SECTION 19

Eye, Ear, Nose, Throat, and Oral Disorders

CHAPTER 241

Eye Emergencies

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INTRODUCTION AND EPIDEMIOLOGY

The breadth of ocular emergencies seen in the ED requires solid examination skills and an understanding of basic differential diagnosis. A recent review[1] of 1400 ED ocular emergencies identified the following conditions: ocular trauma in 27%, of which 73% involved corneal abrasions, 6% involved blunt eye trauma, and 5% involved a corneal foreign body; the second most common condition was conjunctivitis (15%), and retinal problems and glaucoma involved 6%.

This chapter reviews eye anatomy, the essential skills needed for the ED eye examination, and common ophthalmic medications. Common causes of the red eye, ocular infections and inflammation, trauma to the eye, acute visual reduction or loss, and acute cranial nerve palsies are discussed. The principles and advantages of ocular US are summarized.

EYE ANATOMY

The **orbit** is a pyramid of bony walls that converge to an apex posteriorly. The orbit is bordered superiorly by the frontal sinus, medially by the ethmoid sinus, inferiorly by the maxillary sinus, and laterally by the zygomatic bone. The ethmoid bone (lamina papyracea) is paper thin and is the most likely sinus wall to break in blunt eye trauma or to be perforated due to sinusitis with subsequent spread of infection to the orbit. The orbital contents include the ocular muscles, retroseptal fat, and the optic nerve, whereas the globe is considered a separate entity.

The anterior limit of the orbital cavity is the **orbital septum**, which is a layer of fascia extending from the periosteum along the orbital rim to the levator aponeurosis of the upper eyelid and to the edge of the tarsal plate of the lower eyelid. Abnormalities, such as the accumulation of blood or infection, are referred to as "preseptal" or "postseptal." Postseptal conditions are extremely serious. **The septum is generally impervious to bacteria, which serves to limit spread of infection from the facial skin into the orbit (Figure 241-1).** All nerves and vessels of the eye enter through the apex of the orbit, which is also the site of origin for the extraocular muscles. The optic nerve is subject to compression from mass effect due to tumors, abscesses, or hematomas.

The arterial blood supply of the eye and orbit is the ophthalmic artery, the first major branch of the intracranial portion of the internal carotid artery, which enters the orbit beneath the optic nerve. The **central retinal artery** is the first intraorbital branch of the ophthalmic artery and courses through the optic nerve. The venous drainage of the eye and orbit is through the ophthalmic veins, which drain into the central retinal vein. The ophthalmic veins communicate directly to the cavernous sinus. This venous system has no valves, and this fact is the basis for the spread of facial and periorbital infections to the cavernous sinus.

The eye itself is composed of several different layers (**Figure 241-2**). The outermost layer is a thin, transparent mucous membrane (the **bulbar conjunctiva**) that continues onto the posterior surface of the eyelids (the **palpebral conjunctiva**). Deep to the conjunctiva is the **episclera**, a layer of thin, elastic tissue containing blood vessels that nourish the next deepest layer, the sclera. The **sclera** is the collagenous protective coating of the eye, which is the thinnest (and prone to rupture) at the insertion of the rectus muscles.

The **cornea** forms the anterior surface of the eyeball and is attached to the sclera at the limbus. From anterior to posterior, the cornea has five separate layers: epithelium, Bowman layer, stroma, Descemet membrane, and endothelium. The epithelium is five or six cell layers thick and is subject to damage from minor mechanical forces, resulting in corneal abrasion (**Figure 241-3**).

The iris, ciliary body, and choroid (the vascular pigmented layer of the eye between the sclera and retina) make up the **uveal tract**. The uveal tract supplies nutrition to the eye and assists in accommodation and pupillary constriction. The lens separates the aqueous humor in the anterior chamber from the vitreous humor in the remainder of the globe. (See the section **Acute and Painful Vision Reduction or Loss, Acute Angle-Closure Glaucoma** in this chapter for discussion of the production and flow of aqueous humor.) The retina is the sheet of neural tissue containing the rods and cones that lines the posterior two thirds of the inner surface of the globe, extending anteriorly as far as the ciliary body.

EYE EXAMINATION

HISTORY

A detailed history is as important in the patient with an eye complaint as it is for a complaint related to any other organ system. History should first categorize the symptom as vision loss, change in appearance of the eye, eye pain/discomfort, or trauma. The onset (gradual or sudden) of symptoms, duration of the symptoms, and circumstances surrounding the onset are important. For example, a history of sudden, painless monocular vision loss associated with a history of atrial fibrillation or carotid stenosis would suggest a central retinal artery occlusion, but a history of eye pain occurring while hammering metal on metal would suggest a projectile causing corneal abrasion or intraocular foreign body. Eye discomfort should be characterized as pain (aching, burning, throbbing, etc.), pruritus (associated with allergy), or a foreign body sensation as seen with corneal foreign bodies, abrasions, or ulcers. "Flashing lights" and a "curtain or veil" obstructing a portion of the visual field suggest a retinal detachment. In the case of trauma, ask about onset (traumatic iritis occurs 1 to several days after blunt trauma to the eye) and mechanism (globe penetration may occur in association with hammering, grinding, or use of other high-speed machinery). Document tetanus status and give tetanus toxoid as appropriate.

Past medical history is always important and can focus the physical examination and narrow the differential diagnosis. Previous surgery may be the cause of an irregular pupil. Absence of corrective lenses may account for decreased visual acuity and will require modification of the method for testing visual acuity. Use of contact lenses, especially the extended wear type, may be associated with bacterial corneal ulcers. Chronic use of certain ophthalmic medications may cause chemical conjunctivitis and inflammatory changes of the cornea. A history of diabetes or chronic hypertension and acute isolated sixth-nerve palsy suggests an ischemic cranial neuropathy. Monocular diplopia following trauma in a patient with an intraocular lens implant suggests dislocation of the intraocular lens. Always ask about previous instances of similar **symptoms** and the associated diagnosis.

EXAMINATION

The eye examination typically proceeds in a sequential fashion unless the circumstances require otherwise (e.g., **chemical ocular injuries**

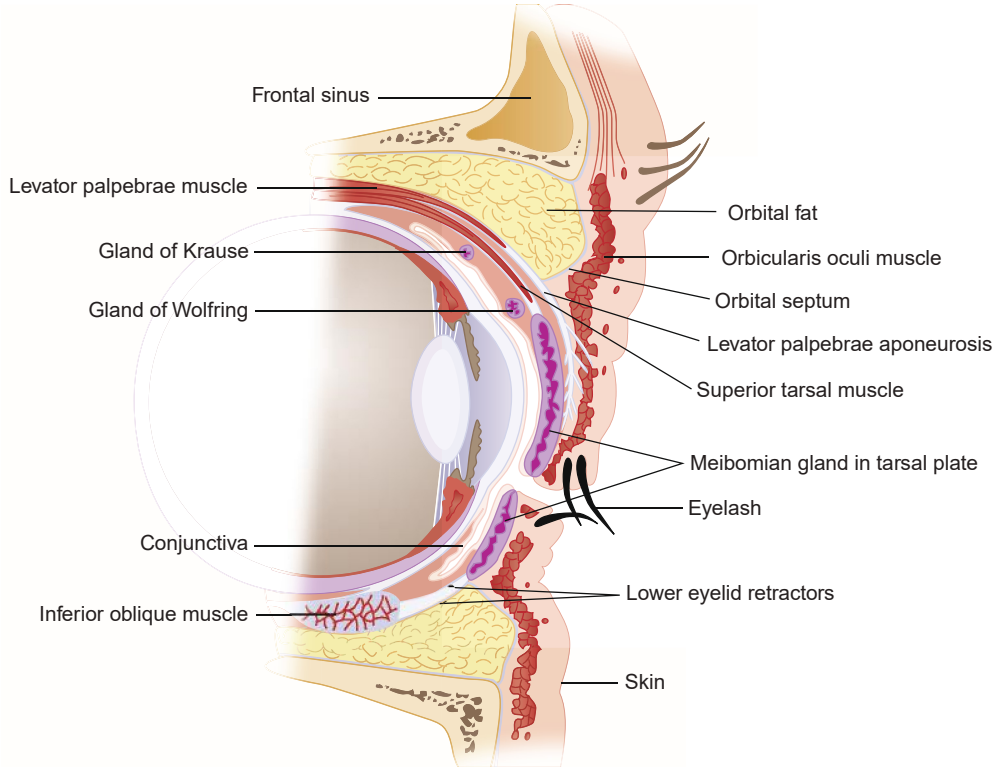


FIGURE 241-1. Cross-section of the eyelids. [Reproduced with permission from Riordan-Eva P, Whitcher J: *Vaughn & Asbury's General Ophthalmology,* 17th ed. New York: Lange Medical Books/McGraw-Hill, 2008.]

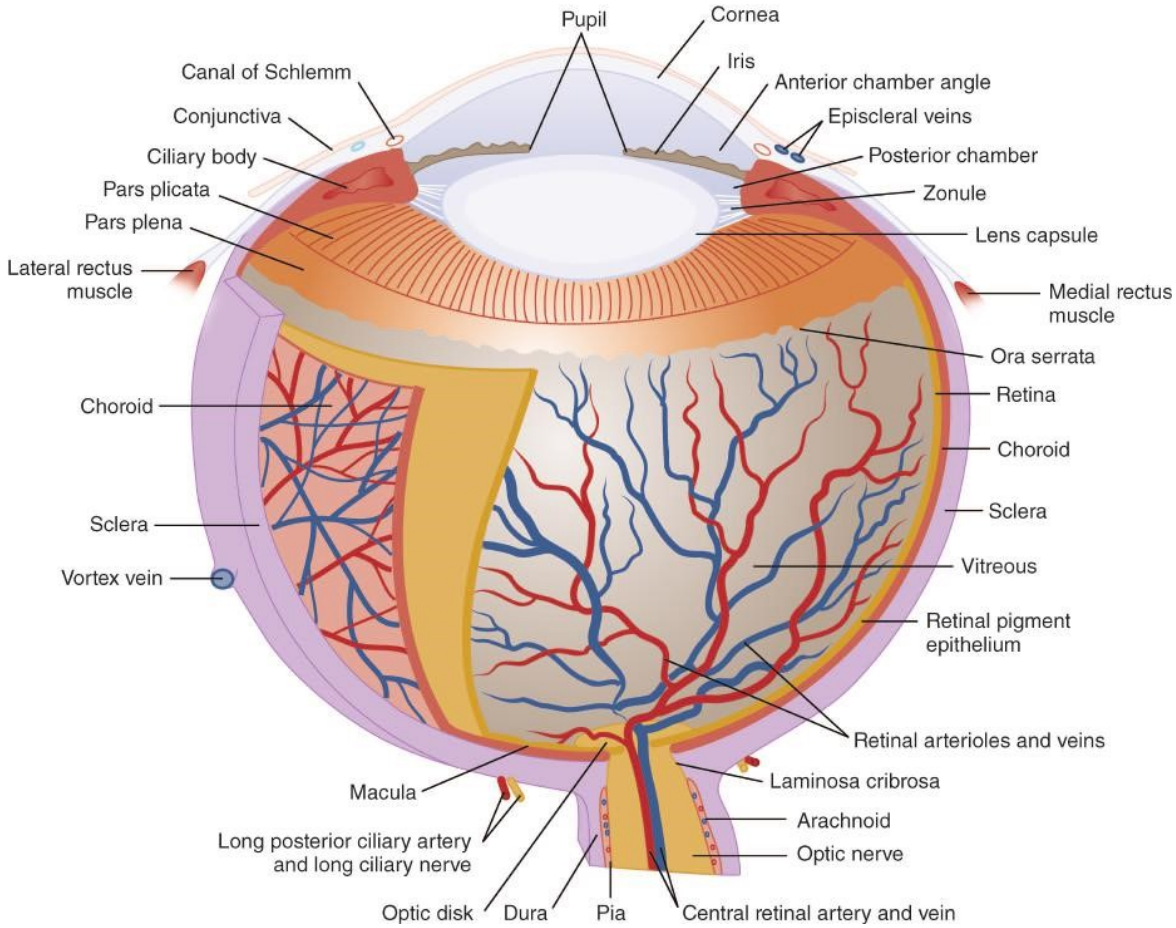


FIGURE 241-2. Internal structures of the human eye. [Reproduced with permission from Riordan-Eva P, Whitcher J: *Vaughn & Asbury's General Ophthalmology,* 17th ed. New York: Lange Medical Books/McGraw-Hill, 2008.]

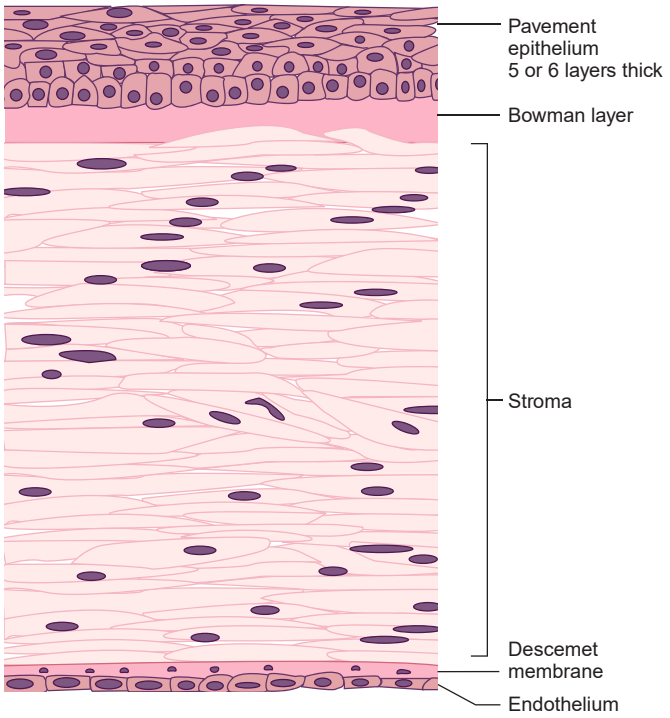


FIGURE 241-3. Transverse section of cornea. [Reproduced with permission from Riordan-Eva P, Whitcher J: *Vaughn & Asbury's General Ophthalmology,* 17th ed. New York: Lange Medical Books/McGraw-Hill, 2008.]

require irrigation *before* assessment of visual acuity). The glossary of terms and abbreviations in **Table 241-1** is helpful when communicating with the ophthalmologist.

Full examination should include the following, generally in the order listed: visual acuity, confrontational visual fields, extraocular movements, pupillary reactions, lids and adnexa, conjunctiva and sclerae, cornea, anterior chamber, iris, lenses, vitreous, intraocular pressure, and funduscopic examination. Measurement of intraocular pressure is done toward the end of the examination because physical touching of the cornea is more irritating and invasive than the rest of the examination. Performance of a thorough funduscopic examination requires dilatation of the pupil, so this part of the examination is performed last. Not all parts of the examination need to be done on every patient. For example, testing visual fields adds little to the evaluation of a corneal foreign body but is essential to the evaluation of acute vision loss.

Visual Acuity Most vision-threatening disorders present with decreased visual acuity. **Visual acuity testing is the vital sign of the eye** and is the first step in any eye examination, even before shining a light in the eye; bright light can temporarily decrease visual acuity. The only exception to this rule is for chemical burns to the eye, where irrigation takes precedence above all else. Test visual acuity with contact lenses or glasses in place if possible. If the patient's glasses or contacts are unavailable, use **pinhole testing** of visual acuity. A commercial pinhole occluder may be used, although a perforated metal eye shield or a note card perforated with an 18-gauge needle are acceptable substitutes. The pinhole allows only parallel light rays to fall on the macula, thereby reducing the refractive error and allowing an estimate of the person's corrected visual acuity. Visual acuity testing is ideally done with a standard wall-mounted visual acuity chart (**Snellen chart**) with the patient standing 20 ft (6 m) from the chart. Record the visual acuity as 20/x, where the numerator is the distance from which the patient can read the line (always 20) and the denominator is the distance from which a person with normal vision can read the same line. **The visual acuity is determined by the smallest line a patient can read with one half of the letters correct.** The number of incorrect letters is listed after the visual acuity as follows: 20/x-y (e.g., 20/40-2). Document best acuity in each eye and whether prosthetic devices were used in testing (glasses, pinhole).

Visual acuity can also be tested with a near card (**Rosenbaum chart**) held 14 in. (36 cm) from the patient. Patients in their mid-40s or older may require reading glasses or bifocals to read a near card because of presbyopia. If the bifocals are not available, use a pinhole occluder.

For patients with visual acuity <20/200, figure counting at a distance (e.g., figure counting at 3 ft or 1 m), perception of hand motion

TABLE 241-1 Glossary of Terms, Abbreviations, and Notations

AC	Anterior chamber, the first portion of the anterior segment.	IOP	Intraocular pressure (mm Hg).
Anisocoria	Unequal pupil size under equal lighting conditions.	Limbus	Circumferential border where clear cornea ends and white sclera begins.
Anterior segment	Consists of the anterior chamber and posterior chamber. Aqueous humor is produced in the posterior chamber of the anterior segment and circulates through the pupil into the anterior chamber of the anterior segment.	NLP	No light perception (blind).
		OD	Oculus dexter (right eye).*
		OS	Oculus sinister (left eye).
APD	Afferent pupillary defect (see **Figure 241-8**).	OU	Oculus uterque (each eye).*
CF	Counting fingers (visual acuity assessment).	PH	Pinhole visual acuity.
CVF	Confrontation visual fields.	RD	Retinal detachment.
EOM	Extraocular muscle. Extraocular movements.	Tono-Pen® (Reichert, Inc., Depew, NY)	A hand-held, pen-shaped device for measuring IOP.
HM	Hand motion (visual acuity assessment).		
Hyphema	RBCs in the anterior chamber.	T_{tono}	Tension (IOP) with subscript representing method used: (tono = Tono-Pen®; S = Schiötz; A = applanation).†
Hypopyon	WBCs in the anterior chamber.		
INO	Internuclear ophthalmoplegia.	V_{Ac}	Visual acuity with correction (glasses or contact lenses).†
IOFB	Intraocular foreign body.	V_{As}	Visual acuity without correction.

*By convention, in documenting the visual acuity (V_A) or IOP, the right eye is listed above the left, as follows:

$T_{tono} < \begin{matrix}14\\15\end{matrix}$ †This represents an IOP of 14 mm Hg in the right eye and 15 mm Hg in the left eye measured by Tono-Pen®.

$V_{Ac} < \begin{matrix}20/20\\20/30\end{matrix}$ †This represents a visual acuity *with* glasses/contacts of 20/20 right eye and 20/30 left eye.

$V_{As} < \begin{matrix}20/400 \rightarrow 20/30\\CF \text{ at } 8 \text{ ft} \rightarrow 20/40\end{matrix}$ This represents a visual acuity *without* glasses/contacts of 20/400 in the right eye, improving to 20/30 with pinhole testing; counting fingers at 8 ft (2.4 m) in the left eye, improving to 20/40 with pinhole testing.

at 1 to 2 ft (0.3 to 0.6 m), and ultimately light perception can be used to document visual acuity. If the patient is unable to detect hand motion, turn off all the lights in the room, fully occlude the contralateral eye, and test for light perception. Illiterate patients can be tested using the direction of the letter **E** on the chart, and a verbal child may be tested with an **Allen chart** (pictures). Corneal abrasions or foreign bodies can cause severe photophobia, pain, and tearing, so a topical anesthetic can reduce discomfort sufficiently to allow a more accurate assessment of visual acuity. In recording the results of visual acuity testing, refer to **Table 241-1.**

When alternating black-and-white lines are passed from one side to another in front of a patient's eyes, **involuntary horizontal nystagmus (optokinetic nystagmus)** will occur. The presence of optokinetic nystagmus excludes blindness in a patient with an otherwise normal examination who claims he or she cannot see (hysterical blindness). The test can be performed by placing thick black lines approximately 1 in. apart on a 2-ft strip of cardiac monitor paper, which is passed back and forth at eye level a distance of 1 ft from the patient.

Confrontation Visual Fields Test all four quadrants of the visual fields by having the patient cover one eye and look at the physician's nose. The examiner closes the opposite eye and holds a finger halfway between the patient and himself or herself. The finger is wiggled as it is moved medially toward the patient. The normal patient should see movement at approximately the same time as the physician does. A visual field defect may represent pathology anywhere from the occipital cortex to the optic nerve (**Figure 241-4**). Bitemporal hemianopia can occur in pituitary adenoma; homonymous hemianopia is associated with some cerebrovascular accidents; and monocular field cuts are sometimes seen with large retinal detachments.

Ocular Motility The normal patient can move the eye through the six cardinal positions of gaze, and the eye movements are controlled by the six extraocular muscles attached to each eye (**Figures 241-5** and **241-6**). Extraocular muscles are innervated by cranial nerves III, IV, and VI. **Cranial nerve IV controls the superior oblique muscle, cranial nerve**

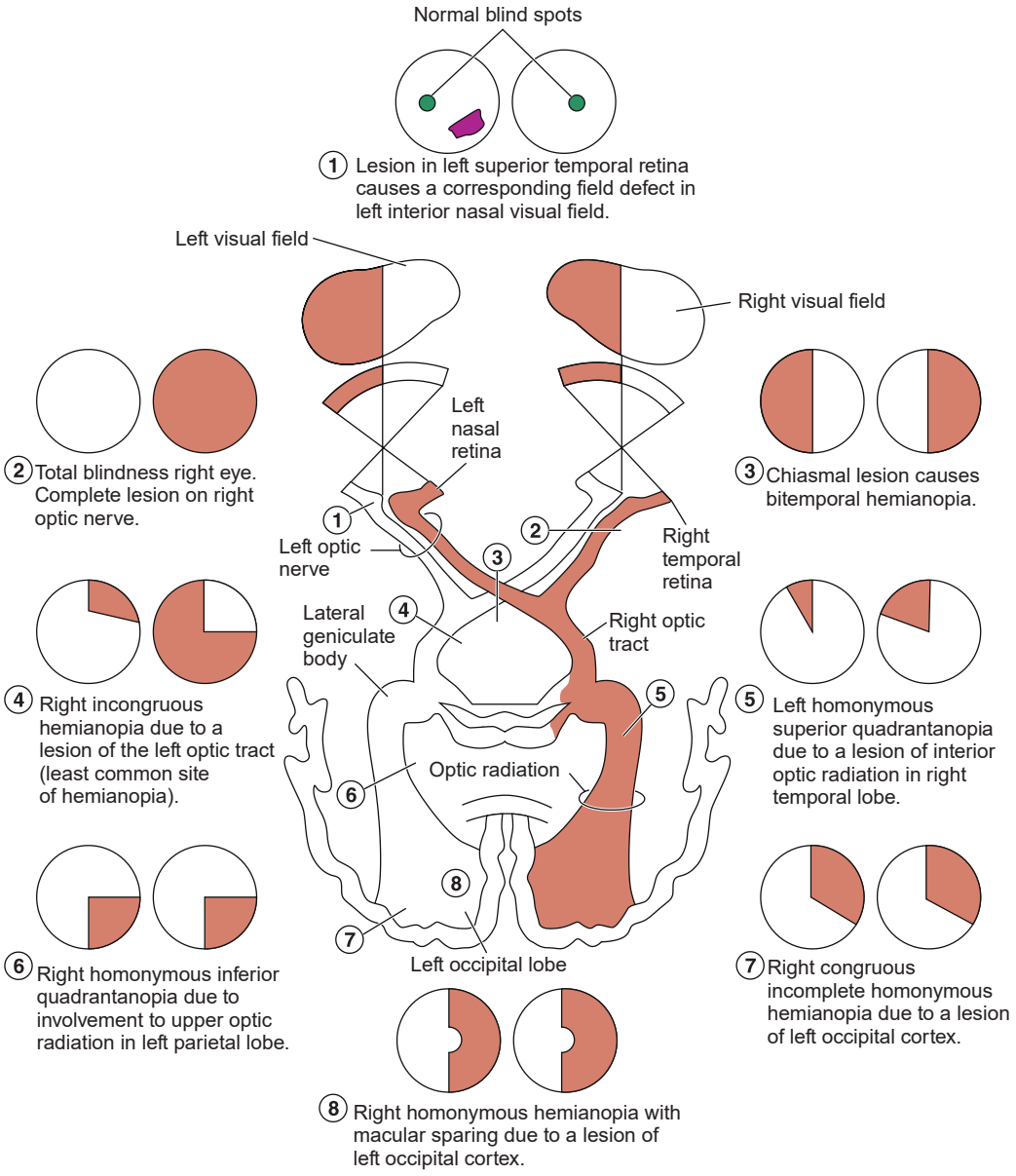


FIGURE 241-4. Visual field defects produced by lesions at various points along the optic pathways: (*1*) field defect caused by retinal lesion, (*2*) total blindness right eye, (*3*) bitemporal hemianopia, (*4*) right incongruous hemianopia, (*5*) left homonymous superior quadrantanopia, (*6*) right homonymous inferior quadrantanopia, (*7*) right congruous incomplete homonymous hemianopia, and (*8*) right homonymous hemianopia with macular sparing. [Reproduced with permission from Riordan-Eva P, Whitcher J: *Vaughn & Asbury's General Ophthalmology,* 17th ed. New York: Lange Medical Books/McGraw-Hill, 2008.]

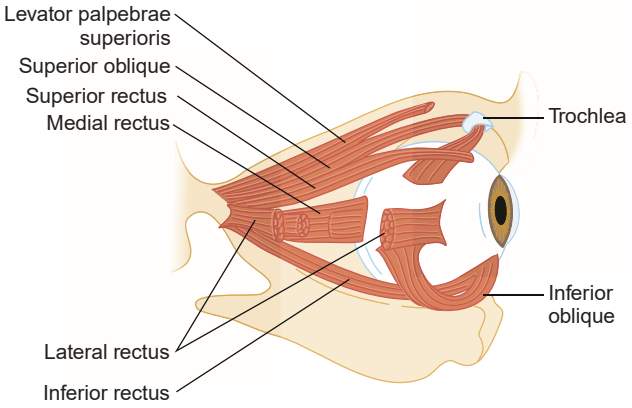


FIGURE 241-5. Extraocular muscles of the eye.

VI controls the lateral rectus muscle, and all other extraocular muscles are controlled by cranial nerve III. Extraocular movement can be impaired by restriction, interrupted or decreased innervation, or trauma. Examples of restriction include thyroid orbitopathy, myositis, and mechanical entrapment of a muscle secondary to an orbital blow-out fracture. Cranial nerve palsies or paresis may be caused by stroke, myasthenia gravis, diabetes, hypertension, tumors, aneurysms, infections, and trauma. Penetrating or blunt traumatic injury to an extraocular muscle also can result in motility disturbance.

Evaluate ocular alignment initially in *primary gaze* (looking straight ahead), and then test eye movements in all fields of gaze. Always ask the patient about diplopia, which may be a subtle sign of problems with extraocular muscles. Diplopia is usually worse when the patient is attempting to look in the direction of the malfunctioning muscle. Ask patients if diplopia persists when one eye is covered (monocular diplopia). **Monocular diplopia** can be caused by corneal irregularity, lens problems, or intraocular lens dislocation, or can be a sign of malingering. Resolution of diplopia when one eye is covered represents pathology of an extraocular muscle or its innervation. Patients with lesions of the superior oblique muscle or the fourth cranial nerve may tilt their head to compensate for the diplopia.

Pupils Note the pupil size in millimeters and test shape and reaction to light. An irregular pupil may occur from prior surgery or remote trauma. The patient will usually be able to relate a previous history of irregular pupil. The classic irregular teardrop-shaped pupil may also be seen in acute blunt or penetrating trauma with rupture of the iris (**Figure 241-7**).

Assess pupils under slightly dim light to test for an afferent pupillary defect (**Figure 241-8**). **A positive afferent pupillary defect indicates an optic nerve disorder.** Any pathology that prevents light from getting to the CNS, such as opacification of the vitreous with blood, retinal pathology, or optic nerve pathology, will cause an afferent pupillary defect, also known as a ***Marcus-Gunn pupil***. The pupils will be equal in size *before* testing because of the consensual light response. Therefore, an afferent pupillary defect does *not* cause a baseline anisocoria and will be discovered only if specifically tested for. Perform the "swinging flashlight test" to detect an afferent pupillary defect. Shine a light in the pupil. The light causes constriction of the ipsilateral pupil and consensual constriction of the opposite pupil. The light is then shined/swung to the opposite pupil. The opposite pupil will dilate if an afferent pupillary defect is present, because the effect of light is not getting through to the CNS.

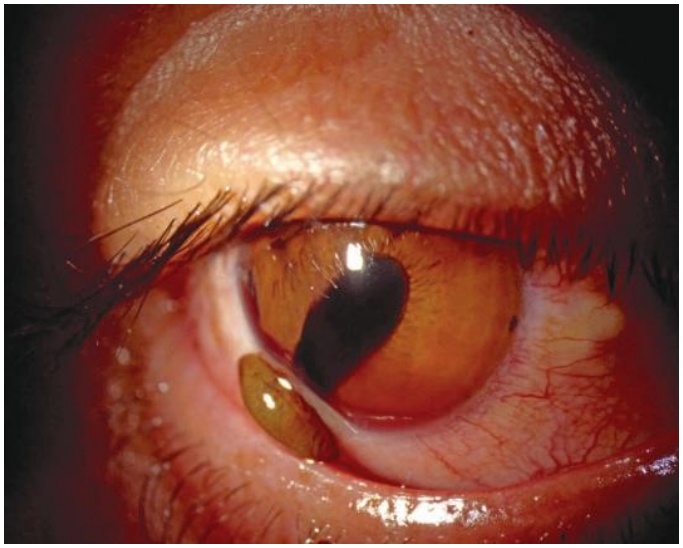


FIGURE 241-7. Prolapse of the iris with the classic teardrop-shaped pupil after penetrating trauma. [Courtesy of Allen R. Katz, Department of Ophthalmology, University of Nebraska Medical Center.]

Causes of unequal pupils (**anisocoria**) can range from an acute emergency (posterior communicating artery aneurysm) to chronic baseline conditions such as previous intraocular trauma or surgery, or they can be idiopathic. **Physiologic anisocoria (difference in pupil size) is the most common cause of asymmetric pupils.** The difference in size is usually <1 mm, and both pupils react normally by constricting to light and dilating in darkness. A single dilated pupil may represent impending **uncal herniation** (from pressure on the third nerve), but uncal herniation is accompanied by an altered level of consciousness and other focal neurologic signs. A single nonreactive dilated pupil may result from a topical cycloplegic agent (scopolamine, cyclopentolate, or atropine) for uveitis or if an anticholinergic medication (such as ipratropium from a nebulization treatment for bronchospasm) is splashed into the eye. A careful history is important to determine whether anisocoria is preexisting. **It is not worthwhile to attempt to "reverse" a suspected chemically altered pupil in the ED as a diagnostic test because the results are not reliable.**

External Eye: Periorbital Skin, Lids, and Adnexa The ocular adnexa include the eyebrows, eyelids, and lacrimal apparatus. Examine the periorbital skin and lids for trauma, infection, dysfunction, deformity, crepitus, or proptosis. Subcutaneous emphysema can be found with blow-out fractures of the medial orbital wall (ethmoid). Palpate the orbital rims for step-off deformities in trauma. Evert the upper eyelid to check for foreign bodies. Use of a cotton applicator is often recommended; however, this technique will only visualize the lower half of the inner upper eyelid (**Figure 241-9**). The edge of an eyelid retractor may be used to tent the upper lid while a second examiner looks under the lid from a caudal direction, so-called *double eversion of the eyelid*. This will allow visualization of the upper half of the inner eyelid. A large

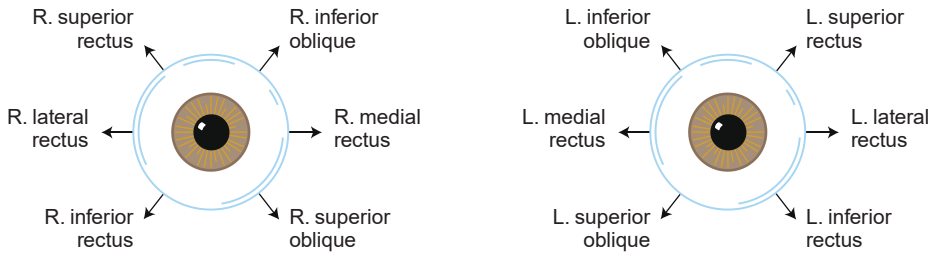


FIGURE 241-6. Arrows indicate direction of ocular movement by each muscle. Cranial nerve IV, superior oblique muscle; cranial nerve VI, lateral rectus muscle; cranial nerve III, superior rectus, inferior rectus, inferior oblique, and medial rectus muscles.

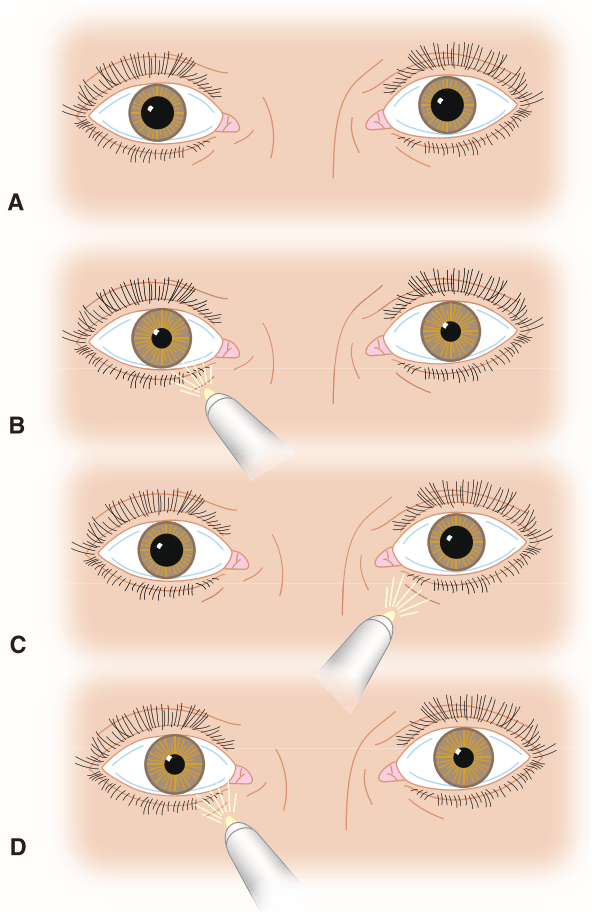


FIGURE 241-8. **A** and **B.** "Swinging flashlight test" revealing an afferent pupillary defect (Marcus-Gunn pupil) of the left eye. **A.** Pupils are normal and equal before light testing. **B.** Both pupils constrict when light is shined into the normal (right) eye. **C** and **D.** The test is positive when the affected pupil (left pupil) dilates in response to light. Conditions with an afferent pupillary defect include optic neuritis and central retinal artery occlusion.

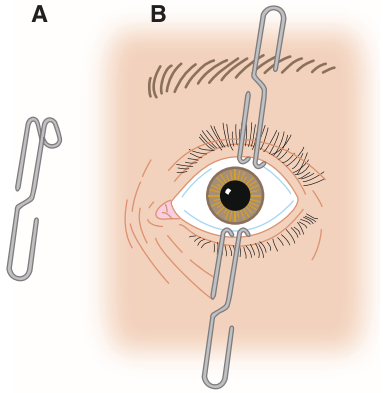


FIGURE 241-10. An alternative to an eyelid retractor. **A.** Unfold a paper clip and bend it into shape with a hemostat. **B.** Paper clips used to retract the eyelids. [Reproduced with permission from Reichman EF, Simon RR: *Emergency Medicine Procedures*. © 2004, Eric F. Reichman, PhD, MD, and Robert R. Simon, MD. McGraw-Hill Professional, Inc.]

paperclip may be bent into the shape of an eyelid retractor for this purpose (**Figure 241-10** and see **Figure 241-38**).

Anterior Segment and the Slit Lamp Examination The palpebral and bulbar conjunctiva, sclerae, cornea, anterior chamber, and iris and ciliary body make up the anterior chamber, and all except the ciliary body may be examined with the slit lamp (**Figure 241-11A**).

The slit lamp is a binocular microscope that affords a highly magnified three-dimensional view of ocular structures. Use the slit lamp to assess eye complaints whenever possible. The patient and examiner should both be seated on adjustable stools so that the examiner's and patient's eyes are at the same level. Cover the chin rest with tissue paper or a washcloth. Adjust the slit lamp height so the patient can lean forward and comfortably place the forehead against the upper plastic bar and the chin on the chin rest. Adjust the height of the chin rest so the patient's lateral canthus is even with the black line on the vertical bar. The oculars and light source are generally straight ahead for the general eye evaluation. Focus is adjusted by the anterior-posterior movement of the ocular and light source in relation to the patient. The joystick generally controls focus by moving the slit lamp closer to or farther away from the patient's eyes. Rotation of the joystick usually controls vertical movement of the light source and oculars. When one looks through the oculars, the focus should be close to correct if a narrow slit of light falls on

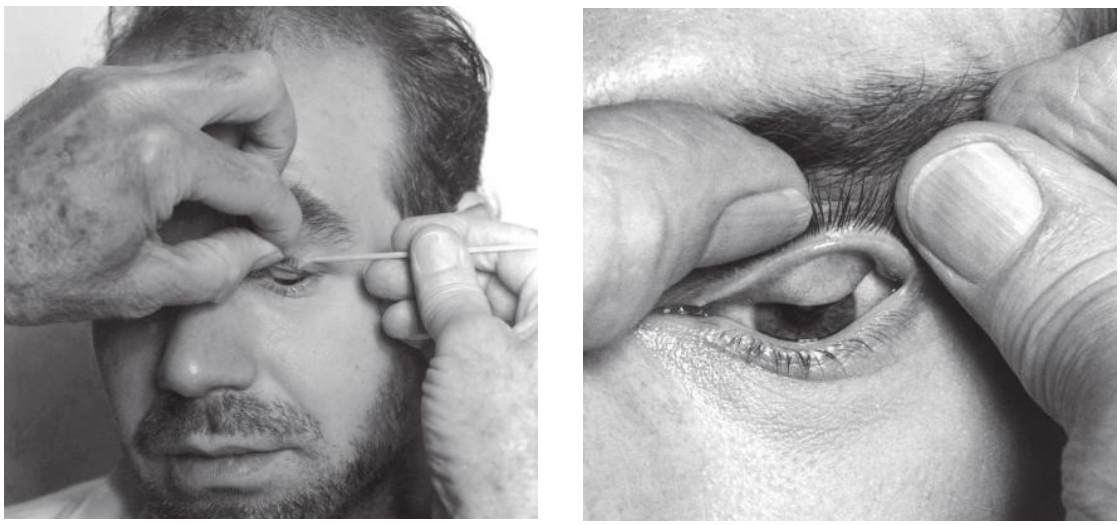


FIGURE 241-9. Single eversion of eyelid when a cotton applicator is used. [Reproduced with permission from Riordan-Eva P, Whitcher J: *Vaughn & Asbury's General Ophthalmology,* 17th ed. New York: Lange Medical Books/McGraw-Hill, 2008.]

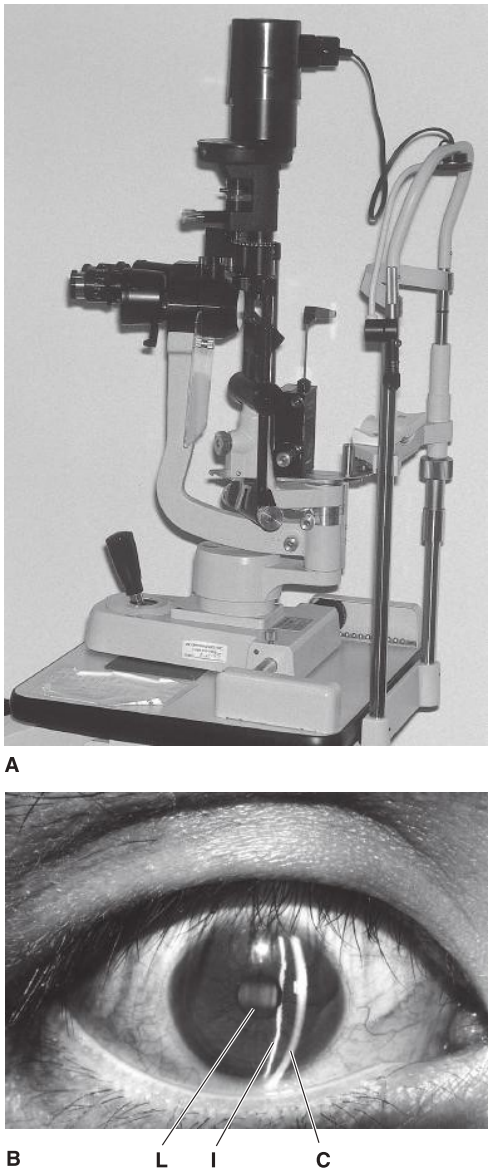


FIGURE 241-11. **A.** The slit lamp provides a magnified view of the eye. **B.** A thin slit can demonstrate the cornea (*C*), iris (*I*), and lens (*L*).

the structures to be examined. Fine adjustments in focus are then made with the joystick. Adjust the vertical light beam to the full height of the cornea with a width of approximately 1 mm (**Figure 241-11B**).

Examine the palpebral and bulbar conjunctivae for follicles (seen with allergic and viral conjunctivitis), chemosis (subconjunctival edema fluid), injection/inflammation, discharge, trauma, and foreign bodies.

To examine the cornea, rotate the light source to a 45-degree angle. The slit lamp can be brought close to the proper focus by looking at the slit of light on the cornea with the naked eye and moving the slit lamp in an anterior-posterior direction to obtain the thinnest and sharpest slit possible. The cornea is assessed by narrowing the light source to produce a slit beam that optically sections the cornea (**Figure 241-12**). Inspect the corneal epithelium for abrasions, ulcers, edema, and foreign bodies. Examine the corneal stroma for edema, scars, and lacerations, and examine the endothelium for precipitates (WBCs on the endothelium characteristic of iritis) and lacerations.

Assess the depth of the anterior chamber by adjusting the angle of the light source on the slit lamp or by shining a penlight onto the iris from a lateral direction. If the iris is bowed forward, as with a shallow anterior chamber, a shadow will be cast on the medial (nasal) iris (**Figure 241-13**).

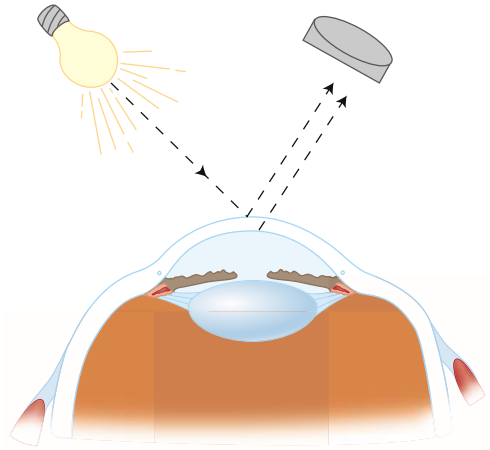


FIGURE 241-12. Optical sectioning. By creating an angle of 45 to 60 degrees between the slit-beam light source and the observer's biomicroscope objective, the cornea can be optically "sectioned" obliquely. This allows a cross-sectional view of the cornea and is helpful in ascertaining the depth of penetration of corneal foreign bodies and injuries.

Assess the anterior chamber for flare and cells as follows: shorten the slit beam to approximately 1 mm, and shut off the room lights. Select the high-magnification position of the oculars. The incident light source should create an angle of 45 to 60 degrees with the objective (similar to optical sectioning). Focus the light beam on the pupillary margin and pull the joystick back to focus on the cornea. One may see keratitic precipitates that are white spots on the undersurface of the corneal epithelium, representing deposits of inflammatory cells in iritis.

Now move the focus inward halfway between the iris and cornea, with the pupillary aperture as a dark backdrop. This will place your focus in the center of the aqueous humor, and the light beam will illuminate WBCs and red blood cells (if present) slowly drifting up and down in the aqueous convection currents, sometimes likened to snowflakes floating through the beam from a car's headlight at night. Iritis may result in WBC layers in the anterior chamber (hypopyon) (**Figure 241-14**). Trauma to the eye may cause red blood cells in the anterior chamber (hyphema or microhyphema). **Hyphema** is layering of the red cells in the anterior chamber visible to the naked eye (**Figure 241-15**). A hyphema may occasionally be clotted (**Figure 241-16**). Flare is described as the appearance of "headlights in a fog" and represents the ability to see the course of the normally transparent light beam through the aqueous humor. **Flare** is caused by increased aqueous protein in the anterior chamber, which is common with inflammatory conditions such as iritis.

Examine the iris for pupil irregularity and pupillary dysfunction. Irregularity of the pupil will occur whenever one portion of the iris is

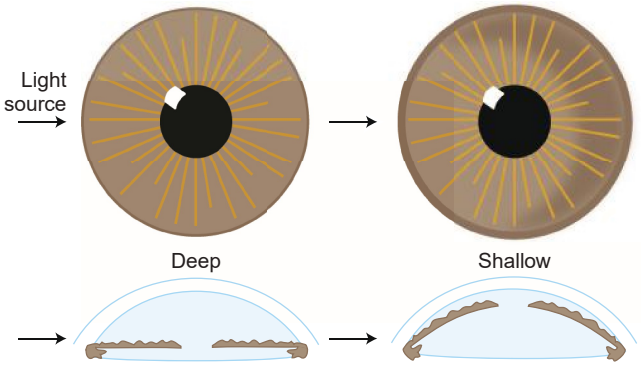


FIGURE 241-13. Estimation of depth of the anterior chamber by oblique illumination. Note the shadow cast in the shallow chamber. [Reproduced with permission from Riordan-Eva P, Whitcher J: *Vaughn & Asbury's General Ophthalmology,* 17th ed. New York: Lange Medical Books/McGraw-Hill, 2008.]

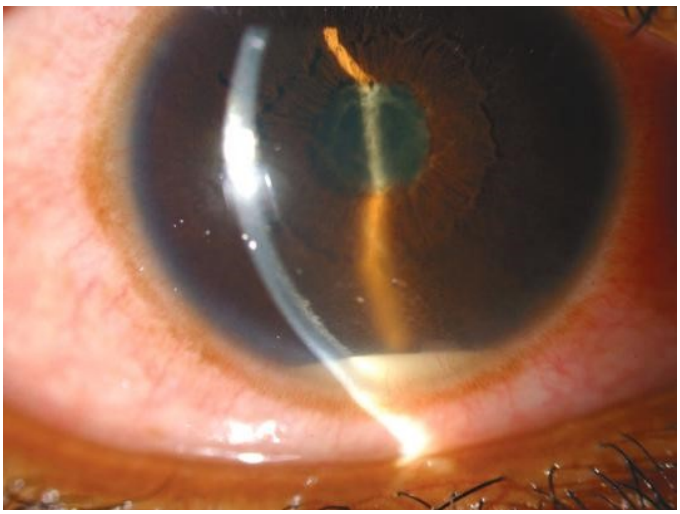


FIGURE 241-14. Hypopyon: a layering of WBCs in the inferior portion of the anterior chamber. [Courtesy of Allen R. Katz, Department of Ophthalmology, University of Nebraska Medical Center.]

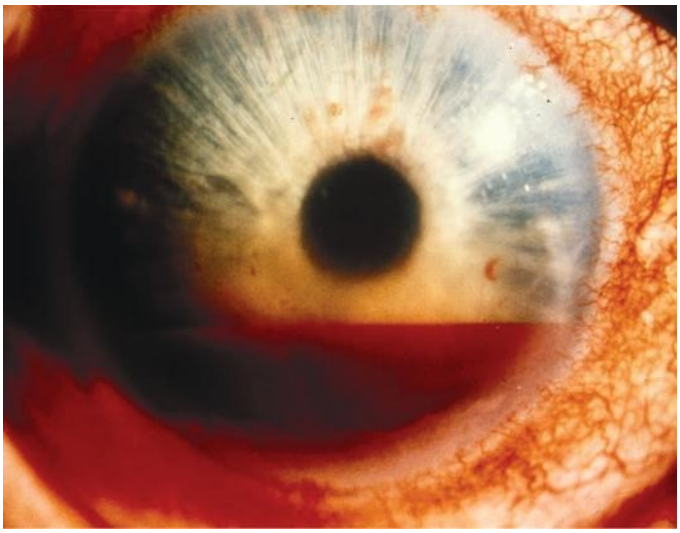


FIGURE 241-15. Hyphema secondary to blunt trauma. Note the blood filling the lower half of the anterior chamber and hazy appearance of cornea suggesting increased intraocular pressure. [Courtesy of Allen R. Katz, Department of Ophthalmology, University of Nebraska Medical Center.]

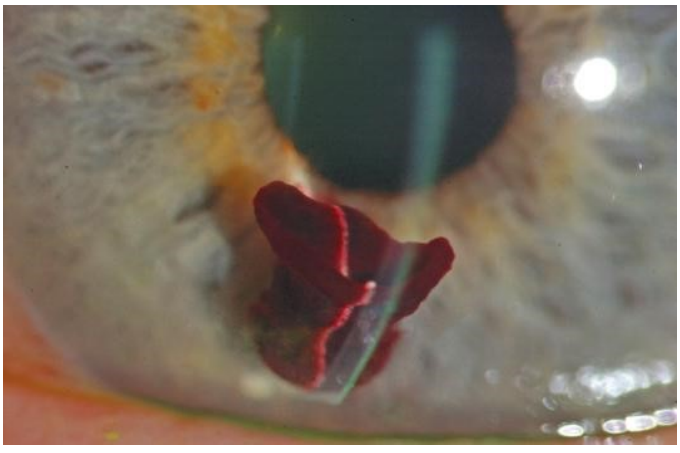


FIGURE 241-16. Hyphema appearing as a clot rather than layering out. [Courtesy of Allen R. Katz, Department of Ophthalmology, University of Nebraska Medical Center.]

tethered into place and may occur from posterior synechiae, where the iris is adhesed to the anterior lens capsule as seen in uveitis, or when any portion of the iris plugs a corneoscleral laceration causing a peaked pupil. Trauma may also cause the iris to tear at the root, termed *iridodialysis*. Assess the lens for opacities, lacerations, and subluxation.

Fluorescein Examination The final part of the slit lamp examination is done after fluorescein is instilled into the eye. Fluorescein binds to damaged corneal epithelium and fluoresces green under a **Wood's lamp** or light through a cobalt-blue filter. **Always remove contact lenses, as fluorescein will cause permanent staining of the lenses.** Touching the fluorescein strip directly to the cornea will cause staining mimicking a linear abrasion. The best way to apply fluorescein dye is to apply several drops of eye-irrigating solution or saline onto a paper fluorescein strip, and then lightly apply the moistened end of the fluorescein strip into the inferior conjunctival fornix. Ask the patient to blink several times to distribute the fluorescein. Then examine the cornea for streaming of fluorescein-tinged aqueous humor (positive Seidel test) seen in full-thickness laceration of the cornea (**Figure 241-17**). **The Seidel test can be negative (no streaming) with a small or spontaneously sealing corneal laceration.** Ask the patient to blink to wash out the fluorescein, and then examine the cornea with the cobalt blue filter on the slit lamp. A corneal abrasion will fluoresce bright green. A Wood's lamp (ultraviolet light) may also be used to look grossly (without the slit lamp) for corneal abrasions, but microscopic/punctate abrasions will be missed without use of a slit lamp.

Funduscopic Examination Note the size, shape, and sharpness of the borders of the optic disk, the cup-to-disk ratio, the size ratio of the arteries to veins (normal 2:3), any nicking where the arteries and veins cross, the texture and color of the retina as well as the presence of lesions (e.g., hemorrhages or exudates) of the retina or vessels (e.g., aneurysms), and the color and size of the macula. To locate lesions of the retina note the direction (e.g., superonasal) and distance from the disk in terms of disk diameters. Opacities of the lens may obscure the view of the retina, and lens opacities appear as black spots of various shapes. Lesions in the vitreous, such as vitreous hemorrhage, will also obscure the view. Vitreous hemorrhage will have an irregular shape and may have a reddish hue.

The **direct hand-held ophthalmoscope** is used to examine the fundus. Pharmacologic dilatation will greatly enhance the view of the disk, macula, and proximal retinal vessels. Dilation is achieved by using one drop of 1% tropicamide in Caucasian patients and one drop each of 1% tropicamide and 2.5% phenylephrine in all others. An **indirect ophthalmoscope** provides an excellent three-dimensional view of the optic nerve and retina but requires extensive practice to use and generally is not a tool for the nonophthalmologist.

The Welch Allyn Panoptic™ direct ophthalmoscope allows a five times larger view of the fundus than the standard direct ophthalmoscope and provides a better view of the fundus with an undilated pupil (**Figure 241-18**). It also allows for more distance between the patient

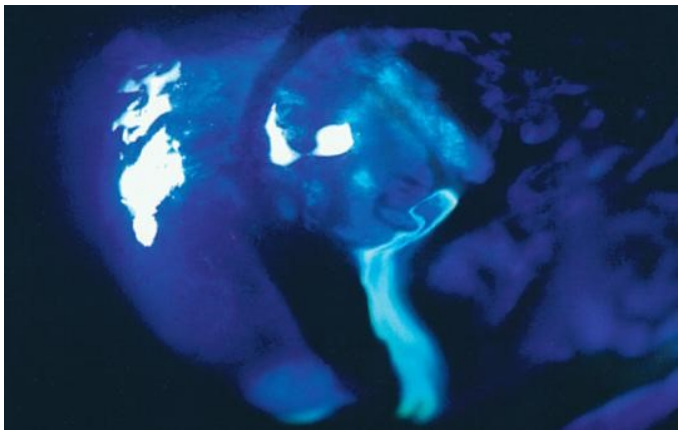


FIGURE 241-17. Positive Seidel test showing aqueous humor leaking through a full-thickness corneal wound. Aqueous humor will turn fluorescein lime-green under a cobalt-blue light as it oozes through the wound while being observed through the slit lamp.

FIGURE 241-18. Welch Allyn Panoptic™ direct ophthalmoscope. Copyright © Welch Allyn, Inc. All rights reserved.

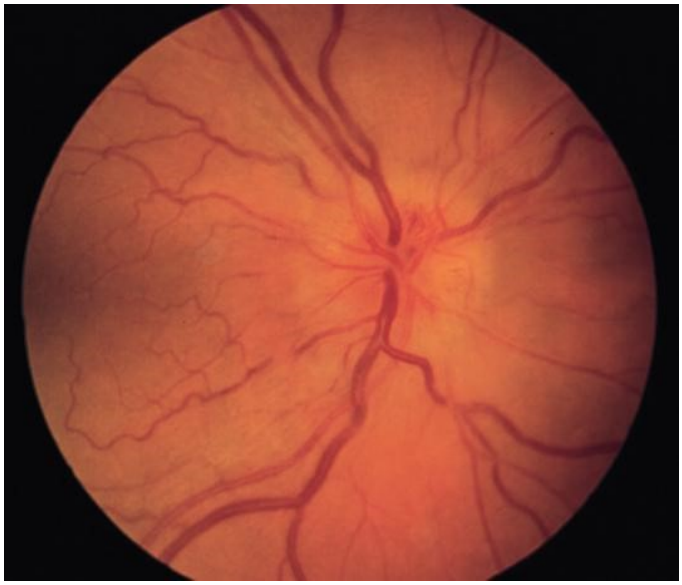


FIGURE 241-19. Optic nerve head edema. Vascular congestion, elevation of the nerve head, and blurred disk margins are characteristically seen in papilledema, papillitis, and compressive lesions of the optic nerve. [Reproduced with permission from Knoop K, Stack L, Storrow A: *Atlas of Emergency Medicine,* 2nd ed. © 2002, McGraw-Hill, New York.]

and examiner, for the comfort of both. The Panoptic™ device attaches to a standard Welch Allyn handle.

Use the Panoptic as follows:

1. Remove your and the patient's glasses; seat the patient upright or with the patient's stretcher set as upright as possible. Position yourself in a direct line of vision to the patient's eyes.
2. With the scope turned off, focus on an object at least 10 ft away.
3. Set the aperture dial to small ("home" position—green line).
4. Turn on the scope and adjust to maximum brightness.
5. Ask the patient to be still and look straight ahead, and tell him or her that the eyecup will touch the brow.
6. Place your hand on the patient's forehead, and position the scope 6 in. away at a 15- to 20-degree angle to the temporal side.
7. Locate the red reflex, and move the scope toward the patient keeping the red reflex in view.
8. Maximum view should be obtained when the eyecup is compressed by half.
9. If you have a dominant eye and prefer to use that for the examination, you may examine the opposite eye without switching your eye on the scope.
10. Make sure you wipe off the eyepiece with antiseptic/antibacterial solution after use, or change eyepieces between patients.

Papilledema **Papilledema** is bilateral edema of the head of the optic nerve due to increased intracranial pressure. Any disease process that increases the intracranial pressure and inhibits vascular or axoplasmic flow in the optic nerve causes congestion and edema of the nerve head. Bilateral papilledema is a common finding in malignant hypertension, pseudotumor cerebri, intracranial tumors, and hydrocephalus. The disk margins are blurred, the cup is diminished or absent, and the nerve head is elevated with vascular congestion (**Figure 241-19**). Frequently, flame-shaped hemorrhages are seen on or adjacent to the nerve head. A distinguishing feature of papilledema is prolonged preservation of visual acuity (frequently patients are visually asymptomatic).

Intraocular Pressure The eye remains consistently "inflated" because of a delicate balance between intraocular aqueous fluid production and outflow. Intraocular pressure can decrease due to reduced ciliary body production (some cases of iritis and uveitis) or loss of globe integrity (perforating injury). Intraocular pressure increases when intraocular fluid production exceeds outflow (glaucoma, hyphema). Measure intraocular pressure in all cases of vision loss, eye pain (suspected glaucoma), and acute or remote trauma. **Do not attempt to measure intraocular pressure if globe rupture from blunt or penetrating trauma is suspected**, as the pressure placed on the globe during pressure measurement may cause extrusion of intraocular contents. **The normal intraocular pressure is 10 to 20 mm Hg.** Digital palpation of the globe may give a rough estimation, using the examiner's eye or tip of the nose as control. Provide topical eye anesthetic when devices are used to measure pressure. To measure pressure, the lid must be open, and the patient must look straight ahead. Hold the lids open, with your fingers compressing the patient's lids against the bony rims of the orbit. Avoid placing any pressure on the globe with your fingers when holding the lids open, because this will cause a falsely high reading. Document the method used for determining intraocular pressure. In recording the pressure, refer to **Table 241-1**.

Schiötz Tonometer The **Schiötz tonometer** is a device using a plunger to indent the cornea. Counterweights are placed on top of the plunger, and the reading off the tonometer scale is correlated to numbers on a chart supplied with the tonometer to give a pressure reading. **The direct Schiötz scale reading is *not* the intraocular pressure.** Schiötz tonometry is inaccurate and not well tolerated by patients, and the instrument is difficult to sterilize, leading to potential spread of infection (**Figure 241-20**). The Tono-Pen□ XL (Reichert, Inc., Depew, NY), Goldman□ applanation tonometer, and pneumatonometer have supplanted the use of the Schiötz tonometer.

Tono-Pen□ The **Tono-Pen□ XL** and similar electronic devices have disposable latex covers and measure pressure by indentation of the cornea. The Tono-Pen□ XL is touched to the cornea 4 to 10 times and will read out an average pressure reading (**Figure 241-21**).

Applanation Tonometer Use of the **Goldman□ applanation tonometer** requires training and practice and is a method used by optometrists and ophthalmologists. **The cone must be sterilized between patients.** The cone of the tonometer is touched to the cornea after topical anesthesia, and fluorescein is instilled into the eye (without irrigation). When looking through the slit lamp, two half circles are seen and properly aligned by

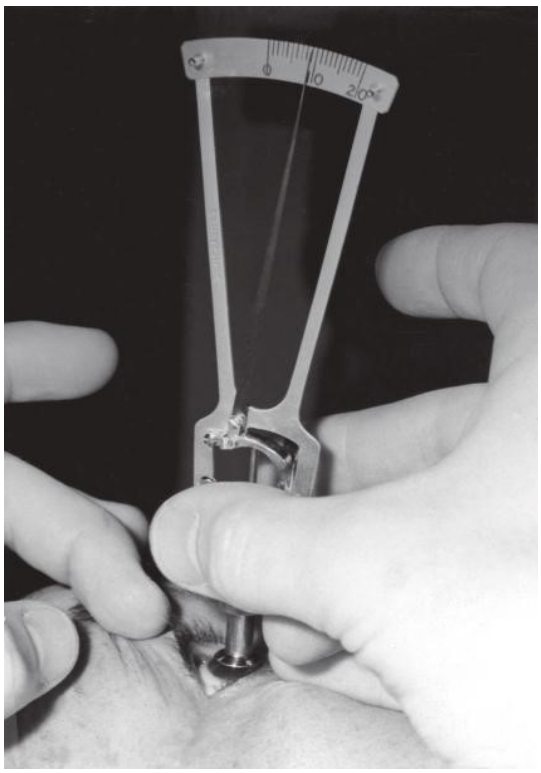


FIGURE 241-20. Schiötz tonometry. [Reproduced with permission from Riordan-Eva P, Whitcher J: *Vaughn & Asbury's General Ophthalmology,* 17th ed. New York: Lange Medical Books/McGraw-Hill, 2008.]

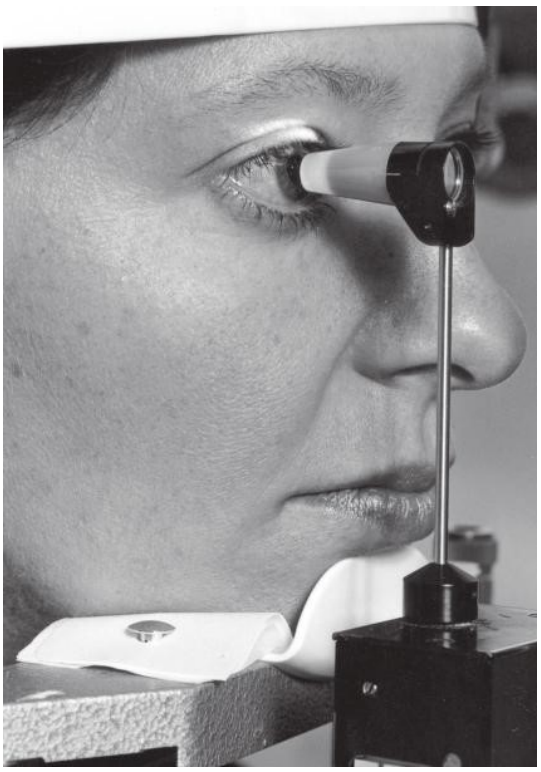


FIGURE 241-22. Goldman□ application tonometry. [Reproduced with permission from Riordan-Eva P, Whitcher J: *Vaughn & Asbury's General Ophthalmology,* 17th ed. New York: Lange Medical Books/McGraw-Hill, 2008.]

FIGURE 241-21. Tono-Pen□ XL (Reichert, Inc., Depew, NY). [Reproduced with permission from Rhee J: *Glaucoma: Color Atlas and Synopsis of Clinical Ophthalmology*. New York: McGraw-Hill, 2003.]

FIGURE 241-23. Appearance of fluorescein semicircles using applanation tonometry under the slit lamp. [Reproduced with permission from Riordan-Eva P, Whitcher J: *Vaughn & Asbury's General Ophthalmology,* 17th ed. New York: Lange Medical Books/McGraw-Hill, 2008.]

adjusting the dial on the tonometer, from which the pressure is read (**Figures 241-22** and **241-23**).

COMMON OPHTHALMIC MEDICATIONS USED IN THE ED

Topical anesthetics are needed for eye examination, intraocular pressure measurement, and corneal foreign body removal. Mydriatics and cycloplegics are needed for a more thorough eye examination. Ophthalmic antibiotics are prescribed for conjunctivitis and corneal abrasions. Antiviral medications are used for herpes simplex. Glaucoma may be treated with various combinations of topical nonselective and β_1-blockers, selective α_2-agonists, carbonic anhydrase inhibitors, cholinergic agents, and prostaglandin analogs. Allergic conjunctivitis is treated with topical nonsteroidal anti-inflammatory drugs, mast cell stabilizers, selective H_1 antagonists, or corticosteroids.

Table 241-2 lists common agents used in the ED and those that the consulting ophthalmologists may ask the emergency medicine physician to prescribe.

THE RED EYE

The differential diagnosis of the red eye is extensive. Key differentiating factors are the presence or absence of pain, itching, photophobia, systemic symptoms, discharge and injections, visual loss, and changes in the cornea, pupils, and intraocular pressure. **Table 241-3** lists various causes of the red eye with key differentiating features. Many of these conditions are discussed more extensively in the text.

TABLE 241.2 Ophthalmic Medications Used in the ED

Type	Generic Name	Trade Name	Indication	Cautions	Usual Dose
Mydriatic-cycloplegic					
Sympathomimetic	2.5% phenylephrine	Mydfrin□ AK Dilate□	Pupil dilation, no cycloplegia, usually adjunctive to an anticholinergic	Hypertension, glaucoma; do not use after chemical injury to the eye	1 drop, onset 20–30 min, duration several hours
Anticholinergic	Cyclopentolate	Cyclogyl□	Short-term mydriasis and cycloplegia for examination	Glaucoma; higher concentrations in children can cause agitation	0.5% in children, one drop; 1% in adults, one drop; onset 30 min, duration ≤24 h
Anticholinergic	Tropicamide	Mydriacyl□ Tropicamide Ophthalmic Solution	Short-term mydriasis and cycloplegia for examination	Glaucoma	One to two drops of 0.5% or 1% solution, onset 20 min; duration of action 6 h
Anticholinergic	Homatropine	Isopto Homatropine□	Intermediate-term pupil dilation, cycloplegia, treatment of iritis	Glaucoma, avoid in children	One to two drops of 2% solution; onset 30 min; duration of action 2–4 d; for iritis one to two drops twice a day
Antihistamine/ decongestant	Naphazoline and pheniramine	Naphcon-A□ Visine A□	Conjunctival congestion/itching	Do not use >72 h; avoid in narrow angle glaucoma; hypertension; do not use with contact lenses in place	One drop three to four times a day
Antihistamine	Olopatadine	Patanol□	Allergic conjunctivitis	Do not administer while contact lenses are present	0.1% solution, one drop twice daily, onset of action 30–60 min, duration 12 h
Topical anesthetics	Tetracaine ophthalmic solution	—	Anesthetic for eye examination, foreign body removal	Sensitivity to ester-type anesthetics; no prolonged use; delays healing	0.5% solution, one to two drops; onset of action 1 min, duration 30 min
	Proparacaine ophthalmic solution	Alcaine□ Ophthetic□	Anesthetic for eye examination foreign body removal		0.5% solution, one to two drops; onset of action 20 s, duration 15 min
Antibiotics	Erythromycin ophthalmic ointment	—	Conjunctivitis Do not use for corneal abrasion if a contact lens wearer	Not agent of choice for contact lens wearers	$^1/_2$ in. applied to lower eyelid two to four times a day
	Ciprofloxacin	Ciloxan□ Ophthalmic Solution and Ointment	Conjunctivitis, corneal abrasion if a contact lens wearer	—	Solution: one to two drops when awake every 2 h for 2 d; ointment, $^1/_2$ in. applied to lower eyelid three times a day for 2 d
	Tobramycin	Tobrex□ Ophthalmic Solution and Ointment	Conjunctivitis, corneal abrasions if a contact lens wearer	—	0.3% solution, one to two drops every 4 h; 0.3% ointment, $^1/_2$ in. applied to lower lid two to three times/d
	Gentamicin	Garamycin□ Genoptic□	Conjunctivitis, corneal abrasion if a contact lens wearer	—	0.3% solution, instill one to two drops every 4 h; 0.3% ointment, $^1/_2$ in. applied to lower lid two to three times/d
	Sulfacetamide sodium	Bleph-10 Ophthalmic Solution 10%□	Conjunctivitis	Do not use for corneal abrasion if a contact lens wearer or if allergic to sulfa	One to two drops four times/d
	Besifloxacin	Besivance Ophthalmic Suspension□	Conjunctivitis, corneal abrasion if a contact lens wearer	—	One drop three times a day for 7 d

(*Continued*)

TABLE 241.2 Ophthalmic Medications Used in the ED (*Continued*)

Type	Generic Name	Trade Name	Indication	Cautions	Usual Dose
Antibiotics (continued)	Levofloxacin	Iquix Ophthalmic Solution□ Quixin Ophthalmic Solution□	Conjunctivitis, corneal abrasion if a contact lens wearer	Corneal ulcer	One to two drops every 30 min to 2 h while awake and every 4–6 h at night
	Moxifloxacin hydrochloride	Moxeza Ophthalmic Solution□ Vigamox Ophthalmic Solution□	Conjunctivitis, corneal abrasion if a contact lens wearer		One drop twice a day for 7 d
	Ofloxacin	Ocuflox Ophthalmic Solution□	Conjunctivitis, corneal abrasion if a contact lens wearer		Conjunctivitis: one to two drops every 2–4 h for 2 d, then one to two drops four times a day for 5 d. Corneal ulcer: one to two drops every 30 min while awake and one to two drops every 4–6 hours after retiring for 2 d, then one to two drops every hour while awake for 5–7 days, then one to two drops four times a day for 2 d or until treatment completion
	Polymyxin b sulfate, trimethoprim sulfate	Polytrim Ophthalmic Solution□	Conjunctivitis	Do not use for corneal abrasion if a contact lens wearer	One drop every 3 h
	Gatifloxacin	Zymaxid Ophthalmic Solution□	Conjunctivitis, corneal abrasion if a contact lens wearer		
Antivirals	Idoxuridine	Dendrid Sterile Ophthalmic Solution□	Herpes simplex keratitis		One drop every hour
	Trifluridine	Viroptic Ophthalmic Solution□	Herpes simplex keratitis		One drop every 2 h
	Ganciclovir	Zirgan Ophthalmic Gel□	Herpes simplex keratitis		One drop five times per day
Antibiotic–steroid combination	Prednisolone acetate, sulfacetamide sodium	Blephamide Ophthalmic Suspension□ Brimonidine Tartrate Ophthalmic Solution□	Conjunctivitis	Do not use for corneal abrasion if a contact lens wearer or if sulfa allergy	Suspension: instill two drops into conjunctival sac every 4 h during the day and at bedtime Ointment: apply ½-in. ribbon into conjunctival sac three to four times a day and two to four times a night
	Dexamethasone, neomycin sulfate, polymyxin b sulfate	Maxitrol Ophthalmic Ointment□ Maxitrol Ophthalmic Suspension□	Conjunctivitis	Do not use for corneal abrasion if a contact lens wearer	Ointment: ½ inch in conjunctival sac(s) up to three to four times a day Suspension: instill one to two drops four to six times a day up to every hour
	Neomycin sulfate, polymyxin b sulfate, prednisolone acetate	Poly-Pred Ophthalmic Suspension□	Conjunctivitis	Do not use for corneal abrasion if a contact lens wearer	One to two drops every 3–4 h
	Dexamethasone, tobramycin	TobraDex Ophthalmic Ointment□ TobraDex Ophthalmic Suspension□ TobraDex ST Ophthalmic Suspension□	Conjunctivitis	Do not use for corneal abrasion if a contact lens wearer	Suspension: one to two drops every 4–6 h ST: one drop every 4–6 h Ointment: ½-in. ribbon three to four times a day
	Loteprednol etabonate, tobramycin	Zylet Ophthalmic Suspension□	Conjunctivitis	Do not use for corneal abrasion if a contact lens wearer	One to two drops every 4–6 h

Nonsteroidal anti-inflammatory drugs	Ketorolac	Acular Ophthalmic Solution□ Acular LS Ophthalmic Solution□ Acular PF Ophthalmic Solution□ Acuvail Ophthalmic Solution□	Allergic conjunctivitis, corneal abrasions, UV keratitis		One drop four times a day for 3–4 d
	Bromfenac	Bromday Ophthalmic Solution□	Allergic conjunctivitis, corneal abrasions, UV keratitis		One drop every day
	Nepafenac	Nevanac Ophthalmic Suspension□	Allergic conjunctivitis, corneal abrasions, UV keratitis		One drop three times a day
	Diclofenac sodium	Voltaren Ophthalmic Solution□	Allergic conjunctivitis, corneal abrasions, UV keratitis		One drop four times a day
Mast cell stabilizers	Nedocromil sodium	Alocril Ophthalmic Solution□	Allergic conjunctivitis		One to two drops twice a day
	Pemirolast potassium	Alamast Ophthalmic Solution□	Allergic conjunctivitis		One to two drops four times a day
	Lodoxamide tromethamine	Alomide Ophthalmic Solution□	Allergic conjunctivitis		One to two drops four times a day
	Cromolyn sodium	Cromolyn Sodium Ophthalmic Solution□	Allergic conjunctivitis		One to two drops four to six times a day
Selective H_1 antagonist	Bepotastine besilate	Bepreve Ophthalmic Solution□	Allergic conjunctivitis		One drop two times a day
	Epinastine hydrochloride	Elestat Ophthalmic Solution□	Allergic conjunctivitis		One drop two times a day
	Emedastine difumarate	Emadine Ophthalmic Solution□	Allergic conjunctivitis		One drop up to four times a day
	Alcaftadine	Lastacaft Ophthalmic Solution□	Allergic conjunctivitis		One drop every day
	Azelastine hydrochloride	Optivar Ophthalmic Solution□	Allergic conjunctivitis		One drop two times a day
Combination mast cell stabilizers–H_1 antagonists	Olopatadine hydrochloride	Pataday Ophthalmic Solution□ Patanol Ophthalmic Solution□	Allergic conjunctivitis		One drop two times a day
Nonselective β-blocker	Levobunolol hydrochloride	Betagan Ophthalmic Solution□	Glaucoma		0.5%: one to two drops once a day; twice a day for more severe or uncontrolled glaucoma 0.25%: one to two drops twice a day
	Timolol hemihydrate	Betimol Ophthalmic Solution□	Glaucoma		One drop 0.25% twice a day; may increase to maximum of one drop 0.5% twice a day
	Timolol maleate	Istalol Ophthalmic Solution□ Timoptic Sterile Ophthalmic Solution□ Timoptic-XE Sterile Ophthalmic Gel Forming Solution□	Glaucoma		One drop once a day
	Carteolol hydrochloride	Carteolol Hydrochloride Ophthalmic Solution□ Ocupress Ophthalmic Solution□	Glaucoma		One drop twice a day
	Metipranolol	OptiPranolol Ophthalmic Solution□	Glaucoma		One drop two times a day

(*Continued*)

TABLE 241_2 Ophthalmic Medications Used in the ED (*Continued*)

Type	Generic Name	Trade Name	Indication	Cautions	Usual Dose
Selective β_1-blocker	Betaxolol hydrochloride	Betaxolol Hydrochloride Ophthalmic Solution□ Betoptic S Ophthalmic Suspension□	Glaucoma		One to two drops twice a day
Selective α_2-agonists	Brimonidine tartrate	Alphagan P Ophthalmic Solution□ Brimonidine Tartrate Ophthalmic Solution□	Glaucoma		One drop every 8 h
	Apraclonidine hydrochloride	Iopidine 0.5% Ophthalmic Solution□ Iopidine Ophthalmic Solution□	Glaucoma		One to two drops three times a day
Combination α_2-agonist–nonselective β-blocker	Brimonidine tartrate, timolol maleate	Combigan Ophthalmic Solution□	Glaucoma		One drop every 12 h
Carbonic anhydrase inhibitors	Brinzolamide	Azopt Ophthalmic Suspension□	Glaucoma		One drop three times a day
	Dorzolamide hydrochloride	Trusopt Sterile Ophthalmic Solution□	Glaucoma		One drop three times a day
Combination carbonic anhydrase inhibitor–selective β-blocker	Dorzolamide hydrochloride, timolol maleate	Cosopt Sterile Ophthalmic Solution□	Glaucoma		One drop every 12 h
Cholinergic agents	Pilocarpine hydrochloride	Isopto Carpine Ophthalmic Solution□	Glaucoma; contraindicated in pupillary block glaucoma		Two drops three to four times a day
Prostaglandin analog	Bimatoprost	Lumigan Ophthalmic Solution□	Glaucoma		One drop once a day
	Travoprost	Travatan Z Ophthalmic Solution□	Glaucoma		One drop once a day
	Latanoprost	Xalatan Ophthalmic Solution□	Glaucoma		One drop once a day
	Loteprednol etabonate	Alrex Ophthalmic Suspension□	Allergic conjunctivitis, uveitis		One drop four times a day
	Difluprednate	Durezol Ophthalmic Emulsion□	Allergic conjunctivitis, uveitis		One drop four times a day
	Fluorometholone acetate	Flarex Ophthalmic Suspension□ FML Forte Ophthalmic Suspension□ FML Ophthalmic Ointment□ FML Ophthalmic Suspension□	Allergic conjunctivitis, uveitis		One to two drops four times a day
	Loteprednol etabonate	Lotemax Ophthalmic Ointment□ Lotemax Ophthalmic Suspension□	Allergic conjunctivitis, uveitis		Ointment: ½ inch four times a day Suspension: one to two drops four times a day
	Prednisolone acetate	Omnipred Ophthalmic Suspension□ Pred Forte Ophthalmic Suspension□ Pred Mild Ophthalmic Suspension□	Allergic conjunctivitis, uveitis		Two drops four times a day
	Rimexolone	Vexol 1% Ophthalmic Suspension□	Allergic conjunctivitis, uveitis		One to two drops every hour while awake

Note: Blue-eyed individuals are sensitive to mydriatics and cycloplegics and tend to have a longer duration of action, whereas brown-eyed individuals may require a double dose for adequate mydriasis.

Abbreviation: UV = ultraviolet.

TABLE 241-3 Differential Diagnosis of the Red Eye

Diagnosis	Pain	Itching	Photophobia	Systemic Symptoms	Visual Acuity	Discharge	Injection	Cornea	Pupils	IOP
Chalazion	Mild-moderate lid	No	No	No	Normal	No	Minimal localized	Normal	Normal	Normal
Hordeolum	Mild-moderate lid	No	No	No	Normal	No	Minimal localized	Normal	Normal	Normal
Blepharitis	Mild – foreign body sensation	Yes	Yes	No	Normal	Morning crusting, tearing	Diffuse	Normal	Normal	Normal
Dacryocystitis	Mild-moderate medial canthus	No	No	Fever if severe	Normal	No	Localized	Normal	Normal	Normal
Ectropion	Irritation	No	No	No	Normal	Watery	Lid margin and diffuse	Normal	Normal	Normal
Corneal abrasion	Yes	No	No unless associated iritis (after several hours)	No	Normal unless central or with associated iritis	Watery	Diffuse	Visible abrasion	Normal or constricted with associated iritis	Normal
Ultraviolet keratitis	Severe	No	No unless associated iritis (after several hours)	No	Decreased	Watery	Diffuse	Punctate lesions	Normal or constricted with associated iritis	Normal
Superficial keratitis	Mild	No	No	No	Normal	Watery	Diffuse	Punctate lesions	Normal	Normal
Corneal ulcer	Moderate	No	No	No	No unless central or with associated iritis	Watery	Diffuse	Visible ulcer	Normal	Normal
Corneal foreign body	Moderate	No	No	No	Normal unless central	Watery	Diffuse	Visible foreign body	Normal	Normal
Chemical burn	Moderate-severe	No	No	No	Normal unless central	Watery	Diffuse—none with severe alkaline burn	Cloudy if severe	Normal	Normal
Bacterial conjunctivitis	None or irritation	No	No	No	Normal	Purulent	Diffuse bulbar and palpebral	Normal, punctate lesions if associated keratitis	Normal	Normal
Viral conjunctivitis	None or irritation, severe with EKC	No	No	Occasional URI symptoms, fever with EKC	Normal	Watery	Diffuse bulbar and palpebral	Normal, punctate lesions if associated keratitis	Normal	Normal
Allergic conjunctivitis	None	Yes	No	Sneezing, rhinorrhea	Normal	Watery	Diffuse bulbar and palpebral	Normal	Normal	Normal
Stevens-Johnson syndrome	Foreign body sensation, burning	Yes	Yes	Fever, tachycardia, hypotension, skin and mucous membranes involved	Decreased	Watery	Diffuse	Punctate lesions, corneal ulcer, neovascularization, hazy, perforation	—	—
Orbital cellulitis	Pain with eye movement	No	No	Fever	Normal, decreased late	No	Yes	Normal	Normal	Occasionally increased
Preseptal cellulitis	Mild—no pain with eye movement	No	No	Fever	Normal	No	Yes	Normal	Normal	Normal
Episcleritis	Mild	No	No	Usually none; occasional rheumatologic symptoms	Normal	Watery	Focal	Normal	Normal	Normal
Scleritis	Severe, tender to palpation	No	No	Usually none; occasional rheumatologic symptoms	Decreased with advanced disease	Watery	Diffuse, occasionally violaceous color	Normal	Normal	Normal, may be increased

(*Continued*)

TABLE 241-3 Differential Diagnosis of the Red Eye (*Continued*)

Diagnosis	Pain	Itching	Photophobia	Systemic Symptoms	Visual Acuity	Discharge	Injection	Cornea	Pupils	IOP
Subconjunctival hemorrhage	None	No	No	No	Normal	No	No	Normal	Normal	Normal
Iritis/uveitis	Yes	No	Yes	Occasional rheumatologic or GI symptoms	Decreased	Watery	Perilimbal (ciliary) flush	Normal, flare and cells in anterior chamber	Constricted, poorly reactive	Usually normal, may be low
Acute angle closure glaucoma	Severe	No	No	Headache, nausea, vomiting	Decreased	Watery	Diffuse	Cloudy or hazy	Midpoint, poorly reactive	Increased
Endophthalmitis	Mild-moderate "ache"	No	Yes	Fever	Decreased	Purulent if present	Diffuse	Hazy, flare and cells anterior chamber, hypopyon	—	—

Abbreviations: EKC = epidemic keratoconjunctivitis; IOP = intraocular pressure; URI = upper respiratory infection.

OCULAR INFECTIONS AND INFLAMMATION

PRESEPTAL (PERIORBITAL) AND POSTSEPTAL (ORBITAL) CELLULITIS

Orbital and periorbital infections exist in a spectrum of increasing severity: preseptal (periorbital) cellulitis, postseptal (orbital) cellulitis, subperiosteal abscess, orbital abscess, and cavernous sinus thrombosis, from least to most severe. Preseptal cellulitis and postseptal cellulitis, although both of an infectious etiology and involving periocular tissues, are very different entities with different morbidities. **Preseptal cellulitis (periorbital cellulitis)** is an infection of the eyelids and periocular tissues that is anterior to the orbital septum. It is generally benign and may be treated in the outpatient setting. **Postseptal cellulitis (orbital cellulitis)** is an infection of the orbital soft tissues posterior to the orbital septum. It may be life- and vision-threatening and must be treated as an inpatient with IV antibiotics and occasionally surgical drainage. **Endophthalmitis** is an infection of the globe and is a completely separate entity.

The outward appearance of the patient with postseptal cellulitis may be very similar to that of the patient with preseptal cellulitis. Preseptal and postseptal cellulitis may both display excessive tearing, fever, erythema, edema, warmth, and tenderness to palpation of the lids and periorbital soft tissues. Laboratory studies do not discriminate between the two conditions, and blood cultures are not helpful. **CT scan differentiates the two conditions.** Differentiation of preseptal and postseptal cellulitis is essential. A misdiagnosis can result in significant neurologic disability and death. The differential diagnosis for preseptal and postseptal cellulitis is listed in **Table 241-4**.

TABLE 241-4 Differential Diagnosis of Preseptal and Postseptal Cellulitis

Preseptal cellulitis
Postseptal cellulitis
Subperiosteal abscess
Orbital abscess
Cavernous sinus thrombosis
Dacryoadenitis
Dacryocystitis
Hordeolum
Bacterial and viral conjunctivitis
Contact dermatitis
Herpes zoster
Herpes simplex

Preseptal Cellulitis **Preseptal cellulitis** is usually associated with upper respiratory tract infections, especially paranasal sinusitis, and may result from eyelid problems such as hordeolum, chalazion, insect bites, and trauma. Preseptal cellulitis is primarily a disease of childhood, with most patients <10 years of age. The most common organisms are *Staphylococcus aureus* and *S. epidermidis*, *Streptococcus* species, and anaerobes. Since the introduction of the *Haemophilus influenzae* type B vaccine, *H. influenzae* has become a rare cause of preseptal cellulitis in children.

Upper respiratory symptoms, low-grade fever, redness and swelling of the eyelid, and excessive tearing (epiphora) are signs and symptoms of preseptal cellulitis. **The eye itself is not involved, and visual acuity and pupillary reaction are maintained, and full painless ocular motility is preserved.**

CT scan of the orbit is not necessary for uncomplicated cases of preseptal cellulitis; however, when there is decreased ocular motility or other signs of orbital involvement, or in the young child in whom examination may be unreliable, or if there is any concern for postseptal cellulitis, obtain a CT scan of the orbit with contrast. MRI is also an option.

The nontoxic, adult patient and older child with mild preseptal cellulitis may be managed as outpatients with oral antibiotics (amoxicillin/clavulanic acid or a first-generation cephalosporin), hot packs, and close follow-up in 24 to 48 hours. For the young child and more severe cases of preseptal cellulitis, consider hospitalization; treatment with a third-generation cephalosporin such as ceftriaxone and vancomycin is often added for the possibility of methicillin-resistant *S. aureus*. Obtain ophthalmology consultation in young children (see Chapter 119, Eye Emergencies in Infants and Children).

Postseptal or Orbital Cellulitis **Postseptal cellulitis** occurs most frequently from the spread of paranasal sinusitis. The ethmoid sinus is most frequently implicated, probably due to perforation of the thin lamina papyracea. Trauma, intraorbital foreign body, spread of periorbital skin infection, seeding from bacteremia, and ocular surgery are also predisposing factors. The infection is often polymicrobial, with *S. aureus*, *S. pneumoniae*, and anaerobes being most common; however, *H. influenzae* should be considered in unimmunized young children and mucormycosis in diabetics and immunocompromised patients.

Orbital cellulitis is characterized by an insidious onset with preceding upper respiratory symptoms, including rhinitis, facial pressure, and fever. The patient will often complain of pain when moving the eyes. There may be a decrease in visual acuity. On examination, signs referable to orbital involvement are present, including limitation of extraocular muscle movement , chemosis, proptosis, abnormal pupillary response, and decreased visual acuity. **Involvement of cranial nerves 3, 4, or 6 suggests cavernous sinus thrombosis.**

Postseptal cellulitis may lead to vision loss and requires an aggressive approach with hospitalization and institution of IV antibiotics (see Chapter 119, Eye Emergencies in Infants and Children). Any patient

suspected of having postseptal cellulitis clinically or by CT scan of the orbit should have immediate ophthalmologic consultation. Antibiotics should be broad spectrum with both aerobic and anaerobic coverage. Choices include second- or third-generation cephalosporins, ampicillin-sulbactam, ticarcillin-clavulanate, and carbapenems. A fluoroquinolone is used for penicillin-allergic patients, and metronidazole or clindamycin is added for anaerobic coverage. Adjuvant therapy includes a topical nasal decongestant such as oxymetazoline. Consider emergent **lateral canthotomy** if the intraocular pressure is elevated or an optic neuropathy is present. Patients with orbital abscess require operative drainage and debridement in addition to antibiotics. Complications include cavernous sinus thrombosis, frontal bone osteomyelitis, meningitis, subdural empyema, epidural abscess, and brain abscess that should be evident on CT or MRI scan.

LIDS

Stye (External Hordeolum) A **stye** is an acute bacterial infection (usually *Staphylococcus*) of the follicle of an eyelash and adjacent sebaceous glands (Zeis) or sweat glands (Moll). It is located at the lash line and has the appearance of a small pustule at the margin of the eyelid (**Figure 241-24**). An **internal hordeolum** is an acute bacterial infection of the meibomian glands associated with the eyelashes. Signs and symptoms are similar to a stye except that the pustule occurs on the inner surface of the tarsal plate. Signs and symptoms include pain, edema, and erythema of the eyelid. Warm compresses and erythromycin ophthalmic ointment twice daily for 7 to 10 days are usually sufficient treatment. Removal of the offending eyelash could be considered. Systemic antibiotics may be necessary if there is significant surrounding cellulitis. Should incision and drainage be considered, refer to an ophthalmologist.

Chalazion A **chalazion** is an acute or chronic inflammation of the eyelid secondary to blockage of one of the meibomian or Zeis oil glands in the tarsal plate (**Figure 241-25**). The condition tends to be subacute to chronic and is associated with a (usually) painless lump that develops in the lid or at the lid margin, occasionally with mild erythema. Clinical differentiation of an acute chalazion from an internal hordeolum may be impossible, but treatment is the same. Treatment of chronic or recurrent chalazia may require injection of corticosteroids into the lesion or incision and curettage/drainage depending on the size. Refer to an ophthalmologist in 1 to 2 weeks.

Blepharitis **Blepharitis** is a common cause of prolonged red eye due to inflammation of the eyelash follicles along the edge of the eyelid. The most common cause is overgrowth of *S. epidermidis*, and the inflation is largely a reaction to the deltalike toxin produced by the bacteria. The disorder is associated with seborrheic dermatitis, atopic dermatitis, and occasionally eyelash infestation with lice or infection with *S. aureus.* Typical symptoms include conjunctival injection; crusting, swollen, pruritic eyelids; and occasional complaint of eye pain. It is most commonly treated with careful daily cleansing of the edges of the eyelids and eyelashes. In severe cases, antibiotic drops or ointment at night may be required.

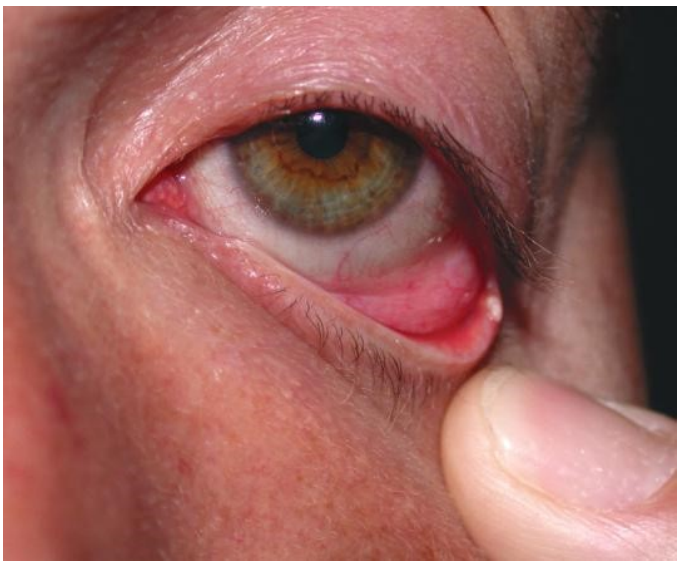


FIGURE 241-24. External hordeolum. [Courtesy of Allen R. Katz, Department of Ophthalmology, University of Nebraska Medical Center.]

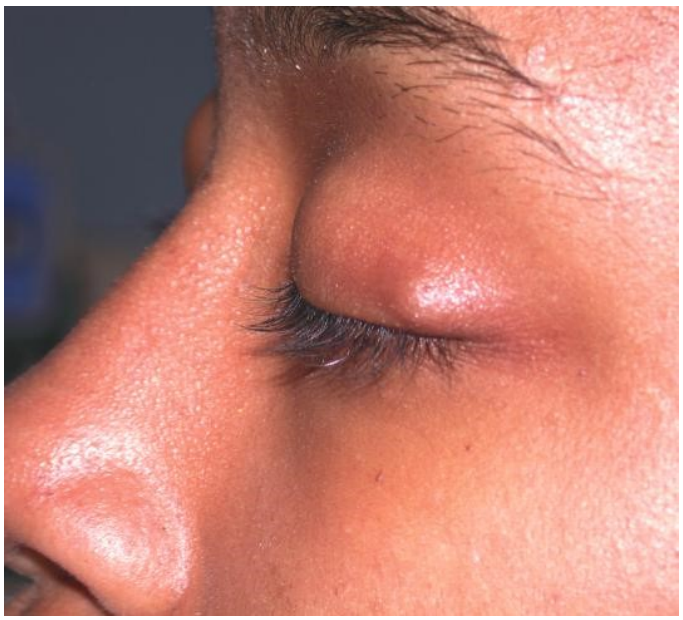


FIGURE 241-25. Chalazion. [Courtesy of Allen R. Katz, Department of Ophthalmology, University of Nebraska Medical Center.]

Conjunctivitis **Conjunctivitis** is an inflammatory condition of the conjunctiva and is a common cause of the red eye. It is usually viral in etiology and is a benign self-limited condition. **The task is to sort out the occasional case of serious bacterial infection or corneal herpetic involvement that may result in vision loss without aggressive treatment.** Causes of conjunctivitis are viral, bacterial (including gonococcal and chlamydial), parasitic, or fungal infections and allergic, toxic, or chemical irritation. **Keratoconjunctivitis** is the term used to indicate corneal involvement, usually in the form of punctuate ulcerations.

Bacterial Conjunctivitis Symptoms of **bacterial conjunctivitis** are painless, unilateral, or bilateral mucopurulent discharge (**Figure 241-26**), frequently causing adherence of the eyelids on awakening. The conjunctiva is injected, and the cornea is clear *without* fluorescein staining. Chemosis (edema of the conjunctiva) is common, and preauricular lymphadenopathy is usually absent, except in gonococcal infections. Typical pathogens are *Staphylococcus* and *Streptococcus* species. Perform fluorescein stain of the cornea (especially in infants) to avoid missing a corneal abrasion, ulcer, or herpetic dendrite. Consider culture and sensitivity of the discharge in severe cases. Treatment consists of a topical ocular antibiotic four times daily for 5 to 7 days. Treatment with a broad-spectrum agent is safe for patients 2 months of age and older. Trimethoprim–polymyxin B is very effective and avoids potential allergies to sulfa and neomycin preparations. Wearers of soft contact lenses should be treated with a fluoroquinolone (besifloxacin, gatifloxacin, levofloxacin, moxifloxacin, or ofloxacin) or aminoglycoside (tobramycin) to treat *Pseudomonas*. Gentamicin is seldom used because of the high incidence of ocular irritation.

Gonococcal conjunctivitis is a cause of ophthalmia neonatorum, and **chlamydial conjunctivitis** is also a disease of the newborn (see Chapter 119, Eye Emergencies in Infants and Children, for discussion of ophthalmia neonatorum and chlamydia conjunctivitis).

Viral Conjunctivitis The most common cause of **viral conjunctivitis** is adenovirus, which generally resolves spontaneously with only symptomatic

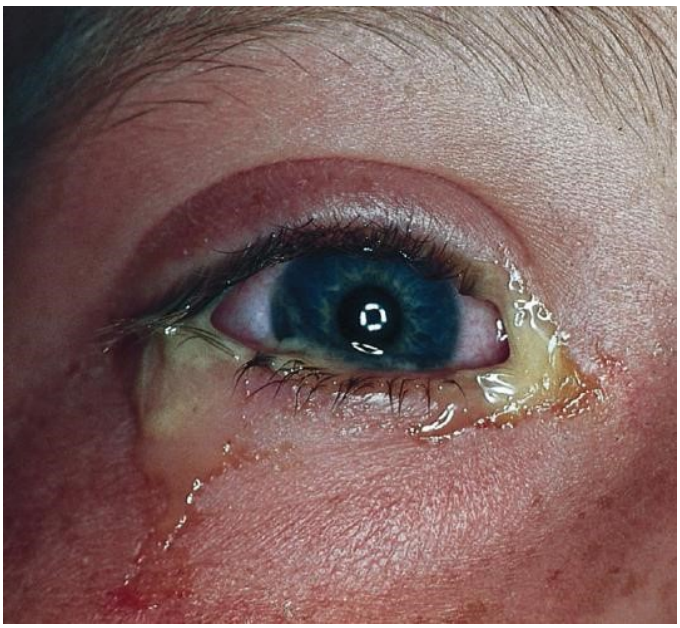


FIGURE 241-26. Bacterial conjunctivitis. Note the mucopurulent discharge, conjunctival injection, and lid edema in a pediatric patient with *Haemophilus influenzae* conjunctivitis. [Reproduced with permission from Knoop K, Stack L, Storrow A: *Atlas of Emergency Medicine,* 2nd ed. © 2002, McGraw-Hill, New York.]

treatment. Several systemic viral diseases, such as measles, influenza, and mumps, may also cause conjunctival injection. Viral keratoconjunctivitis may be caused by **herpes simplex** and **herpes zoster**, and, if left untreated, may result in corneal scarring and loss of vision. **Epidemic keratoconjunctivitis** (**Figure 241-27**) is a more severe type of adenovirus infection that is highly contagious and tends to occur in epidemics. It is often preceded by cough, high fever, malaise, and myalgias. Symptoms are marked eye redness, photophobia, foreign body sensation, and tearing. Physical examination findings are similar to viral conjunctivitis, except they are more severe.

Viral conjunctivitis is often preceded by an upper respiratory infection. Symptoms include a complaint of "red eye" and mild to moderate watery discharge. There is no eye pain unless there is some degree of keratitis. Generally, one eye will be involved initially, with the other eye becoming involved within days. Physical examination reveals unilateral or bilateral conjunctival injection, occasional chemosis and small subconjunctival hemorrhages, and preauricular lymphadenopathy. Slit lamp examination demonstrates follicles (small, regular, translucent bumps) on the inferior palpebral conjunctiva (**Figure 241-27**). Punctate fluorescein staining represents keratitis (**Figure 241-28**). Make sure to examine the cornea with fluorescein to avoid missing a herpetic dendrite.

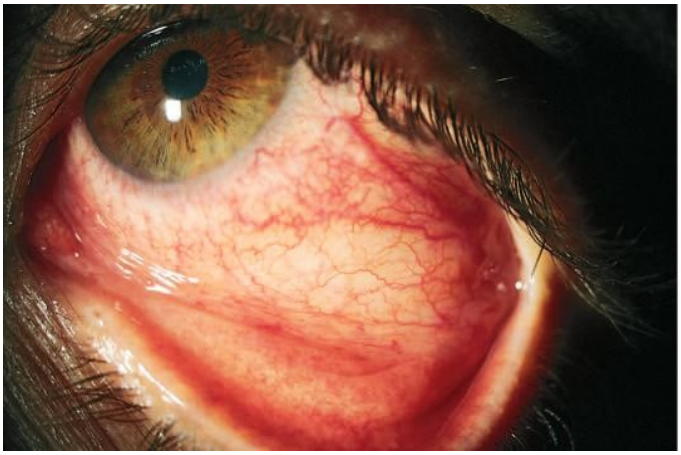
Treatment consists of cool compresses; ocular decongestants such as Naphcon-A®, one drop three times daily as needed for redness and conjunctival congestion; and artificial tears five or six times a day. Viral conjunctivitis can take 1 to 3 weeks to run its course and is very contagious. Instruct the patient to wash hands frequently and use separate towels. The examiner should wear gloves to avoid self-contamination, and the slit lamp, exam table, and exam chair should be disinfected after patient contact. If, after a history and physical examination, it is still uncertain if the conjunctivitis is viral or bacterial, prescribe ocular antibiotics until the patient is reexamined by an ophthalmologist.

Allergic Conjunctivitis Allergens can cause watery discharge, redness, and itching. Physical findings may include erythematous swollen eyelids and injected and edematous conjunctiva with papillae (irregular mounds of tissue with a central vascular tuft) on the inferior conjunctival fornix (**Figure 241-29**). Try to identify and eliminate the offending allergen. Treatment is cool compresses four times daily and topical drops, depending on the severity of symptoms. Mild symptoms may be treated with artificial tears alone. Moderate symptoms may additionally require a topical antihistamine/decongestant, mast cell stabilizers, or nonsteroidal anti-inflammatory drugs, and severe symptoms may justify the use of topical steroids. We generally recommend against the use of ocular steroids except by an ophthalmologist because occult herpetic infection is always a possibility (see following section on herpes simplex virus infection of the cornea). **Should ocular steroids be chosen as a treatment option, consult an ophthalmologist first.**

Subconjunctival Hemorrhage The fragile conjunctival vessels can rupture from trauma, sudden increased venous pressure related to Valsalva maneuvers (sneezing, coughing, vomiting, straining), hypertension, or spontaneously (**Figure 241-30**). The eye examination is normal other than the presence of the subconjunctival hemorrhage. Reassurance is the only treatment necessary, and the hemorrhage usually resolves within 2 weeks. If multiple recurrent episodes occur, coagulation studies and further investigation are warranted.

CORNEA

Herpes Simplex Keratoconjunctivitis **Herpes simplex virus** can affect the eyelids, conjunctiva, and cornea. The patient may give a history of oral or genital herpes infection and complain of photophobia,



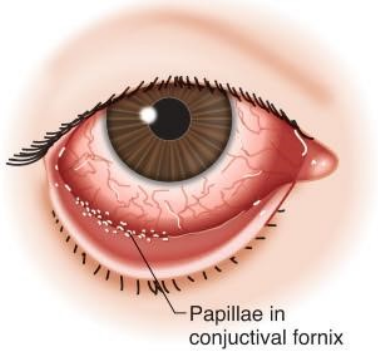


FIGURE 241-27. Epidemic keratoconjunctivitis (EKC). Diffuse bulbar conjunctival injection and inferior palpebral papillae as seen in EKC. The erythema is usually much more intense than in this photo. [Reproduced with permission from Knoop K, Stack L, Storrow A: *Atlas of Emergency Medicine,* 2nd ed. © 2002, McGraw-Hill, New York.]

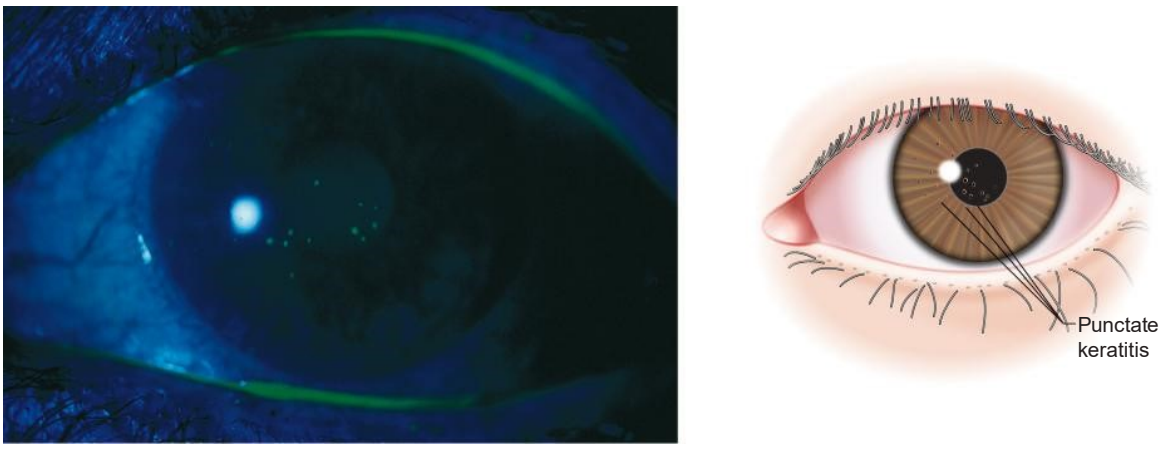


FIGURE 241-28. Fluorescein stain demonstrating punctate staining as seen with epithelial keratitis in epidemic keratoconjunctivitis. The keratitis is usually much more widespread than demonstrated here. [Reproduced with permission from Knoop K, Stack L, Storrow A: *Atlas of Emergency Medicine,* 2nd ed. © 2002, McGraw-Hill, New York.]

pain (which may be mild), eye redness, and decreased vision. The eyelid may have the typical herpetic vesicular eruptions. The infection tends to be unilateral with a palpable preauricular node. The conjunctiva can be injected, but ocular herpes simplex frequently presents with only corneal findings on physical examination. The findings can be subtle and are easily missed. The dendrite of herpes keratitis is an epithelial defect that can be seen with fluorescein staining and classically has a linear branching pattern with terminal bulbs (**Figure 241-31**), or may be a "geographic ulcer," which is an amoeba-shaped ulceration with dendrites at the edge. **Herpetic infection may be very difficult to diagnose**, as infection may present as involvement of the cornea with a neurotrophic ulceration, which is a smooth-edged ulcer over an area of underlying stromal disease; as stromal edema and/or white infiltrates with intact epithelium and an associated mild iritis with keratitic precipitates; or as an isolated uveitis, without epithelial or stromal involvement and with an elevated intraocular pressure. Corneal sensation may be decreased and should be checked before instillation of anesthetic drops. The diagnosis of herpes simplex is usually clinical, but check with the ophthalmologic consultant to see if cultures are desired.

Herpes simplex keratitis can progress to corneal scarring and requires prompt treatment with topical antiviral agents. **An initial outbreak of herpes simplex virus involving the lids is treated with an oral acyclovir derivative such as acyclovir (Zovirax□) or famciclovir (Famvir□). For conjunctival involvement, prescribe topical trifluridine (Viroptic□), one drop nine times a day.** Idoxuridine (Dendrid□), one drop every 1 hour during the day and every 2 hours at night, can be substituted for those who are allergic. Erythromycin ophthalmic ointment can be added to prevent secondary infection.

Oral acyclovir or other derivative agents do not prevent progression of corneal epithelial disease to deeper involvement of the stroma or uveal tract. Do not prescribe topical steroids, and refer patients to an ophthalmologist in 24 to 48 hours.

Herpes Zoster Ophthalmicus **Herpes zoster ophthalmicus** is shingles involving the first division (V1) of the trigeminal nerve distribution with ocular involvement. The rash usually does not cross the midline and involves only the upper eyelid, although rarely the cheek (V2) and mandible (V3) may be affected. **Involvement of the nasociliary nerve is associated with cutaneous lesions on the tip of the nose (Hutchinson sign) and predicts a high likelihood of ocular involvement.** Symptoms that may precede the rash are pain and paresthesias in a dermatomal distribution; fever, headache, and malaise; and red eye, blurred vision, and eye pain/photophobia. Eye involvement may take the form of epithelial keratitis, stromal keratitis, uveitis, retinitis, and choroiditis. Optic neuritis and other cranial nerve palsies may occur, as well as elevated intraocular pressure. The cornea can have a pseudodendrite, which is a poorly staining mucous plaque with no epithelial erosion (unlike herpes simplex virus, which has a true dendrite with epithelial

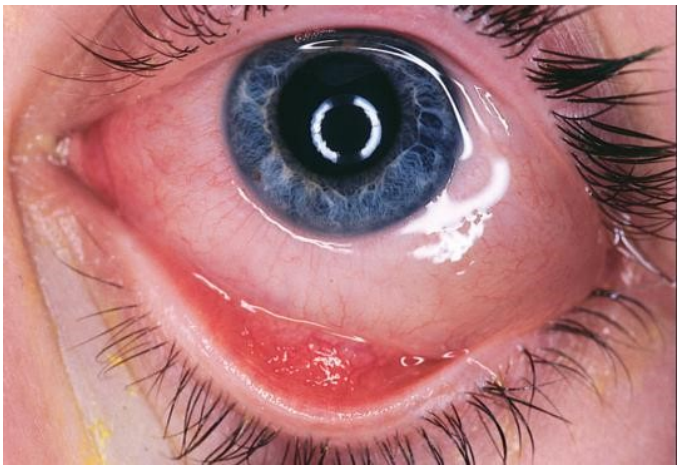


FIGURE 241-29. Prominent chemosis may be seen in allergic conjunctivitis. [Reproduced with permission from Knoop K, Stack L, Storrow A: *Atlas of Emergency Medicine,* 2nd ed. © 2002, McGraw-Hill, New York.]

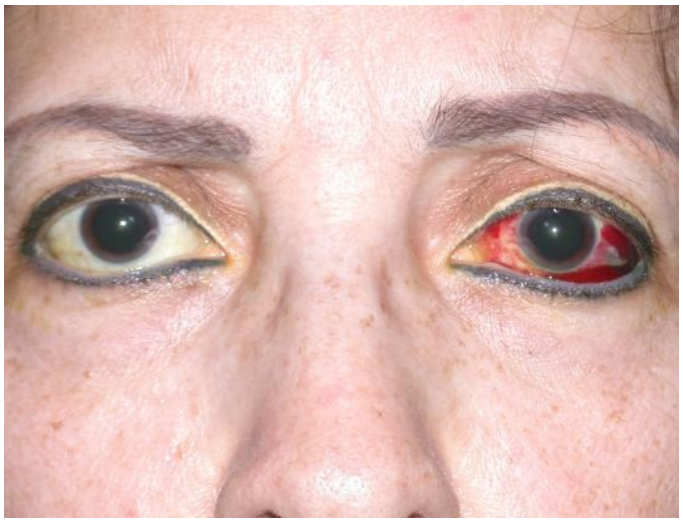


FIGURE 241-30. Spontaneous subconjunctival hemorrhage. [Courtesy of Allen R. Katz, Department of Ophthalmology, University of Nebraska Medical Center.]

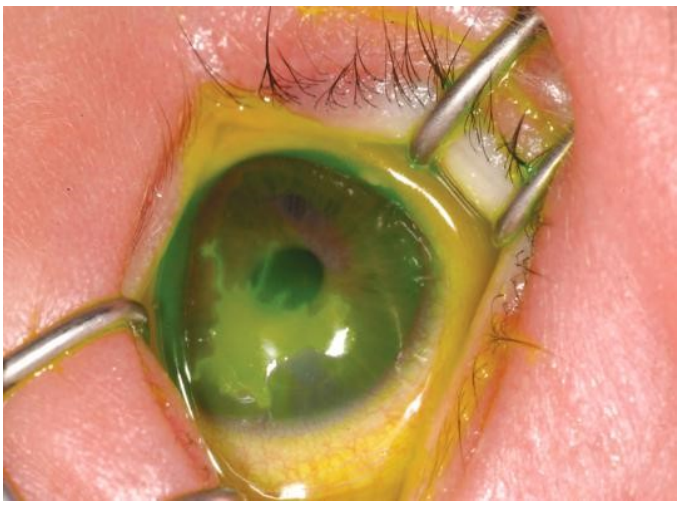


FIGURE 241-31. Herpes simplex corneal dendrite in an infant seen with fluorescein staining. [Courtesy of Allen R. Katz, Department of Ophthalmology, University of Nebraska Medical Center.]

erosion and staining). The anterior chamber on slit lamp examination can show the cells and flare of iritis. Consider the possibility of associated immunocompromise in patients <40 years old.

Skin involvement is treated with cool compresses, and patients presenting with a rash for <1 week should be treated with oral antiviral medications for 7 to 10 days. Choices include acyclovir, 800 milligrams five times a day; famciclovir, 500 milligrams three times a day; or valacyclovir, 1000 milligrams three times a day. Cutaneous lesions are treated with bacitracin or erythromycin ointment to prevent secondary bacterial infection. Conjunctivitis is treated with erythromycin ophthalmic ointment twice a day. Iritis can be treated with topical steroids such as prednisolone acetate 1%, one drop four to five times a day, but consultation with an ophthalmologist is recommended first. Pain reduction can be achieved with topical cycloplegic agents (cyclopentolate 1%, one drop three times daily). If herpes zoster ophthalmicus is diagnosed, in particular, when the orbit, optic nerve, or cranial nerves are involved, or if the patient is immunocompromised or systemically ill, consider admission and IV acyclovir.

Corneal Ulcer A **corneal ulcer** is a serious infection involving multiple layers of the cornea and develops secondary to breaks in the epithelial barrier, so that infectious agents invade the underlying corneal stroma. The initial disruption of the epithelial layer can be due to desquamation, trauma, or direct microbial invasion. Exposure keratitis from incomplete lid closure secondary to **Bell's palsy** can cause corneal desiccation and sloughing of the epithelium, allowing bacteria to gain access to the underlying stroma and create an ulcer. Trauma can also breach the epithelium and inoculate the cornea. *S. pneumoniae* and *S. aureus* are common causes of corneal ulceration. Contact lens users can develop *Pseudomonas* infection (**Table 241-5**). Wearing of soft contact lenses is a very common cause of corneal ulcers, and the incidence increases dramatically in those who use extended-wear lenses and wearers who sleep with them in place. Fungi and viruses have also become a more common cause of corneal ulcer due to the widespread use of both topical and systemic immunosuppressant medications.

The history should include an inquiry into the use of contact lenses, previous ocular surgery or injury, recent trauma, and presence or history of genital herpes. Medication history should include questions about the use of topical or systemic steroids or other immunosuppressants.

Patients may complain of redness and swelling of lids and conjunctivae, discharge from the eye, ocular pain or foreign body sensation, photophobia, or blurred vision. Visual acuity is decreased if the ulcer is located in the central visual axis or if uveal tract inflammation is present. The eyelids and conjunctiva may be erythematous with a mucopurulent discharge. Associated iritis may cause a miotic pupil and consensual photophobia due to ciliary spasm. Examination of the cornea reveals a round or irregular ulcer with a white, hazy base extending into the underlying stroma due to WBC infiltration, or with heaped-up edges (**Figure 241-32**). Slit lamp examination reveals flare and cell from iritis and occasionally a hypopyon.

TABLE 241-5 Causative Organisms in Corneal Ulcer

Bacteria
Pseudomonas aeruginosa
Streptococcus pneumoniae
Staphylococcus species
Moraxella species
Viruses
Herpes simplex
Varicella zoster
Fungi
Candida
Aspergillus
Penicillium
Cephalosporium

Diagnosis is made by the clinical appearance. The organism is identified by scraping of the ulcer and culture of the offending organism, generally done by the ophthalmologist. The differential diagnosis as to causative organisms is listed in **Table 241-5**.

Corneal ulcers need to be treated aggressively with topical antibiotics. Emergent ophthalmologic consultation for culture of the ulcer and institution of appropriate antibiotics should be considered. Once cultures are obtained, topical antibiotics are started. A fluoroquinolone such as ciprofloxacin (Ciloxan®) or ofloxacin (Ocuflox®), one drop every hour in the affected eye, is the current recommended treatment. If the suspicion is high for viral or fungal infection, a topical antiviral medication (e.g., natamycin, amphotericin B, or fluconazole) should be given.

Cycloplegic drops such as cyclopentolate 1% are often used due to pain from accompanying iritis. Topical steroids are relatively contraindicated in viral infections but may decrease the incidence of scarring and perforation. Steroid eye drops should not be initiated by the emergency physician unless advised to do so by the ophthalmologist. **Do not patch the eye because of the risk of *Pseudomonas* infection, which can cause rapid, aggressive ulceration with corneal melting and perforation.** All corneal ulcers should be referred to an ophthalmologist to be seen within 12 to 24 hours. Complications of corneal ulcers include corneal scarring, corneal perforation, development of anterior and posterior synechiae, glaucoma, and cataracts.

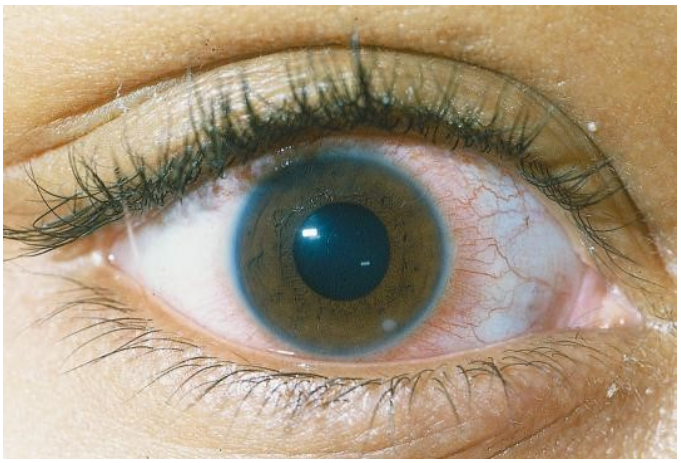


FIGURE 241-32. Corneal ulcer is seen at 5 o'clock. [Courtesy of Allen R. Katz, Department of Ophthalmology, University of Nebraska Medical Center.]

Ultraviolet Keratitis Light in the ultraviolet range can cause death of corneal epithelial cells. Classically described as "snow blindness," **ultraviolet keratitis** is also caused by unprotected exposure to arc welders and tanning beds and is often called "*welder's flash*." Ultraviolet keratitis may occur if protective glasses are not applied tightly to the face, allowing light to hit the cornea obliquely. Effects are cumulative, so multiple short exposures are the same as one long exposure. Corneal cells do not die immediately, so symptoms develop after a delay of up to 6 to 12 hours with slow onset of foreign body sensation and mild photophobia, progressing to severe pain and photophobia. Patients sometimes are awakened from sleep after midnight by pain. Physical examination may reveal blepharospasm, conjunctival injection, and prominent tearing. Topical anesthetic drops are often required to complete the eye examination. Slit lamp examination reveals diffuse punctate corneal edema, and instillation of fluorescein reveals diffuse punctate corneal abrasions. Treatment may include double patching of both eyes if the patient requests, and the use of cycloplegics, topical antibiotics (erythromycin), and oral analgesics. Healing occurs in 24 to 36 hours.

UVEAL TRACT

Uveitis/Iritis Iritis is inflammation of the anterior segment of the uveal tract. It is not a true ocular emergency, but does require follow-up by an ophthalmologist. Iritis is caused by inflammation of various local and systemic etiologies. Pain in iritis is caused by irritation of the ciliary nerves and ciliary muscle spasm. Ciliary spasm irritates the trigeminal nerve and can cause photophobia. Keratitic precipitates are deposits of inflammatory cells on the corneal endothelium. A proteinaceous transudate from uveal vessels occurs in the anterior chamber and causes flare seen with the slit lamp. WBCs released from the uveal vessels may be seen in the anterior chamber with the slit lamp and are termed *cells* (as in "flare and cells"). Cells appear as snowflakes in a headlight beam at night.

The patient will complain of unilateral pain, although the pain may be bilateral with systemic disease. There may be complaints of conjunctival injection, photophobia, and decreased vision. There is usually no discharge. Complaints of systemic symptoms, including arthritis, urethritis, and recurrent GI symptoms, are not unusual. Past medical history should include exposure to tuberculosis, history of genital herpes, or history of previous similar symptoms, and the associated diagnosis. Ask about recent trauma or exposure to welding without protective goggles.

Inspection of the eye may reveal a perilimbal flush (injection is greatest around the limbus) or diffuse conjunctival injection without mucopurulent discharge. Photophobia is usually present. **Consensual photophobia (shining light on the unaffected eye causes pain in the affected eye) is highly suggestive of iritis.** The pupil is usually miotic and poorly reactive. Visual acuity may be decreased with severe inflammation and clouding of the aqueous humor. Slit lamp examination will reveal flare and cells in the anterior chamber, culminating in a hypopyon with severe disease. Intraocular pressure may be decreased if the ciliary body is involved secondary to decreased production of aqueous humor. Fluorescein staining of the cornea may show abrasions, ulcerations, or dendritic lesions.

The diagnosis of iritis is based on the typical history and the finding of flare and cells in the anterior chamber on slit lamp examination. The differential diagnosis is listed in **Table 241-6**.

Blocking the pupillary sphincter and ciliary body with a long-acting cycloplegic agent, such as homatropine (duration 2 to 4 days) or tropicamide (duration 24 hours), will decrease pain. Refer the patient to an ophthalmologist in 24 to 48 hours.

TABLE 241-6 Differential Diagnosis of Iritis

Systemic diseases	Malignancies
Juvenile rheumatoid arthritis	Leukemia
Ankylosing spondylitis	Lymphoma
Ulcerative colitis	Malignant melanoma
Reiter syndrome	Trauma/environmental
Behçet's syndrome	Corneal foreign body
Sarcoidosis	Post-traumatic (blunt trauma)
Infectious	Ultraviolet keratitis
Tuberculosis	
Lyme disease	
Herpes simplex	
Toxoplasmosis	
Varicella zoster	
Syphilis	
Adenovirus	

VITREOUS HUMOR

Endophthalmitis Endophthalmitis is inflammation (usually infectious) of the aqueous or vitreous humor that frequently leads to loss of vision. The most frequent cause is postsurgical, followed by penetrating ocular injuries and, rarely, hematogenous spread. History may include ocular surgery, hammering with steel, working with high-speed machinery such as grinders or weed whackers, or ocular trauma. Symptoms may include headache, eye pain, photophobia, vision loss, and ocular discharge. Physical examination may reveal erythema and swelling of the lids, conjunctival and scleral injection, chemosis, hypopyon, and evidence of uveitis. If suspected, immediate ophthalmologic consultation is required. Treatment may include aspiration of the vitreous or pars plana vitrectomy, and administration of intravitreal antibiotics and steroids, in addition to systemic antibiotics. Admission is required except for postoperative cases.

Vitreous Detachment and Hemorrhage The vitreous is avascular and attached firmly to the anterior eye at the ora serrata, posteriorly at the optic nerve head, and along the major retinal vessels. Liquefaction of the vitreous can cause detachment from the retina, with or without accompanying hemorrhage. Symptoms are sudden onset of floaters, especially with eye movement. Traction at vascular areas due to trauma or pathologic neovascularization can cause vitreous hemorrhage. The most common causes of vitreous hemorrhage are proliferative diabetic retinopathy, posterior vitreous detachment in the elderly, and ocular trauma such as shaken baby syndrome in infants. An unusual cause is subhyaloid hemorrhage associated with subarachnoid hemorrhage. **History includes sudden painless vision loss and sudden appearance of black spots, cobwebs, or generalized unilateral hazy vision.** Past medical history may include diabetes or sickle cell disease. Examination of the retina of the affected eye may be impossible due to the hemorrhage. Examination of the contralateral retina may give clues to the diagnosis. Important differential diagnoses include sickle cell disease, diabetic retinopathy, retinal detachment, central retinal vein occlusion, subarachnoid hemorrhage, and lupus erythematosus. Consult ophthalmology if the diagnosis is suspected. Check the INR of patients receiving warfarin (Coumadin®) and withhold antiplatelet therapy. Ocular US can rule out retinal detachment.

TRAUMA TO THE EYE

CONJUNCTIVAL ABRASION, LACERATION, AND FOREIGN BODY

The conjunctiva has less innervation than the cornea, so **conjunctival abrasions** are far less symptomatic than corneal abrasions. The patient may complain of a scratchy foreign body sensation, mild pain, tearing, and, rarely, photophobia. Vision should not be affected unless there is a full-thickness conjunctival laceration with globe penetration. Physical examination may reveal mild conjunctival injection or subconjunctival hemorrhage. A conjunctival abrasion is seen with fluorescein staining.

Conjunctival lacerations may bleed, the edges of the bulbar conjunctiva may retract with underlying sclera visible to the naked eye, and fluorescein stain may pool in the defect. Perform the Seidel test to exclude globe perforation. **The Seidel test can be negative if a full-thickness laceration is small or has spontaneously closed.** Inspect the conjunctiva for a foreign body. Conjunctival foreign bodies usually can

be removed with a moistened, cotton-tipped applicator after anesthetizing the eye with a topical anesthetic. Evert the upper eyelid and inspect under the highest magnification available to avoid missing any additional foreign bodies. Frequently, small wooden particles such as sawdust will blend into the conjunctiva when moistened by the tears and be difficult to find without slit lamp magnification.

Superficial conjunctival abrasions and lacerations without any other associated ocular injury only require erythromycin ophthalmic ointment 0.5% four times a day for 2 to 3 days or no treatment if very small. Suturing of lacerations is almost never required. Any suspicion of globe laceration requires immediate ophthalmologic referral.

CORNEAL ABRASION, LACERATION, AND FOREIGN BODY

The corneal epithelium is fragile and easily damaged. It is richly innervated and therefore very painful when injury occurs. Corneal epithelium regenerates quickly, so healing time for abrasions is short, usually within 24 to 48 hours. Intact corneal epithelium is resistant to infection, but damaged epithelium is a portal of entry for bacteria, viruses, and fungi. Most abrasions not treated immediately will develop an associated inflammatory iritis.

Corneal Abrasion Abrasions may be caused by contact lens wear, fingernails, makeup brushes, and foreign objects blown into eyes while driving or on windy days or that drop into the eye while working overhead (construction) or under a car (mechanics). Injury to the cornea causes intense pain that may be delayed several hours after the inciting event. Initial symptoms are a foreign body sensation, photophobia, and tearing. Ask about the work environment and the mechanism of injury if known, because corneal abrasions sustained using high-speed machinery, such as grinders, lawn mowers, or weed whackers, and hammering metal against metal are associated with corneal laceration and perforation of the globe.

Inspection of the eye may reveal conjunctival injection, tearing, and lid swelling. Blepharospasm may occur with severe pain, requiring a topical anesthetic to accomplish the examination and obtain the visual acuity. Relief of pain with topical anesthesia is virtually diagnostic of corneal abrasion. Photophobia may be evident when shining a light into the affected or the opposite eye. Decreased visual acuity may occur if the abrasion is in the central visual axis or if there is an associated iritis, but otherwise vision should be normal. The corneal abrasion is often visible to the naked eye as an irregular area of light reflection off the cornea.

Slit lamp examination may show flare and cells from iritis if the abrasion is large and >24 hours old, but there is no corneal infiltrate. Examine the entire thickness of the cornea for full-thickness laceration, and the Seidel test should be negative. The abrasion usually appears as a superficial, irregular corneal defect appearing bright green under the cobalt blue light after instillation of fluorescein (**Figure 241-33**). A series of small, fine-lined vertical corneal abrasions seen with fluorescein staining suggests the presence of a foreign body embedded in the tarsal conjunctiva of the upper lid. Multiple linear corneal abrasions or punctuate keratitis also suggest a retained foreign body under the upper lid.

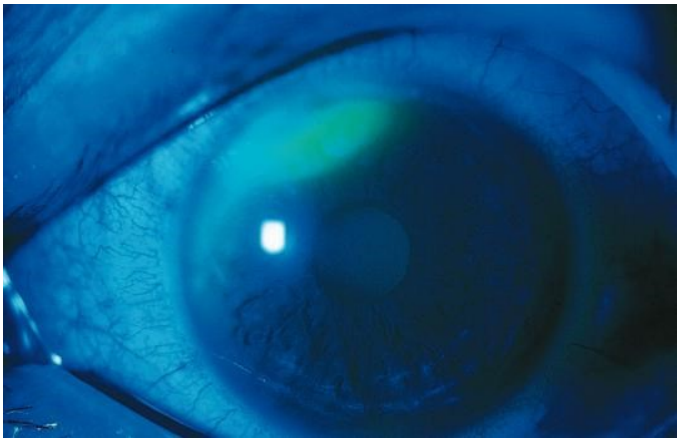


FIGURE 241-33. Corneal abrasion. [Reproduced with permission from Knoop K, Stack L, Storrow A: *Atlas of Emergency Medicine,* 2nd ed. © 2002, McGraw-Hill, New York.]

Some slit lamps (Haag-Streit) have a measuring dial attached to the mechanism that varies the length of the slit beam. If your slit lamp is equipped with this feature, you can vary the length of the slit beam on the cornea until it corresponds to the length or width of the abrasion. The reading on the wheel equals the length of the slit beam in millimeters. This additional feature allows you to document the dimensions of the abrasion precisely, thereby enabling subsequent examiners to evaluate the wound's healing response objectively.

Because the majority of corneal abrasions heal spontaneously, treatment is aimed at relieving pain and preventing infection. Cycloplegics relax the ciliary body and relieve pain from spasm as well as decreasing secondary iritis. Patching the eye does not promote healing, but some patients feel better with the eye patched. Loss of depth perception results from patching one eye, so patients should not drive a car. Abrasions from fingernails, vegetable matter, or a contact lens should not be patched, as they are at higher risk of infection.

If an abrasion is >2 mm or very painful, prescribe a **cycloplegic agent** (cyclopentolate 1% or homatropine 5%), one drop three to four times a day at home, to help control discomfort. The duration of action for each agent is much shorter in the inflamed eye, so a several times a day dosing schedule is recommended. **Cyclopentolate** 1%, one drop three times daily, wears off within 24 hours. Homatropine should be reserved for very large, painful abrasions and lasts several days. **Avoid atropine because the effect lasts for approximately 2 weeks.** Topical nonsteroidal anti-inflammatory drugs such as ketorolac and diclofenac give some degree of pain relief and do not impair healing in patients with corneal abrasions. Topical antibiotics can be provided (**Table 241-7**).

Large abrasions or abrasions in the central visual axis should be checked by an ophthalmologist in 24 hours; small abrasions should be checked in 48 to 72 hours. **Never prescribe topical anesthetics, as they inhibit corneal healing and obliterate the normal corneal protective mechanism** (blinking when material gets into the eye).

Applying an Eye Patch An eye patch may be preferred for comfort for some patients with corneal abrasions or may be needed to prevent keratitis in a patient with Bell's palsy. When an eye patch is properly applied, the eyelid will not move under the eye patch. Assemble supplies—two cotton oval eye pads and multiple pieces of tape pretorn to approximately 5 in. (13 cm). To properly apply an eye patch, have the patient sit with both eyes closed. Have the patient keep both eyes closed until the patch is applied and secure. Take one cotton oval eye pad, fold it in half, and hold the eye pad gently but firmly over the closed lid of the eye to be patched. Then take another eye patch, do not fold it in half, and place it over the first patch. Very deep set eyes may require a third patch. Keep holding firmly but gently. Then tape the patch in place, applying the tape in an X-fashion over the eye pad. Now have the patient open his or her eyes. If the patch is properly placed and taped, the lid under the patch will remain closed. If the patient says the lid can open, remove the patch and try again.

Corneal Laceration Full-thickness **corneal lacerations** can be identified by a misshapen iris, macro- or microhyphema, decrease in visual acuity, and shallow anterior chamber. The Seidel test should be positive. **However, small corneal lacerations can close spontaneously, the**

TABLE 241-7 Suggested Ophthalmic Antibiotics for Corneal Abrasions

Situation	Antibiotic
Not related to contact lens wear	Erythromycin ophthalmic ointment three to four times a day
Related to contact lens wear	Ciprofloxacin, ofloxacin, or tobramycin ointment three to four times a day
Organic source	Erythromycin ointment three to four times a day

Seidel test will be negative, and there may be no gross distortion of globe anatomy (see Figure 241-48). Corneal lacerations occur in young children from a wide variety of objects—sharp sticks, fingernails, thorns, broken glass, or sharp toys.[2] Objects as diverse as bungee cords and eyelash curlers[3,4] can cause globe perforation. A history of eye irritation while working with metal fragments or high-speed machinery suggests the possibility of a corneal laceration. Pain out of proportion to physical findings, decrease in visual acuity, or other unexplained ocular symptoms may be the only symptoms or signs of a small full-thickness corneal laceration. Evaluate the entire thickness of the cornea during slit lamp examination to identify a corneal laceration.

If there is any suspicion of penetrating injury, obtain a CT of the orbit to identify changes in globe anatomy or contour or a foreign body within the globe, and consult ophthalmology. The sensitivity of CT for the detection of occult globe perforation is reported as 56% to 68%,[5] further emphasizing the need for a high index of suspicion. Unrecognized corneal lacerations can quickly result in endophthalmitis or traumatic cataract. Once endophthalmitis develops, vision is at great risk.

Corneal Foreign Bodies Corneal foreign bodies are usually superficial and benign, but penetration of a foreign body into the globe can cause loss of vision. Foreign bodies are generally small pieces of metal, wood, or plastic that become embedded in the cornea. The presence of a corneal foreign body causes an inflammatory reaction, dilating blood vessels of the conjunctiva and causing edema of the lids, conjunctiva, and cornea. When the foreign body is present for >24 hours, WBCs may migrate into the cornea and anterior chamber as a sign of iritis.

The patient will usually complain of a "foreign body" sensation during blinking. Tearing, blurred vision, and photophobia are common. Ask about details surrounding the onset of symptoms, including patient activity. If a cause is not obvious, ask about all activities in the previous 24 hours, especially activities that cause high-velocity projectiles. High-velocity globe penetration injuries include grinding, hammering metal on metal, or operation of other high-speed machinery. Visual acuity should be normal. Inspection of the eye may reveal edema of the eyelid and diffuse or focal/perilimbal conjunctival injection.

Occasionally, the foreign body may be visible with the naked eye. Evert the lid to identify and remove other foreign bodies that may be present. When a metallic foreign body is present for more than a few hours, a **rust ring** (**Figure 241-34**) develops around the metal. Foreign bodies present for >24 hours may be surrounded by a white ring representing a WBC infiltrate. Anterior chamber flare and cells and a corneal foreign body are identifiable on slit lamp examination (**Figure 241-35**). **The presence of a gross hyphema or a microhyphema evident in the anterior chamber on slit lamp examination suggests globe perforation.** If the foreign body has penetrated the cornea, the tract of the projectile *may* be seen. The Seidel test *may* be positive with penetration of the globe.

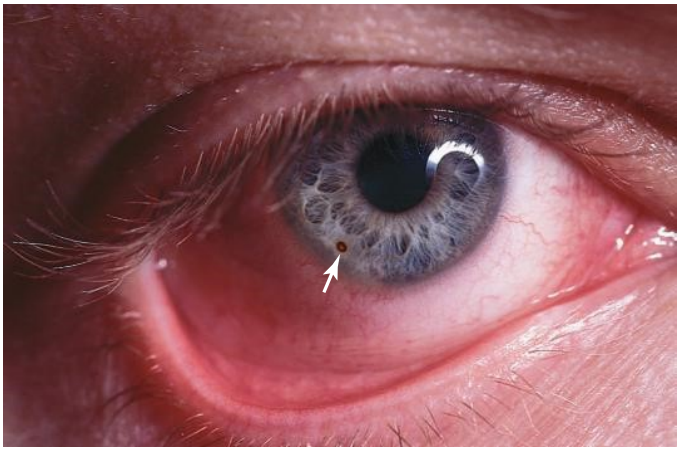


FIGURE 241-34. Rust ring. [Reproduced with permission from Knoop K, Stack L, Storrow A: *Atlas of Emergency Medicine,* 2nd ed. © 2002, McGraw-Hill, New York.]

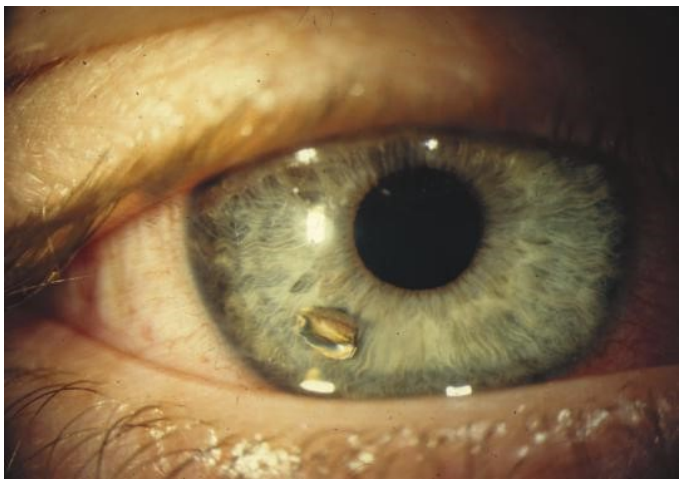


FIGURE 241-35. Corneal foreign body. [Courtesy of Allen R. Katz, Department of Ophthalmology, University of Nebraska Medical Center.]

Corneal Foreign Body Removal Corneal foreign bodies should be removed carefully, with the patient and physician seated at each other's eye level, and under the best magnification available. Anesthetize the cornea with a local anesthetic such as 0.5% proparacaine. Sometimes, anesthetizing both eyes is helpful, because that can eliminate reflex blinking during attempts at foreign body removal. Irrigate with normal saline first, as a very superficial foreign body may be irrigated off the cornea. Next, try to dislodge the foreign body with a moistened cotton applicator. Efforts at removal may themselves cause corneal abrasion.

If the foreign body is tightly adherent to or embedded in the cornea, inspect the cornea using optical sectioning to assess the depth of penetration (**Figure 241-12**). **Full-thickness corneal foreign bodies should be removed by an ophthalmologist.**

For superficial foreign bodies, a 25-gauge needle (using needle bevel up) or a sterile foreign body spud (1 mm diameter) on an **Alger brush** (a low-speed, low-torque, battery-operated hand-held drill) can be used to remove the foreign body. Use slit lamp magnification to ensure safe foreign body removal. Before the removal, prepare a moistened cotton applicator and set it aside on the slit lamp table. The upper lid can be held open by an assistant or by the clinician's nondominant hand. Many slit lamps have an attached "fixation light" that can be moved in front of the unaffected eye to give the patient a steady target to concentrate on. Using either the 25-gauge needle or the Alger brush, place the tip into the slit lamp beam using the naked eye. With the tip close to the cornea, look through the slit lamp and move the tip into contact with the cornea. Using the bevel-up edge of the tip of the 25-gauge needle, hook the edge of the foreign body and dislodge it. You may then lift it off the cornea using the previously moistened cotton applicator. Alternatively, using the spinning tip of the Alger brush, the foreign body may be dislodged and removed with the cotton applicator as above.

After successful foreign body removal, discharge the patient with a prescription for topical antibiotics, cycloplegics, and oral analgesics. Administer tetanus toxoid as appropriate. Provide ophthalmology follow-up the next day if the foreign body is in the central visual axis or if there is a residual rust ring. Otherwise, after complete removal of the foreign body, advise follow-up if symptoms persist at 48 hours.

Rust Ring Removal Metallic foreign bodies can create rust rings that are toxic to the corneal tissue (**Figure 241-34**). If a rust ring is present, the spud or an ophthalmic burr can remove superficial rust, but rust often reaccumulates by the next day, requiring additional burring. **It is therefore not necessary to remove a rust ring in the ED if the patient can be seen by an ophthalmologist the next day.** Once the metallic foreign body is removed, the rust ring area softens overnight and can be more easily removed in the office the next day. The deeper the stromal involvement, the higher is the risk of corneal scarring, so if rust ring removal is done in the ED, only perform superficial burring. **No ED drill burring should take place if the rust ring is located in the visual**

axis (pupil) owing to the risk of causing visually significant scarring. Such conditions require that an ophthalmologist remove the stromal rust in the office within 24 hours.

LID LACERATIONS

Eyelid lacerations that involve the lid margin, those within 6 to 8 mm of the medial canthus or involving the lacrimal duct or sac, those involving the inner surface of the lid, wounds associated with ptosis, and those involving the tarsal plate or levator palpebrae muscle need repair by an oculoplastic specialist. Lacerations medial to the lacrimal puncta are at high risk of canalicular involvement. Suspect involvement of the levator palpebral muscle in the presence of a horizontal laceration with ptosis or when orbital fat is seen protruding through the laceration, indicating a breach of the orbital septum.

Consider the possibility of corneal laceration and globe rupture in all full-thickness lid lacerations. Ocular injuries such as corneal abrasion, traumatic hyphema, and globe rupture are seen with lid lacerations in up to two thirds of cases. Lid lacerations require a thorough examination using a slit lamp to exclude other associated ocular injuries.

Deep lacerations medial to the punctum potentially can transect the canalicular system. These injuries need to be seen by an ophthalmologist for evaluation of the nasolacrimal duct system's integrity. Instillation of fluorescein dye in the eye with subsequent appearance in the wound indicates loss of canalicular integrity. If a canalicular laceration is discovered, the patient will need to go to the operating room within 24 to 36 hours for repair and Silastic® tube stenting. Because a meticulous repair by an experienced eye surgery team is preferable, it is not unreasonable for the ophthalmologist to discharge a patient seen late in the evening or on the weekend with arrangements for surgical repair to take place within the next 36 hours. Patients discharged pending repair should be placed on oral and topical antibiotics and told to use cold compresses. Oral cephalexin (Keflex®), 500 milligrams twice or four times daily, and topical erythromycin ophthalmic ointment four times daily are reasonable choices.

Partial-thickness lid lacerations not meeting the preceding criteria can usually be repaired in the ED, with referral for ophthalmologic evaluation in 2 to 3 days. Use a soft, absorbable or nonabsorbable 6-0 or 7-0 suture. Have the suture ends closest to the cornea tucked under more distant sutures to avoid corneal irritation (**Figure 241-36**). Cut the ends of each suture 1 cm long. When the second suture is tied, take the long ends of the first suture into the loop of the knot of the second suture. This keeps the ends of the first suture secure. Do this for every successive suture. Do not incorporate the ends of the suture into the wound itself, but make sure the suture ends are kept to the side of the wound margin. Sutures are removed from the bottom.

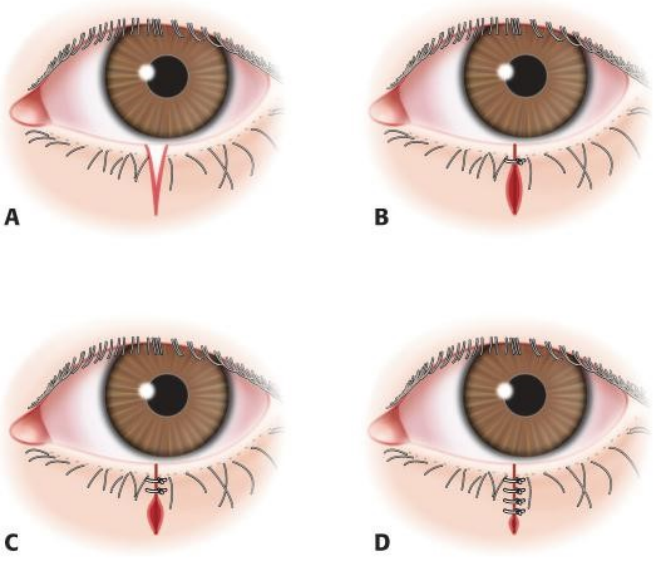


FIGURE 241-36. **A** through **D.** Each suture holds down the ends of the adjacent suture to prevent corneal irritation or abrasion. On this drawing, the lighter shade sutures are above the skin. Note that this illustration shows a laceration at the lid margin. Lid margin lacerations >1 mm need repair by an oculoplastic specialist.

Lacerations at the Lid Margin **Very small lacerations (<1 mm) at the lid edge only do not need suturing and can heal spontaneously.** Any laceration >1 mm at the lid edge needs repair by a specialist. Proper alignment of the lid margin during repair under magnification (loupe or microscope) is essential to preserve proper lid function and even corneal wetting with each blink. Notching of the lid can result in improper lid closure.

If there is no opportunity for the patient to see an ophthalmologist, repair should be performed as in **Figure 241-37**. Soft (gut or chromic) sutures 6-0 or smaller should be used for all repairs. One vertical mattress suture, using the meibomian gland orifices as a landmark, or two 6-0 soft sutures (one approximating the anterior and the other the posterior lamella) are used to repair the lid margin. The initial suture can be used for traction to extend the lid and facilitate the repair. The tarsus should be repaired with 6-0 absorbable suture (polyglactin) from the external side so as to approximate the wound without the need for sutures on the conjunctival side of the lid (which would abrade the cornea with each blink). Skin closure can be performed with 6-0 or smaller soft nonabsorbable suture.

BLUNT EYE TRAUMA

The first steps are assessment of the visual acuity, anterior chamber, and integrity of the globe. The eyelids frequently swell shut, making visualization of the globe difficult. Prying the eyelids open with the fingers is difficult, usually yields an unsatisfactory view of the globe, and can raise intraocular pressure. Insertion of a paperclip bent in an appropriate shape (**Figure 241-38**) or an eyelid speculum (**Figure 241-39**) provides a significantly improved view of the cornea and anterior chamber. Use of an eyelid retractor allows your hands to remain free for examination of the globe using the slit lamp.

If the anterior chamber is flat, a ruptured globe is certain, so stop the examination, place a metal shield over the injured eye, and consult ophthalmology. A **hyphema** is also evidence of significant ocular trauma and necessitates an ophthalmology consult. If the globe appears intact and vision is preserved, check ocular motility. Restricted upgaze or lateral gaze suggests a **blow-out fracture** with entrapment (see later section, Orbital Blow-Out Fractures), and a CT scan of facial bones should be obtained. A head CT scan may be indicated to assess for associated intracranial injury. Feel the orbital rim above and below for step-off deformities. Test for cutaneous sensation along the distribution of the inferior orbital nerve (below the eye and ipsilateral side of the nose). **Perform a slit lamp examination with fluorescein staining to check for abrasions, lacerations, foreign bodies, hyphema, iritis, and lens dislocation.** Measure intraocular pressure if there are no signs of a ruptured globe. Traumatic iritis is common, causing cell and flare to be seen on slit lamp examination. The pupil can be constricted or dilated after sustaining trauma. It is important to look for pupillary irregularity because the pupil often will peak toward the site of a penetration or rupture. If the anterior chamber is of normal depth and not shallow, apply a mydriatic. Nonwhite, brown-eyed individuals frequently will require an additional drop of a mydriatic to achieve adequate dilation. If vision and ocular anatomy and function are preserved, outpatient follow-up by an ophthalmologist in the next 48 hours should be planned. If a ruptured globe is suspected due to loss of visual acuity, flat anterior chamber, obvious full-thickness laceration, or intraocular foreign body, do not manipulate the eye or measure intraocular pressure. Consult ophthalmology immediately.

Hyphema A **hyphema** is blood or blood clots in the anterior chamber (**Figures 241-15** and **241-16**). Hyphemas are traumatic or spontaneous. A traumatic hyphema usually results from bleeding from a ruptured iris root vessel. Spontaneous hyphemas frequently are associated with sickle cell disease. In addition to standard ophthalmologic history, inquire as to the use of any anticoagulant or antiplatelet medications or history of a bleeding diathesis. **A hyphema may layer out posteriorly when the patient is**

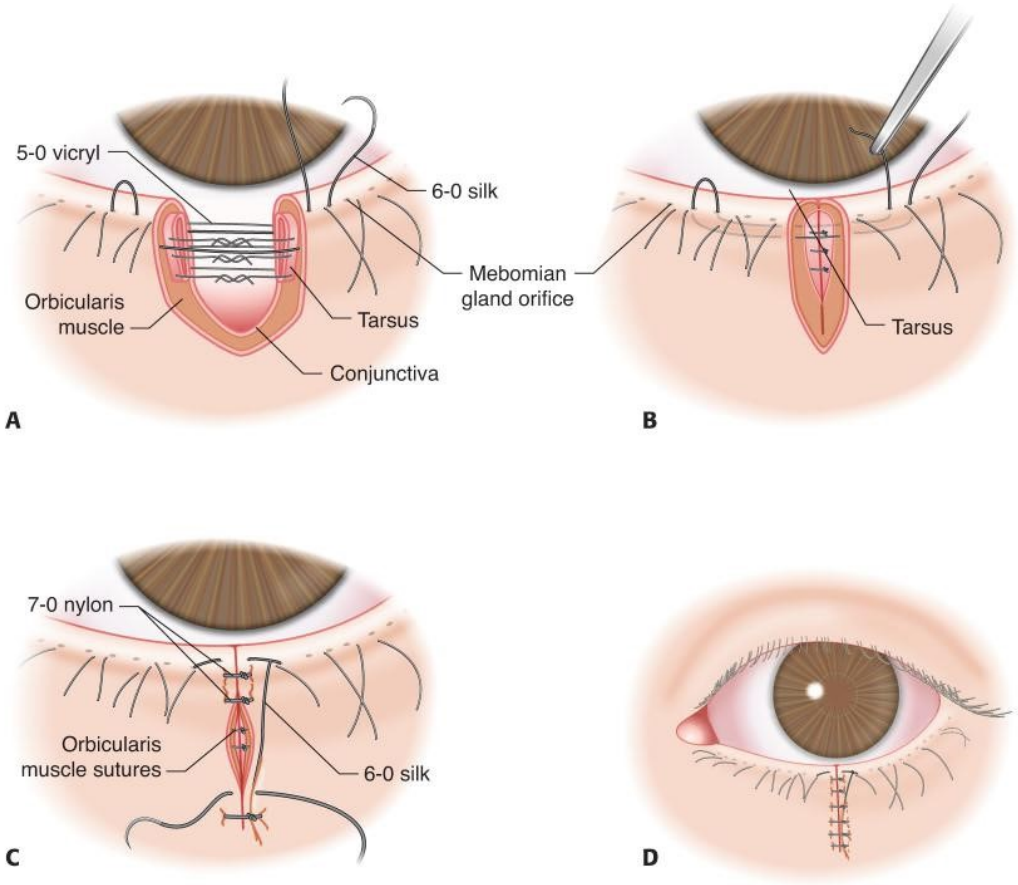


FIGURE 241-37. **A** through **D.** Full-thickness lid repair. 6-0 silk is used for lid margin. 5-0 Vicryl□ is used to approximate the tarsal plate. The Vicryl□ sutures should not pass through the conjunctiva on the inside of the eyelid to avoid mechanical abrasion of the cornea during blinking. 7-0 nylon is used for skin closure, and the lid margin silk suture tail can be incorporated into these sutures to avoid corneal irritation.

lying flat and may only become grossly evident when the patient is sitting upright. The complications of hyphema include increased intraocular pressure, rebleeding, peripheral anterior synechiae, corneal staining, optic atrophy, and accommodative impairment. Patients with large hyphemas, sickle cell disease, and bleeding tendency are more likely to develop vision loss. A **microhyphema** is suspension of red blood cells in the anterior chamber without the formation of a layered blood clot. It is generally seen with a slit lamp and can progress into a hyphema. The most significant complications of microhyphema include rebleeding and intraocular pressure elevation. Patients with sickle cell disease are more likely to develop these complications. The treatment of microhyphema is somewhat controversial, but the principles of management are the same as those for the hyphema.

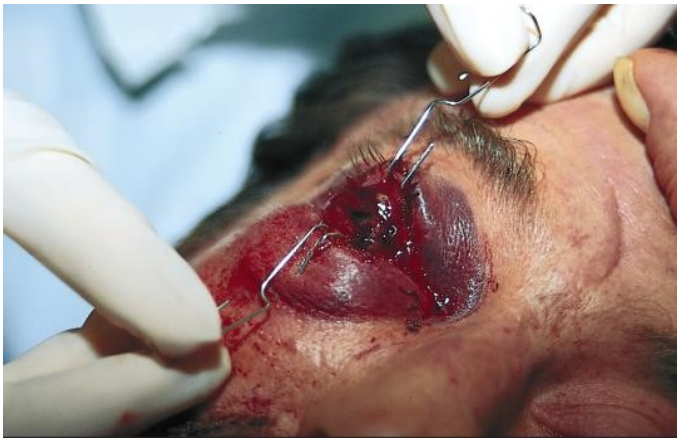


FIGURE 241-38. Eyelid retractors fashioned from paperclips. [Reproduced with permission from Knoop K, Stack L, Storrow A: *Atlas of Emergency Medicine,* 2nd ed. © 2002, McGraw-Hill, New York.]

Hyphemas should be evaluated by an ophthalmologist in the ED. Patients at high risk for complications include those with suspected ruptured globe, those with sickle cell disease, those taking anticoagulants, or those with a bleeding diathesis.

Treatment consists of the prevention of rebleeding and intraocular hypertension. Elevate the patient's head to 45 degrees to promote settling of suspended red blood cells inferiorly to prevent occlusion of the trabecular meshwork. After consultation with the ophthalmologist, dilate the pupil to avoid "pupillary play" (constriction and dilation movements of the iris in response to changing lighting conditions), which can stretch the involved iris vessel, causing additional bleeding. Pupillary dilation *does not* compromise the angle and aqueous outflow in normal individuals, and some ophthalmologists choose to dilate hyphemas to prevent pupillary activity. Control of intraocular pressure consists of topical β-blockers, IV mannitol, topical α-adrenergic agonists (apraclonidine), and oral, topical, or IV carbonic anhydrase inhibitors such as Diamox□. **Do not give carbonic anhydrase inhibitors to patients with sickle cell disease.** Carbonic anhydrase inhibitors lower the aqueous pH in the anterior chamber, causing the red blood cells to sickle and become less flexible, thereby clogging outflow through the trabecular meshwork and increasing intraocular pressure.

Rebleeding can occur 3 to 5 days later in up to 30% of cases, sometimes causing severe elevation of intraocular pressure and necessitating surgical anterior chamber "washouts." Because of this risk, some

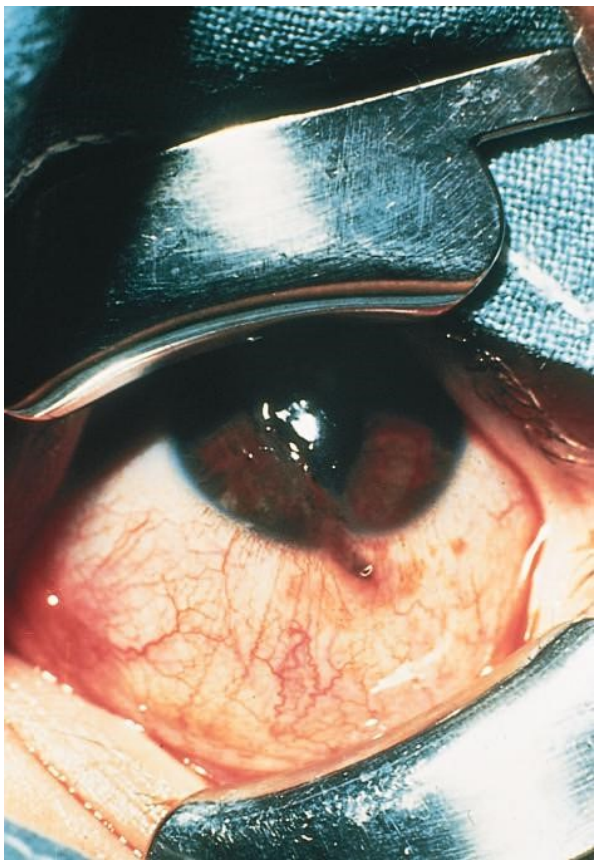


FIGURE 241-39. Commercial eyelid retractors. [Reproduced with permission from Knoop K, Stack L, Storrow A: *Atlas of Emergency Medicine,* 2nd ed. © 2002, McGraw-Hill, New York.]

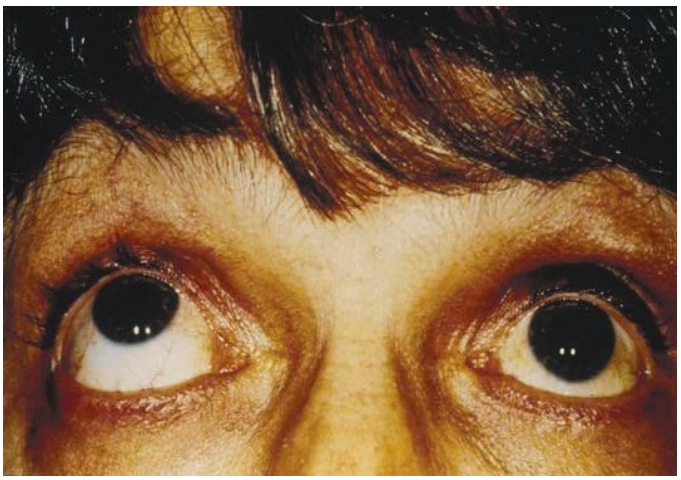


FIGURE 241-40. Inferior wall blow-out fracture of the left eye with entrapment of the inferior rectus muscle. The patient's right eye is unable to look upward, causing diplopia on upward gaze. [Courtesy of Allen R. Katz, Department of Ophthalmology, University of Nebraska Medical Center.]

ophthalmologists believe in admitting all patients with hyphemas, whereas others will choose to follow them closely as outpatients. Generally, patients with hyphemas occupying one third or less of the anterior chamber can be followed closely as outpatients. The disposition decision should be made by the ophthalmologist after examining the patient. Lower risk of rebleeding has been reported in patients who receive topical glucocorticoids. Additionally, topical steroids may prevent posterior synechiae and treat iridocyclitis. Close follow-up and serial examinations by an ophthalmologist are recommended in patients receiving topical ocular steroids to ensure no infection or corneal perforation occurs.

Orbital Blow-Out Fractures The most frequent sites of **orbital blow-out fractures** are the inferior wall (maxillary sinus) and medial wall (ethmoid sinus through the lamina papyracea). Fractures of the medial wall can be associated with subcutaneous emphysema, sometimes exacerbated by sneezing or blowing the nose. Fractures of the inferior wall with entrapment of the inferior rectus muscle can cause restriction of upgaze and diplopia (**Figure 241-40**). Orbital wall fractures are suspected on clinical examination and confirmed by CT scanning. About one third of blow-out fractures are associated with ocular trauma (abrasion, traumatic iritis, hyphema, lens dislocation/subluxation, retinal tear, or detachment); therefore, a careful eye examination in the ED is necessary. **All blow-out fractures with normal initial eye examination in the ED should be referred to an ophthalmologist for an outpatient fully dilated examination to rule out any unidentified retinal tears or detachments.** Orbital blow-out fractures without any evidence of serious eye injury do not require admission.

Isolated blow-out fractures with or without entrapment and without any eye injury do not require immediate surgery and can be referred to ophthalmology, plastic surgery, oral maxillofacial surgery, or otolaryngology (depending on the local referral patterns) for repair within the next 3 to 10 days. Oral antibiotics (cephalexin, 250 to 500 milligrams PO four times daily for 10 days) are often recommended because of the presence of sinus wall fractures.

Ruptured Globe Rupture of the globe is a vision-threatening emergency that may be easily missed. The patient will usually complain of eye pain but may not have a decrease in visual acuity. Rupture of the globe presenting with a large subconjunctival hemorrhage is easily recognized, but a penetrating wound of the cornea caused by a tiny piece of metal launched from a grinder may be easily overlooked and requires a high index of suspicion to detect. Periorbital ecchymosis and maxillofacial fractures, including blow-out fracture with limitation of extraocular muscle movement , should raise one's suspicion for globe rupture.

Scleral rupture may occur from blunt or penetrating trauma. Blunt trauma directly to the eyeball (for example, a blow by a fist) will cause a sudden elevation of intraocular pressure, with the globe tending to rupture at the thinnest points of the sclera, the limbus, and at the insertion of the extraocular muscles. Any object that impacts the orbital rim at a high velocity and causes a seal around the orbit (tennis balls, racquetballs, etc.) will also cause a sudden peak in intraocular pressure and may result in rupture. A history of ocular surgery or previous ocular injury may predispose to globe rupture. Penetrating trauma may occur from bullets, BB pellets, knives, sticks, darts, needles, hammering, and lawn mower projectiles. **Any projectile injury has the potential for penetrating the eye.** The bony canal protects the globe from posterior and oblique injuries, but the eyelids afford little protection anteriorly. **Suspect globe penetration with any puncture or laceration of the eyelid or periorbital area,** and make sure to conduct a thorough slit lamp examination**.** The smaller the diameter of the offending object, the higher is the likelihood of occult injury. **Corneal abrasions occurring when hammering metal on metal; associated with the use of high-speed machinery such as lawn mowers, line trimmers (weed whackers), grinders, or drills; and sustained during explosions should always be investigated for occult globe penetration.**

Whenever globe rupture is obvious or strongly suspected, cover the eye with a metal eye shield or make a shield from a paper cup (Figures 241-41 and 241-42), and consult ophthalmology immediately without further manipulation. Elevate the head of the bed to 45 degrees. Administer broad-spectrum IV antibiotics, and give tetanus toxoid as appropriate. Provide sedation and analgesia, and administer antiemetics to prevent increased intraocular pressure and extrusion of intraocular contents from vomiting. Avoid any topical eye solutions. Give the patient nothing by mouth, anticipating surgery.

Most cases, however, will require an initial eye examination to determine the type and extent of injury before consulting ophthalmology. **If at any step of the examination globe rupture is suspected, stop the examination, place a protective shield over the eye, and consult**

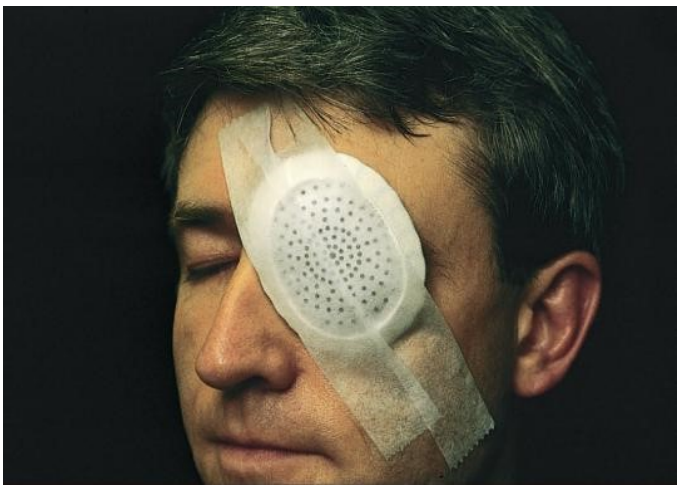


FIGURE 241-41. Metal eye shield placed in suspected globe rupture to prevent inadvertent pressure on the eye. [Reproduced with permission from Knoop K, Stack L, Storrow A: *Atlas of Emergency Medicine,* 2nd ed. © 2002, McGraw-Hill, New York.]

ophthalmology. Do not measure intraocular pressure as this may result in extrusion of globe contents.

Eye examination must be careful and gentle. Manufactured or homemade eyelid retractors should be used to gently retract the lids to examine the eye and to avoid increasing intraocular pressure during examination.

Examination of the eye may reveal decreased visual acuity, an irregular or teardrop-shaped pupil, an afferent pupillary defect, shallow anterior chamber, hyphema, positive Seidel test, and lens dislocation (**Figure 241-43**). Presence of a large subconjunctival hemorrhage involving the entire sclera or hemorrhagic chemosis (bullous, raised subconjunctival hemorrhage) is very suspicious for rupture of the globe (**Figures 241-44** and **241-45**). Uveal prolapse through a scleral wound may appear as a brownish-black discoloration against the white sclera (**Figure 241-46**). One may occasionally visualize a corneal laceration (**Figure 241-47**) or an intraocular foreign body on slit lamp examination (**Figure 241-48**). The Seidel test may or may not be positive with a small corneal laceration. Funduscopic examination may reveal a poor view of the optic nerve and posterior pole due to vitreous hemorrhage. **Unfortunately, the examination may be nearly normal after globe rupture from a tiny high-speed projectile.**

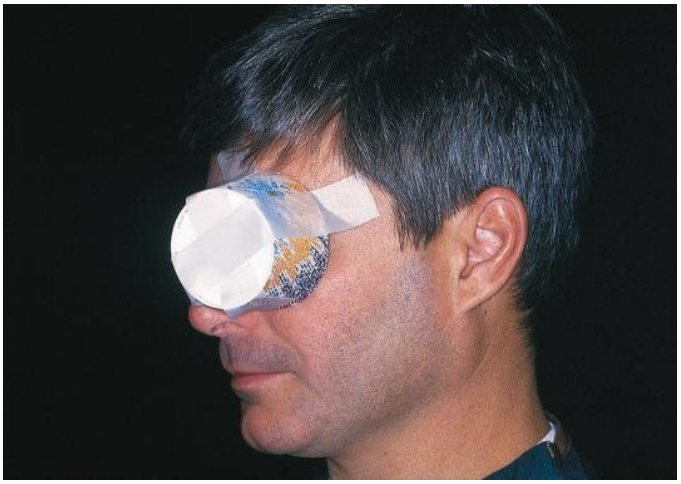


FIGURE 241-42. Protective eye shield fashioned from a paper cup. [Reproduced with permission from Knoop K, Stack L, Storrow A: *Atlas of Emergency Medicine,* 2nd ed. © 2002, McGraw-Hill, New York.]

The diagnosis is made based on a combination of history, physical examination, and selected radiologic studies. CT scan of the eye is the preferred imaging modality to detect occult open globe rupture and associated optic nerve injury. CT scan of the orbit using 2- to 3-mm cuts in both the axial and coronal planes will also localize small intraocular foreign bodies. US and both direct and indirect ophthalmoscopy after pupillary dilatation may assist in the diagnosis and can be helpful in locating and confirming the presence of orbital and intraocular foreign bodies. MRI is contraindicated if a metallic foreign body is possible.

For patients in the ED with multiple trauma and a possible ruptured globe who require rapid-sequence intubation, there is no clear consensus on the best agent to use because quick airway stabilization takes priority. Although a depolarizing agent like succinylcholine is associated with an increase in intraocular pressure, the underlying mechanism is not clear. Some studies suggest that intraocular pressure increases because of extraocular muscle contraction, although others suggest it increases because succinylcholine has a cycloplegic effect on the ciliary muscle. Pretreatment with a nondepolarizing muscle relaxant or a pretreatment dose of succinylcholine does not necessarily attenuate the increase in intracranial pressure. Use of a nondepolarizing agent like rocuronium can mitigate increases in intraocular pressure, but disadvantages are a longer onset and longer duration of action[6] (see Chapter 29, Intubation and Mechanical Ventilation).

Orbital Hemorrhage: Preseptal and Postseptal Severe blunt trauma to the orbit can occasionally cause an orbital hemorrhage. **Preseptal hemorrhage** is dramatic in appearance, but not as vision threatening as a **postseptal hemorrhage** (often called **retrobulbar hematoma)**, which can cause an **orbital compartment syndrome**. A postseptal hematoma can cause an abrupt increase in intraocular pressure, resulting in decreased blood flow to the optic nerve and its blood supply and loss of vision.

Traumatic periorbital hematomas (black eyes) are common, but the extension of bleeding into the postseptal compartment is a true emergency. Differentiation of preseptal versus postseptal hematoma depends on the examination and noncontrast orbital CT findings. Clinical findings of postseptal hemorrhage are eye pain, proptosis, impaired extraocular movements, decreased vision, possibly an afferent pupillary defect, and elevated intraocular pressure. An intraocular pressure >40 mm Hg is a consideration for emergency lateral canthotomy. No matter what the intraocular pressure, if postseptal hematoma is suspected or confirmed, request emergency ophthalmology consultation. Preseptal hematomas can be observed to make sure that the hematoma is not expanding.

Lateral Canthotomy The goals of **lateral canthotomy** are to release pressure on the globe and to reduce intraocular pressure to reestablish retinal artery blood flow. To perform the procedure, place the patient in the supine position and anesthetize the lateral canthus area with 1% to 2% lidocaine with epinephrine. Place a straight Kelly clamp horizontally across the lateral canthus for about 1 to 2 minutes to crush the tissues and minimize bleeding. Remove the clamp, and with sterile scissors, make a 1- to 2-cm lateral incision in the compressed tissue at the clamp site. Then retract the lower lid to expose the lateral canthus tendon. With the scissors directed inferoposteriorly toward the lateral orbital rim, cut the inferior crus of the lateral canthus tendon. This critical incision is generally 1 to 2 cm in depth and length. If the procedure is successful, the intraocular pressure should be less than 40 mm Hg and the visual acuity should improve. If the intraocular pressure continues to remain elevated, the superior crus of the lateral canthus tendon can be cut in a similar fashion. The complications of lateral canthotomy include hemorrhage, infection, and mechanical injury. These complications generally respond to treatment better than does retinal injury from prolonged ischemia. Lateral canthotomy incisions usually heal well without suturing or significant scar formation.

Ocular Hemorrhage and Antithrombotic Therapy The use of anticoagulant and antiplatelet agents is very common in current clinical practice and can complicate the management of ocular hemorrhage from trauma. Spontaneous ocular hemorrhage has also been reported in patients taking these agents. Ocular complications in patients on oral anticoagulant therapy include subconjunctival hemorrhage, hyphema, vitreous hemorrhage, subretinal

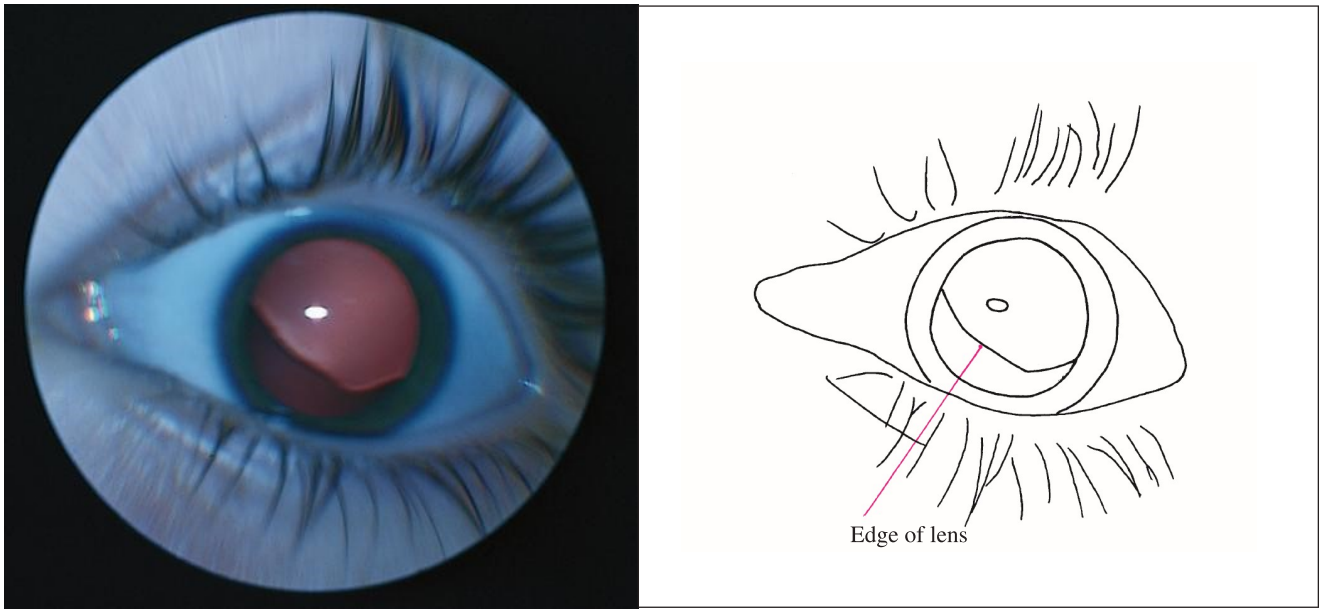


FIGURE 241-43. Lens dislocation. [Reproduced with permission from Knoop et al., *The Atlas of Emergency Medicine*, 3rd edition © 2010 McGraw-Hill Inc.; Photo contributed by Department of Ophthalmology, Naval Medical Center, Portsmouth, VA.]

hemorrhage, and choroidal hemorrhage. A higher incidence of hemorrhagic complications has been reported in patients with macular degeneration using anticoagulants or other antithrombotic drugs. Ocular hemorrhages in patients taking warfarin can potentially be vision-threatening events, but frequently they are benign and resolve without any sequelae.

There are no clear guidelines to guide treatment of ocular hemorrhage in patients taking anticoagulant and antiplatelet therapy. The severity of bleeding, INR levels, and benefits of using these agents should be taken into consideration when managing these patients. Ophthalmologic and hematology consultations should be obtained. These medications should be stopped in patients with active bleeding. Prothrombin complex concentrate, fresh frozen plasma, and vitamin K should be considered in patients taking warfarin (see chapter 239 for detailed discussion). Platelet transfusions might be beneficial to stop active bleeding in patients with thrombocytopenia or taking antiplatelet drugs. Currently, no specific reversal agent for dabigatran is available. Activated prothrombin complex concentrates, recombinant factor VIIa, or concentrates of coagulation factors II, IX, or X and dialysis should be considered in patients taking dabigatran with active hemorrhage.

CHEMICAL OCULAR INJURY

Chemical burns to the eye are a true ocular emergency. Complications of chemical burns to the eye include scarring of the cornea with permanent loss of vision and loss of the eye due to corneal perforation. Irrigation of the eyes with 1 to 2 L of normal saline must be done immediately and before any examination, including testing of vision.

Alkali and Acid Injuries

Alkali injuries occur more frequently than acid injuries, due to the presence of alkaline substances in household cleaning agents and in building materials. The most serious alkali injuries are associated with ammonia, found in many household cleaners, and lye, a common ingredient in drain cleaners. Lye is also a component of concrete. Alkali injuries tend to be much more serious than acid injuries because they cause a liquefaction necrosis, characterized by denaturing of proteins and saponification of fats, allowing deep penetration into tissue. Acid, on the other hand, causes coagulation necrosis, with denaturing of protein forming a coagulum that acts as a barrier to further tissue penetration.

Irrigation should begin at the scene and continue in the ED. Instill a topical anesthetic and continue irrigation for at least 30 minutes. Then

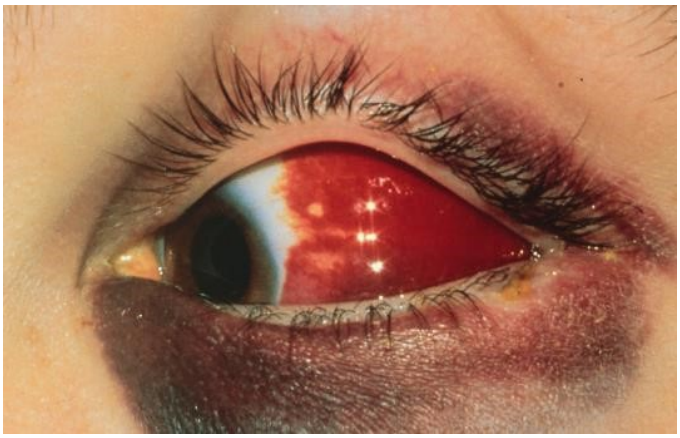


FIGURE 241-44. Large subconjunctival hemorrhage. Note that it is flat, not raised. [Reproduced with permission from Knoop K, Stack L, Storrow A: *Atlas of Emergency Medicine,* 2nd ed. © 2002, McGraw-Hill, New York.]

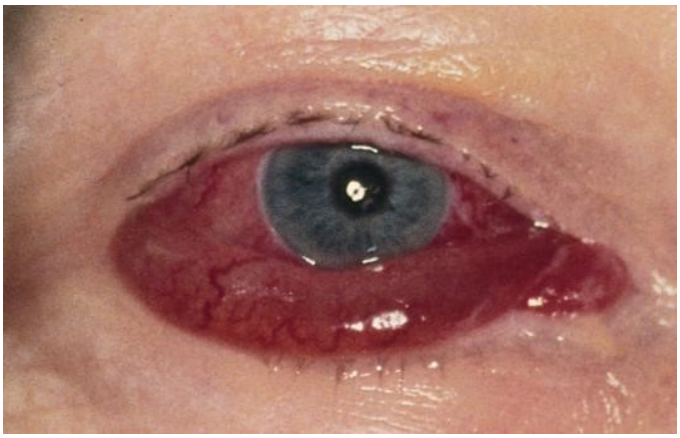


FIGURE 241-45. Bloody chemosis. Note the raised or bullous appearance. [Reproduced with permission from Knoop K, Stack L, Storrow A: *Atlas of Emergency Medicine,* 2nd ed. © 2002, McGraw-Hill, New York.]

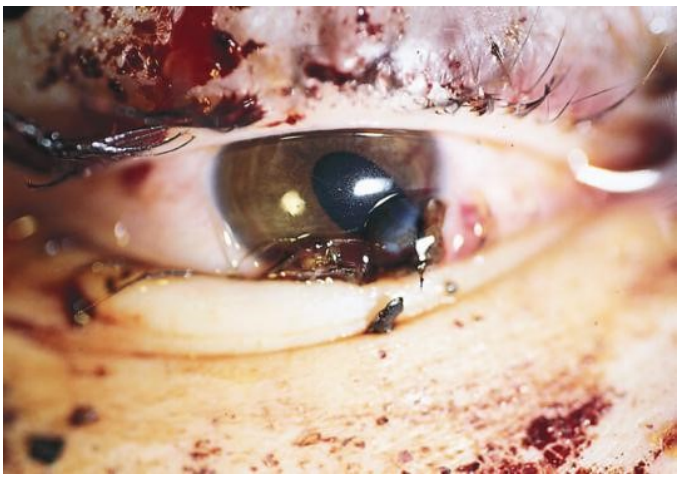


FIGURE 241-46. Uveal prolapse with globe rupture and teardrop pupil. [Reproduced with permission from Knoop K, Stack L, Storrow A: *Atlas of Emergency Medicine,* 2nd ed. © 2002, McGraw-Hill, New York.]

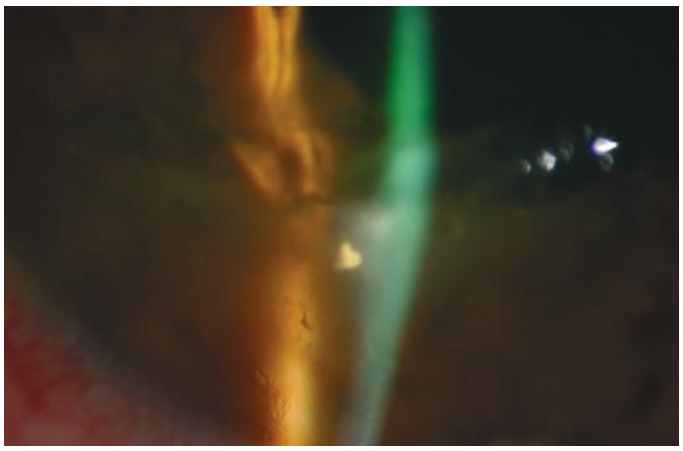


FIGURE 241-48. Intraocular foreign body associated with laceration in Figure 241-47 as seen on slit lamp examination. [Courtesy of Allen R. Katz, Department of Ophthalmology, University of Nebraska Medical Center.]

check pH by touching a strip of litmus paper to the inferior conjunctival fornix. **If the pH is >7.4, continue irrigation until the pH remains neutral 30 minutes after the last irrigation.** Irrigation should be with sterile normal saline or other isotonic solution and may be instilled into the eye by hand, using bottles of eye-irrigating solution, or by a Morgan Lens□ (MorTan Inc., Missoula, MT) (**Figure 241-49**) attached to a bag of an isotonic IV solution.

After irrigation and maintainance of ocular pH >7.4, perform the eye examination. Inspect the facial skin and eyelids for burns. Evert the eyelids and remove any particulate matter with a cotton applicator.

Exposure to chemical agents can cause conjunctival injection and chemosis, but severe chemical burns can cause scleral whitening, secondary to ischemia and blood vessel injury. Document visual acuity and measure intraocular pressure. Intraocular pressure may be increased if the trabecular meshwork has been damaged. Use the slit lamp to evaluate corneal injury and to detect for cells and flare in the anterior chamber. Injury to the cornea may range from punctuate defects to complete loss of epithelium. The cornea may become cloudy with severe burns (**Figure 241-50**).

After irrigation, and once time permits, identify the substance. The pH is usually listed on bottles of household cleaners. The U.S. Occupational Health and Safety Administration requires the patient's workplace to maintain Material Data Safety Sheets, a list of all the physical properties, including pH, of chemicals used at the site. Data on the pH of

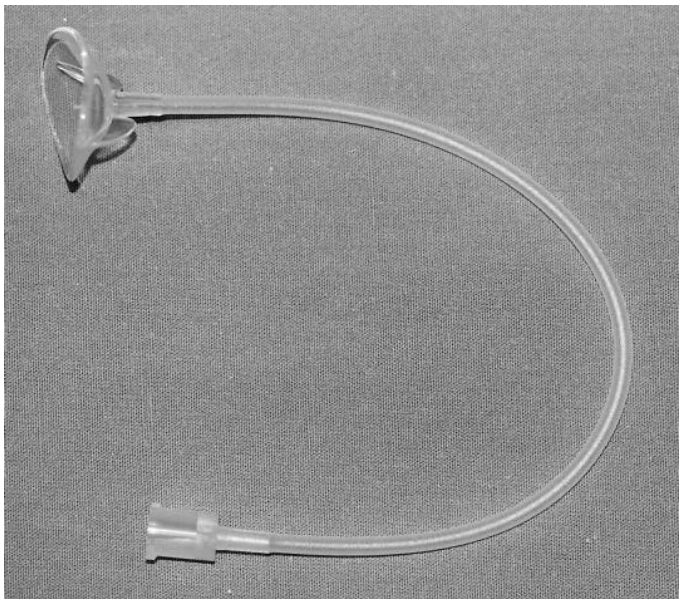


FIGURE 241-49. Eye irrigation with a Morgan Lens□ (MorTan Inc., Missoula, MT). [Reproduced with permission from Reichman EF, Simon RR: *Emergency Medicine Procedures*. © 2004, Eric F. Reichman, PhD, MD, and Robert R. Simon, MD. McGraw-Hill, Inc.]

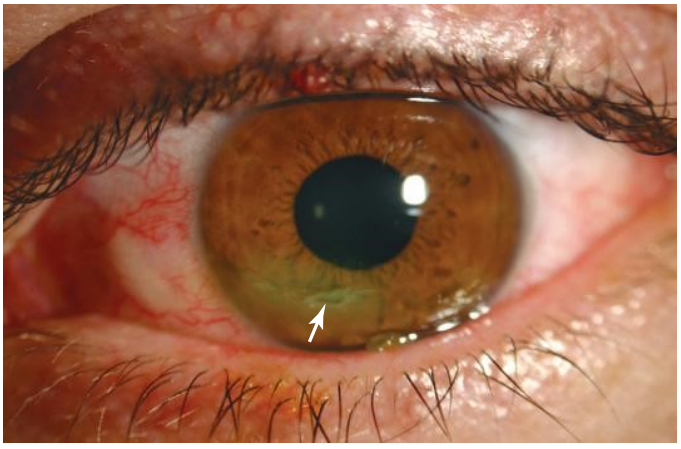


FIGURE 241-47. Corneal laceration due to hammering concrete. [Courtesy of Allen R. Katz, Department of Ophthalmology, University of Nebraska Medical Center.]

FIGURE 241-50. Severe lye burn with corneal opacification. [Reproduced with permission from Knoop K, Stack L, Storrow A: *Atlas of Emergency Medicine,* 2nd ed. © 2002, McGraw-Hill, New York.]

known chemicals can also be obtained from a poison control center or from Poisindex®. Alkaline substances with pH <12 or acidic substances with pH >2 are thought not to cause serious injury, but duration of exposure can increase severity of injury. Circumstances surrounding the injury (e.g., battery explosion) should be determined to identify any other associated ocular or facial injuries.

Obtain ophthalmology consultation for all but minor burns: Any patient with corneal clouding or an epithelial defect after irrigation should receive prompt ophthalmology referral.

Patients with chemosis (edema of the bulbar conjunctiva overlying the white sclera) and no corneal or anterior chamber findings should be treated after irrigation with erythromycin ointment four times daily and referred for an ophthalmologic examination in 24 to 48 hours. These patients are considered to have "**chemical conjunctivitis**."

A topical cycloplegic agent should be used three times daily for pain reduction if an epithelial defect is present. **Avoid phenylephrine as a cycloplegic**, as it will constrict blood vessels, causing further ischemia to the limbus. Apply erythromycin ophthalmic ointment four times daily to affected eyes. Administer tetanus toxoid as appropriate. Consider prescribing topical corticosteroids after consultation with an ophthalmologist to control inflammation.

CYANOACRYLATE (SUPER GLUE/CRAZY GLUE)

Accidental instillation of cyanoacrylate adhesives into the eye and adnexa can cause adherence of the lids and clumps of adhesive to form on the cornea. Medicinal-grade cyanoacrylates are occasionally used to seal corneal perforations and are not toxic to the cornea, so there is rarely permanent damage to the eye. The mechanical abrasive effect of hard, irregular glue aggregates rubbing against the cornea with eye movement and blinking may cause corneal abrasions. To remove crazy glue, instill generous amounts of **erythromycin ointment** onto the eye and on the surface of the eyelids to moisten, lubricate, and provide antibiotic coverage. Clumps of glue on the surface should begin to loosen. Remove only those pieces that are easily removable. Gentle traction may separate the lids. The glue will loosen and become easier to remove in a few days.

Refer to an ophthalmologist within 24 hours for complete removal.

ACUTE VISUAL REDUCTION OR LOSS

Acute visual loss is usually divided into painful and painless visual loss for diagnostic categorization. Other differentiating features include presence or absence of a relative afferent pupillary defect, the rapidity of onset, funduscopic exam, and various physical exam and historical features. The differential diagnosis of visual loss is listed in **Table 241-8**. Many of the diagnoses are discussed in further detail in the text.

ACUTE AND PAINFUL VISION REDUCTION OR LOSS

Acute Angle-Closure Glaucoma • *Pathophysiology* Glaucoma is a group of ocular disorders characterized by increased intraocular

TABLE 241-8 Differential Diagnosis of Visual loss

Diagnosis	Eye Pain	Relative Afferent Pupillary Defect	Onset	Fundoscopic Exam	Other Findings
Central retinal artery occlusion	No	Yes	Sudden	Pale retina, cherry red spot	
Central retinal vein occlusion	No	+/–	Sudden	"Blood and thunder"/"ketchup" fundus	
Acute ischemic optic neuropathy	No	Yes	Gradual	Swollen pale disk	Signs of temporal arteritis
Acute angle-closure glaucoma	Yes	+/–	Sudden	Difficult to visualize the fundus due to corneal edema	Painful red eye, hazy cornea, midpoint pupil, narrow anterior chamber, firm globe
Optic neuritis	Yes	Yes	Gradual	Papilledema	Painful EOM, young female patient
Giant cell arteritis	Possible retro-orbital headache/pain	Yes	Gradual	Normal	Headache, myalgias
Cataract	No	+/–	Gradual	Often unable to visualize fundus	Opacity in the lens
Uveitis	Yes	No	Gradual	Normal	Flare and cells in anterior chamber, ciliary flush, consensual photophobia
Vitreous hemorrhage	No	+/–	Sudden	Opacity in the vitreous	Floaters, cobwebs
Amaurosis fugax	No	No	Sudden	Normal	Transient monocular vision loss
Transient ischemic attack	No	No	Sudden	Normal	Transient binocular vision loss
Cortical blindness	No	+/–	Sudden or gradual	Possible papilledema	Complete visual loss or homonymous hemianopsia, headache
Migraine headache	Possible retro-orbital headache/pain	No	Sudden	Normal	Visual scotomata, nausea, vomiting
Retinal detachment	No	+/–	Sudden	Retina may be difficult to visualize (portion of retina out of focus)	Possible localized visual field defect, "cloudy veil," "window shade"; suspect by history
Diabetic retinopathy	No	+/–	Gradual	Neovascularization, retinal hemorrhages	History of diabetes mellitus
Macular degeneration	No	+/–	Gradual	Drusen, macular pigment clumps	Spots in visual field
Cytomegalovirus retinitis	No	+/–	Sudden or gradual	"Tomato and cheese" pizza (retinal necrosis), retinal hemorrhages	History of human immunodeficiency virus or other immunosuppression
Methanol	No	No	Gradual	Normal	Headache, nausea, vomiting, history of ingestion
Functional visual loss	No	No	Sudden or gradual	Normal	Optokinetic nystagmus

Abbreviation: EOM = extraocular muscle movement.

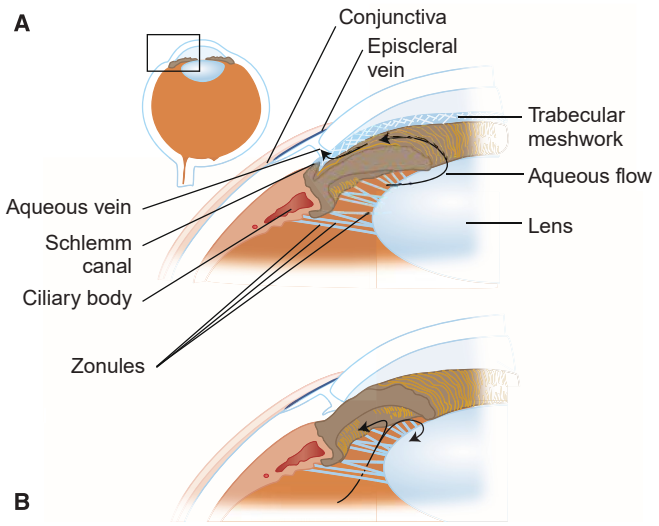


FIGURE 241-51. **A.** Normal flow of aqueous from ciliary body, through the pupil, and out through the trabecular meshwork and Schlemm canal located in the anterior chamber angle. **B.** Angle-closure glaucoma with pupillary block. Iris leaflet bows forward, blocking the chamber angle and prohibiting aqueous outflow. Meanwhile, aqueous production continues, and intraocular pressure rises.

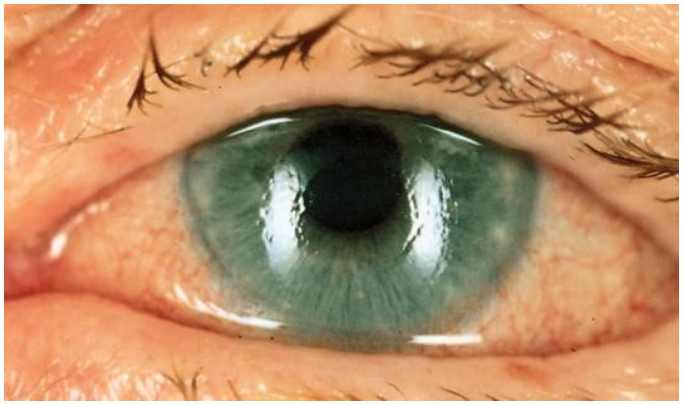


FIGURE 241-52. Acute angle-closure glaucoma. The cornea is cloudy, and there is marked conjunctival injection. [Reproduced with permission from Knoop K, Stack L, Storrow A: *Atlas of Emergency Medicine,* 2nd ed. © 2002, McGraw-Hill, New York.]

pressure causing optic neuropathy and vision loss if left untreated. Obstruction to aqueous humor outflow is the basic underlying problem in glaucoma. In acute angle-closure glaucoma, the lens or the peripheral iris blocks the trabecular meshwork, obstructing the outflow of aqueous humor. This occurs more easily in persons whose eyes have shallow anterior chambers where the angle between the cornea and iris is reduced. A shallow anterior chamber results in a greater area of contact between the lens and iris, impeding flow of aqueous humor from the posterior to anterior chamber. This results in a pressure differential between the posterior and anterior chamber (referred to as *pupillary block*) and causes forward bowing of the iris, further narrowing the angle (**Figure 241-51**).

An acute attack is usually precipitated by pupillary dilation. Dilation increases contact between the iris and lens as the iris becomes thicker. When the pupil is mid-dilated, relative pupillary block and peripheral laxity of the iris are maximal. This increases the degree of pupillary block, increasing pressure in the posterior chamber and causing the iris to bulge forward (iris bombe). The angle between the peripheral iris, trabecular meshwork, and cornea becomes acutely closed, resulting in a precipitous increase in intraocular pressure. Intraocular pressure eventually exceeds the capacity of the corneal pump mechanism, causing the cornea to become edematous and less transparent, thus explaining the foggy vision or halos patients complain of and the hazy appearance of the cornea on physical examination.

As people age, the lens becomes less elastic and thicker, or cataracts may develop. These events can push the iris forward into greater contact with the lens, increasing the degree of pupillary block. Hypermetropic (farsighted) eyes have a shorter anterior to posterior length, a flatter cornea, and a narrower angle, increasing the risk of acute angle-closure glaucoma.

Anything causing pupillary dilatation can trigger an acute attack. The use of topical or systemic parasympatholytic agents (mydriatics, antihistamines) or sympathomimetics (epinephrine, pseudoephedrine), dim illumination, and emotionally upsetting events have all been implicated. Precipitation of acute angle-closure glaucoma has been reported with therapeutic use, and abuse of, intranasal cocaine, as well as therapeutic use of nebulized β-sympathomimetic and anticholinergic medications (e.g., albuterol and ipratropium).

Clinical Features Acute angle-closure glaucoma is abrupt in onset, painful, and may result in severe visual impairment if not treated quickly. Patients complain of sudden onset of severe eye pain or frontal or supraorbital headache. Associated symptoms include blurred vision, nausea, and vomiting. Rarely, acute angle-closure glaucoma may result in painless monocular vision loss. Acute angle-closure glaucoma may be misdiagnosed as migraine, temporal arteritis, subarachnoid hemorrhage, or intra-abdominal emergency.

Examination reveals a fixed, midposition pupil and a hazy (cloudy/steamy) cornea with conjunctival injection (**Figure 241-52**), most prominent at the limbus. The affected eye is rock hard. Measure the intraocular pressure to establish the diagnosis (see earlier section, Intraocular Pressure), although the presence of the characteristic symptom complex—a cloudy cornea, fixed midposition pupil, and rock hard globe—is diagnostic. Normal intraocular pressure is 10 to 20 mm Hg, and may exceed 60 to 80 mm Hg in an acute attack.

Treatment There have been no controlled studies regarding medical treatment for acute angle-closure glaucoma. Treatment of acute angle-closure glaucoma involves lowering the intraocular pressure by blocking production of aqueous humor, facilitating outflow of aqueous humor, and reducing the volume of vitreous humor (**Table 241-9**). IV mannitol quickly lowers intraocular pressure and should be given if there are no contraindications. Pilocarpine frequently will not cause the iris to constrict during the acute attack until the pressure is reduced, due to pressure-induced ischemic paralysis of the iris. Topical steroids are frequently recommended. Pilocarpine 1% or 2% should be instilled into the affected eye once the pressure is lowered (usually not effective until the intraocular pressure is <50 mm Hg) to make the pupil miotic, thereby pulling the peripheral iris away from the angle. Begin treatment immediately, simultaneously with consulting ophthalmology. Definitive treatment is laser iridectomy.

Concern exists regarding precipitation of acute angle-closure glaucoma in patients with shallow anterior chambers who undergo diagnostic mydriasis, but the incidence is low. Some clinicians believe that, if

TABLE 241-9 Treatment of Acute Glaucoma

Treatment	Effect
Topical β-blocker (timolol 0.5%), one drop	Blocks production of aqueous humor
Topical α_2-agonist (apraclonidine 1%), one drop	Blocks production of aqueous humor
Carbonic anhydrase inhibitor (acetazolamide), 500 milligrams IV or PO	Blocks production of aqueous humor
Mannitol, 1–2 grams/kg IV	Reduces volume of aqueous humor
Recheck IOP hourly	—
Topical pilocarpine 1%–2%, one drop every 15 min for two doses once IOP is below 40 mm Hg, then four times daily	Facilitates outflow of aqueous humor

Abbreviation: IOP = intraocular pressure.

necessary, it may be wise to dilate these patients in the ED because precipitation of an acute attack would ideally occur when the patient can get acute care.

ACUTE PAINLESS VISUAL LOSS

Optic Neuritis Acute optic neuritis is often painless but can be painful, especially with eye movement. Visual acuity can range from mildly reduced to profound loss with no light perception. Reduction of vision occurs most commonly over days, but occasionally over hours. Visual loss is usually unilateral, but can be bilateral. Color vision is affected more commonly than visual acuity, and there may be visual field deficits.

The red desaturation test is helpful in identifying optic neuropathies. This test is performed by having the patient look with one eye at a dark red object and then testing the other eye to see if the object looks the same color. The affected eye often will see the red object as pink or lighter red. An afferent pupillary defect (**Figure 241-8**) is commonly present. Funduscopic examination will reveal a swollen and edematous optic disk (papillitis) in approximately 30% of patients. If the head of the optic nerve is normal in appearance, the patient is said to have retrobulbar neuritis.

Optic neuritis can be idiopathic or an initial presentation of multiple sclerosis. Other causes of optic neuritis include postchildhood vaccination; viral infections such as measles, mumps, chickenpox, encephalitis, herpes zoster, and mononucleosis; inflammation of structures contiguous with the optic nerve such as the meninges, orbit, and sinuses; and other infections, including syphilis, tuberculosis, *Cryptococcus*, and sarcoidosis. The differential diagnosis includes ischemic optic neuropathy (sudden onset and painless), papilledema (bilateral, painless with preserved visual acuity), hypertensive retinopathy, orbital tumor compressing the optic nerve (proptosis frequent), intracranial tumor compressing the visual pathway (seen on CT), and toxic or metabolic optic neuropathy from alcohol or various toxins such as the heavy metals or chloroquine.

Neurology and ophthalmology consultation is needed to establish a diagnosis. MRI results are important prognosticators for optic neuritis.

Central Retinal Artery Occlusion The first branch off the internal carotid artery is the ophthalmic artery, which supplies the central retinal artery, which, in turn, provides the blood supply to the inner retina. The ciliary arteries also originate from the ophthalmic artery distal to the central retinal artery and supply the outer retina by the choriocapillaries of the choroid. If the central retinal artery becomes occluded, the inner retina will infarct and become pale, less transparent, and edematous. The macula is the thinnest portion of the retina, and the intact underlying choroidal circulation remains visible through this section of retina, creating the illusion of a "**cherry red spot**." The macular area maintains its normal color, and the surrounding ischemic retina turns pale, thus causing this classic finding on funduscopy (**Figure 241-53**). Causes include carotid or cardiac embolus, retinal artery thrombosis, giant cell arteritis, vasculitis (lupus, polyarteritis nodosa), sickle cell disease, trauma, vasospasm (migraine), elevated intraocular pressure (glaucoma), hypercoagulable states, and low retinal blood flow (carotid stenosis or hypotension).

Sudden (occurring over seconds), profound, painless, monocular loss of vision is characteristic of a central retinal artery occlusion. The event is often preceded by episodes of amaurosis fugax. Physical examination will often reveal an afferent pupillary defect in addition to the pale retina and cherry red macula. **Evidence-based treatment and information about the course of the disease are lacking.** Central retinal artery occlusion is rare, thought to account for 1/10,000 ophthalmic visits.[7] There are no data on ED visits. There is no evidence supporting or refuting the success of maneuvers such as digital massage, intraocular pressure–lowering drugs, and breathing into a paper bag to increase partial pressure of arterial carbon dioxide.[8,9] There are reports discussing the use of intra-arterial tissue plasminogen activator within 20 hours of onset of symptoms.[7] **Consult an ophthalmologist and neurologist immediately when suspecting the condition, and follow institutional protocols for treatment.** Irreversible loss of visual function usually occurs after 4 hours of ischemia.

Central Retinal Vein Occlusion Thrombosis of the central retinal vein causes retinal venous stasis, edema, and hemorrhage. Risk factors include diabetes, hypertension, cerebrovascular disease, cardiovascular disease, dyslipidemia, hypercoagulable states, vasculitis, glaucoma, and compression of the vein in thyroid disease and orbital tumors. Loss of vision is variable, ranging from vague blurring to rapid, painless, and monocular loss of vision. Funduscopic examination typically reveals optic disk edema and diffuse retinal hemorrhages in all quadrants ("blood-and-thunder fundus") (**Figure 241-54**). The contralateral optic nerve and fundus generally are normal, which helps distinguish central

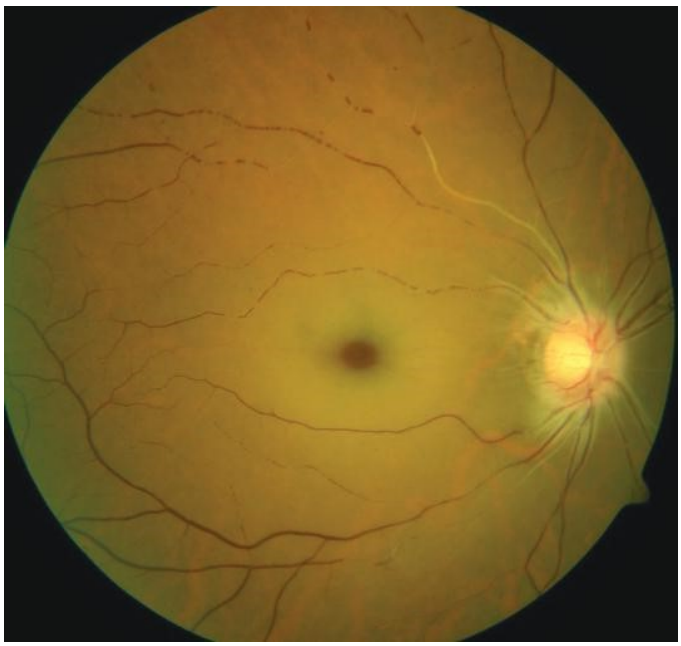


FIGURE 241-53. Central retinal artery occlusion. Note macular "cherry red spot" and retinal pallor as well as the plaques visible in the retinal vessels. [Courtesy of Allen R. Katz, Department of Ophthalmology, University of Nebraska Medical Center.]

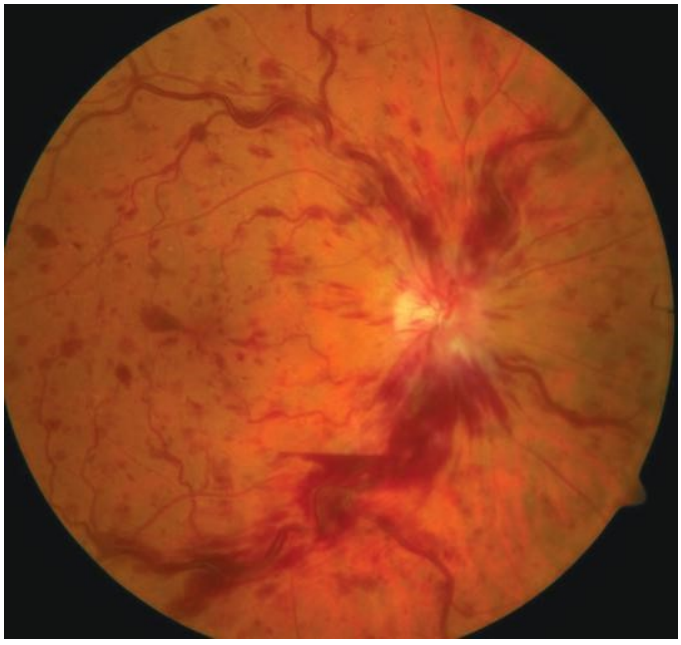


FIGURE 241-54. Central retinal vein occlusion. The disk margin is blurred, the veins are dilated and tortuous, and there is a large amount of hemorrhage typical of the "blood-and-thunder fundus." [Courtesy of Allen R. Katz, Department of Ophthalmology, University of Nebraska Medical Center.]

retinal vein occlusion from papilledema, and the diffuse retinal hemorrhages help distinguish it from optic neuritis (the peripheral retina is normal in optic neuritis). No specific treatment is available. Consult neurology and ophthalmology.

Flashing Lights and Floaters/Retinal Detachment Complaints about new-onset flashing lights and/or floaters commonly cause patients to seek urgent medical attention. The first distinction to make is if the symptoms are monocular or binocular. **Binocular complaints are almost always intracranial (i.e., ophthalmic migraines), whereas monocular complaints are almost always related to the symptomatic eye.**

The posterior segment of the eye is a large cavity filled with vitreous gel. As a person ages, this gel eventually contracts centrally and separates from the posterior wall of the eye. The vitreous is very sticky and tugs on the retina before separation, stimulating the retina, which the brain perceives as light. The average age of onset is 55 years old, but it can occur as early as the 20s in severely nearsighted people. If the vitreous gel separates successfully, then floaters occur, which may persist for years until the gel liquefies enough for the floaters to sink below the visual axis. If the gel creates enough traction on the retina before separation that it tears a hole in the retina, then fluid can go through the retinal hole and start to peel the retina off like wallpaper.

Symptoms may include flashes of light, floaters, a dark veil or curtain in the field of vision, and decreased peripheral and/or central visual acuity. This is an emergent condition requiring a retina specialist to evaluate and treat the patient. **Diagnosing a retinal detachment or tear or vitreous detachment requires a dilated indirect ophthalmoscopic evaluation by an ophthalmologist within 24 hours.** Most tears occur in the peripheral retina, which is not visualized on the direct funduscopic examination. A large retinal detachment will appear as a pale billowing parachute on dilated funduscopic examination. The diagnosis is mainly by history and confirmed by the ophthalmologist.

Temporal Arteritis **Temporal arteritis**, also called **giant cell** or **cranial arteritis**, is a systemic vasculitis involving medium-sized and large arteries. The temporal artery is the most common vessel involved. The disease causes a painless ischemic optic neuropathy with profound visual loss and contralateral ocular involvement in days to weeks if not diagnosed and treated promptly. Patients are generally >50 years of age and frequently have a history of polymyalgia rheumatica. Symptoms may include headache, jaw claudication, myalgias, fatigue, fever, anorexia, and temporal artery tenderness. Many patients may have associated symptoms of transient ischemic attacks or stroke. The physical examination frequently will reveal an afferent pupillary defect if the optic nerve circulation is involved. An elevated sedimentation rate is usually present, with the majority of biopsy-proven cases in the range of 70 to 110 mm/h. The added presence of an elevated C-reactive protein also suggests the diagnosis. Treatment consists of several doses of IV steroids followed by oral steroids. Steroids should not be delayed while waiting for a temporal artery biopsy to be performed. Biopsies will still be positive a week after initiation of steroid therapy.

CRANIAL NERVE PALSIES

BELL'S PALSY AND GENU VII BELL'S PALSY

Bell's palsy is a dysfunction of peripheral cranial nerve VII commonly of viral origin. It is palsy of the ipsilateral upper and lower face. The orbicularis muscles are involved, resulting in incomplete closure of the eyelids on the affected side and leading to corneal exposure keratitis. Prescribe viscous topical wetting agents to keep the corneal epithelium from breaking down, and patch the affected eye. Ophthalmology referral for outpatient monitoring of the cornea is warranted.

Treatment of Bell's palsy remains controversial. As of this writing, the most recent Cochrane review seems to conclude that antivirals provide no benefit over placebo in the treatment of Bell's palsy. Corticosteroids alone and antivirals with corticosteroids confer treatment benefit.[10] Consequently, the best evidence at present is to consider the administration of both antivirals and steroids, but not antivirals alone.[10]

Genu VII Bell's palsy is a stroke, masquerading as a peripheral seventh-nerve Bell's palsy, involving cranial nerve VI and the ipsilateral cranial nerve VII as it "genuflects" around the sixth-nerve nucleus. **This results in a cranial nerve VII palsy identical to a typical Bell's palsy (affecting the upper and lower face ipsilaterally) but with the added finding of the patient's inability to abduct the ipsilateral eye (cranial nerve VI palsy).** This underscores the importance of extraocular muscle testing in all Bell's palsy patients.

DIABETIC/HYPERTENSIVE CRANIAL NERVE PALSIES

Chronic diabetes and hypertension eventually can create vascular compromise to the vasa nervorum of any cranial nerve. **The pupil is spared in acute diabetic cranial nerve III palsy** due to vascular compromise of the central nerve fibers (the efferent pupillomotor fibers run in the periphery of the nerve) (**Figure 241-55**). Extraocular muscle testing will reveal an inhibition of ipsilateral medial gaze, upward gaze, and downward gaze as well as ptosis in an acute cranial nerve III palsy. Lateral gaze (abduction) will be preserved, and diplopia will be worse when the patient attempts to look toward the contralateral side due to the inability to adduct the eye (medial rectus dysfunction). In an acute cranial nerve VI palsy, lateral gaze will be diminished (abduction) on the ipsilateral side, and diplopia will be worse when the patient is trying to look to the affected side (lateral rectus dysfunction). Neuroimaging is needed in the ED to rule out an intracranial lesion.

If no other associated neurologic symptoms or findings are present, the blood sugar and blood pressure are under control, and neuroimaging does not suggest an alternative diagnosis, the patient can be discharged with ophthalmology and/or neurology follow-up.

POSTERIOR COMMUNICATING ARTERY ANEURYSM

Acute cranial nerve III palsy with ipsilateral pupillary dilatation is a posterior communicating artery aneurysm until proven otherwise. Concomitant headache is a frequent but not absolute finding. Expansion of an aneurysm of the posterior communicating artery frequently causes compression of the outer fibers of cranial nerve III. The pupillomotor fibers are located in the outer portion of cranial nerve III; therefore, the pupil becomes dilated on the affected side (**Figure 241-55**). Treatment is emergent blood pressure reduction if hypertensive, neuroimaging, and neurosurgical consultation.

HORNER'S SYNDROME

The physical findings of ipsilateral ptosis and miosis and anhydrosis are characteristic of Horner's syndrome (Figure 241-56). Interruption of the sympathetic nerve impulses controlling the Mueller muscle in the upper eyelid and the iris dilators causes these classic findings. Interruption can occur anywhere along the pathway from the brainstem to the sympathetic plexus surrounding the carotid artery (**Figure 241-57**). ED

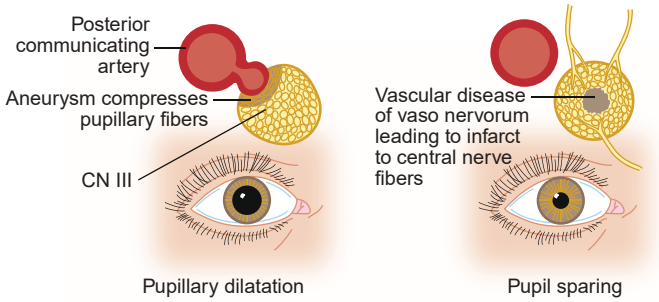


FIGURE 241-55. Posterior communicating artery aneurysm compresses the peripherally located pupillomotor fibers of cranial nerve (CN) III, causing a nerve palsy and pupillary dilatation. Diabetes and hypertension can cause microvascular compromise of the central nerve fibers, causing a nerve palsy with pupil sparing.

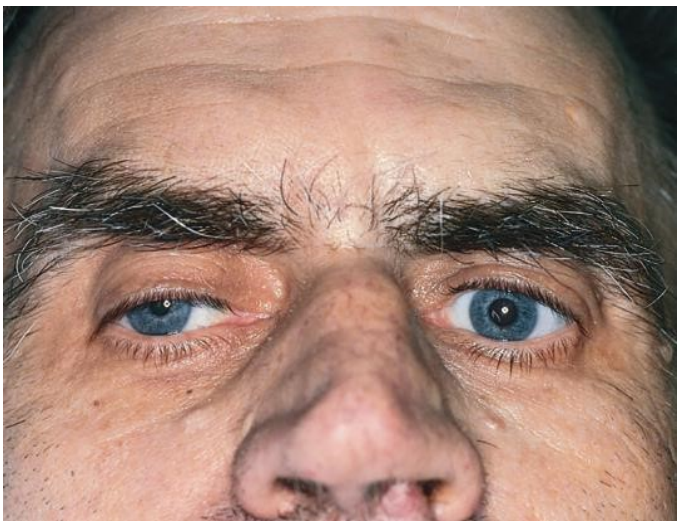


FIGURE 241-56. Horner's syndrome. Note the ptosis and miosis of the right eye. [Reproduced with permission from Knoop K, Stack L, Storrow A: *Atlas of Emergency Medicine,* 2nd ed. © 2002, McGraw-Hill, New York.]

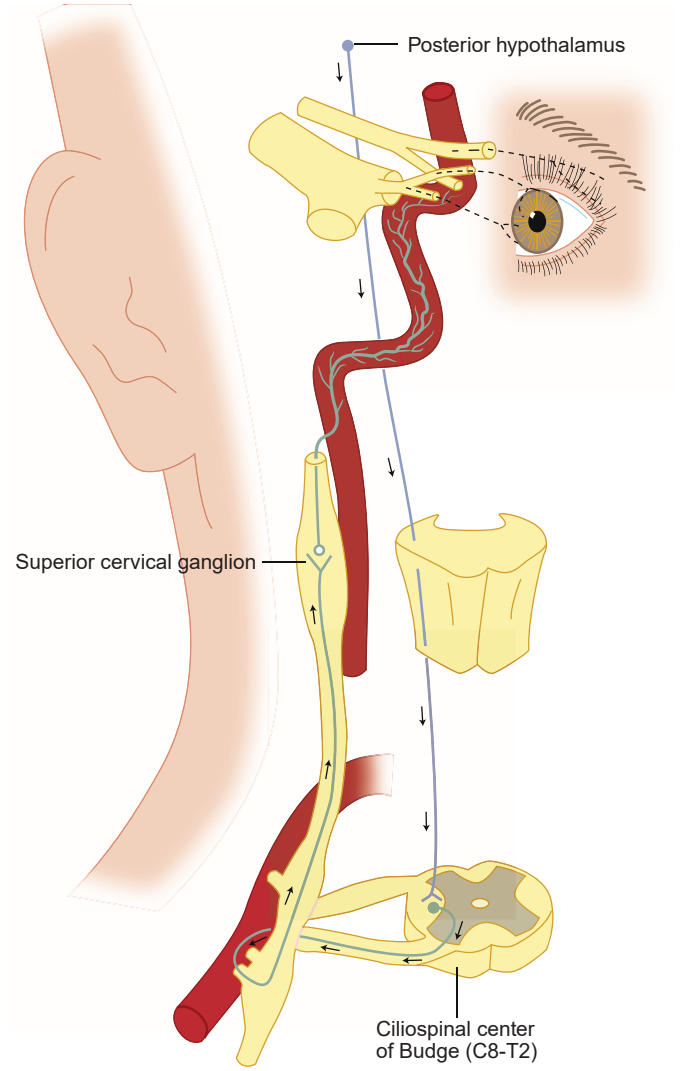


FIGURE 241-57. Sympathetic nerve pathway of the eye. An interruption anywhere along this pathway can cause Horner's syndrome.

evaluation includes a chest x-ray, CT scan of the brain and cervical region, and CT angiogram or magnetic resonance angiography of the head and neck vessels for carotid dissection. Institutional protocols determine if CT angiogram or magnetic resonance angiography is preferred.

In adults, causes of Horner's syndrome include cerebrovascular accidents, tumors, internal carotid artery dissection, herpes zoster, and trauma. In children, the causes include neuroblastoma, lymphoma, and metastasis. **Neck pain and acute Horner's syndrome suggest carotid dissection and can occur spontaneously (usually >30 years old) or as a result of blunt or penetrating neck injury.**

PSEUDOTUMOR CEREBRI (IDIOPATHIC INTRACRANIAL HYPERTENSION)

Increased intracranial pressure, papilledema, normal cerebrospinal fluid, and normal CT/MRI characterize pseudotumor cerebri. This condition can occur at any age. Patients complain of nausea, vomiting, headaches, and blurring of vision. **Patients can develop cranial nerve VI paresis, causing horizontal diplopia (double vision on lateral gaze).** A key component of examination is the identification of **visual field defects**. If CT/MRI is normal, perform lumbar puncture and record opening pressure; send cerebrospinal fluid for routine diagnostics. Consult neurosurgery for the treatment plan. Initial treatment is acetazolamide, 500 milligrams PO twice daily, and outpatient visual field monitoring.

OCULAR ULTRASONOGRAPHY

Direct visualization of intraocular structures can be limited by periorbital edema, corneal abrasions, hyphema, and cataracts. The superficial location of the eye and its cystic composition make US ideal in the assessment of a variety of ocular disorders. Ocular ultrasonography can expedite the diagnosis and management of several ocular emergencies, including retinal detachment, retrobulbar hematoma, globe perforation, lens dislocation, vitreous hemorrhage, and intraocular foreign body.[11-13] The indications for ocular ultrasonography include loss of vision/decreased vision, eye pain, eye trauma, suspected intraocular foreign body, and head injury. The ability of emergency physicians to accurately diagnose ocular pathology using bedside ocular ultrasonography has been well documented in emergency medicine literature.[11,12]

SCANNING TECHNIQUE AND NORMAL ANATOMY

Use a 7.5-to 10-mHz linear array transducer and set the unit to 'Ocular' for lower energy. Place the patient in a supine or partially upright position and have the patient keep the eyes closed (**Figure 241-58**). Apply a large amount of standard water-soluble US gel to the patient's closed eyelid so that the transducer does not touch the eyelid. US gel is not detrimental to the eye. Without applying any pressure, and asking the patient to look straight ahead with the eyes closed, scan the globe in sagittal and transverse planes. Stabilize the scanning hand over the bridge of the nose or on the forehead. Scan both eyes through closed eyelids for comparison. Examine the eyes in the neutral position and during gentle eye movements from side to side and up and down to thoroughly evaluate the orbit. Dynamic examination is crucial to identify adhesions, detachments, and membranes. The probe can also be moved side to side in both scanning planes to demonstrate the full extent of the structures in the eye. Adjust the depth so that the image fills the screen. The gain also needs to be adjusted multiple times during the examination to identify subtle abnormalities and avoid artifacts. Because the eye is a fluid-filled organ, it provides a perfect acoustic window for scanning and obtaining excellent images.[14-16]

The normal eye appears as a circular hypoechoic organ. The cornea is seen as a thin hyperechoic layer parallel to the eyelid attached to the sclera at the periphery. The iris is identified as a linear echogenic line extending from the periphery toward the lens on both sides. The anterior chamber contains anechoic fluid and is bordered by the cornea, iris, and anterior reflection of the lens. The normal lens has anterior and posterior boundary echoes with an anechoic center and is biconvex in

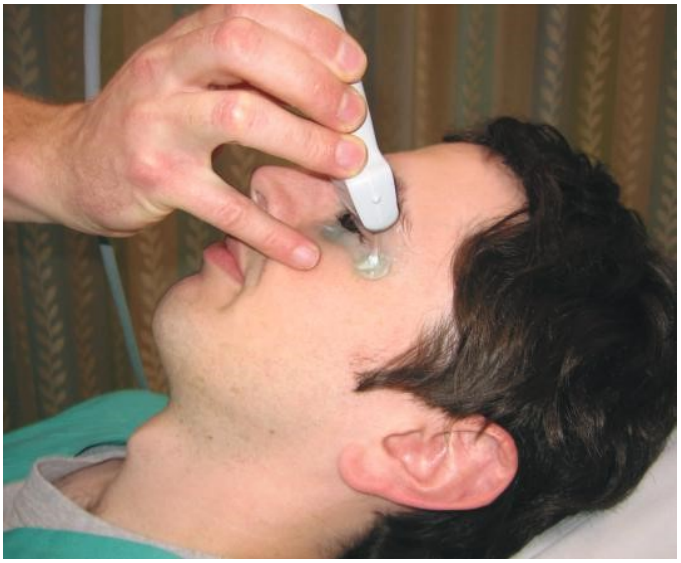


FIGURE 241-58. A high-resolution linear array US transducer is being applied to the closed eyelid. [Courtesy of Allen R. Katz, Department of Ophthalmology, University of Nebraska Medical Center.]

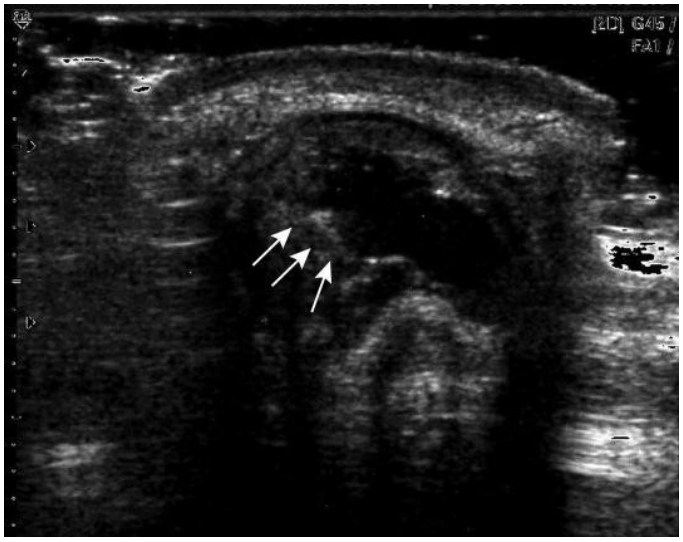


FIGURE 241-60. Extensive globe rupture from trauma. US shows abnormal, irregular shape of the eye with ocular contents displaced posteriorly (*arrows*). [Courtesy of M. Blaivas and Allen R. Katz, Department of Ophthalmology, University of Nebraska Medical Center.]

shape. The normal vitreous chamber is filled with anechoic fluid. Vitreous is relatively echo free in a young, healthy eye. Sonographically, the normal retina cannot be differentiated from the other posterior layers such as choroid and sclera (**Figure 241-59**). The evaluation of the retrobulbar space includes optic nerve, extraocular muscles, and bony orbit. The retro-orbital fat appears very echogenic. The optic nerve is visible as a hypoechoic linear region extending away from the globe posteriorly. Minor manipulations in the angulation of the probe are necessary to visualize the optic nerve. The central retinal artery and central retinal vein can be identified using Doppler.[14,15]

OCULAR TRAUMA

Assessment of patients with ocular trauma by US is of particular value when pain, soft tissue edema, and abnormalities like corneal edema, hyphema, or cataract make direct visualization of the posterior segment of the eye difficult. Ultrasound can detect a wide variety of ocular pathology from trauma including dislocated lens, globe disruption, vitreous hemorrhage, hyphema, retinal detachment, orbital emphysema, and retrobulbar hemorrhage. **Suspected ruptured globe is a relative contraindication to US examination, due to the risk of extruding globe contents associated with direct pressure on and around the eye.**[12]

US findings of globe rupture include distortion of the normal shape of the globe, decrease in the size of the globe, anterior chamber collapse, and vitreous hemorrhage.[11] Ocular trauma can lead to buckling of the sclera and scleral folds, which appear sonographically as irregularities in globe contour, highly reflective at their top and shadowing the orbital tissues (**Figure 241-60**). Sonographically, **lens dislocation** is seen as a highly reflective oval mass moving independently of the surrounding structures with eye movements. The dislocation is more readily visualized if it is complete or a cataract has formed. **Hyphema** can be identified on US as an echoic structure of variable echogenicity depending on the age of the bleed. Fresh hyphema will have low echogenicity and becomes more echogenic as it becomes organized. A **retrobulbar (pre- or postseptal) hematoma** is visualized as an echoluceny posterior to the globe.[17]

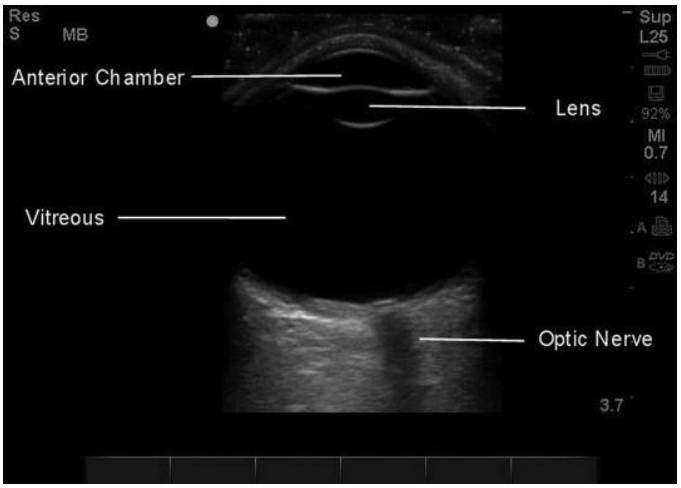


FIGURE 241-59. Normal eye in transverse view showing the anterior chamber, lens, vitreous, and optic nerve. [Courtesy of Allen R. Katz, Department of Ophthalmology, University of Nebraska Medical Center.]

INTRAOCULAR FOREIGN BODY

The presence of an intraocular foreign body may not always be apparent on clinical examination. US is a useful adjunct for detecting and localizing intraocular foreign bodies, but visualization of intraocular foreign bodies depends on their intrinsic echogenicity. Metallic objects are especially visible because of their bright echogenic acoustic profile and associated shadowing or reverberation artifacts in the echolucent vitreous, whereas materials such as wood are more difficult to detect (**Figure 241-61**). Sonographic patterns of shadowing and comet tail artifacts may help distinguish different foreign body materials. The dynamic nature of the US examination is also helpful in anatomic localization, determination of the size, and analysis of the composition of the foreign body.[18-20] With penetrating ocular injuries, a track of hemorrhage may be seen outlining the route of passage of a foreign body.

RETINAL DETACHMENT

Retinal detachment can be difficult to detect on physical examination, especially when the detachment is small. Bedside US reliably detects retinal detachment and is particularly useful when the examiner's view to the retina is obscured by periorbital edema, blood, or other opacities. For the detection of retinal detachment, ultrasonography performed by

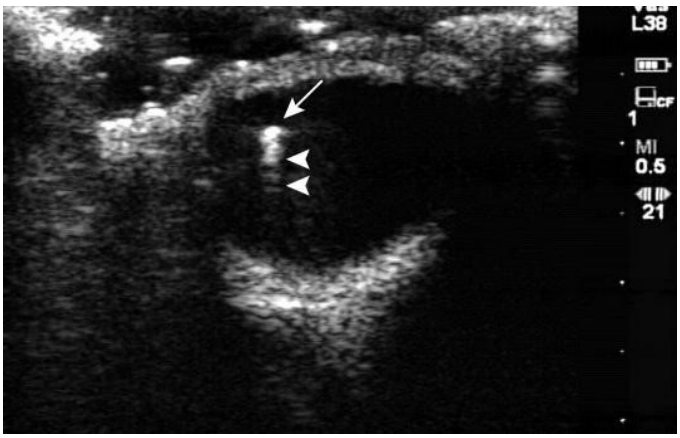


FIGURE 241-61. A hyperechoic foreign body (*arrow*) in the eye. Note the bright echogenic reverberation artifact (*arrowheads*). [Courtesy of M. Blaivas and Allen R. Katz, Department of Ophthalmology, University of Nebraska Medical Center.]

an emergency physician has a sensitivity of 97% to 100% and a specificity of 83% to 92%.[21,22]

A retinal detachment is seen as an echogenic undulating membrane in the posterior to lateral globe, protruding into vitreous (**Figure 241-62**). Even in complete retinal detachments, the typically folded surface remains bound to the ora serrata anteriorly and the optic nerve head posteriorly. Unlike choroidal detachment that doesn't change with ocular movements, retinal detachment moves with eye movements.[16] A shallow cuff of subretinal fluid may also be seen along with the detachment. On occasion, retinal detachments are also accompanied by vitreous hemorrhages.

Vitreous Hemorrhage Vitreous hemorrhage can be spontaneous or associated with trauma. Vitreous hemorrhage can interfere with vision and, if it is large, can potentially cause blindness. It appears as echogenic material in the posterior chamber. The sonographic appearance of vitreous hemorrhage depends on its age and the severity of the bleed. Increasing gain is helpful for detecting acute hemorrhages because they are often minimally echogenic. Early mild hemorrhages are seen as small dots or scattered low-amplitude reflective mobile opacities in the vitreous. As the hemorrhage matures and organizes, thick mobile membranes are formed in the vitreous. Sonographically, this is seen as vitreous filled with multiple large echoes (**Figure 241-63**). Due to gravitational forces, these opacities may also layer inferiorly. Echogenic

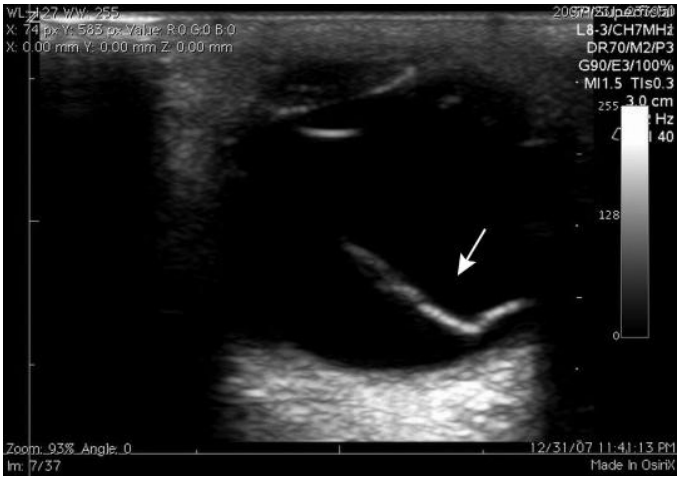


FIGURE 241-62. Retinal detachment is seen as a hyperechoic membrane in the posterior aspect of the globe (*arrow*). [Courtesy of D. Chandwani and Allen R. Katz, Department of Ophthalmology, University of Nebraska Medical Center.]

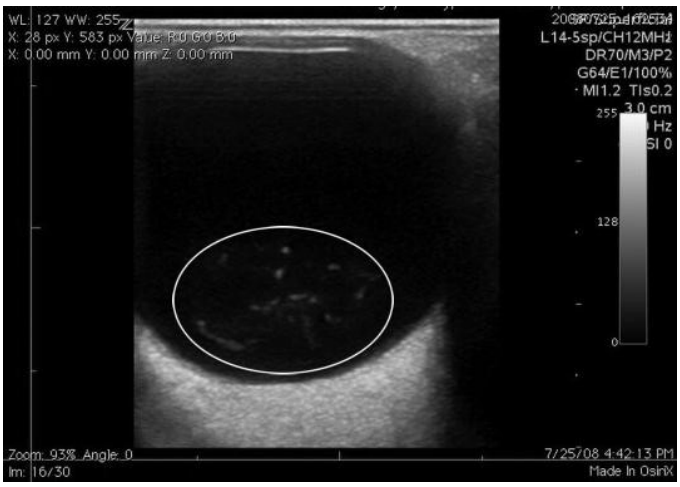


FIGURE 241-63. Bright echoes (enclosed in oval) in the posterior chamber demonstrating vitreous hemorrhage. [Courtesy of D. Chandwani and Allen R. Katz, Department of Ophthalmology, University of Nebraska Medical Center.]

stranding and scarring of the vitreous occurs as time progresses.[16,23,24] As vitreous hemorrhage becomes more echogenic, it can mimic the appearance of a retinal detachment. The main characteristics that can help in differentiating vitreous hemorrhage from retinal detachment are as follows: (1) Retinal detachment moves with eye movements unlike vitreous hemorrhage, which will remain horizontal; and (2) vitreous hemorrhages are frequently seen in the middle portion of the posterior chamber and retinal detachments almost always occur at the posterior-most portion of the eye, adjacent to the optic disk and macula.[21]

Elevated Intracranial Pressure—Optic Nerve Sheath Measurement Another novel use of bedside ocular US is the evaluation of the optic nerve sheath diameter to assess possible elevated intracranial pressure. Multiple studies have shown good correlation between intracranial pressure and sonographic optic nerve sheath diameter.[25-32] On US, a normal optic nerve sheath measures up to 5.0 mm in diameter in adults, 4.5 mm in children, and 4.0 mm in infants.[33] The measurement is obtained 3 mm posterior to the globe for both eyes. A position of 3 mm behind the globe is selected because the US contrast is greatest at this point, and the measurements are more reproducible (**Figure 241-64**). Typically, three measurements are

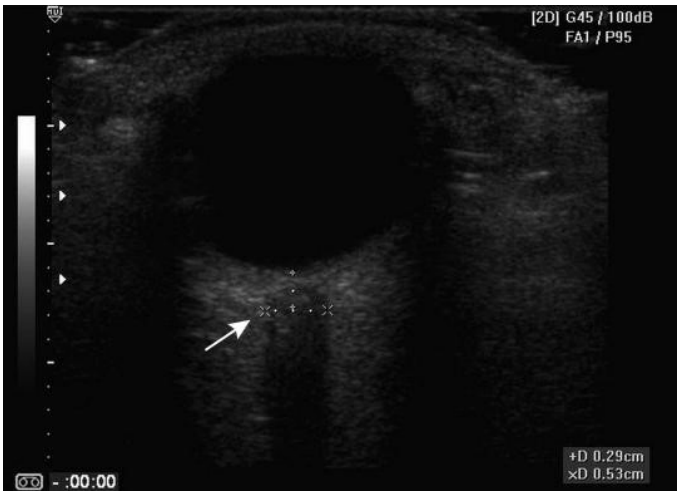


FIGURE 241-64. An optic nerve sheath measuring 5.3 mm in a patient with head injury is shown. One set of calipers measures 3 mm behind the globe, and the second measures the optic nerve sheath diameter (*arrow*). [Courtesy of M. Blaivas and Allen R. Katz, Department of Ophthalmology, University of Nebraska Medical Center.]

averaged. Current literature suggests that the cut-off value that provides the best accuracy for the prediction of intracranial pressure >20 mm Hg is 5.7 to 6.0 mm, and increased intracranial pressure should be suspected with values above this threshold. The sensitivity and specificity in detecting increased intracranial pressure using a cut-off value of 5.7 to 6.0 mm are in the range of 87% to 95% and 79% to 100%, respectively.[34]

CAUTIONS WITH OCULAR US

- **If globe rupture is suspected, avoid any manipulation or pressure upon the globe or eyelid.**
- Limit the duration of ocular US examination, especially when using spectral and color Doppler, and **set the US unit for 'ocular imaging'**. The recommended exposure limits are half that of fetal imaging.
- Various artifacts may interfere with ocular ultrasound examination. Orbital emphysema can make it difficult to visualize contents of the orbit.
- Air bubbles within the vitreous, which may appear in the setting of trauma to the globe, may resemble an intraocular foreign body.

Acknowledgment: The authors gratefully acknowledge the contributions of John D. Mitchell, the author of this chapter in the previous edition.

REFERENCES

The complete reference list is available online at www.TintinalliEM.com.

CHAPTER 242

Ear Disorders

Kathleen Hosmer

This chapter discusses common nontraumatic conditions affecting the external, middle, and inner ear. Selected traumatic conditions include auricular hematoma, burns, and frostbite. Lacerations to the ear are discussed in the chapter 40, "Face and Scalp Lacerations." Ear disorders in children are discussed in chapter 115, "Ear and Mastoid Disorders in Infants and Children."

ANATOMY

EXTERNAL EAR

The auricle, or pinna, is the visible external portion of the ear, whose trumpet shape enables it to collect air vibrations. It consists of a thin plate of elastic cartilage with a tightly adherent covering of skin. The external auditory canal is an S-shaped skin-lined tube that extends from the auricle to the tympanic membrane (TM). The outer one third of the external auditory canal is composed of an incomplete cartilaginous tube. Its thick skin supports hair follicles plus apocrine and sebaceous glands. The inner two thirds of the canal is composed of bone covered by a thin layer of tightly adherent skin, which is easily torn by minimal trauma.

The blood supply to the external ear is derived from the posterior auricular, superficial temporal, and deep auricular arteries. Venous drainage of the external ear is into the superficial temporal and posterior auricular veins, which then drain into the external jugular vein. The posterior auricular vein frequently connects to the sigmoid sinus, providing a route for extension of infected material into the intracranial cavity.

MIDDLE EAR

The middle ear is an air-containing cavity in the petrous temporal bone. It contains the auditory ossicles, which transmit vibrations of the TM to the perilymph of the internal ear. It communicates with the nasopharynx anteriorly via the eustachian tube and with the mastoid air spaces posteriorly via the aditus ad antrum (**Figure 242-1**).

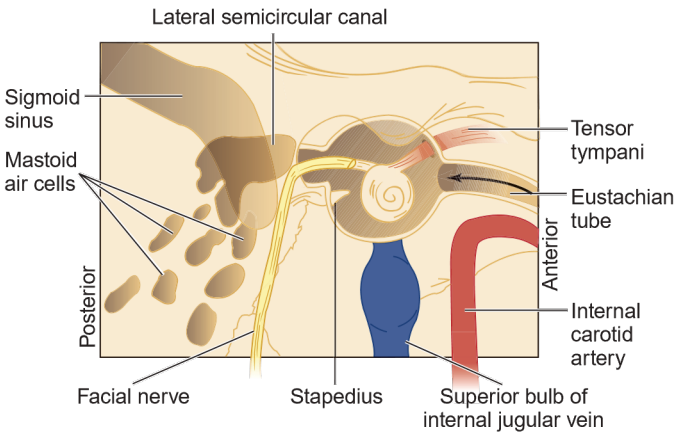


FIGURE 242-1. Sagittal section of the middle ear and related structures.

The TM is a thin, pearly gray, fibrous membrane that produces a cone-shaped light reflex anteroinferiorly when illuminated. Superiorly, the pars flaccida is the relatively slack portion of the membrane between the malleolar folds; the remainder of the membrane is tense and is called the pars tensa. The auditory ossicles are the malleus, incus, and stapes. Both the incus and the handle and lateral processes of the malleus are typically visible through the TM (**Figure 242-2**). Figure 242-1 shows the relationships of the facial nerve, sigmoid sinus, and internal carotid artery to the middle ear.

INNER EAR

The inner ear consists of the cochlea, which contains the auditory sensory receptors, and the vestibular labyrinth, which contains balance receptors. Cristae in the semicircular canals detect angular acceleration, and macules detect linear acceleration. Afferent nerves from the vestibular labyrinth connect to brainstem nuclei to maintain smooth movement of the eyes during head movement and to the cerebellum to control oculomotor and postural functions. Blood supply is from the vertebrobasilar system (**Figure 242-3**). The otolithic organs (utricle and saccule) lie in the vestibule. The internal auditory artery divides into the common cochlear artery and the anterior vestibular artery. The anterior vestibular artery provides the blood supply to the anterior and horizontal semicircular canals but not to the cochlea. Isolated occlusion of the anterior vestibular artery may therefore cause acute vestibular syndrome without hearing loss.

OTALGIA

Primary otalgia is caused by auricular and periauricular disease, whereas referred otalgia is caused by disease originating from remote structures.[1] Referred otalgia is common because the ear and several structures of the

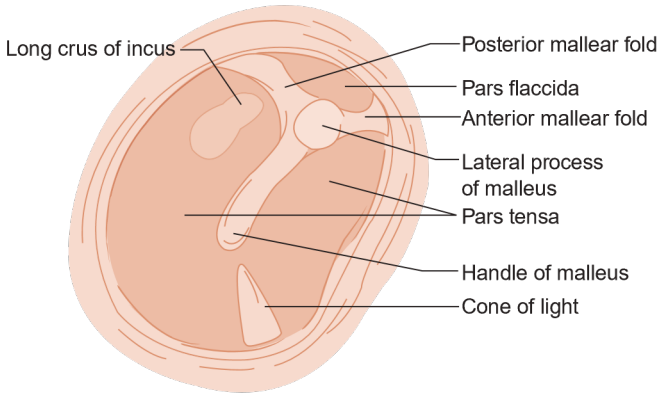


FIGURE 242-2. Right tympanic membrane as seen through the otoscope.