (such as fracture of the ethmoid bone, which allows air from the sinus to enter the orbital tissue, displacing soft tissue and the eyeball). Exophthalmos may also stem from hemorrhage, varicosities, thrombosis, and edema, all of which similarly displace one or both eyeballs.

Other systemic and ocular causes include:

- infection—orbital cellulitis, panophthalmitis, and infection of the lacrimal gland or orbital tissues
- parasitic cysts—in surrounding tissue
- pseudoexophthalmos paralysis of extraocular muscles—relaxation of eyeball retractors, congenital macrophthalmia, and high myopia
- tumors and neoplastic diseases—in children, rhabdomyosarcomas, leukemia, gliomas of the optic nerve, dermoid cysts, teratomas, metastatic neuroblastomas, and lymphoma; in adults, lacrimal gland tumors, mucoceles, cavernous hemangioma, meningiomas, metastatic carcinomas, and lymphoma.

Signs and symptoms

The obvious effect is a bulging eyeball, commonly with diplopia, if extraocular muscle edema causes misalignment. (See *Recognizing exophthalmos*.) A rim of the sclera may be visible below the upper lid as lid retraction occurs, and the patient may blink infrequently. Other symptoms depend on the cause: pain may accompany traumatic exophthalmos; a tumor may produce conjunctival hyperemia or chemosis; retraction of the upper lid predisposes to exposure keratitis. If exophthalmos is associated with cavernous sinus thrombosis, the patient may exhibit paresis of the muscles supplied by cranial nerves III, IV, and VI; limited ocular movement; and a septictype (high) fever.

Diagnosis

Exophthalmos is usually obvious on physical examination; exophthalmometer readings confirm diagnosis by showing the degree of anterior projection and asymmetry between the eyes (normal bar readings range from 12 to 20 mm). The following diagnostic measures identify the cause:

- Computed tomography scan or magnetic resonance imaging detects swollen extraocular muscles or lesions within the orbit.
- Culture of discharge determines the infecting organism; sensitivity testing indicates appropriate antibiotic therapy.
- Biopsy of orbital tissue may be necessary if initial treatment fails.

Treatment

Eye trauma may require cold compresses for the first 24 hours, followed by warm compresses, and prophylactic antibiotic therapy. After edema subsides, surgery may be necessary in a small percentage of cases. Eye infection requires treatment with broad-spectrum antibiotics during the 24 hours preceding positive identification of the organism, followed by specific antibiotics. A patient with exophthalmos resulting from an orbital tumor may initially benefit from antibiotic or corticosteroid therapy. Eventually, surgical exploration of the orbit and excision of the

tumor, enucleation, or exenteration may be necessary. Radiation and chemotherapy may be used when primary orbital tumors can't be fully excised as encapsulated lesions, such as in rhabdomyosarcoma lesions.

Treatment for Graves' disease may include antithyroid drug therapy or partial or total thyroidectomy to control hyperthyroidism; initial high doses of systemic corticosteroids, such as prednisone, for optic neuropathy and, if lid retraction is severe, protective lubricants.

Surgery may include orbital decompression (removal of the superior and lateral orbital walls) if vision is threatened, followed by lid (blepharoplasty) and muscle surgery.

Special considerations

- Administer medication as ordered.
- Record the patient's response to therapy.
- Apply cold and warm compresses, as ordered, for fracture or other trauma.
- Provide postoperative care.
- Explain tests and procedures and give emotional support.
- Protect the exposed cornea with lubricants to prevent corneal drying.

Ptosis

Ptosis (drooping of the upper eyelid) may be congenital or acquired, unilateral or bilateral, and constant or intermittent. Severe ptosis usually responds well to treatment; slight ptosis may require no treatment at all.

Causes and incidence

Congenital ptosis is transmitted as an autosomal dominant trait or results from a congenital anomaly in which the levator muscles of the eyelids fail to develop. This condition is usually unilateral.

Acquired ptosis may result from any of the following:

- advanced age (involutional ptosis, the most common form, usually seen in older patients following cataract surgery)
- mechanical factors that make the eyelid heavy, such as swelling caused by a foreign body on the palpebral surface of the eyelid or by edema, inflammation produced by a tumor or pseudotumor, or an extra fatty fold
- myogenic factors, such as muscular dystrophy or myasthenia gravis (in which the defect appears to be in humoral transmission at the myoneural junction)
- neurogenic (paralytic) factors from interference in innervation of the eyelid by the oculomotor nerve (cranial nerve III), most commonly due to trauma, diabetes, or carotid aneurysm
- nutritional factors, such as thiamine deficiency in chronic alcoholism, hyperemesis gravidarum, and other malnutritionproducing states.

Risk factors for ptosis include aging, diabetes, stroke, Horner's syndrome, myasthenia gravis, and cancer that affects nerve or muscle response.

Complication

- Lazy eye in children

Signs and symptoms

PEDIATRIC TIP

An infant with congenital ptosis has a smooth, flat upper eyelid, without the eyelid fold normally caused by the pull of the levator muscle; associated weakness of the superior rectus muscle isn't uncommon.

The child with unilateral ptosis that covers the pupil can develop an amblyopic eye from disuse or lack of eye stimulation. In bilateral ptosis, the child may elevate his brow in an attempt to compensate, wrinkling his

forehead in an effort to raise the upper lid. Also, the child may tilt his head backward to see.

In myasthenia gravis, ptosis results from fatigue and characteristically appears in the evening, but is relieved by rest.

Ptosis due to oculomotor nerve damage produces a fixed, dilated pupil; divergent strabismus; and slight depression of the eyeball.

Diagnosis

A physical examination reveals upper lid retraction, and examination with the Hertel exophthalmometer reveals the degree of proptosis. Diagnosis also includes these tests to determine the underlying cause:

- digital subtraction angiography or magnetic resonance imaging—aneurysm
- glucose tolerance test—diabetes
- ophthalmologic examination—foreign bodies
- patient history—chronic alcoholism
- Tensilon test—myasthenia gravis (in acquired ptosis with no history of trauma).

Treatment

Slight ptosis that doesn't produce deformity or loss of vision requires no treatment. Severe ptosis that interferes with vision or is cosmetically undesirable usually necessitates resection of the weak levator muscles. Surgery to correct congenital ptosis is usually performed at age 3 or 4, but it may be done earlier if ptosis is unilateral because the completely occluded pupil may cause an amblyopic eye. If surgery is undesirable, special glasses with an attached suspended crutch on the frames may elevate the eyelid.

Effective treatment for ptosis also requires treatment for the underlying cause. For example, in patients with myasthenia gravis, neostigmine or

steroids may be prescribed to increase the effect of acetylcholine and aid transmission of nerve impulses to muscles.

Special considerations

- Report any bleeding immediately. After surgery to correct ptosis, watch for blood on the pressure patch. (Some surgical procedures may not require a patch.) Apply ointment to the sutures as prescribed.
- Emphasize to the patient and his family the need to prevent accidental trauma to the surgical site until healing is complete (6 weeks). Suture line damage can precipitate recurrence of ptosis.

Orbital cellulitis

Orbital cellulitis is an acute infection of the orbital tissues and eyelids that doesn't involve the eyeball. With treatment, the prognosis is good; if untreated, the infection may spread to the cavernous sinus or the meninges, where it can be life-threatening.

Causes and incidence

Orbital cellulitis may result from bacterial, fungal, or parasitic infection. It can develop from direct inoculation, via the bloodstream, or spread from adjacent structures. Periorbital tissues may be inoculated as a result of surgery, foreign body trauma, and even animal or insect bites.

PEDIATRIC TIP

The most common pathogens in children are Haemophilus influenzae, Streptococcus pneumoniae, *and* Staphylococcus aureus. *In young children, it's spread from adjacent sinuses (especially the ethmoid air cells) and accounts for the majority of postseptal cellulitis cases.*

Immunosuppressed patients are also susceptible. The incidence has decreased because of the use of Hib vaccine.

Complications

- Cavernous sinus thrombosis
- Hearing loss
- Septicemia
- Meningitis

- Optic nerve damage

Signs and symptoms

Orbital cellulitis generally produces unilateral eyelid edema, hyperemia of the orbital tissues, reddened eyelids, and matted lashes. Although the eyeball is initially unaffected, proptosis develops later (because of edematous tissues within the bony confines of the orbit). Other indications include extreme orbital pain, impaired eye movement, chemosis, and purulent discharge from indurated areas. The severity of associated systemic symptoms (chills, fever, and malaise) varies according to the cause.

Complications include posterior extension, causing cavernous sinus thrombosis, panophthalmitis, meningitis, or brain abscess and, rarely, atrophy and subsequent loss of vision secondary to optic neuritis.

Diagnosis

Typical clinical features establish diagnosis. Computed tomography scan or magnetic resonance imaging of the sinuses and orbit tissues will determine if the cause of the cellulitis is preseptal or if deeper structures are involved, or if a tumor is the cause of swelling. Usually the patient will also be febrile with this type of infection. Wound culture and sensitivity testing determine the causative organism and specific antibiotic therapy. Other tests include white blood cell count, and ophthalmologic examination.

Treatment

Prompt treatment is necessary to prevent complications. Primary treatment consists of antibiotic therapy. Systemic antibiotics (I.V. or oral) and eyedrops or ointment will be ordered. Supportive therapy

consists of fluids; warm, moist compresses; and bed rest. The patient should be monitored closely. If during the initial 48 to 72 hours of treatment no improvement is seen, adjustment of antibiotics guided by drug sensitivity should be considered. If an orbital abscess is present, surgical incision and drainage may be necessary.

Special considerations

- Monitor vital signs at least every 4 hours, and maintain fluid and electrolyte balance.
- Have the patient instill antibiotic eyedrops frequently during the day and apply ointment at night.
- Apply compresses every 3 to 4 hours to localize inflammation and relieve discomfort. Teach the patient to apply these compresses. Give pain medication, as ordered, after assessing pain level.
- Before discharge, stress the importance of completing prescribed antibiotic therapy. To prevent orbital cellulitis, tell the patient to maintain good general hygiene and to carefully clean abrasions and cuts that occur near the orbit.

PREVENTION

- *Use Hib vaccination to prevent* Hemophilus *infection in children.*
- *Administer rifampin (Rifadin) to children in same household.*
- *Treat sinus and dental infections early to decrease spread to the eye.*

Dacryocystitis

Dacryocystitis is an infection of the lacrimal sac. In infants, dacryocystitis results from congenital atresia of the nasolacrimal duct; in adults, it results from an obstruction (dacryostenosis) of the nasolacrimal duct (most common in women older than age 40). It can be acute, chronic, or congenital.

Causes and incidence

Atresia of the nasolacrimal ducts results from failure of canalization or, in the first few months of life, from blockage when the membrane that separates the lower part of the nasolacrimal duct and the inferior nasal meatus fails to open spontaneously before tear secretion. Bony obstruction of the duct may also occur.

In acute dacryocystitis, *Staphylococcus aureus* and, occasionally, beta-hemolytic streptococci are the cause. In chronic dacryocystitis, *Streptococcus pneumoniae* or, sometimes, a fungus—such as *Actinomyces* or *Candida albicans*—is the causative organism.

Primary lumps and secondary tumors from sinuses, nose, and orbits have also been reported as causes.

Complication

- Orbital cellulitis

Signs and symptoms

Dacryocystitis is extremely painful for the patient. The hallmark of both the acute and chronic forms of dacryocystitis is constant tearing. Other symptoms of dacryocystitis include inflammation and tenderness over the nasolacrimal sac; pressure over this area may fail to produce purulent discharge from the punctum.

Diagnosis

Clinical features and a physical examination suggest dacryocystitis. Culture of the discharged material demonstrates *Staphylococcus aureus* and, occasionally, beta-hemolytic streptococci in acute dacryocystitis, and *Streptococcus pneumoniae* or *C. albicans* in the chronic form. The white blood cell count may be elevated in the acute form; in the chronic form, it's generally normal. An X-ray after injection of a radiopaque medium (dacryocystography) locates the atresia in infants.

Treatment

Treatment for acute dacryocystitis consists of warm compresses, topical and systemic antibiotic therapy and, occasionally, incision and drainage. Chronic dacryocystitis may eventually require dacryocystorhinostomy. Laser-assisted endoscopic dacryocystorhinostomy and balloon dilatation or probing of the nasolacrimal system may also be used.

Therapy for nasolacrimal duct obstruction in an infant consists of careful massage of the area over the lacrimal sac four times a day for 6 to 9 months. If this fails to open the duct, dilation of the punctum and probing of the duct are necessary.

Special considerations

- Check the patient history for possible allergy to antibiotics before administration. Emphasize the importance of precise compliance with the prescribed antibiotic regimen.
- Tell the adult patient what to expect after surgery. He'll have ice compresses over the surgical site and will have bruising and swelling.
- Monitor blood loss by counting dressings used to collect the blood.
- Apply ice compresses postoperatively. A small adhesive bandage may be placed over the suture line to protect it from damage.

Chalazion

A chalazion is a chronic granulomatous inflammation of a meibomian gland or gland of Zeis in the upper or lower eyelid. (There are approximately 100 of these glands located near the eyelashes.) This common eye disorder is characterized by localized swelling within the tarsal plate, or it may break through the conjunctival or skin side. Mild irritation and blurred vision usually develop slowly over several weeks. (See *Recognizing chalazion*, page 662.) A chalazion may become large enough to press on the eyeball, causing astigmatism. A large chalazion seldom subsides spontaneously. It's generally benign and chronic, and can occur at any age. In some patients, it's apt to recur.

Causes and incidence

Obstruction of the meibomian (sebaceous) gland duct causes a chalazion.

Complication

- Astigmatism

Signs and symptoms

A chalazion occurs as a painless, hard lump that usually points *toward* the conjunctival side of the eyelid. Eversion of the lid reveals a red or red-yellow elevated area on the conjunctival surface. Otherwise, it's seen as an indurated bump under the skin of the upper eyelid.

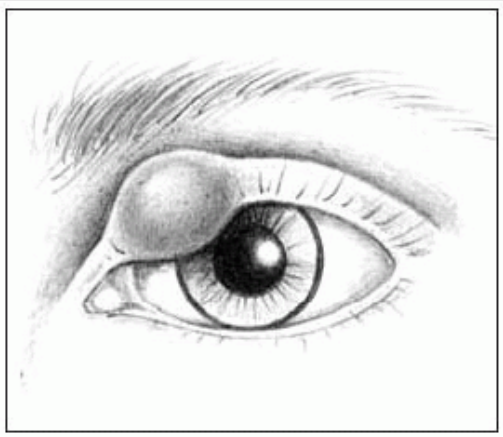
Diagnosis

Diagnosis requires visual examination and palpation of the eyelid, revealing a small bump or nodule. Persistently recurrent

chalazions, especially in an adult, necessitate biopsy to rule out meibomian cancer.

RECOGNIZING CHALAZION

A chalazion is a nontender granulomatous inflammation of a meibomian gland on the upper or lower eyelid.



Treatment

Initial treatment consists of application of warm compresses for 10 to 15 minutes at least four times a day to open the lumen of the gland, soften the hardened oils blocking the duct, and promote drainage and healing. If such therapy fails, or if the chalazion presses on the eyeball or causes a severe cosmetic problem, steroid injection or incision and curettage under local anesthetic may be necessary. After such surgery, a pressure eye patch applied for 4 to 6 hours controls bleeding and swelling. After removal of the patch, treatment again consists of warm compresses. Antibiotic eyedrops are occasionally prescribed before and after cyst removal, but otherwise are of little value.

Special considerations

- Instruct the patient how to properly apply warm compresses: Tell him to take special care to avoid burning the skin, to always use a clean cloth, and to discard used compresses. Also tell him to start applying warm compresses at the first sign of lid irritation to increase the blood supply and keep the lumen open.
- Teach the patient how to instill antibiotic eyedrops.

Stye

A localized, purulent staphylococcal infection, a stye (or hordeolum) can occur externally (in the lumen of the smaller glands of Zeis or in Moll's glands) or internally (in the larger meibomian gland). A stye can occur at any age. Generally, styes are self-limiting and respond well to hot, moist compresses. More than one may occur at the same time. If untreated, a stye can eventually lead to cellulitis of the eyelid. Styes can also develop into a chalazion if gland ducts are fully blocked.

Causes and incidence

Skin bacteria that enter eyelash hair follicles and cause inflammation can result in stye formation. Risk factors include blepharitis, diabetes and other chronic debilitating illnesses, and seborrhea.

Complication

- Cellulitis of the eyelid

Signs and symptoms

Typically, a stye produces redness, swelling, and pain. An abscess frequently forms at the lid margin, with an eyelash pointing outward from its center. (See *Recognizing a stye*.)

Diagnosis

Visual examination generally confirms this infection. Culture of purulent material from the abscess usually reveals a staphylococcal organism.

Treatment

Treatment consists of warm compresses applied for 10 to 15 minutes, four times a day for 3 to 4 days, to facilitate drainage of the abscess, to relieve pain and inflammation, and to promote suppuration. Drug therapy includes a topical sulfonamide or antibiotic eyedrops or ointment and, occasionally, a systemic antibiotic for secondary eyelid cellulitis. If conservative treatment fails, incision and drainage may be necessary.

Special considerations

- Instruct the patient to use a clean cloth for each application of warm compresses and to dispose of it or launder it separately.
- Warn against squeezing the stye; this spreads the infection and may cause cellulitis.
- Teach the patient or his family members the proper technique for instilling eyedrops or ointments into the cul-de-sac of the lower eyelid.

PREVENTION

Teach proper eye hygiene, such as washing hands and using clean towels, to prevent recurrent infections.

CONJUNCTIVAL DISORDERS

Inclusion conjunctivitis

Inclusion conjunctivitis (also called *inclusion blennorrhea*) is an acute ocular inflammation resulting from infection by *Chlamydia trachomatis*. Although inclusion conjunctivitis occasionally becomes chronic, the prognosis is usually good.

Causes and incidence

C. trachomatis is an obligate intracellular organism of the lymphogranuloma venereum serotype group. Serotypes D through K are sexually transmitted, and secondary eye involvement in adults occurs in about 1 in 300 genital cases. Because contaminated cervical secretions infect the eyes of the neonate during birth, inclusion conjunctivitis is an important cause of ophthalmia neonatorum. Ocular chlamydial disease occurs most frequently in adults between ages 18 and 30.

Complications

- Otitis media
- Blindness

RECOGNIZING A STYE

A stye is a localized red, swollen, and tender abscess of the lid glands.

Signs and symptoms

Inclusion conjunctivitis develops 5 to 12 days after contamination (it takes longer to develop than gonococcal ophthalmia). In a neonate, reddened eyelids and tearing with moderate mucoid discharge are presenting symptoms. In neonates, pseudomembranes may form, which can lead to conjunctival scarring. In adults, follicles appear inside the lower eyelids; such follicles don't form in infants because the lymphoid tissue isn't yet well developed. Children and adults also develop preauricular lymphadenopathy, and children may develop otitis media as a complication. Inclusion conjunctivitis may persist for weeks or months, possibly with superficial corneal involvement.

Diagnosis

Clinical features and a history of sexual contact with an infected individual suggest inclusion conjunctivitis.

CONFIRMING DIAGNOSIS

Examination of Giemsa-stained conjunctival scraping reveals cytoplasmic inclusion bodies in conjunctival epithelial cells, and is effective in detecting chlamydial infection in infants. The direct fluorescent monoclonal antibody and enzyme-linked immunosorbent assay are most effective in adults.

Treatment

Because infection isn't limited to the eye in neonates, infants, or adults, systemic antimicrobial treatment is necessary. In infants, effective therapy is achieved with erythromycin. Adults may be given tetracycline, doxycycline, or erythromycin.

Prophylactic tetracycline or erythromycin ointment is applied once, 1 hour after delivery. However, this treatment hasn't been found to be significantly more effective than Credé's method (1% silver nitrate).

Special considerations

- Keep the patient's eyes as clean as possible, using sterile technique. Clean the eyes from the inner to the outer canthus. Apply warm soaks as needed. Record the amount and color of drainage.
- Remind the patient not to rub his eyes, which can irritate them.
- If the patient's eyes are sensitive to light, keep the room dark or suggest that he wear dark glasses.

PREVENTION

Take these actions to prevent further spread of inclusion conjunctivitis:

- *Wash hands thoroughly before and after administering eye medications.*
- *Suggest genital examination of the mother of an infected neonate or of any adult with inclusion conjunctivitis.*
- *Obtain a history of recent sexual contacts, so they can be examined for chlamydial infection.*

Conjunctivitis

Conjunctivitis is characterized by hyperemia of the conjunctiva due to infection, allergy, or chemical reactions. (See *Recognizing conjunctivitis*.) This disorder usually occurs as benign, self-limiting pinkeye; it may also be chronic, possibly indicating degenerative changes or damage from repeated acute attacks.

Causes and incidence

The most common causative organisms include:

- bacterial—*Staphylococcus aureus, Streptococcus pneumoniae, Neisseria gonorrhoeae, Neisseria meningitidis*
- chlamydial—*Chlamydia trachomatis* (inclusion conjunctivitis)

- viral—adenovirus types 3, 7, and 8; herpes simplex virus, type 1.

Other causes include allergic reactions to pollen, grass, topical medications, air pollutants, smoke, or unknown seasonal allergens (vernal conjunctivitis); environmental (wind, dust, and smoke) and occupational irritants (acids and alkalies); and a hypersensitivity to contact lenses or solutions.

Vernal conjunctivitis (so-called because symptoms tend to be worse in the spring) is a severe form of immunoglobulin Emediated mast cell hypersensitivity reaction. This form of conjunctivitis is bilateral. It usually begins at age 3 to 5 years and persists for about 10 years. It's sometimes associated with other signs of allergy commonly related to pollens, asthma, and allergic rhinitis.

Epidemic keratoconjunctivitis is an acute, highly contagious viral conjunctivitis caused by adenovirus types 8 and 19. It's commonly complicated by visual loss due to corneal subepithelial infiltrates. Health care providers must be careful to wash their hands and sterilize equipment to prevent the spread of this disease.

In the Western hemisphere, conjunctivitis is probably the most common eye disorder.

Complications

- Tic
- Corneal infiltrates
- Corneal ulcers
- Eye loss
- Reinfection

Signs and symptoms

Conjunctivitis commonly produces hyperemia of the conjunctiva, sometimes accompanied by discharge, tearing and, with corneal involvement, pain and photophobia. It generally doesn't affect vision. Conjunctivitis usually begins in one eye and rapidly spreads to the other by contamination

of towels, washcloths, or the patient's own hand.

Acute bacterial conjunctivitis (pinkeye) usually lasts only 2 weeks. The patient typically complains of itching, burning, and the sensation of a foreign body in his eye. The eyelids show a crust of sticky, mucopurulent discharge. If the disorder is due to *N. gonorrhoeae,* however, the patient exhibits a profuse, purulent discharge.

Viral conjunctivitis produces copious tearing with minimal exudate, and enlargement of the preauricular lymph node. Some viruses follow a chronic course and produce severe disabling disease; others last 2 to 3 weeks and are self-limiting.

Diagnosis

Physical examination reveals peripheral injection of the bulbar conjunctival vessels. In children, possible systemic symptoms include sore throat or fever, if the conjunctivitis is suspected of being of adenoviral origin.

Lymphocytes are predominant in stained smears of conjunctival scrapings if conjunctivitis is caused by a virus. Polymorphonuclear cells (neutrophils) predominate if conjunctivitis is due to bacteria; eosinophils, if it's allergy-related. Culture and sensitivity tests identify the causative bacterial organism and indicate appropriate antibiotic therapy.

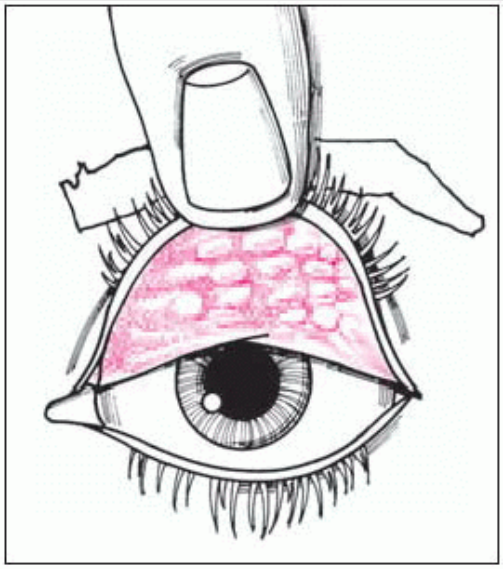
Treatment

Treatment for conjunctivitis varies with the cause. Bacterial conjunctivitis requires topical application of the appropriate broad-spectrum antibiotic. Although viral conjunctivitis resists treatment, a sulfonamide or broad-spectrum antibiotic eyedrops may prevent a secondary infection. Patients may be contagious for several weeks after onset. The most important aspect of treatment is preventing transmission. Herpes simplex infection generally responds to treatment with trifluridine drops or vidarabine ointment or oral acyclovir, but the infection may persist for 2 to 3 weeks. Treatment for vernal (allergic) conjunctivitis includes administration of corticosteroid drops followed by

cromolyn sodium, cold compresses to relieve itching and, occasionally, oral antihistamines.

RECOGNIZING CONJUNCTIVITIS

Itching is the hallmark of allergy. Giant papillae resembling cobblestones may be seen on the palpebral conjunctiva, as shown here.



Instillation of a one-time dose of erythromycin or 1% silver nitrate solution (Credé's procedure) into the eyes of neonates prevents gonococcal conjunctivitis.

Special considerations

- Apply compresses and therapeutic ointment or drops, as ordered. Don't irrigate the eye, as this will spread the infection. Have the patient wash his hands before he uses the medication. Tell him to use clean washcloths or towels frequently so he doesn't infect his other eye. (See *Preventing conjunctivitis,* page 666.)
- Teach the patient to instill eyedrops and ointments correctly—without touching the bottle tip to his eye or lashes.
- Remind the patient that the ointment will blur his vision.

- Stress the importance of safety glasses for the patient who works near chemical irritants.
- Notify public health authorities if cultures show *N. gonorrhoeae*.

PREVENTION

PREVENTING CONJUNCTIVITIS

To prevent conjunctivitis from occurring or recurring, teach your patient to practice good hygiene. Encourage the following prevention tips.

Practice good hygiene

To encourage good eye hygiene, teach proper hand-washing technique because bacterial and viral conjunctivitis are highly contagious. Stress the risk of spreading infection to family members by sharing washcloths, towels, and pillows. Suggest the use of tissues or disposable wipes to reduce the risk of transmission from contaminated linens. Caution the patient against rubbing his infected eye, which could spread infection to his other eye.

Use cosmetics carefully

If the patient uses eye cosmetics, instruct her not to share them. Also, encourage her to replace eye cosmetics regularly.

Keep contact lenses clean

If the patient wears contact lenses, teach him to handle and clean contact lenses properly. Also, while his eyes are infected, he should stop wearing the lenses until the infection clears.

Avoid contact with contagious people

Because conjunctivitis is highly contagious, particularly among children, infected children should avoid close contact with other children. Warn the patient with “cold

sores” to avoid kissing others on the eyelids to prevent the spread of the disease.

Trachoma

The most common cause of preventable blindness in underdeveloped areas of the world, trachoma is a chronic form of keratoconjunctivitis. This infection is usually confined to the eye but may have a systemic component. Although trachoma itself is self-limiting, it causes permanent damage to the cornea and conjunctiva by scarring the lids, and it results in secondary infections that can lead to blindness. (See *What happens in trachoma*.) Early diagnosis and treatment (before trachoma results in scar formation) ensure recovery but without immunity to reinfection.

Causes and incidence

Trachoma results from infection with *Chlamydia trachomatis,* a gram-negative obligate-intracellular bacterium. These organisms are transmitted from eye to eye by flies and gnats and through hand-to-eye contact in endemic areas.

Trachoma is spread by close contact between family members or among schoolchildren. It's prevalent in Africa, Latin America, and Asia, particularly in children. It is rare in the United States, but it can be found among Native Americans of the Southwest. Other predisposing factors include poverty and poor hygiene due to lack of water. Patients in hot, dusty climates are at greater risk.

Complications

- Conjunctival and corneal scarring
- Deformities of the eyelid
- Vision loss

Signs and symptoms

Trachoma begins with a mild infection resembling bacterial conjunctivitis (visible conjunctival follicles, red and edematous eyelids,

pain, photophobia, tearing, and exudation).

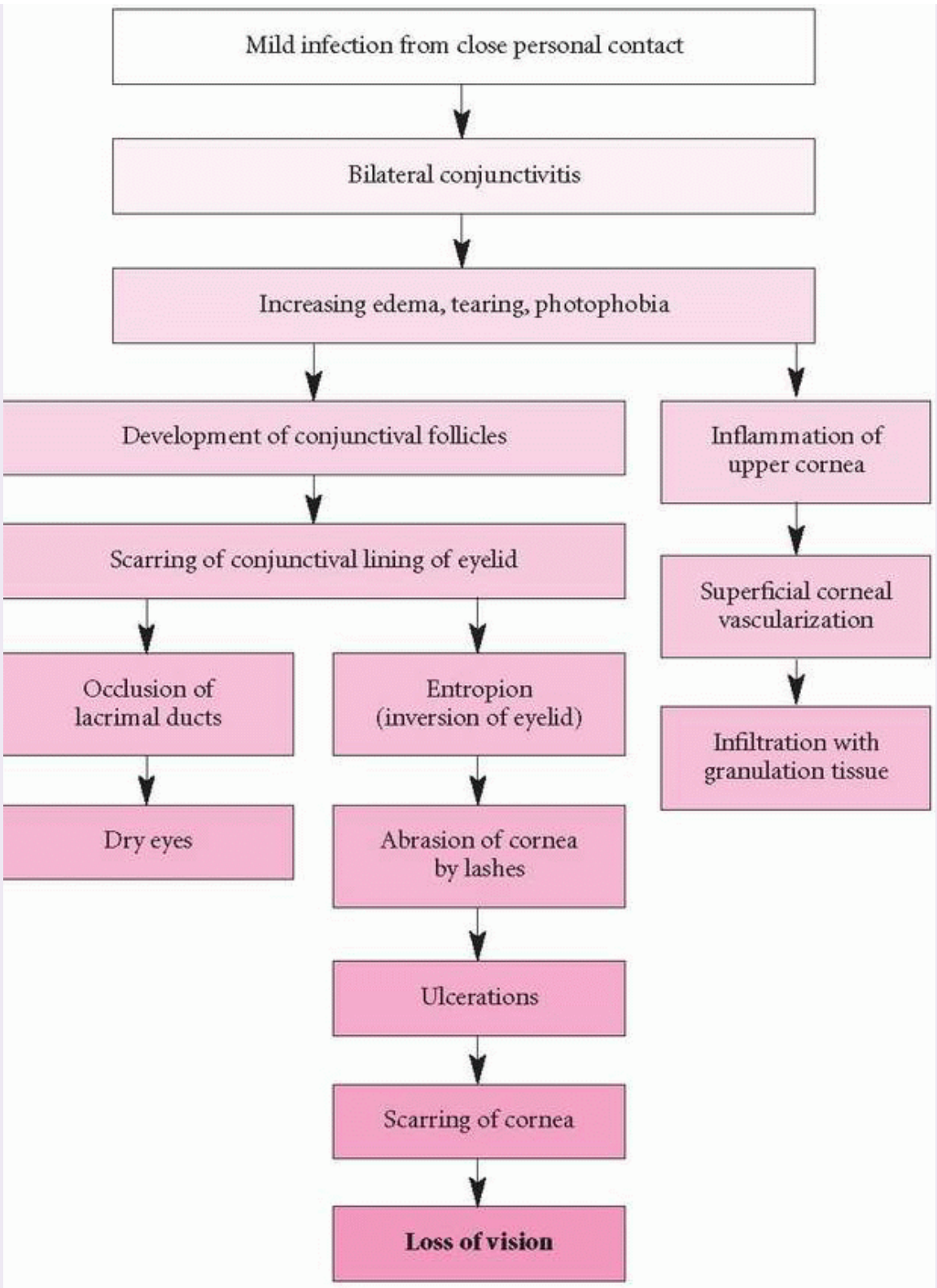
After about 1 month, if the infection is untreated, conjunctival follicles enlarge into inflamed papillae that later become yellow or gray. At this stage, small blood

vessels invade the cornea under the upper lid.

PATHOPHYSIOLOGY

WHAT HAPPENS IN TRACHOMA

Trachoma results from infection with *Chlamydia trachomatis* and in its early stages resembles bacterial conjunctivitis. If untreated, this chronic infection can spread to the cornea and lead to scarring and, eventually, to blindness.


Mild infection from close personal contact
Bilateral conjunctivitis
Increasing edema, tearing, photophobia
Development of conjunctival follicles
Scarring of conjunctival lining of eyelid
Occlusion of lacrimal ducts
Dry eyes
Entropion (inversion of eyelid)
Abrasion of cornea by lashes
Ulcerations
Scarring of cornea
Loss of vision
Inflammation of upper cornea
Superficial corneal vascularization
Infiltration with granulation tissue

Eventually, severe scarring and contraction of the eyelids cause entropion; the eyelids turn inward and the lashes rub against the cornea, producing corneal scarring and visual distortion. In late stages, severe conjunctival scarring may obstruct the lacrimal ducts and cause dry eyes.

Diagnosis

Follicular conjunctivitis with corneal infiltration, and upper lid or conjunctival scarring suggest trachoma, especially in endemic areas, when these symptoms persist longer than 3 weeks.

CONFIRMING DIAGNOSIS

Microscopic examination of a Giemsa-stained conjunctival scraping confirms the diagnosis by showing cytoplasmic inclusion bodies, some polymorphonuclear reaction, plasma cells, large macrophages containing phagocytosed debris, and follicle cells.

Treatment

Primary treatment for trachoma consists of topical or systemic antibiotic therapy with erythromycin (and its derivatives), doxycycline, or sulfonamides. Severe entropion requires surgical correction.

ALERT

Tetracycline is contraindicated in pregnant women, because it may adversely affect the fetus, and in children younger than age 7, in whom it may permanently discolor teeth.

Because trachoma is contagious and reinfection is common, some physicians suggest treating entire villages with high incidence rates with antibiotic therapy.

Special considerations

Patient teaching is essential:

- Emphasize the importance of hand washing and making the best use of available water supplies to maintain good personal hygiene.
- Because no definitive preventive measure exists (vaccines offer temporary and partial protection, at best), stress the need for strict compliance with the prescribed drug therapy.
- If ordered, teach the patient or his family how to instill eyedrops correctly.
- Stress the importance of not sharing contaminated items, such as towels, handkerchiefs, and eye makeup.
- Emphasize the need for facial cleanliness.

PREVENTION

To prevent trachoma, warn the patient not to allow flies or gnats to settle around his eyes.

CORNEAL DISORDERS

Keratitis

An inflammation of the cornea, keratitis may result from bacterial, fungal, or viral infection. If untreated, the infection can lead to blindness.

Causes and incidence

The most common cause of keratitis is infection by herpes simplex virus, type 1 (known as *dendritic corneal ulcer* because of a characteristic branched lesion of the cornea resembling the veins of a leaf). Bacterial corneal ulcers frequently occur as a result of an infected corneal abrasion or a contaminated contact lens. Fungal keratitis is more frequently encountered in tropical climates. Poor lid closure can result in exposure keratitis. Chemicals accidentally splashed into the eye and exposure to ultraviolet light (sunlamps, sunlight, or welding arcs) also can produce keratitis. Vaccinial keratitis may result when the patient has red eye or periocular vesicles coinciding with a history of recent vaccine exposure (such as smallpox vaccination or close contact with a vaccine recipient).

Complications

- Blindness
- Corneal scarring or perforation

Signs and symptoms

Keratitis is usually unilateral. The patient presents with decreased vision, discomfort ranging from mild irritation to acute pain, tearing, and photophobia. On gross examination

with a penlight, the corneal light reflex may appear distorted. When keratitis results from exposure, it usually affects the lower portion of the cornea.

Diagnosis

CONFIRMING DIAGNOSIS

Visual acuity may be decreased if the lesion is central. Slit-lamp examination confirms keratitis. Staining the eye with a sterile fluorescein strip enables the examiner to discern the extent and depth of the corneal lesion.

Patient history may reveal a recent infection of the upper respiratory tract accompanied by cold sores, or eye irritation with the wearing of contact lenses. Culture may identify the virus.

Treatment

Treatment for acute keratitis due to herpes simplex virus consists of trifluridine eyedrops, vidarabine ointment, or oral acyclovir. A broad-spectrum antibiotic may prevent secondary bacterial infection. Dendritic keratitis may become chronic with recurrent episodes. Bacterial corneal ulcers require intense topical eyedrop instillation every half hour for the first 48 hours with two broad-spectrum antibiotics. Long-term topical therapy may be necessary. (Corticosteroid therapy is contraindicated in dendritic keratitis or any other viral or fungal disease of the cornea.) Fungal keratitis is treated with natamycin.

Exposure keratitis is treated with ointment at night and frequent instillation of artificial tears during the day. A plastic bubble shield may prevent tear evaporation. Vision may be restored by penetrating keratoplasty (corneal transplant) in blindness resulting from corneal scarring.

Special considerations

- Protect the exposed corneas of unconscious patients by cleaning the eyes daily, applying moisturizing ointment, or covering the eyes with an eye shield.
- Be aware that the patient with a red eye may have keratitis. Check for a history of contact lens wear, cold sores, or recent foreign-body sensation. Refer the patient for slit-lamp examination as soon as possible for intense treatment.

Corneal abrasion

A corneal abrasion is a scratch on the surface epithelium of the cornea. An abrasion, or foreign body in the eye, is the most common eye injury. With treatment, the prognosis is usually good.

Causes and incidence

A corneal abrasion usually results from a foreign body, such as a cinder or a piece of dust, dirt, or grit that becomes embedded under the eyelid. Even if the foreign body is washed out by tears, it may still injure the cornea. Small pieces of metal that get in the eyes of workers who don't wear protective glasses quickly form a rust ring on the cornea and cause corneal abrasion. Such abrasions also commonly occur in the eyes of people who fall asleep wearing hard contact lenses or whose lenses aren't fitted properly.

A corneal scratch produced by a fingernail, a piece of paper, or other organic substance may cause a persistent lesion. The epithelium doesn't always heal properly, and a recurrent corneal erosion may develop, with delayed effects more severe than the original injury.

In the United States, corneal abrasions are a common ophthalmologic cause of emergency department visits. Incidence is highest among younger, physically active individuals; corneal abrasions are rare in elderly people.

Complications

- Corneal erosion
- Corneal ulceration
- Permanent vision loss

Signs and symptoms

A corneal abrasion typically produces redness, increased tearing, discomfort with blinking, a sensation of “something in the eye” and, because the cornea is richly endowed with nerve endings from the trigeminal nerve (cranial nerve V), pain disproportionate to the size of the injury. It

may also affect visual acuity, depending on the size and location of the injury.

Diagnosis

History of eye trauma or prolonged wearing of contact lenses and typical symptoms suggest corneal abrasion.

CONFIRMING DIAGNOSIS

Staining the cornea with fluorescein stain confirms the diagnosis: The injured area appears green when examined with a flashlight. Slit-lamp examination discloses depth and allows measurement of the abrasion.

Examining the eye with a flashlight may reveal a foreign body on the cornea; the eyelid must be everted to check for a foreign body embedded under the lid.

Before beginning treatment, a test to determine visual acuity provides a medical baseline and a legal safeguard.

Treatment

Topical anesthetic eyedrops are instilled in the affected eye before removal of a superficial foreign body, using a foreign body spud. A rust ring on the cornea must be removed with an ophthalmic burr. When only partial removal is possible, reepithelialization lifts the ring again to the surface and allows complete removal the following day.

Treatment also includes instillation of broad-spectrum antibiotic eyedrops in the affected eye every 3 to 4 hours. Application of a pressure patch prevents further corneal irritation when the patient blinks. If the patient wears contact lenses, it may be advisable for him to abstain from wearing the lenses until the corneal abrasion heals.

Special considerations

- Assist with examination of the eye. Check visual acuity before beginning treatment.
- If a foreign body is visible, carefully irrigate with normal saline solution.
- Tell the patient with an eye patch to leave it in place for 6 to 12 hours. Warn that a patch alters depth perception, so advise caution in daily activities, such as climbing stairs or stepping off a curb.
- Reassure the patient that the corneal epithelium usually heals in 24 to 48 hours.
- Stress the importance of instilling antibiotic eyedrops, as ordered, because an untreated corneal abrasion, if infected, can lead to a corneal ulcer and permanent vision loss. Teach the patient the proper way to instill eye medications.

PREVENTION

Emphasize the importance that safety glasses be worn to protect a worker's eyes from flying fragments. Also review instructions for wearing and caring for contact lenses, to prevent further trauma. Encourage use of sunglasses.

Corneal ulcers

A major cause of blindness worldwide, ulcers produce corneal scarring or perforation. They occur in the central or marginal areas of the cornea, vary in shape and size, and may be singular or multiple. Marginal ulcers are the most common form. Prompt treatment (within hours of onset) can prevent visual impairment.

Causes and incidence

Corneal ulcers generally result from protozoan, bacterial, viral, or fungal infections. Common bacterial sources include *Staphylococcus aureus, Pseudomonas aeruginosa, Streptococcus viridans, Streptococcus (Diplococcus) pneumoniae,* and *Moraxella liquefaciens;* viral sources comprise herpes simplex type 1, variola, vaccinia, and varicella-zoster viruses; and common fungal sources are *Candida, Fusarium,* and *Cephalosporium*.

Other causes include trauma, exposure, reactions to bacterial infections, toxins, trichiasis, entropion, allergens, and wearing of contact lenses. (See *What happens in corneal ulceration*.) Tuberculoprotein causes a classic phlyctenular keratoconjunctivitis, vitamin A deficiency results in xerophthalmia, and fifth cranial nerve lesions lead to neurotropic ulcers.

Complications

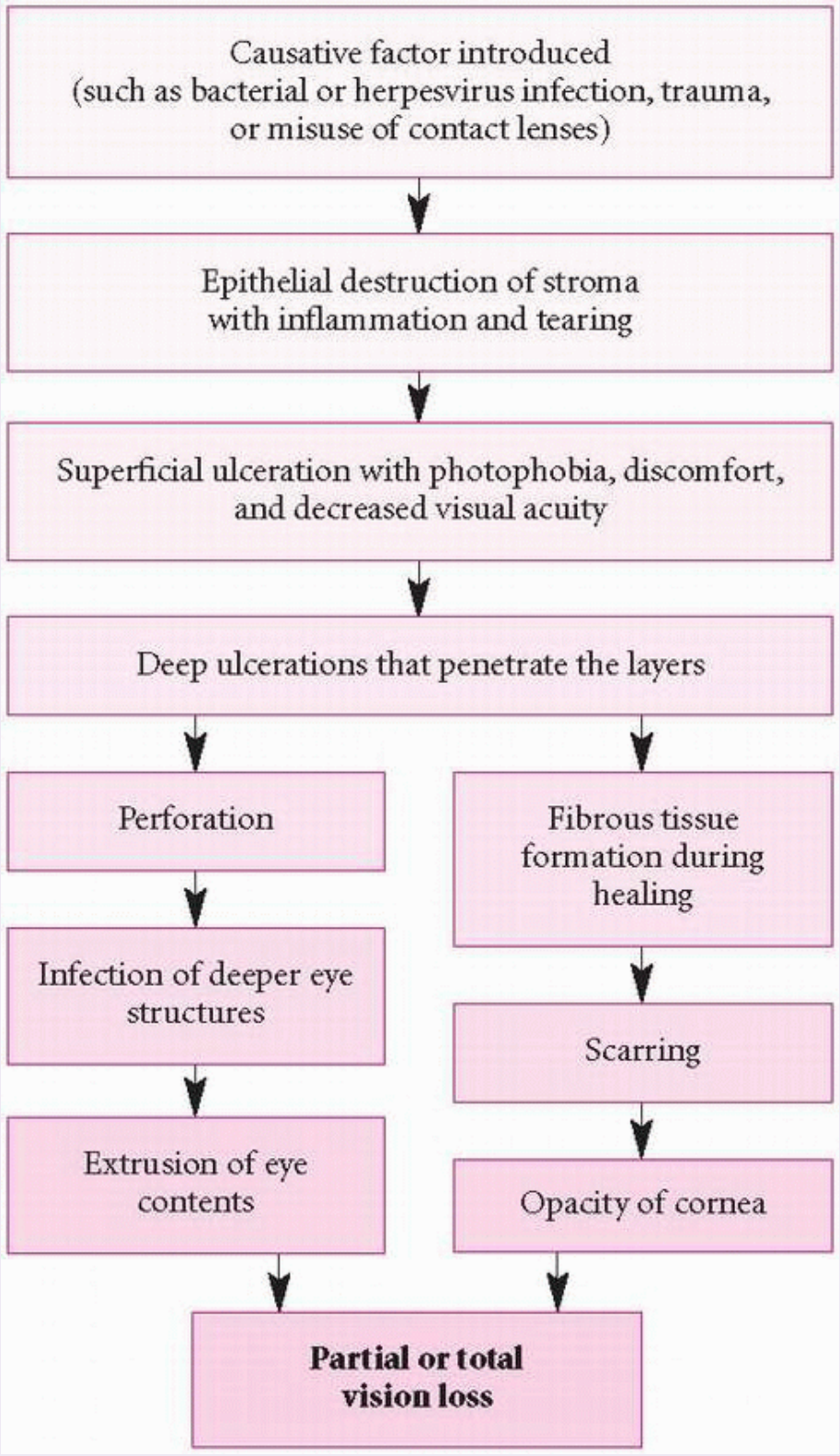
- Corneal scarring
- Loss of the eye
- Vision loss

PATHOPHYSIOLOGY

WHAT HAPPENS IN CORNEAL ULCERATION

Corneal ulcers can be caused by infection (protozoan, bacterial, viral, or fungal), trauma, exposure, toxins, contact lenses, or allergens. Scarring or perforation can

cause changes in the eye structure and can lead to partial or total vision loss.



Signs and symptoms

Typically, corneal ulceration begins with pain (aggravated by blinking) and photophobia, followed by increased tearing. Eventually, central corneal ulceration produces pronounced visual blurring. The eye may appear injected. If a bacterial ulcer is present, purulent discharge is possible.

Diagnosis

A history of trauma or use of contact lenses and flashlight examination that reveals irregular corneal surface suggest corneal ulcer. Exudate may be present on the cornea, and a hypopyon (accumulation of white cells in the anterior chamber) may appear as a white crescent moon that moves when the head is tilted.

CONFIRMING DIAGNOSIS

Fluorescein dye, instilled in the conjunctival sac, stains the outline of the ulcer and confirms the diagnosis.

Culture and sensitivity testing of corneal scrapings may identify the causative bacteria or fungus, and may indicate appropriate antibiotic or antifungal therapy.

Treatment

Prompt treatment is essential for all forms of corneal ulcer to prevent complications and permanent visual impairment. Treatment usually consists of systemic and topical broad-spectrum antibiotics until culture results identify the causative organism. The goals of treatment are to eliminate the underlying cause of the ulcer and to relieve pain:

- Fungi—topical instillation of natamycin for *Fusarium*, *Cephalosporium*, and *Candida*.
- Herpes simplex type 1 virus—topical application of trifluridine drops or vidarabine ointment. Corneal ulcers resulting from a viral infection often recur, requiring further treatment with trifluridine.

- Hypovitaminosis A—correction of dietary deficiency or GI malabsorption of vitamin A.
- Infection by *P. aeruginosa*—polymyxin B and gentamicin, administered topically and by subconjunctival injection, or carbenicillin and tobramycin I.V. Because this type of corneal ulcer spreads so rapidly, it can cause corneal perforation and loss of the eye within 48 hours. Immediate treatment and isolation of hospitalized patients are required.

ALERT

Treatment for a corneal ulcer due to bacterial infection should never include an eye patch because patching creates the dark, warm, moist environment ideal for bacterial growth.

- Neurotropic ulcers or exposure keratitis —frequent instillation of artificial tears or lubricating ointments and use of a plastic bubble eye shield.
- Varicella-zoster virus—topical sulfonamide ointment applied three to four times daily to prevent secondary infection. These lesions are unilateral, following the pathway of the fifth cranial nerve, and are typically quite painful. Give analgesics as ordered. Associated anterior uveitis requires cycloplegic eyedrops. Watch for signs of secondary glaucoma (transient vision loss and halos around lights).

Special considerations

- Keep the room darkened and orient the patient as necessary.
- Teach the patient how to properly clean and wear his contact lenses to prevent a recurrence.

PREVENTION

Encourage yor patient to:

- *seek treatment early for eye infections*
- *wash hands before handling contact lenses*
- *avoid wearing contact lenses overnight.*

UVEAL TRACT, RETINAL, AND LENS DISORDERS

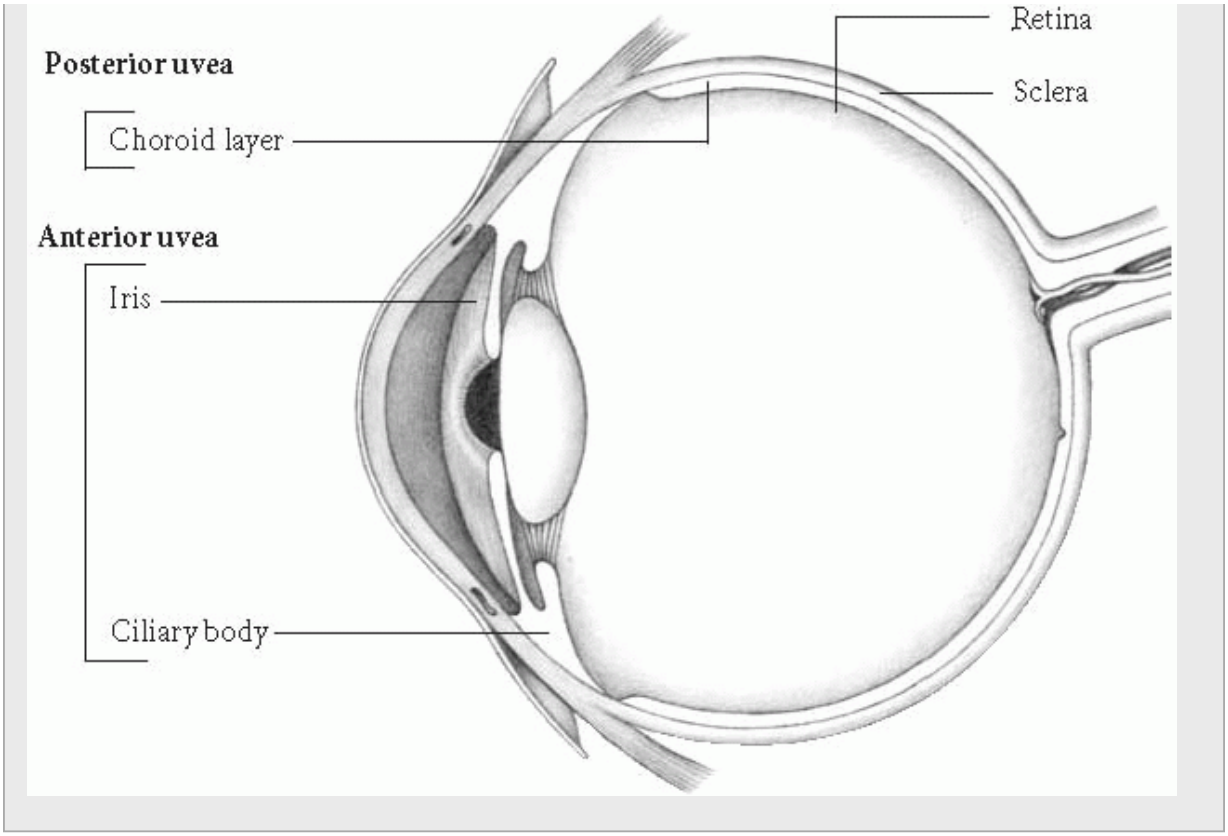
Uveitis

Uveitis is inflammation of the uveal tract. It occurs as anterior uveitis, which affects the iris (iritis) or both the iris and the ciliary body (iridocyclitis); as posterior uveitis, which affects the choroid (choroiditis) or both the choroid and the retina (chorioretinitis); or as panuveitis, which affects the entire uveal tract. Although clinical distinction isn't always possible, anterior uveitis occurs in two forms—granulomatous and nongranulomatous. (See *Anatomy of the uveal tract*.)

Granulomatous uveitis was once thought to be caused by tuberculosis bacilli; nongranulomatous uveitis, by streptococci. Although this isn't true, the terms are still used. (See *Granulomatous and nongranulomatous uveitis*, page 674.) Untreated anterior uveitis may result in elevated intraocular pressure (IOP), leading to vision loss. With immediate treatment, anterior uveitis usually subsides after a few days to several weeks; however, recurrence can occur. Posterior uveitis may lead to vision loss if the macula is involved.

ANATOMY OF THE UVEAL TRACT

The uveal tract, which is the middle layer of the eye, is pigmented and vascular. It consists of the anterior uvea, the posterior uvea, and the retina. The anterior uvea is made up of the ciliary body and the iris. The posterior uvea contains the choroid layer.



Causes and incidence

Typically, uveitis is idiopathic. However, it can result from allergy, bacteria, viruses, fungi, chemicals, trauma, or surgery; or it may be associated with systemic diseases, such as rheumatoid arthritis, ankylosing spondylitis, and toxoplasmosis. Laser-assisted in situ keratomileusis (LASIK) surgery may worsen the condition.

Uveitis occurs in 15 of every 100,000 people.

Complications

- Cataracts
- Glaucoma
- Retinal detachment
- Vision loss

Signs and symptoms

Anterior uveitis produces moderate to severe unilateral eye pain; severe ciliary injection; photophobia; tearing; a small, nonre-active pupil; and blurred vision (due to the increased number of cells in the aqueous humor). It sometimes produces deposits called keratic precipitates on the back of the cornea, which may be seen in the anterior chamber. The iris may adhere to the lens, causing posterior synechiae and pupillary distortion; pain and photophobia may occur. Onset may be acute or insidious.

Posterior uveitis begins insidiously, with complaints of slightly decreased or blurred vision or floating spots. Posterior uveitis may be acute or chronic, and it may affect one or both eyes. Retinal damage caused by lesions from toxoplasmosis and retinal detachments may occur. Refer the patient to an ophthalmologist for dilated fundus examination and treatment for local systemic diseases.

GRANULOMATOUS AND NONGRANULOMATOUS UVEITIS

Various characteristics that differentiate granulomatous and nongranulomatous uveitis are listed below.

Characteristic	Granulomatous	Nongranulomatous
Location	▪ Usually, posterior part of uveal tract	▪ Anterior part of the iris and ciliary body
Onset	▪ Insidious	▪ Acute
Pain	▪ None or slight	▪ Marked
Photophobia	▪ Slight	▪ Marked
Course	▪ Chronic	▪ Acute
Prognosis	▪ Fair to poor	▪ Good
Recurrence	▪ Occasional	▪ Common
Blurred vision	▪ Marked	▪ Moderate

Diagnosis

CONFIRMING DIAGNOSIS

In anterior and posterior uveitis, a slit-lamp examination shows a "flare and cell" pattern, which looks like particles dancing in a sunbeam. With a special lens, slit-lamp and ophthalmoscopic examination can also identify active inflammatory fundus lesions involving the retina and choroid, although a hazy vitreous may obscure the view.

In posterior uveitis, serologic tests may be used to rule out toxoplasmosis.

Treatment

Uveitis requires vigorous and prompt management, which includes treatment for any known underlying cause—corticosteroids with antibiotic therapy for infectious diseases and suppression therapy for autoimmune diseases—and application of a topical cycloplegic, such as 1% atropine sulfate, and of topical corticosteroids applied three to four times daily. For severe uveitis, therapy includes oral systemic corticosteroids.

ALERT

Long-term steroid therapy can cause a rise in IOP or cataracts. Carefully monitor IOP during acute inflammation. If IOP rises, therapy should include an antiglaucoma medication, such as brimonidine (Alphagan), an alpha$_2$-adrenergic agonist, or dorzolamide (Trusopt), a sulfonamide.

Occasionally, posterior uveitis requires systemic immunosuppression with azathioprine or cyclosporine.

Special considerations

- Encourage rest during the acute phase.

- Teach the patient the proper method of instilling eyedrops.
- Suggest the use of dark glasses to ease the discomfort of photophobia.
- Instruct the patient to watch for and report adverse effects of systemic corticosteroid therapy (for example, edema or muscle weakness).
- Stress the importance of follow-up care for IOP checks while the patient is taking steroids. Tell the patient to seek treatment immediately at the first sign of iritis.

Retinal detachment

Retinal detachment occurs when the outer retinal pigment epithelium splits from the neural retina, creating subretinal space. This space then fills with fluid, called *subretinal fluid*. Retinal detachment usually involves only one eye, but may later involve the other eye. Surgical reattachment is usually successful. However, the prognosis for good vision depends on which area of the retina has been affected.

Causes and incidence

Any retinal tear or hole allows the liquid vitreous to seep between the retinal layers, separating the retina from its choroidal blood supply. Predisposing factors include myopia, intraocular surgery, and trauma. In adults, retinal detachment usually results from degenerative changes of aging, which cause a spontaneous retinal hole. Perhaps the influence of trauma explains why retinal detachment is twice as common in males. Retinal detachment may also result from seepage of fluid into the subretinal space (because of inflammation, tumors, or systemic diseases) or from traction that's placed on the retina by vitreous bands or membranes (due to proliferative diabetic retinopathy, posterior uveitis, or a traumatic intraocular foreign body).

Retinal detachment is rare in children, but occasionally can develop as a result of retinopathy of prematurity, tumors (retinoblastomas), trauma, or myopia (which tends to run in families).

In the United States, about 10,000 people per year are affected by retinal detachments.

Complications

- Severe vision impairment
- Blindness

Signs and symptoms

Initially, the patient may complain of floating spots and recurrent flashes of light (photopsia). However, as detachment progresses, gradual, painless vision loss may be described as a veil, curtain, or cobweb that eliminates a portion of the visual field.

Diagnosis

CONFIRMING DIAGNOSIS

Diagnosis depends on ophthalmoscopy after full pupil dilation. Examination shows the usually transparent retina as gray and opaque; in severe detachment, it reveals folds in the retina and ballooning out of the area. Indirect ophthalmoscopy is used to search for retinal tears. Ultrasound is performed if the lens is opaque.

Treatment

Treatment depends on the location and severity of the detachment. It may include restriction of eye movements and complete bed rest until surgical reattachment is done. A hole in the peripheral retina can be treated with cryotherapy; in the posterior portion, with laser therapy. Retinal detachment usually requires a scleral buckling procedure or a vitrectomy to reattach the retina. Basic salt solution is used to replace the retina while the vitreous is removed.

Certain types of uncomplicated retinal detachment may be treated by pneumatic retinopexy, in which an expansile gas is initially injected into the vitreous cavity and the patient's head is positioned to facilitate retina reattachment. This procedure can be performed under local anesthesia.

Special considerations

- Provide emotional support because the patient may be understandably distraught about his loss of vision.
- During transportation, position the patient's head so that the detached portion of the retina will fall back with the aid of gravity.
- To prepare for surgery, wash the patient's face with no-tears shampoo. Give antibiotics and cycloplegic-mydriatic eyedrops.
- Postoperatively, position the patient facedown on his right or left side and with the head of the bed raised. Discourage straining at stool, bending down, hard coughing, sneezing, or vomiting, which can raise intraocular pressure. Antiemetics may be indicated.
- Protect the patient's eye with a shield or glasses.
- To reduce edema and discomfort, apply ice packs as ordered. Administer pain medication, as ordered, for eye pain.

- After removing the eye shield, gently clean the eye with cycloplegic eyedrops and administer steroid-antibiotic eyedrops, as ordered. Use cold compresses to decrease swelling and pain.
- Administer analgesics as needed, and report persistent pain. Teach the patient how to properly instill eyedrops, and emphasize compliance and follow-up care. Suggest dark glasses to compensate for light sensitivity caused by cycloplegia.

Vascular retinopathies

Vascular retinopathies are noninflammatory retinal disorders that result from interference with the blood supply to the eyes. The five distinct types of vascular retinopathy are central retinal artery occlusion, central retinal vein occlusion, diabetic retinopathy, hypertensive retinopathy, and sickle cell retinopathy.

Causes and incidence

When one of the arteries maintaining blood circulation in the retina becomes obstructed, the diminished blood flow causes visual deficits. (See *Anatomy of vascular retinopathy*.)

Central retinal artery occlusion may be idiopathic or may result from embolism, atherosclerosis, infection, or conditions that retard blood flow, such as temporal arteritis, carotid occlusion, and heart failure. This occlusion is rare, occurs unilaterally, and usually affects elderly patients. However, if it occurs in a younger person, the obstruction may have originated in the heart (such as embolization from plaque material from valve vegetations) and should be investigated accordingly.

Causes of central retinal vein occlusion include atherosclerosis, hypertension, optic disk edema, hypercoagulable states (polycythemia, leukemia, or sickle cell disease), glaucoma, retrobulbar compression (such as an orbital tumor), and drugs such as hormonal contraceptives. This form of vascular retinopathy is most prevalent in elderly patients and is characterized by impaired venous outflow.

Diabetic retinopathy results from juvenile or adult diabetes. Microcirculatory changes occur more rapidly when diabetes is poorly controlled. About 90% of patients with juvenile diabetes develop retinopathy within 20 years of onset of diabetes. In adults with diabetes, incidence increases with the duration of diabetes; 80% of patients who have had diabetes for 20 to 25 years develop retinopathy. This condition is a leading cause of acquired adult blindness.

Hypertensive retinopathy results from prolonged hypertensive disease, producing retinal vasospasm, and consequent damage and arteriolar narrowing.

Sickle cell retinopathy results from impaired ability of the sickled cell to pass through microvasculature, producing vasoocclusion. This leads to microaneurysms, chorioretinal infarction, and retinal detachment.

Complications

- Blindness
- Secondary glaucoma

Signs and symptoms

Central retinal artery occlusion produces sudden, painless, unilateral loss of vision (partial or complete). It may follow amaurosis fugax or transient episodes of unilateral loss of vision lasting from a few seconds to minutes, probably due to vasospasm. This condition typically causes permanent blindness. However, some patients experience spontaneous resolution within hours and regain partial vision.

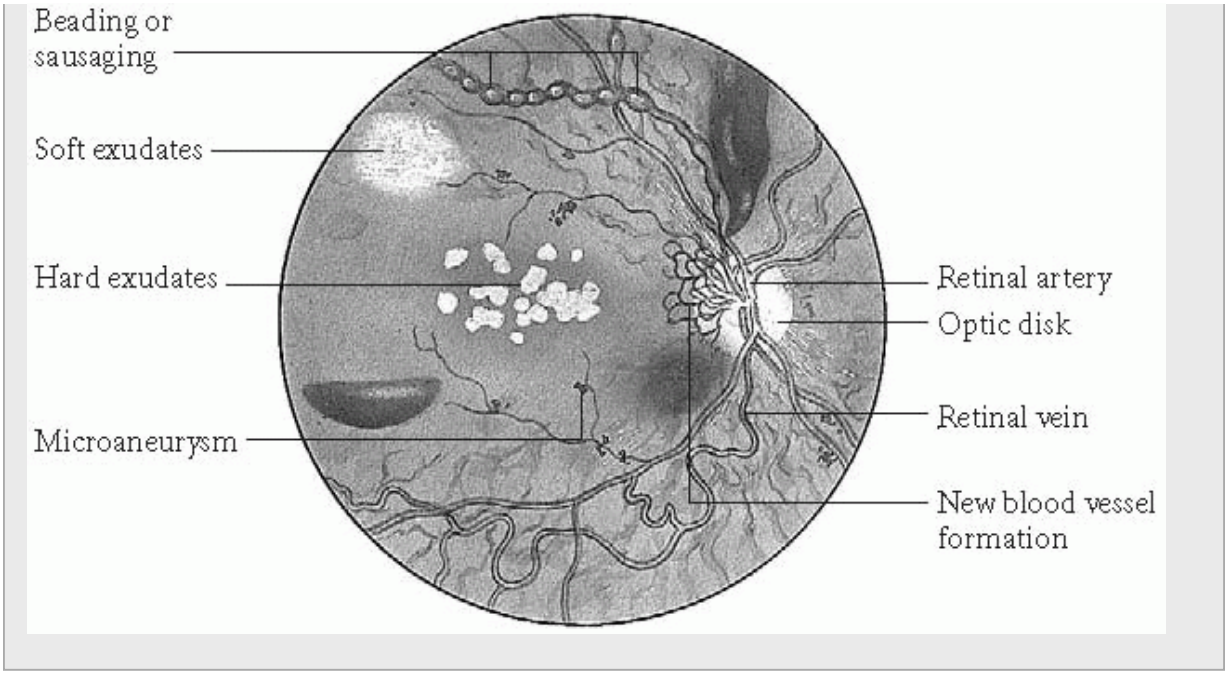
Central retinal vein occlusion causes reduced visual acuity, allowing perception of only hand movement and light. This condition is painless, except when it results in secondary neovascular glaucoma (uncontrolled proliferation of weak blood vessels). The prognosis is poor—some patients with this condition develop secondary glaucoma within 3 to 4 months after occlusion.

Nonproliferative diabetic retinopathy produces changes in the lining of the retinal blood vessels that cause the vessels to leak plasma or fatty substances, which decrease or block blood flow (nonperfusion) within the retina. This disorder may also produce microaneurysms and small hemorrhages. Nonproliferative retinopathy

causes no symptoms in some patients; in others, leakage of fluid into the macular region causes significant loss of central visual acuity (necessary for reading and driving) and diminished night vision.

ANATOMY OF VASCULAR RETINOPATHY

Vascular changes that occur with retinopathy, as seen by an ophthalmoscope, are depicted below.



Proliferative diabetic retinopathy produces fragile new blood vessels on the disk (neovascularization) and elsewhere in the fundus. These vessels can grow into the vitreous and then rupture, causing vitreous hemorrhage with corresponding sudden vision loss. Scar tissue that may form along the new blood vessels can pull on the retina, causing it to tear or even detach.

Symptoms of hypertensive retinopathy include blurred vision, often accompanied by headache. Ophthalmoscopic examination may reveal diffuse binocular narrowing, venular tortuosity, silver wire reflexes, macular stars, and swelling of the head of the optic nerve (disk edema). Severe, prolonged disease eventually produces blindness; mild, prolonged disease, visual defects.

Symptoms of sickle cell retinopathy include peripheral arteriolar occlusions, peripheral arteriovenous anastomoses, sea fan neurovascular fronds, vitreous hemorrhage as tractional forces and vitreous collapse tear fragile neovascular membranes and, with advanced disease, severe vitreous traction and retinal detachment.

Diagnosis

Check visual acuity and then vital signs, including blood pressure. Diagnosis is made on fundal examination with an ophthalmoscope.

Determine if female patients are pregnant; hypertensive retinopathy may be an early sign of preeclampsia. (See *Diagnostic tests for vascular retinopathies*, page 678.)

Treatment

No treatment has been shown to control central retinal artery occlusion. However, an attempt is made to release the occlusion into the peripheral circulation. To reduce intraocular pressure, therapy includes acetazolamide I.V., eyeball massage, thrombolysis by intra-arterial injection or I.V., high concentrations of inhaled oxygen, and anterior chamber paracentesis (to try to

move the arterial obstruction into the peripheral field).

DIAGNOSTIC TESTS FOR VASCULAR RETINOPATHIES

Central retinal artery occlusion

- Ophthalmoscopy (direct or indirect): shows blockage of retinal arterioles during a transient attack.
- Retinal examination: within 2 hours of onset, shows clumps or segmentation in the artery; later, milky white retina around the disk due to swelling and necrosis of ganglion cells caused by reduced blood supply; also shows a cherry-red spot in the macula that subsides after several weeks.
- Color Doppler tests: evaluate carotid occlusion with no need for arteriography.
- Physical examination: reveals the underlying cause of vascular retinopathy, for example, diabetes or hypertension.

Central retinal vein occlusion

- Ophthalmoscopy (direct or indirect): shows flame-shaped hemorrhages, retinal vein engorgement, white

patches among hemorrhages, and edema around the disk.
- Color Doppler tests: confirm or rule out occlusion of blood vessels.
- Physical examination: reveals the underlying cause.

Diabetic retinopathy

- Indirect ophthalmoscopic examination: shows retinal changes such as microaneurysms (earliest change), retinal hemorrhages and edema, venous dilation and beading, lipid exudates, fibrous bands in the vitreous, and growth of new blood vessels. Infarcts of the nerve fiber layer are observed.
- Fluorescein angiography: shows leakage of fluorescein from weak-walled vessels and "lights up" microaneurysms, differentiating them from true hemorrhages.
- History: of diabetes.

Hypertensive retinopathy

- Ophthalmoscopy (direct or indirect): in early stages, shows hard, shiny deposits; flame-shaped hemorrhages; silver wire appearance of narrowed arterioles; and nicking of veins where arteries cross them (atrioventricular nicking). In late stages, shows cotton wool patches, lipid exudates, retinal edema, papilledema due to ischemia and capillary insufficiency, hemorrhages, and microaneurysms in both eyes.
- Physical examination: reveals elevated blood pressure.
- History: of decreased vision, headache, and nausea.
- History: of hypertension, usually acute or malignant.

Therapy for central retinal vein occlusion may include aspirin, which acts as a mild anticoagulant. Patients with central retinal vein occlusion

have reported improved vision after direct injection of tissue plasminogen activator into the retinal venous system. Laser photocoagulation can reduce the risk of neovascular glaucoma for some patients whose eyes have widespread capillary nonperfusion.

Treatment for nonproliferative diabetic retinopathy is prophylactic. Careful control of blood glucose levels may reduce the severity of the retinopathy or delay its onset. Patients with early symptoms of microaneurysms should have frequent eye examinations (three to four times per year); children with diabetes should have an annual eye examination.

Treatment for proliferative diabetic retinopathy or severe macular edema is laser photocoagulation, which cauterizes the leaking blood vessels. Laser treatment may be focal (aimed at new blood vessels) or panretinal (placing burns throughout the peripheral retina). Despite treatment,

neovascularization continues to proliferate, and vitreous hemorrhage, with or without retinal detachment, may follow. If the blood isn't absorbed in 6 weeks to 3 months, vitrectomy may restore partial vision.

Treatment for hypertensive retinopathy includes control of blood pressure with appropriate drugs, diet, and exercise. Treating the systemic hypertension should improve the condition of the eyes. If left untreated, hypertensive retinopathy results in severe vision loss.

The treatment goal of sickle cell retinopathy is to reduce the risk of, or prevent or eliminate, retinal neovascularization. Patients with symptoms should be followed twice a year with ocular examinations and dilated retinal evaluation. Proliferative disease should be treated with fluorescein angiography and panretinal photocoagulation. Cryotherapy hasn't been proven to be effective and has a high complication rate.

Special considerations

- Be sure to monitor a patient's blood pressure if he complains of occipital headache and blurred vision.

ALERT

Arrange for immediate ophthalmologic evaluation when a patient complains of sudden, unilateral loss of vision. Blindness may be permanent if treatment is delayed.

- Encourage a diabetic patient to comply with the prescribed regimen.
- For a patient with hypertensive retinopathy, stress the importance of complying with antihypertensive therapy.

Age-related macular degeneration

Macular degeneration is the atrophy or degeneration of the macular region of the retina. Two types of age-related macular degeneration occur. The dry or atrophic form is characterized by atrophic pigment epithelial changes and is most often associated with a slow, progressive, and mild vision loss. The wet, exudative form causes progressive visual distortion leading to vision loss. It's characterized by subretinal neovascularization that causes leakage, hemorrhage, and fibrovascular scar formation, which produce significant loss of central vision.

Causes and incidence

Age-related macular degeneration results from underlying pathologic changes that occur primarily at the level of the retinal pigment epithelium, Bruch's membrane, and the choriocapillaris in the macular region. Drusen (bumps), which are common in elderly people, appear as yellow deposits beneath the pigment epithelium and may be prominent in the macula. No predisposing conditions have been identified; however, some forms of the disorder are hereditary.

Macular degeneration is the most common cause of legal blindness in adults, accounting for about 12% of blindness cases in the United States and for about 17% of new blindness cases. It's also one of the causes of severe irreversible loss of central vision in elderly people—by age 75, almost 15% of people have this condition. Whites have the highest incidence. Other risk factors are family history and cigarette smoking.

Complications

- Blindness

- Nystagmus

Signs and symptoms

The patient notices a change in central vision. Initially, straight lines (for example, of buildings) become distorted; later, a blank area appears in the center of a printed page (central scotoma).

Diagnosis

- Indirect ophthalmoscopy—fundus examination through a dilated pupil may reveal gross macular changes.
- I.V. fluorescein angiography—sequential photographs may show leaking vessels as fluorescein dye flows into the tissues from the subretinal neovascular net.
- Amsler's grid—used to monitor visual field loss.

Treatment

Laser photocoagulation reduces the incidence of severe vision loss in the patient with subretinal neovascularization, turning serous age-related macular degeneration to the dry form.

Photodynamic therapy, which can be performed in a physician's office, is an option for the patient with wet macular degeneration. In this procedure, verteporfin (a light-sensitive medication) is injected into a vein in the patient's arm and allowed to circulate to the eyes. The physician then shines a laser into the eyes, and the verteporfin produces a chemical reaction that destroys abnormal blood vessels. If the vessels regrow, the procedure can be repeated.

Special considerations

- Inform the patient with bilateral central vision loss of the visual rehabilitation services available to him.
- Special devices, such as low-vision optical aids, are available to improve the quality of life in the patient with good peripheral vision.

PREVENTION

Encourage early detection through regular eye examinations.

Cataract

The most common cause of correctable vision loss, a cataract is a gradually developing opacity of the lens or lens capsule of the eye. Cataracts commonly occur bilaterally, with each progressing independently. Exceptions are traumatic cataracts, which are usually unilateral, and congenital cataracts, which may remain stationary. The prognosis is generally good; surgery improves vision in 95% of affected people.

Causes and incidence

Cataracts have various causes:

- Senile cataracts develop in elderly patients, probably because of degenerative changes in the chemical state of lens proteins.
- Congenital cataracts occur in neonates as genetic defects or as a sequela of maternal rubella during the first trimester. They acquire them through autosomal dominant inheritance, which will occur even if only one parent passes it along. Fifty percent of children in such families are affected.
- Traumatic cataracts develop after a foreign body injures the lens with sufficient force to allow aqueous or vitreous humor to enter the lens capsule. Trauma may also dislocate the lens.
- Complicated cataracts develop as secondary effects in patients with uveitis, glaucoma, or retinitis pigmentosa, or in the course of a systemic disease, such as diabetes, hypoparathyroidism, or atopic dermatitis. They can also result from exposure to ionizing radiation or infrared rays.
- Toxic cataracts result from drug or chemical toxicity with prednisone, ergot alkaloids, dinitrophenol, naphthalene, phenothiazines, or pilocarpine or from extended exposure to ultraviolet rays.

Cataracts occur as part of the aging process and are most prevalent in people older than age 70.

Complication

- Vision loss

Signs and symptoms

Characteristically, a patient with a cataract experiences painless, gradual blurring and loss of vision. As the cataract progresses, the normally black pupil appears hazy, and when a mature cataract develops, the white lens may be seen through the pupil. Some patients complain of blinding glare from headlights when they drive at night; others complain of poor reading vision, and of an unpleasant glare and poor vision in bright sunlight. Patients with central opacities report better vision in dim light than in bright light because the cataract is nuclear and, as the pupils dilate, patients can see around the lens opacity.

Diagnosis

On examination, visual acuity is decreased, and the lens opacity remains unnoticeable until the cataract is advanced.

CONFIRMING DIAGNOSIS

Ophthalmoscopy or slit-lamp examination confirms the diagnosis by

revealing a dark area in the normally homogeneous red reflex.

Treatment

Treatment consists of surgical extraction of the cataractous lens opacity and intraoper-ative correction of visual deficits. The current trend is to perform the surgery as a same-day procedure. Surgical procedures include the following:

- Extracapsular cataract extraction (ECCE) removes the anterior lens capsule and cortex, leaving the posterior capsule intact. With this procedure, a posterior chamber intraocular lens (IOL) is implanted where the patient's own lens used to be. (A posterior chamber IOL is currently the most common type used in the United States.) This procedure is appropriate for use in patients of all ages.
- Phacoemulsification uses ultrasonic vibrations to fragment and then emulsify the lens, which is then aspirated through a small incision.
- Intracapsular cataract extraction removes the entire lens within the intact capsule. This procedure is seldom performed today. ECCE with phacoemulsification has replaced it as the most commonly performed procedure.
- Discission and aspiration can still be used for children with soft cataracts, but this procedure has largely been replaced by phacoemulsification.

Infection is the most serious complication of intraocular surgery. Wound dehiscence can occur but is seldom a complication because of the small incision and minute sutures that are used. Hyphema, pupillary block glaucoma, and retinal detachment still occasionally occur.

The patient with an IOL implant may experience improved vision shortly after surgery if there's no corneal or retinal pathology. Most IOLs correct for distance vision, but new IOLs are multifocal. However, the majority of patients will need either corrective reading glasses or a corrective contact lens, which will be fitted sometime between 4 and 6 weeks after surgery.

Where no IOL has been implanted, the patient may be given temporary aphakic cataract glasses; in about 4 to 8 weeks, he'll be refracted for his own glasses.

Some patients who have an extracapsular cataract extraction develop a secondary membrane in the posterior lens capsule (which has been left intact), which causes decreased visual acuity. This membrane can be removed by the Nd:YAG laser, which cuts an area out of the center of the membrane, thereby restoring vision. Laser therapy isn't used to remove a cataract.

Posterior capsular opacification occurs in approximately 15% to 20% of all patients within 2 years after cataract surgery.

Special considerations

After surgery to extract a cataract:

- Because the patient will be discharged after he recovers from anesthesia, remind him to return for a checkup the next day, and warn him to avoid activities that increase intraocular pressure such as straining.
- Urge the patient to protect the eye from accidental injury at night by wearing a plastic or metal shield with perforations; a shield or glasses should be worn for protection during the day.
- Before discharge, teach the patient to administer antibiotic ointment or drops to prevent infection and steroids to reduce inflammation; combination steroid-antibiotic eyedrops can also be used.
- Advise the patient to watch for the development of complications, such as a sharp pain in the eye uncontrolled by analgesics as a result of hyphema, or clouding in the anterior chamber (which may herald an infection), and to report them immediately.
- Caution the patient about activity restrictions, and advise him that it will take several weeks for him to receive his corrective reading glasses or lenses.

PREVENTION

Encourage your patient to:

- *quit smoking*
- *wear sunglasses.*

Retinitis pigmentosa

Retinitis pigmentosa is a group of hereditary disorders whose common feature is a

gradual deterioration of the light-sensitive cells of the retina.

Postmortem examination of the eyes reveals pigment cells that have clumped together as a result of the pigment epithelium budding off and settling within the layers of the retina. Retinitis pigmentosa often accompanies other hereditary disorders in several distinct syndromes—including Usher's syndrome, in which sight and hearing are both affected; and Laurence-Moon-Biedl syndrome (most common), which is typified by visual destruction from retinitis pigmentosa, with obesity, mental retardation, polydactyly, hypogenitalism, and spastic paraplegia.

Causes and incidence

Retinitis pigmentosa can be classified according to its inheritance pattern: autosomal dominant, autosomal recessive, and X-linked. Typically, in all forms of retinitis pigmentosa, the retinal rods slowly deteriorate. Clumps of pigment resembling bone corpuscles aggregate in the peripheral region of the retina and later involve the macular and peripheral areas. Visual symptoms usually appear between ages 10 and 30, though some children may become blind within the first year of life.

Retinitis pigmentosa affects 1 of every 4,000 people in the United States.

Signs and symptoms

Typically, night blindness occurs while the patient is in his teens. As the disease progresses, his visual field gradually constricts, causing tunnel or “gun-barrel” vision. Many people retain this tunnel of useful vision until quite late in life. The speed of vision loss varies considerably from person to person. However, blindness follows invasion of the macular region.

Diagnosis

A detailed family history may imply predisposition to retinitis pigmentosa. In the patient whose history suggests this condition, the following tests help confirm diagnosis.

- Electroretinography shows a slower than normal or absent retinal response time.

- Fluorescein angiography visualizes white dots (areas of dyspigmentation) in the epithelium.
- Ophthalmoscopy may initially show normal fundi but later shows black pigmentary disturbance and white dots (dyspigmentation) in the epithelium.
- Visual field testing (using a tangent screen) detects ring scotomata.

Treatment

No cure exists for retinitis pigmentosa. However, vitamin A and E supplementation may slow degeneration. Researchers are working on a procedure in which fetal retinal tissue is transplanted into people with retinitis pigmentosa, but its potential efficacy is unknown.

Special considerations

- Teach the patient and his family about retinitis pigmentosa.
- Encourage the patient to use sunglasses to protect the retina from ultraviolet light and to help preserve vision.
- Explain that the disorder is hereditary, and suggest genetic counseling for adults who risk transmitting it to their children.
- Encourage annual eye examinations to monitor the progress of the disease.
- Warn the patient that he might not be able to drive a car safely at night.
- Refer the patient to a social service agency or to the National Retinitis Pigmentosa Foundation for information and for counseling to prepare him for eventual blindness.
- Because the prospect of blindness is frightening, it's important that you provide emotional support and guidance.

PREVENTION

Encourage the patient to seek genetic counseling.

MISCELLANEOUS DISORDERS

Optic atrophy

Optic atrophy, or degeneration of the optic nerve, can develop spontaneously (primary) or can follow inflammation or edema of the nerve head (secondary). Some forms of this condition may subside without

treatment, but degeneration of the optic nerve is irreversible.

Causes and incidence

Optic atrophy usually results from central nervous system disorders (such as chiasmal tumors, syphilis, ischemic optic neuropathy, drugs, retinal vascular disease, or degenerative disease) or from end-stage glaucoma. Other causes include retinitis pigmentosa; chronic papilledema and papillitis; glaucoma; trauma; central retinal artery or vein occlusion that interrupts the blood supply to the optic nerve, causing degeneration of ganglion cells; ingestion of toxins, such as methanol and quinine; and deficiencies of vitamin B_{12}, amino acids, and zinc.

There are several rare forms of hereditary optic atrophy that can affect children and young adults.

Signs and symptoms

Optic atrophy causes abrupt or gradual painless loss of visual field or visual acuity, with subtle changes in color vision.

Diagnosis

CONFIRMING DIAGNOSIS

Visual acuity testing reveals poor vision. An afferent pupillary defect is noted when pupils are examined. Fundus examination through a dilated pupil with an ophthalmoscope shows pallor of the nerve head from loss of microvascular circulation in the disk and deposit of fibrous or glial tissue. Visual field testing reveals a scotoma and, possibly, major visual field impairment.

Treatment

Optic atrophy is irreversible, so treatment aims to correct the underlying cause and prevent further vision loss. Steroids may be given to decrease inflammation and swelling, if the cause is found to be ischemic neuropathy. If a space-occupying lesion is the cause, neurosurgery may be required. In multiple sclerosis, optic neuritis often subsides spontaneously but may recur and improve repeatedly.

Special considerations

- Provide symptomatic care during diagnostic procedures and treatment. Assist the patient who's visually compromised to perform daily activities.
- Explain procedures, to minimize anxiety. Offer emotional support to help the patient deal with loss of vision.

Extraocular motor nerve palsies

Extraocular motor nerve palsies are dysfunctions of the third, fourth, and sixth cranial nerves. The oculomotor (third cranial) nerve innervates the inferior, medial, and superior rectus muscles; the inferior oblique extraocular muscles; the pupilloconstrictor muscles; and the levator palpebrae muscles. The trochlear (fourth cranial) nerve innervates the superior oblique muscles. The abducens (sixth cranial) nerve innervates the lateral rectus muscles. The superior oblique muscles control downward rotation, intorsion, and abduction of the eye. Complete dysfunction of the third cranial nerve is called total oculomotor ophthalmoplegia and may be associated with trauma, diabetes, or an intracranial aneurysm.

Causes and incidence

The most common extraocular motor nerve palsy affects the superior oblique muscle as a result of trauma. Other causes of these disorders vary, depending on the cranial nerve involved:

- Third nerve (oculomotor) palsy (acute ophthalmoplegia) may be congenital or acquired. Causes include oculomotor involvement

resulting from intracranial tumors or aneurysms; diabetic neuropathy; and trauma.

- Sixth nerve (abducens) palsy commonly has an unknown etiology. Strokes are a common cause. Brain stem lesions, elevated intracranial pressure, inflamed petrous pyramid due to otitis media, cavernous sinus, orbital involvement with tumor and inflammation, or thyroid eye disease may be responsible for sixth nerve palsy.

There are approximately 4,500 cases of acquired extraocular motor nerve palsies in the United States. The incidence of congenital extraocular nerve palsies is unknown.

Signs and symptoms

The most characteristic clinical effect of extraocular motor nerve palsies is diplopia of recent onset, which varies in different visual fields, depending on the muscles affected.

Typically, the patient with third nerve palsy exhibits ptosis, exotropia (eye looks outward), pupil dilation, and unresponsiveness to light; the eye is unable to move and can't accommodate.

The patient with fourth nerve palsy displays diplopia and an inability to rotate the eye downward or upward. The head is tilted to the side opposite the involved area in superior oblique palsy.

Sixth nerve palsy causes one eye to turn; the eye can't abduct beyond the midline. To compensate for diplopia, the patient turns his head to the unaffected side and can develop torticollis.

Diagnosis

Diagnosis necessitates an orthoptic examination to isolate the involved muscle, a complete neuro-ophthalmologic examination, and a thorough patient history. Differential diagnosis of third, fourth, or sixth nerve palsy depends on the specific motor defect exhibited by the patient.

For all extraocular motor nerve palsies, a computed tomography scan or magnetic resonance imaging rules out tumors and may help detect the cause of the palsy, such as the cause of increased intracranial pressure.

The patient is also evaluated for an aneurysm or diabetes. If sixth nerve palsy results from infection, culture and sensitivity tests identify the causative organism, and specific antibiotic therapy can be determined.

Treatment

Identification of the underlying cause is essential because treatment for extraocular motor nerve palsies varies accordingly. Neurosurgery is necessary if the cause is a brain tumor or an aneurysm. For infection, massive I.V. doses of antibiotics may be appropriate.

Special considerations

- If the palsy results from thyroid eye disease, the patient must have normal thyroid levels before eye muscle surgery is attempted.

Glaucoma

Glaucoma is a group of disorders characterized by an abnormally high intraocular pressure (IOP), which can damage the optic nerve. If untreated, it can lead to gradual peripheral vision loss and, ultimately, blindness. (See *Blindness*.) Glaucoma occurs in several forms: chronic open-angle (primary), acute angle-closure, congenital (inherited as an autosomal recessive trait), and secondary to other causes. The prognosis for maintaining vision is good with early treatment.

Causes and incidence

Chronic open-angle glaucoma results from overproduction of aqueous humor or obstruction to its outflow through the trabecular meshwork or the canal of Schlemm. (See *Normal flow of aqueous humor,* page 686.) This form of glaucoma, which is estimated to be present in 1% to 2% of people older than age 40, is frequently familial in origin and affects 90% of all patients with glaucoma. Diabetes and systemic hypertension have also been associated with this form of glaucoma.

Acute angle-closure (narrow-angle) glaucoma results from obstruction to the outflow of aqueous humor due to anatomically narrow angles between the anterior iris and the posterior corneal surface, shallow anterior chambers, a thickened iris that causes angle closure on pupil

dilation, or a bulging iris that presses on the trabeculae, closing the angle (peripheral anterior synechiae).

Blacks are four times more likely to have this disorder than Whites, and people with a family history of open-angle glaucoma are twice as likely to develop it than people

without a family history of this disorder. The use of systemic anticholinergic medications, such as atropine or eye dilation drops, in a person who's already at high-risk for acute glaucoma increases the risk. Other risk factors include farsightedness and age-related changes that create an increase in intraocular pressure.

Congenital glaucoma occurs when there is an abnormal fluid drainage angle of the eye. It may be caused by congenital infections such as TORCH virus (*t*oxoplasmosis, *o*ther [varicella, mumps, parvovirus, human immunodeficiency virus], *r*ubella, *c*ytomegalovirus, and *h*erpes), Sturge-Weber syndrome, or retinopathy of prematurity.

Secondary glaucoma can result from uveitis, trauma, or drugs (such as steroids). Neovascularization in the angle can result from vein occlusion or diabetes.

Complications

- Blindness
- Vision loss

Signs and symptoms

Chronic open-angle glaucoma is usually bilateral, with insidious onset and a slowly progressive course. Symptoms appear late in the disease and include mild aching in the eyes, loss of peripheral vision, seeing halos around lights, and reduced visual acuity (especially at night) that isn't correctable with glasses.

Acute angle-closure glaucoma typically has a rapid onset, constituting an ophthalmic emergency. Symptoms include acute pain in a unilaterally inflamed eye, with pressure over the eye, moderate pupil dilation that's nonreactive to light, a cloudy cornea, blurring and decreased visual acuity, photophobia, and seeing halos around lights. Increased IOP may

induce nausea and vomiting, which may cause glaucoma to be misinterpreted as GI distress. Unless treated promptly, this acute form of glaucoma produces blindness in 3 to 5 days.

Diagnosis

CONFIRMING DIAGNOSIS

Loss of peripheral vision and disk changes confirm that glaucoma is present. Diagnosis is made by:

BLINDNESS

Blindness affects 28 million people worldwide. In the United States, blindness is legally defined as optimal visual acuity of 20/200 or less in the better eye after best correction, or a visual field of 20 degrees or less in the better eye.

According to the World Health Organization, the most common causes of preventable blindness worldwide are trachoma, cataracts, onchocerciasis (microfilarial infection transmitted by a blackfly and other species of *Simulium*), and xerophthalmia (dryness of conjunctiva and cornea from vitamin A deficiency).

In the United States, the most common causes of acquired blindness are glaucoma, age-related macular degeneration, and diabetic retinopathy. However, the incidence of blindness from glaucoma is decreasing owing to early detection and treatment. Rarer causes of acquired blindness include herpes simplex keratitis, cataracts, and retinal detachment.

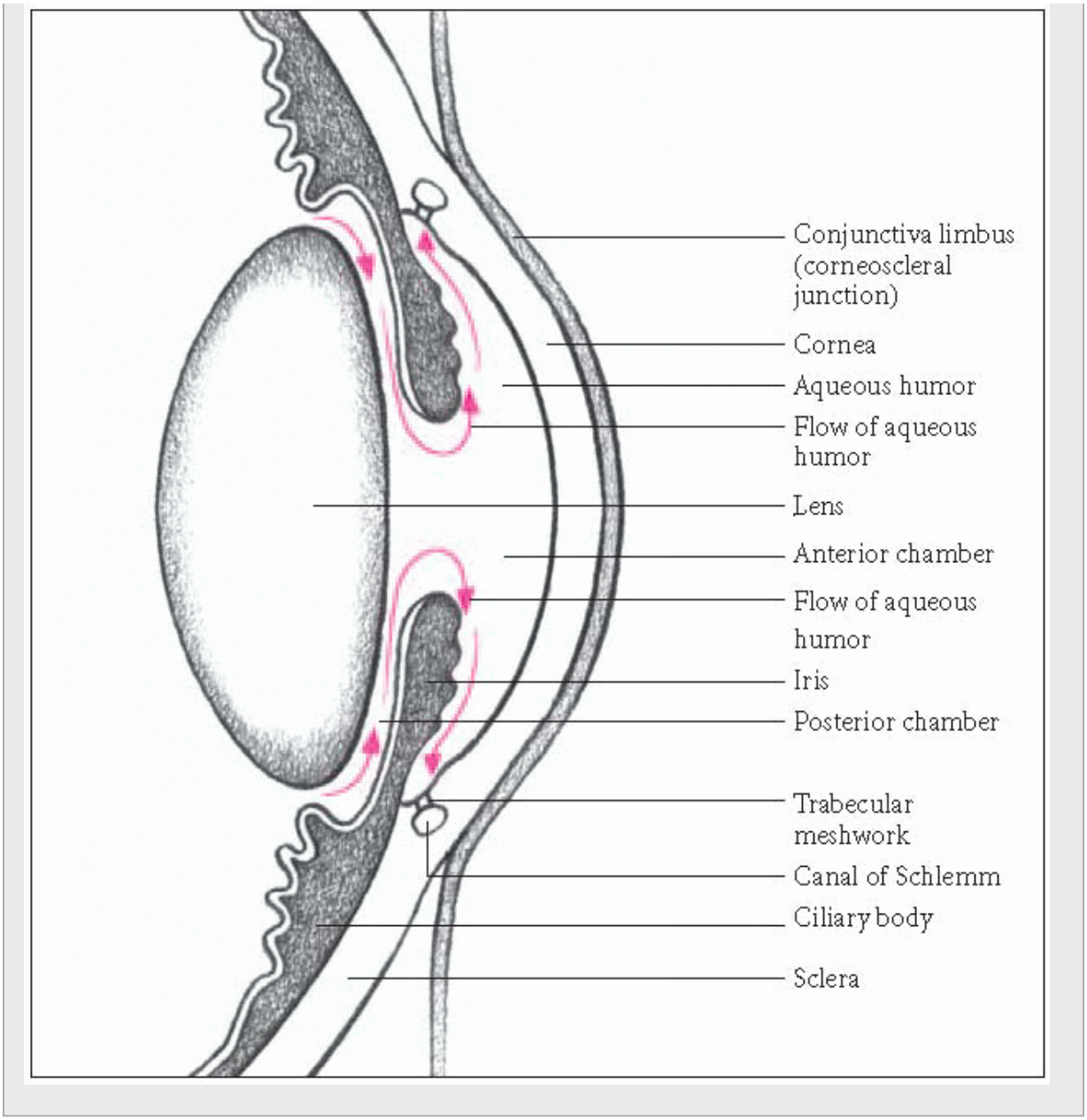
- *testing IOP*
- *measuring the visual field and noting changes, such as an enlarged blind spot and loss of peripheral vision field*
- *observing changes in the cup/disk ratio of the optic nerve head.*

Relevant diagnostic tests include:

- Tonometry (using an applanation tonometer [Tonopen] or air puff tonometer)—This test measures the IOP and provides a baseline for reference. Normal IOP ranges from 8 to 21 mm Hg. However, patients who fall within this normal range can develop signs and symptoms of glaucoma, and patients who have abnormally high pressure may have no clinical effects. Fingertip tension is another way to measure IOP. On gentle palpation of closed eyelids, one eye feels harder than the other in acute angle-closure glaucoma.

NORMAL FLOW OF AQUEOUS HUMOR

Aqueous humor, a plasmalike fluid produced by the ciliary epithelium of the ciliary body, flows from the posterior chamber to the anterior chamber through the pupil. Here it flows peripherally and filters through the trabecular meshwork to the canal of Schlemm, through which the fluid ultimately enters venous circulation.



- Slit-lamp examination—The slit lamp facilitates examination of the anterior structures of the eye: the cornea, iris, and lens.
- Gonioscopy—By determining the angle of the anterior chamber of the eye, this test enables differentiation between chronic open-angle glaucoma and acute angle-closure glaucoma. The angle is normal in chronic open-angle glaucoma. However, in older patients, partial closure of the angle may occur, so that two forms of glaucoma may co-exist.

- Ophthalmoscopy—This test enables the examiner to look at the fundus to establish if there are any cup/disk ratio changes. (See *Optic disk changes*.) These changes appear later in chronic glaucoma if the disease isn't brought under control.
- Fundus photography—Pictures of the optic nerve head are made to track changes.
- Perimetry or visual field tests—These reveal the extent of damage to the optic neurons, signaled by an enlarged blind spot and loss of peripheral vision.

Treatment

For chronic open-angle glaucoma, treatment initially decreases IOP through the use of an alpha antagonist, brimonidine tartrate (Alphagan), and then beta blockers, such as timolol (contraindicated for asthmatics or patients with bradycardia) or betaxolol (Betoptic) to reduce aqueous humor production. A topical anhydrase inhibitor is used in preference to a systemic anhydrase inhibitor such as acetazolamide. A tubo-plast or tube shunt or valve may also be used. Miotic eyedrops such as pilocarpine facilitate the outflow of aqueous humor.

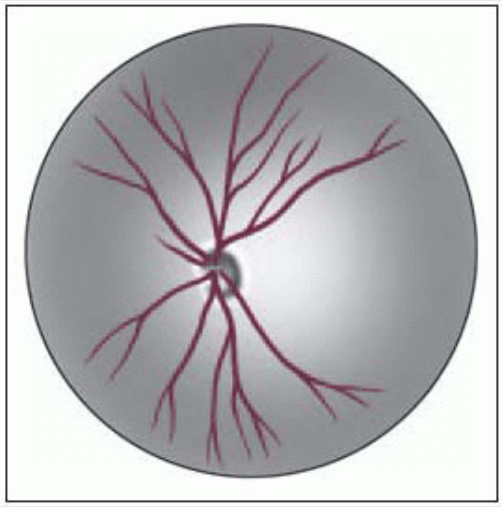
Patients who are unresponsive to drug therapy may be candidates for argon laser trabeculoplasty (ALT) or a surgical filtering procedure called trabeculectomy, which creates an opening for aqueous outflow. In ALT, an argon laser beam is focused on the trabecular meshwork of an open angle. This produces a thermal burn that changes the surface of the meshwork and increases the outflow of aqueous humor. In trabeculectomy, a flap of sclera is dissected free to expose the trabecular meshwork. Then this discrete tissue block is removed and a peripheral iridectomy is performed. This produces an opening for aqueous outflow under the conjunctiva, creating a filtering bleb. In chronic refractory glaucoma, a tubo-plast or tube shunt or valve is used to keep IOP within normal limits.

Acute angle-closure glaucoma is an ocular emergency requiring immediate treatment to lower the high IOP. Preoperative drug therapy lowers IOP with I.V. acetazolamide, pilocarpine (constricts the pupil, forcing the iris away from the trabeculae, allowing fluid to escape),

timolol, and a topical steroid to quiet the inflammatory response, along with I.V. mannitol (20%) or oral glycerin (50%) to force fluid from the eye by making the blood hypertonic. Latanoprost is a topical medication that helps drain the aqueous outflow from the eye and lower the IOP. Oral medication or topical drops may be prescribed separately or in combination. Severe pain may necessitate administration of opioid analgesics. If pressure doesn't decrease with drug therapy, laser iridotomy or surgical peripheral iridectomy must be performed promptly to save the patient's vision. Iridectomy relieves pressure by excising part of the iris to reestablish aqueous humor outflow. A prophylactic iridectomy is performed a few days later on the other eye to prevent an acute episode of glaucoma in the normal eye.

OPTIC DISK CHANGES

Ophthalmoscopy and slit-lamp examination show cupping of the optic disk, characteristic of chronic glaucoma.



Special considerations

- Stress the importance of meticulous compliance with prescribed drug therapy to prevent an increase in IOP, resulting in disk changes and loss of vision.
- For the patient with acute angle-closure glaucoma, give medications as ordered, and prepare him physically and psychologically for laser iridotomy or surgery.

- Postoperative care after peripheral iridectomy includes cycloplegic eyedrops to relax the ciliary muscle and to decrease inflammation, thus preventing adhesions.

ALERT

Cycloplegics must be used only in the affected eye. The use of these drops in the normal eye may

precipitate an attack of acute angle-closure glaucoma in this eye, threatening the patient's residual vision.

- Encourage ambulation immediately after surgery.
- Following surgical filtering, postoperative care includes dilation and topical steroids to rest the pupil.
- Stress the importance of glaucoma screening for early detection and prevention. All people older than age 35, especially those with family histories of glaucoma, should have an annual tonometric examination.

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