CHAPTER 61

Ophthalmology

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The emergent conditions that affect the eye, its surrounding tissues, and the act of seeing itself are myriad and broad in scope and include trauma, inflammatory conditions, infections, hydrostatic issues (such as, glaucoma), vascular events, structural issues, optical derangements, and neurological developments (such as, visual field cuts, anisocoria, nystagmus, diplopia). The evaluation of a chief complaint of "eye problem" may take the clinician in any of a variety of directions. Figure 61.1 represents a general orientation to non-neurological and nontraumatic ophthalmological emergencies, insofar as they incorporate four key symptoms of pain, redness, disordered vision, and swelling.

TRAUMATIC CONDITIONS

Principles

The evaluation of a traumatic injury to the eye should be anatomically methodical, considered from superficial (eyelids, conjunctival, corneal, and anterior segment) structures to deep (including ocular, retrobulbar, and periorbital) structures, keeping in mind that deep injuries may be present with minimal superficial manifestations. Many extraocular structures are found in close proximity, and concomitant, non-ocular injury is common (traumatic facial injury is discussed in more detail in Chapter 35). A detailed examination for additional injury is therefore important.

Periorbital Contusions and Eyelid Lacerations

Clinical Features and Differential Diagnosis

Periorbital contusions and eyelid lacerations present very evidently, but it is important to determine whether additional ocular injury such as a globe perforation, an orbital septal injury (suggested by prolapsed fat), a canalicular laceration (suggested by a laterally displaced puncta) (Fig. 61.2), a levator or canthal tendon laceration, or an intraorbital foreign body is present, because all require consultation in the emergency department (ED) with an ophthalmic surgeon. An eyelid or periorbital injury should not be a distraction from less visible underlying injuries to the eye itself (see sections on deeper injuries later for clinical features).

Diagnostic Testing

The emergency clinician should undertake best attempts to thoroughly examine all ocular structures, even those hidden by swollen eyelids and periorbital tissue, and evaluate visual function. With delay, swelling can increase and limit visualization. Early examination, gentle pressure to displace fluid, or use of ice can improve ability to open the eyelids, whereupon an assessment for deeper injuries can be made. Use of eyelid retractors, such as the Desmarres, can help avoid increasing intraocular pressure (IOP). However, if there is concern for a ruptured or perforated globe (with or without a foreign body present) and the globe cannot be safely and properly examined, computed tomography (CT) imaging, with or without consultation with an ophthalmologist, is advised (see Intraocular Foreign Bodies and Globe Rupture later).

Management and Disposition

Isolated soft tissue injury to the eyelids and the surrounding area is treated with symptomatic management such as head elevation and cool compresses, as most will resolve. Patients should be instructed to seek follow-up care for any increase in pain or swelling, decreased vision, double vision, or significant flashing lights or floaters as these may be indications of retinal injury (see Posterior Segment/Ocular Injuries: Commotio Retinae, Retinal Detachment, Intraocular Foreign Body, and Perforated Globe later). If there are no additional complications, one can manage simple lid lacerations that are parallel to relaxed skin tension lines (exceptions for damage to important structures noted earlier) with primary closure with 6-0 or 7-0 nylon or Prolene interrupted sutures, removed within a week.

Conjunctival and Scleral Injuries: Subconjunctival Hemorrhage, Conjunctival Laceration, and Scleral Laceration

Clinical Features and Differential Diagnosis

Injuries to the conjunctiva include a subconjunctival hemorrhage (very common with blunt or penetrating injury) and conjunctival laceration. A subconjunctival hemorrhage develops when subconjunctival blood vessels bleed, either spontaneously or after a sudden acute venous congestion of the head, such as from a Valsalva maneuver or vigorous coughing. The hemorrhage smoothly and minimally raises the overlying conjunctiva, with no vessels visible behind the blood, and is often incidentally discovered by the patient upon looking in the mirror (Fig. 61.3). Symptoms, if any are present, may include a very mild, diffuse foreign body sensation (from the size and location of the hemorrhage), with no change in visual acuity.[1] Subconjunctival hemorrhages from minor instigations (such as, coughing) are typically self-limited, but those from more direct trauma may be complicated by underlying injury. A 360-degree area of involvement associated with chemosis or pain, decreased visual acuity, or sensitivity to light, should prompt an evaluation for globe perforation. A conjunctival laceration will present with significantly more discomfort than a subconjunctival hemorrhage, and if present, should prompt an evaluation for globe perforation—and retained foreign body if indicated. A globe perforation from a related foreign body may present in an occult fashion, with only a mild-appearing conjunctival laceration or scleral "bruise," and should be screened for if the mechanism of injury (such as, in an injury from a compressed air tool or gun, a nail or object deflected by a hammer, or a significant impact directly on the eye) suggests energy sufficient for globe penetration. A patient may also present with a scleral laceration, a laceration of the thick white envelope that provides the structural integrity of the globe, which should also prompt consideration of globe perforation.

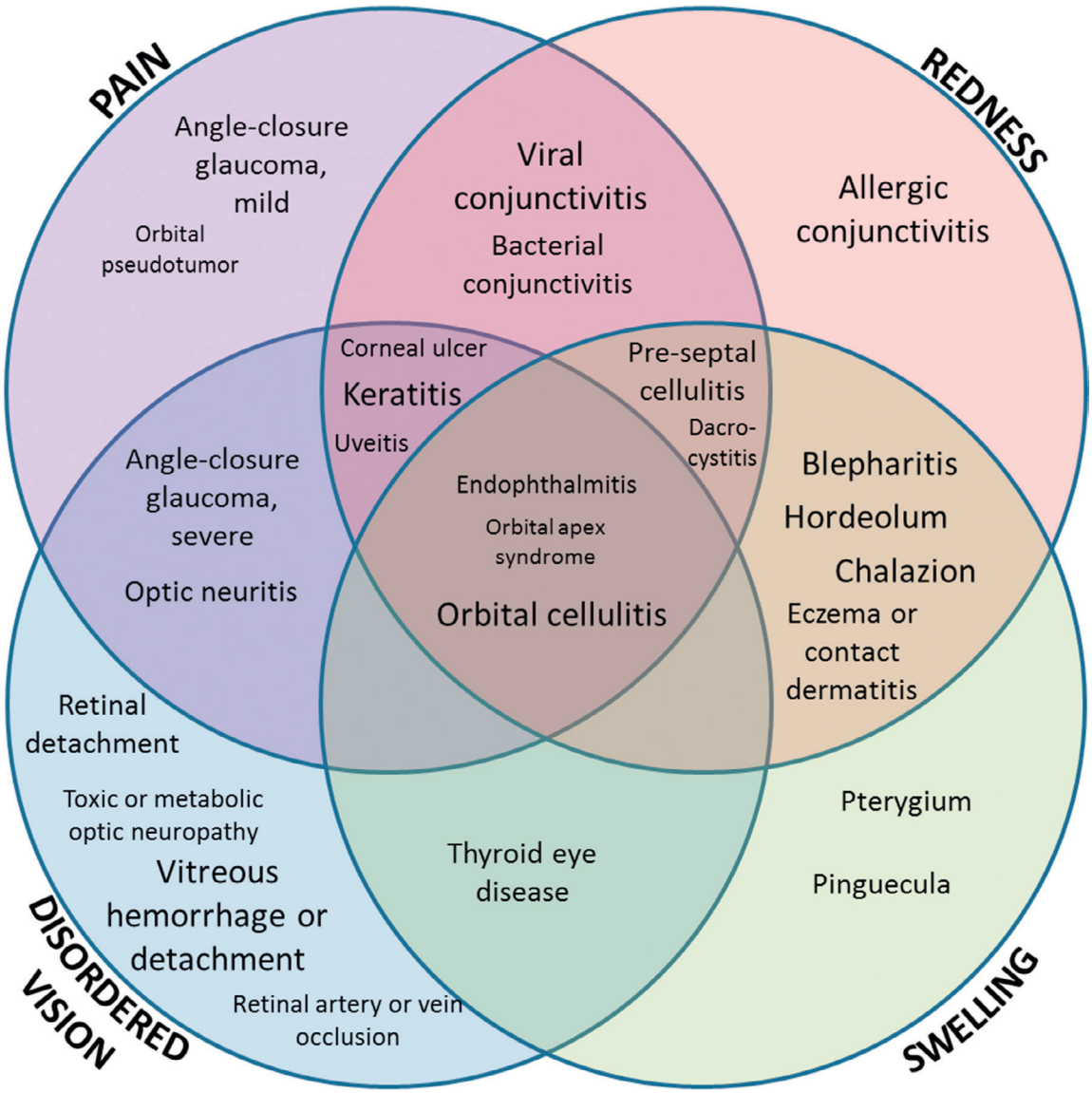


Fig. 61.1. An overview of nontraumatic, non-neurological ophthalmological conditions, arranged by primary presentation in the emergency department (ED). Relative text size is a general representation of the incidence or relatively likelihood of that entity in relation to others. (Note: The actual incidence or likelihood of the entity in any one ED will vary with the local disease patterns and patient population being treated in the department.)

Fig. 61.2. Canalicular laceration. **A,** This patient experienced a laceration of the upper canaliculus. **B,** Canalicular laceration extending from forehead and brow. (**A,** From Zitelli BJ, Davis HW, editors: Atlas of pediatric physical diagnosis, ed 5, St Louis, 2002, Mosby. **B,** Courtesy Jeffrey Lee, MD, University of California San Diego.)

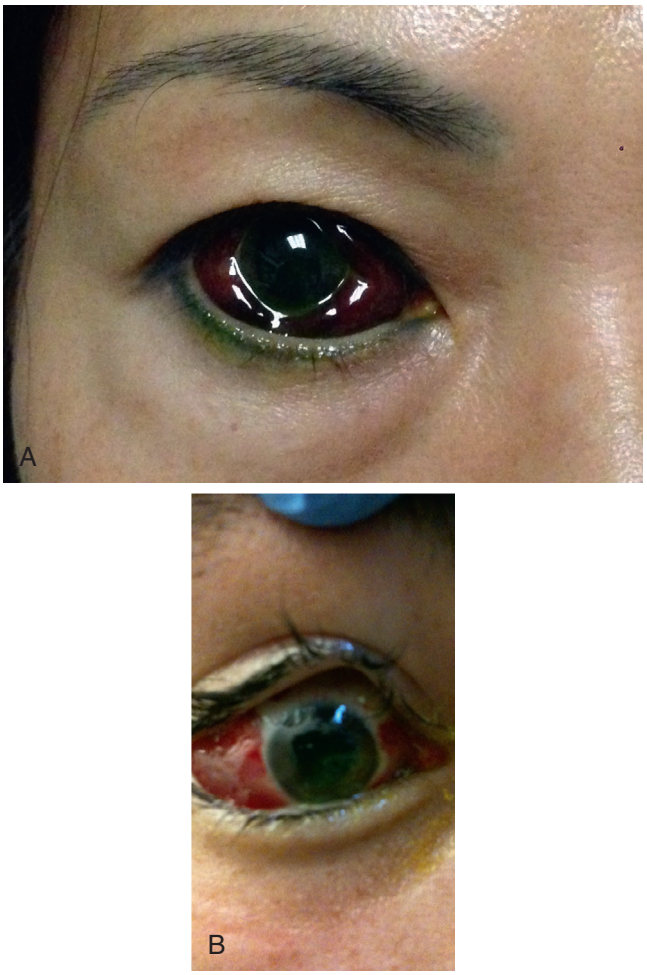


Fig. 61.3. **A** and **B,** Subconjunctival hemorrhage. (Courtesy Jeffrey Lee, MD, University of Calfornia San Diego.)

Diagnostic Testing

An important aspect of the examination for subconjunctival hemorrhage, conjunctival laceration, or scleral laceration (or any injury to the eye for that matter) is examination of the conjunctiva, sclera, cornea, and underlying anterior chamber with a slit lamp, and application of fluorescein dye. This is especially helpful in three ways: (1) identification of corneal abrasions, (2) screening for evidence of a globe perforation, and (3) identification of tarsal foreign bodies. In using fluorescein, the conjunctival and corneal surface is painted with a 10% fluorescein strip (with anesthetic or saline solution). Fluorescein is not taken up by intact corneal epithelium but will stain areas of injured or lost epithelium bright green. If the fluorescein staining has a pattern of repeated lines (Fig. 61.4), this suggests the possibility of a retained tarsal foreign body hidden under the upper or lower lid. If the fluorescein dye on the conjunctival surface is focally displaced by leakage of nonfluorescent aqueous fluid (called a *positive Seidel's test*), or if there is brownish black uveal tissue visible in the scleral wound (Fig. 61.5), a globe perforation is present (see Intraocular Foreign Bodies and Globe Rupture). If no globe perforation is suspected, the eyelids should be everted and the conjunctival fornices inspected for a hidden foreign body.

Management and Disposition

Subconjunctival hemorrhages typically resolve spontaneously without treatment. Cool compresses are often recommended, but there is no evidence that they hasten recovery or improve outcome.

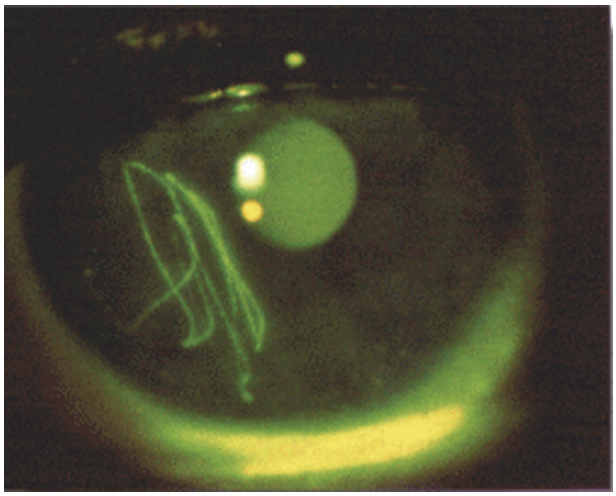


Fig. 61.4. Corneal abrasion demonstrated by slit-lamp examination. (Courtesy www.perret-optic.ch/optometrie/symptomes_diagnostiques/symptomes/opto_symfor_gb.htm#abrasion.)

Fig. 61.5. Scleral laceration with penetrating globe injury. Note care being taken not to increase intraocular pressure (IOP) with examiner's fingers.

There is no evidence to support the use of artificial tears or lubricants. Patients with simple subconjunctival hemorrhage are advised regarding the gradual resolution of their hemorrhage over 10 to 14 days, with the typical colorations associated with resolving hemorrhage. No follow-up is required unless the patient develops recurrent hemorrhages or new symptoms. Although most conjunctival lacerations do not require repair, lacerations that are more than 1 cm should be evaluated by an ophthalmologist in the ED for possible repair. Although there is no evidence to support the practice, topical antibiotic prophylaxis for 3 to 5 days (usually in ointment form; Table 61.1) is common practice. A deeper scleral laceration requires emergent consultation with an ophthalmologist, and may predispose to endophthalmitis. A full-thickness scleral laceration should be treated as a globe perforation.

Corneal Injuries: Corneal Abrasions, Foreign Bodies, and Lacerations

Clinical Features and Differential Diagnosis

Traumatic corneal injuries come in three varieties: (1) abrasions, (2) foreign bodies, and (3) lacerations (which may also go into

TABLE 61.1

Useful Topical Ophthalmic Medications and Their Dosages

MEDICATION	DOSAGE
TOPICAL ANTIBIOTICS	
Erythromycin 0.5% ointment	½-inch ribbon, four times per day for 7 days
Polymyxin B/trimethoprim solution	1 drop, four times per day for 7 days
Sulfacetamide 10% solution	1 to 2 drops, four times per day for 7 days
Azithromycin ophthalmic 1% solution	1 drop twice a day for 2 days, then daily for 5 days
ANTI-PSEUDOMONAL TOPICAL ANTIBIOTICS	
Ciprofloxacin 0.3% ointment	½-inch ribbon, four times per day for 7 days
Ciprofloxacin 0.3% solution	1 to 2 drops, four times per day for 7 days
Moxifloxacin 0.5% solution	1 to 2 drops, three times per day for 7 days
Gentamicin 0.3% ointment	½-inch ribbon, two to three times per day for 7 days
Gentamicin 0.3% solution	1 to 2 drops, four times per day for 7 days
Ofloxacin 0.3% solution	1 to 2 drops, four times per day for 7 days
TOPICAL CYCLOPLEGICS	
Cyclopentolate 1%	1 drop (may repeat in 5 minutes if needed), three times a day, for up to 4 days
Homatropine 5%	1 drop, four times per day, for up to 4 days
TOPICAL ANESTHETICS[a]	
Tetracaine hydrochloride 0.5% or 1%	1 drop, every 30 minutes as needed, for 24 hours only
Proparacaine 0.05% (10 times dilution[b])	2 to 4 drops, every 30 minutes as needed, for 48 hours only
TOPICAL NONSTEROIDAL ANTIINFLAMMATORY DRUGS	
Diclofenac 0.1%	1 drop, four times per day for 2 to 3 days
Ketorolac 0.4%	1 drop, four times per day for 2 to 3 days
TOPICAL ANTIHISTAMINES (ALLERGY)	
Azelastine 0.05%	1 drop twice a day
Emedastine 0.05%	1 drop up to four times per day
TOPICAL STEROIDS	
Prednisolone acetate 1%	2 drops every 15 to 30 minutes four times, then four times per day for 2 to 3 days

[a]For corneal abrasions—use only in reliable patients who have been educated on use.
[b]Can use undiluted 0.5% for diagnostic purposes in emergency department (ED).
Azari AA, Barney NP: Conjunctivitis: a systematic review of diagnosis and treatment. JAMA 310(16):1721-1729, 2013; Wipperman JL, Dorsch JN: Evaluation and management of corneal abrasions. Am Fam Physician 87(2):114–120, 2013.

the sclera). A corneal abrasion is typically sustained when an object or part of an object is dragged across the eye, such as seen with a fingernail injury to the eye or an attempt to pull off an adherent contact lens. A corneal foreign body is typically sustained when a small dense object travelling at high velocity impacts the cornea and becomes embedded, such as seen with debris impact from a grinding tool. A corneal laceration is typically sustained from a direct lancinating injury to the surface of the eye, such as by an infant's fingernail sweeping by the eye of its mother. All of these injuries present with more intensity than a conjunctival or isolated scleral injury, with significant foreign body sensation, pain, tearing, and a decrease in visual acuity if the abrasion, object, or laceration infringes upon the visual axis. A foreign body may actually be conjunctival and not imbedded in the cornea, presenting with more diffuse irritation, or sensation reported by the patient as "something under the eyelid," but may cause a corneal abrasion and more intense, localized foreign body symptoms upon subsequently being moved across the surface of the eye by blinking or rubbing the eye. As with a conjunctival and scleral injury, a perforated globe should be considered in the differential diagnosis of a corneal injury, suggested by symptoms and signs (such as, chemosis, deep eye pain, decreased visual acuity, and/or photophobia) and injury mechanism.

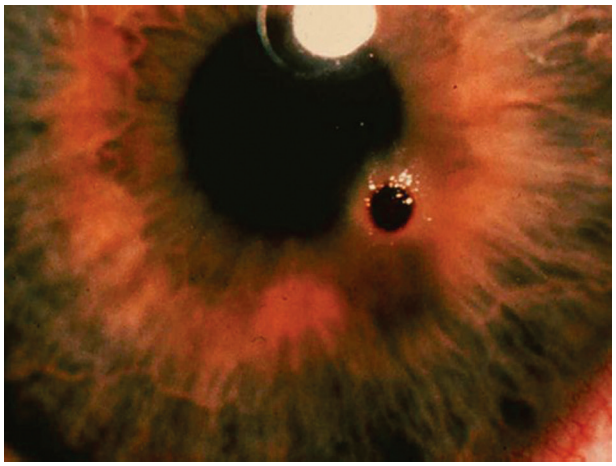


Fig. 61.6. Corneal foreign body seen on slit-lamp examination. (Courtesy www.tedmontgomery.com.)

Diagnostic Testing

With corneal abrasions and foreign bodies, topical anesthesia (see Table 61.1) can not only facilitate patient cooperation, but it helps localize the extent of injury; focal pain that is completely abolished by topical anesthesia, with no signs of deeper injury present, suggests an injury confined to the superficial layers of the cornea. This does not obviate the need for a detailed examination with a slit lamp and fluorescein staining, with diagnostic goals as outlined with conjunctival injuries earlier. A corneal foreign body should be readily evident on slit-lamp evaluation (Fig. 61.6). If the foreign body spans the full-thickness of the cornea, it is considered a globe perforation until proven otherwise and should trigger emergent consultation with an ophthalmologist.

A corneal laceration may be difficult to characterize (or may even be hidden) on slit-lamp examination, and the primary diagnostic goal is identifying it, and determining whether or not it is through-and-through (ie, representing an open globe). Signs suggesting the latter are loss of anterior chamber depth, prolapsed iris (Fig. 61.7A), an irregular or teardrop-shaped pupil (see Fig. 61.7B), blood in the anterior chamber (Fig. 61.8), and a 360-degree subconjunctival hemorrhage. Testing for a positive Seidel's test with fluorescein, as described for conjunctival injuries earlier, can be used to confirm (but not exclude) an open globe. A critical point is that corneal perforation is a form of open globe, and once this is found, the examination ends (so as to prevent the additional extrusion of globe contents, worsening visual outcome) until the

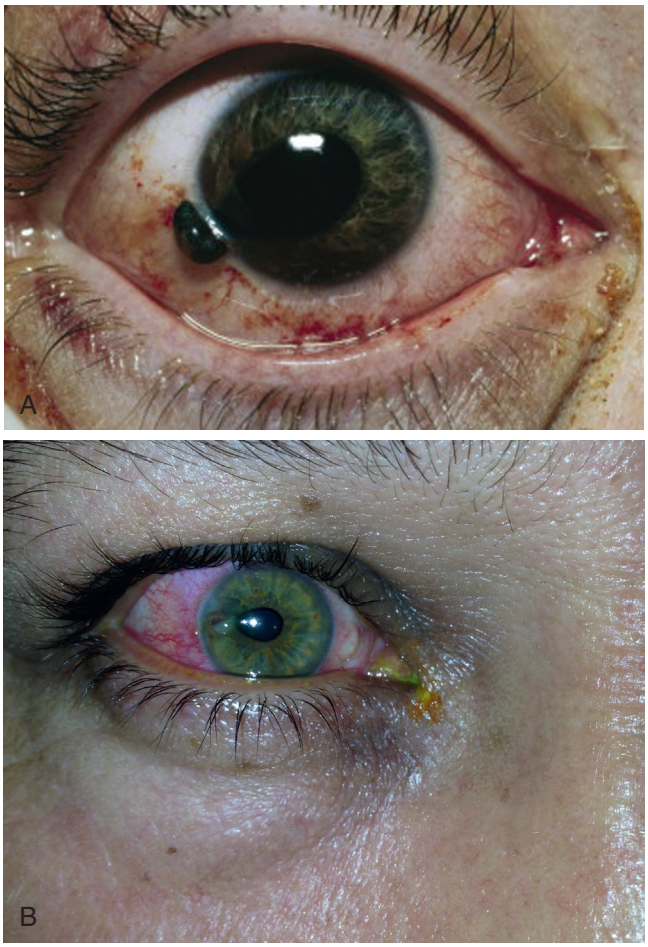


Fig. 61.7. Corneal laceration with prolapse of the iris. **A,** The pupil is irregular and teardrop shaped, pointing toward the laceration. **B,** Corneal laceration with tear drop teardrop pupil, pointing toward the laceration. (**A,** From Roberts JR, Hedges JR, editors: Clinical procedures in emergency medicine, ed 5, Philadelphia, 2010, Saunders. **B,** Courtesy Jeffrey Lee MD, University of California San Diego.)

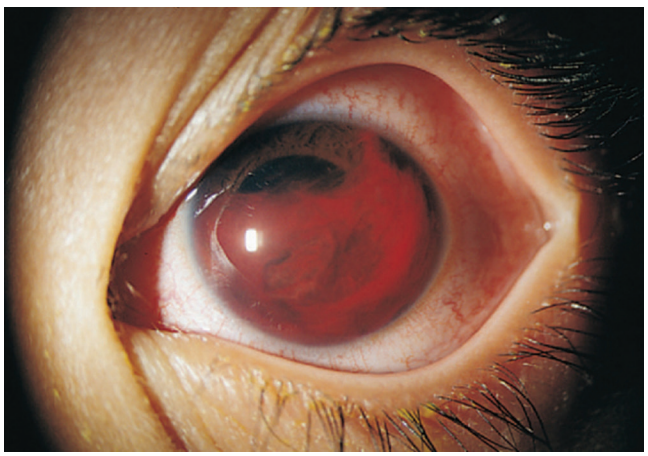


Fig. 61.8. Hyphema in the anterior chamber. (Courtesy Steve Chalfin, MD, University of Texas Health Science Center, San Antonio, TX.)

patient can be further examined under controlled conditions by an ophthalmologist (see Intraocular Foreign Bodies and Globe Rupture later).

If the mechanism of injury involves a high-velocity projectile and no foreign body is seen on slit-lamp examination but is suspected, then an evaluation for an intraocular foreign body as outlined in Intraocular Foreign Bodies and Globe Rupture later should be initiated.

Management and Disposition

Corneal Abrasions. There is no evidence that treatment of corneal abrasions with topical antibiotics, as often recommended, has any beneficial effect. Furthermore, the infection rate of untreated corneal abrasions is low—at 0.7%—and prophylactic antibiotic use is not warranted. We recommend that antibiotics not be used for uncomplicated corneal abrasions (that are not deep, not imparted by a heavily contaminated object, and not in high-risk patients), especially because the indiscriminate prescribing of these agents introduces the risk of toxic and allergic medication reactions in the eye. It is reasonable, however, to empirically treat a corneal abrasion in a contact lens wearer or immunocompromised patient with an anti-pseudomonal agent such as tobramycin, ciprofloxacin, or ofloxacin, as outlined in Table 61.1 (although for a shorter course than would be prescribed for an already established infection; ie, 3 to 5 days).[2]

A major goal in the treatment of a corneal abrasion is managing the pain. Topical nonsteroidal antiinflammatory drugs (NSAIDs), such as ketorolac or diclofenac (see Table 61.1) are options.[3] Prospective trials have revealed that topical anesthetics self-administered as needed for a short duration of time by ED patients for corneal abrasion pain results in significant pain relief without complications.[4,5] Meta-analyses incorporating these trials as well as postoperative ophthalmology literature support this finding.[6,7] A patient with a corneal abrasion can therefore be provided a limited 24- to 48-hour course of a topical anesthetic (see Table 61.1), because the most intense pain occurs in the first 24 hours. This being said, uninformed patients using over-the-counter topical anesthetics for otherwise simple corneal injuries can develop an erosive keratopathy.[8] Regardless of whether this association is due to masking the pain of an emerging infection or due to a direct toxic effect from a misused anesthetic, it is important to counsel patients on the correct and limited use of these agents. Eye patches are not recommended, because they can mask a worsening infection. Urgent ophthalmologic consultation is warranted with signs of active infection, such as a corneal infiltrate (whitening of the cornea) or ulceration (see Corneal Ulcers and Infiltrates later); otherwise, the patient can follow up with an ophthalmologist in 24 to 48 hours.

Corneal Foreign Bodies. Foreign bodies of the cornea and conjunctiva—especially those containing iron (given the propensity for worsening rust deposition over time)—should be removed in the ED if possible, using a topical anesthetic (see Table 61.1) for comfort. Corneal foreign bodies can be removed by moist cotton-tipped applicator or, if needed, by using a 25-gauge or 27-gauge needle carefully applied in a plane tangential to the surface of the cornea, under slit-lamp visualization. Non-corneal conjunctival foreign bodies can be removed by irrigation, a wet cotton-tipped applicator, or fine forceps if needed. For metallic foreign bodies, residual rust rings can be removed 24 to 48 hours later, because the rust will migrate toward the corneal surface and be more accessible. Ophthalmologic consultation is recommended for deep and large corneal foreign bodies or if the central area of the visual axis is involved. Considerations for topical antibiotic prophylaxis are the same as for corneal abrasions (see earlier).

Corneal Lacerations. Large but partial corneal lacerations should be evaluated by ophthalmology for potential closure in the operating room versus observation with medical therapy (topical cycloplegics and antibiotics; see Table 61.1), pressure patch, and possible tissue grafts. Smaller corneal lacerations can be evaluated and treated as corneal abrasions (with the only modification being

they should be empirically treated with topical antibiotic prophylaxis given their depth). A complication of a corneal laceration or abrasion is an infected corneal ulcer (see Corneal Ulcers and Infiltrates), which may develop days to weeks later.

Anterior Segment Injuries: Traumatic Hyphema, Iritis, Cyclodialysis, and Lens Dislocation

Clinical Features and Differential Diagnosis

Any blunt or penetrating injury of the eye may result in injury to the underlying anterior chamber, the iris, lens, and associated anterior segment structures of the eye. A traumatic hyphema (blood in the anterior chamber from ruptured vessels in the ciliary body or iris) can present with a spectrum of severity, from microhyphema (where red blood cells only visible by slit-lamp examination are suspended in the anterior chamber aqueous), to a layered hyphema (where a layer of blood may be observed grossly in the lower anterior chamber), or to a full or total hyphema (in which the entire anterior chamber is filled with blood). A microhyphema may not be visible to the naked eye but will be associated with suggestive clinical features that include peri-limbal conjunctival injection (ciliary flush), abnormal pupil size (larger, smaller, or irregular), and dilated pupils (traumatic mydriasis) (Fig. 61.9). Symptomatically, patients may experience blurry vision and photophobia (from ciliary spasms) from a simultaneous traumatic iritis (iridocyclitis). A traumatic hyphema from a blunt injury may also be associated with a cyclodialysis, a tear in which the ciliary muscle is avulsed from a scleral spur.[9,10]

A blunt injury to the eye can also weaken the lens zonule complex and result in a lens dislocation (Fig. 61.10A). Predisposing factors for lens dislocation include Marfan's syndrome, homocystinuria, and high degrees of myopia. Patients may complain of monocular diplopia, distorted images, and blurred vision, and have other signs related to the trauma, such as traumatic mydriasis, iridocyclitis, and hyphema.

Diagnostic Testing

A diagnosis of a traumatic hyphema can often be missed without the illumination and magnification with a slit lamp. One sequela is increased IOP, and therefore the pressure should be measured (assuming there are no signs of globe rupture). An elevated IOP occurs up to 3 days after the initial injury in approximately one-fourth of patients; it is typically self-limited, but in certain instances can be high and persist and can lead to optic nerve injury and vision loss. Photophobia in the injured eye, especially if occurring on illumination of the opposite eye, strongly suggests a traumatic iritis. A cyclodialysis will be evident on slit-lamp examination as a separation of the edge of the iris away from the limbal margin.

A lens dislocation may be evident on slit-lamp examination; one may see the edge of the natural lens (which is normally not visible whether or not the patient is dilated) (see Fig. 61.10B), phacodonesis (shimmering of the lens with eye movement), and iridodonesis (shimmering of the iris with eye movements), which are evidence that the lens zonules have been compromised. A displaced crystalline lens may also be visible on ultrasound (using a large amount of gel to minimize any pressure from the ultrasound probe on the eye).[11] Depending on the lens location, additional findings including increased IOP (secondary to angle closure glaucoma), corneal swelling, and intraocular inflammation may be present.

Management and Disposition

Traumatic Iritis, Hyphema, and Cyclodialysis. With isolated traumatic iritis, the primary goals of treatment are minimizing scarring, decreasing inflammation, and pain relief. This is often best achieved with a paralytic agent for the iris and ciliary body (eg, homatropine or cyclopentolate; see Table 61.1). Topical ophthalmic steroid drops (eg, prednisolone acetate; see Table 61.1) are considered in cases where significant post traumatic inflammation is present; however, caution should be applied in cases of corneal abrasions. Close follow-up with ophthalmology within 48 hours is warranted to ensure injury does not result in other ocular issues, such as glaucoma, corneal damage, and hypotony.

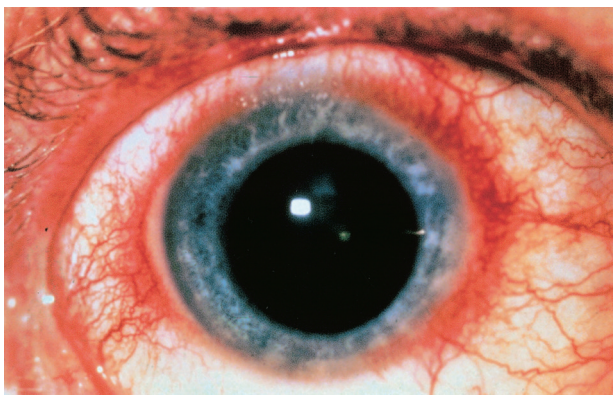
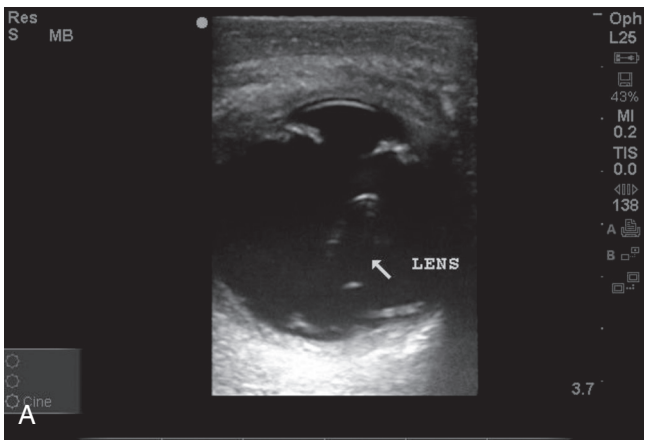


Fig. 61.9. Ciliary flush. Note that conjunctival injection is most prominent immediately around the limbus. (Courtesy Douglas Brunette, MD.)



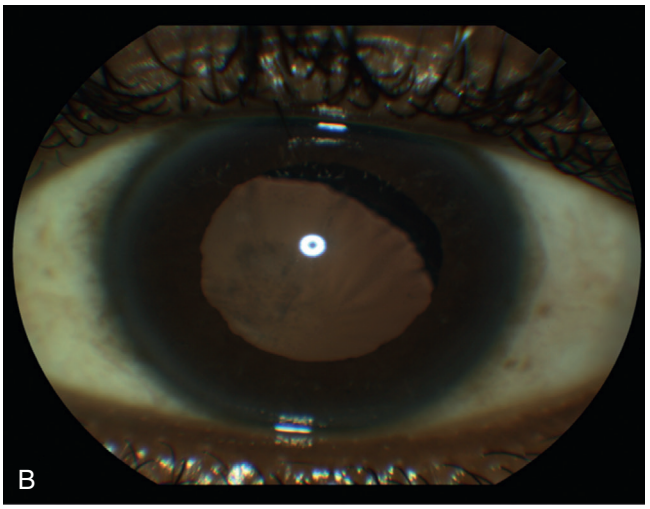


Fig. 61.10. **A,** Ocular ultrasound showing lens dislocation. **B,** Dislocated lens with edge visible. (**A,** Courtesy Douglas Brunette, MD. **B,** Courtesy Jeffrey Lee, MD, University of California San Diego.)

Both easily visible (see Fig. 61.8) and subtle traumatic hyphema deserves attention in follow-up to help minimize the likelihood of long-term complications. These complications include corneal staining and elevated IOP, which should be monitored and followed up to ensure proper resolution. In the past, recommended treatments for hyphema included strict bedrest with the head of bed elevated at least 30 degrees (presumably to allow blood to settle and filter out inferiorly) and restriction of work requiring near-vision (since accommodation may stress injured blood vessels). However there is no evidence that strict bedrest has any effect on outcome or that head of bed position affects resorption rate in a consistent manner, so a patient with an uncomplicated hyphema can be discharged home with gentle ambulation allowed, with head of bed elevation simply to keep hyphema that is larger from clotting in the visual axis.[12,13] The recommendations for pediatric patients with uncomplicated traumatic hyphema are identical; admission to hospital is not warranted, because it results in no discernable benefit.[14,15]

Admission is recommended, however, for patients with hyphema of greater than 50% (in which the large volumes of blood can lead to severely high IOP and permanent corneal damage), sickle cell trait (in whom sickling of red cells in the naturally hypoxic and acidotic anterior chamber prevents egress of aqueous humor and blood products), uncontrolled IOPs, and anticoagulated patients (in whom there is concern for re-bleed). Topical and oral agents to lower IOP are appropriate, but carbonic anhydrase inhibitors should be avoided in sickle cell patients. Antifibrinolytics, such as systemic or topical aminocaproic acid, may also be considered, because they have been shown to reduce the rate of recurrent bleeding even if they do not affect final visual acuity.[12] Initiation of these treatments are best deferred to an ophthalmologist.

For patients discharged with a traumatic hyphema, next-day follow-up with an ophthalmologist is recommended to assess IOP and to evaluate for indications for urgent paracentesis of the anterior chamber or an emergent trabeculotomy, such as inability to clear the hyphema, uncontrolled IOP, or rebleeding (which occurs in 6% to 33% of patients within 2 to 5 days).[12,16] Patients—particularly those with 20/200 vision or less in whom a rebleed might go unnoticed—should be followed for 3 days to identify these developments. Patents with underlying traumatic iritis may be prescribed cycloplegics (homatropine; see Table 61.1), but there is little evidence to support their use for hyphema without iritis; there is also no evidence that eye patching—postulated to prevent photosensitization of the corneal endothelium by protoporphyrin, a blood product—has any effect.[12]

The patient with a cyclodialysis can be treated with topical cycloplegics (see Table 61.1) to relax the iris and ciliary body and be referred to an ophthalmologist for routine outpatient follow-up; small tears tend to resolve spontaneously and can be managed conservatively, whereas large ones may be permanent and—especially if causing visual issues due to hypotony—may require surgery to re-attach.[10]

Lens Subluxation and Dislocation. Lens subluxations or dislocations can be vision-threatening emergencies and should be evaluated by an ophthalmologist in the ED for potential treatment, both medical and surgical.

Posterior Segment/Ocular Injuries: Commotio Retinae, Retinal Detachment, Intraocular Foreign Body, and Perforated Globe

Clinical Features and Differential Diagnosis

A more significant mechanism increases the possibility of a deeper ocular injury, and the spectrum of injury includes commotio retinae, a retinal or vitreous hemorrhage, a retinal detachment, an intraocular foreign body and a perforated globe.

Three main sequelae of a blunt injury to the retina are commotio retinae, retinal or vitreous hemorrhage, and retinal detachment or tear. Commotio retinae, also known as *Berlin's edema,* can occur after ocular trauma. One study showed that it was present in approximately 6% of patients who had surgically treated orbital blowout fractures.[17] With a retinal or vitreous hemorrhage, patients can experience vision compromise ranging widely from dark floaters to vision blackout. A retinal detachment can present with floaters and/or flashes of light (photopsia) and eventually cause a curtain-like blocking of the patient's vision as it evolves into a retinal detachment. Because the retina has no pain receptors, patients are usually pain-free.

An important evaluation after blunt trauma to the orbit is to determine if a globe perforation (ruptured globe) or intraocular foreign body are present. Besides pain and decrease in vision, other signs as previously mentioned (loss of anterior chamber depth, irregular or tear drop-shaped pupil, blood in the anterior chamber, 360 degree subconjunctival hemorrhage, and/or prolapse of uveal tissue) should be noted. When vector forces of the trauma are not enough to blow out the orbit, the forces are directed on the globe itself. Muscle insertion sites and the limbus are the most common sites of scleral rupture because these are the thinnest areas (see Fig. 61.5). Significant collateral damage of the adjacent tissue structures such as the lens, retina, uvea, and optic nerve can also occur. The patient with a globe injury may develop sympathetic ophthalmia, a vision-threatening autoimmune response to the remaining healthy eye triggered by the exposure of the previously naïve immune system to intraocular contents from the ruptured eye.

Diagnostic Evaluation

In some cases of retinal injury, visual acuity testing can even be normal. Commotio retinae may appear as whitening of the retina on funduscopy. The diagnostic approach to retinal detachment is discussed in more detail in the Primary Disorders of Vision section later in this chapter. In instances of a penetrating injury to the globe or orbit, careful evaluation for a retained foreign body is important. Initially, it may not be clear if a foreign body is intraocular or just intraorbital. If the suspicion for an intraocular foreign body or ruptured globe is high but the examination cannot be performed without minimal manipulation, the examination should be deferred to one performed under anesthesia and tonometry avoided. Prior to this, a CT can be performed to provide structural integrity of the globe and orbit (and location of foreign bodies); however, because it has a sensitivity of between 56% to 75% for open globe injury, it cannot be relied upon alone, and formal surgical exploration may be needed.

Management and Disposition

Retinal Injuries. A traumatic retinal detachment, if detected and treated early before the macula is involved, carries a good prognosis. About 10% of all retinal detachments are from blunt trauma. The management of retinal detachment and vitreous hemorrhage is discussed in more detail in the Primary Disorders of Vision section later in this chapter. The decreased visual acuity from commotio retinae is self-limited and resolves in a few weeks.[18] Evaluation by an ophthalmologist in the ED is recommended, however, because the commotio can mask a retinal tear.

Intraocular Foreign Bodies and Globe Rupture. For a globe perforation (open globe) with or without an intraocular foreign body, an ophthalmologist should be consulted emergently, and the examination stops until the patient can be taken to the

operating room. In anticipation of potential surgery, a protective shield should be placed (so that nothing touches the eye), tetanus vaccine administered, the patient kept *nil per os* (nothing by mouth) (NPO), and pain and nausea controlled. The incidence of endophthalmitis (an infection of the globe; see Infectious Conditions later) following open-globe injury ranges from 2.6% to 30%, and systemic antibiotics (cefazolin or vancomycin and a fourth-generation fluoroquinolone—due to its vitreal penetration) to cover common culprits of traumatic endophthalmitis (*Bacillus* species, *Staphylococcus aureus, Pseudomonas* species, gram-negative organisms, and anaerobes) should be administered.[19] Foreign bodies, especially non-metallic foreign bodies, pose the highest risk of infection.

In general, almost all intraocular foreign bodies need to be removed. For intraorbital foreign bodies, the type and location of the material influences the necessity to remove them. Removal of inert plastic, glass, and metals may cause more damage than their permanent presence, whereas organic foreign bodies typically need to be removed because of their propensity to cause infection. Siderous oxidation of ocular tissues is a late complication of iron-containing intraocular foreign bodies and can lead to visual loss. Chalcosis, a sterile inflammatory reaction to copper-containing compounds, may occur, necessitating removal of the offending object.

It is controversial whether it is necessary to avoid the use of succinylcholine for rapid sequence intubation in open globe injury because of the very theoretical possibility of a rise in IOP from succinylcholine causing further extrusion of globe contents.[20] Where time and circumstances permit, we recommend rocuronium for intubation of patients with open globe injuries, but there is no hard evidence that succinylcholine will cause harm.

Retrobulbar and Peribulbar Injuries: Orbital Wall Fracture, Retrobulbar Hemorrhage, and Optic Nerve Injury

Clinical Features and Differential Diagnosis

Trauma to the eye can result in disruption to tissue around the globe and should be suspected based on the mechanism and recognition of specific clinical features. Clinical signs that could indicate an acute orbital wall fracture include ecchymoses, tissue swelling, hypesthesia of the trigeminal nerve, double vision, blurry vision, enophthalmos (posterior displacement of the eyeball within the orbit), and ptosis. In an orbital floor fracture, a medially-hinged bony "trapdoor" fragment may have transiently displaced inferiorly, allowing herniation of orbital contents into the maxillary sinus. Associated globe injuries occur in 10% to 25% of patients with orbital floor fractures. A patient can develop a vision-threatening injury without an orbital wall fracture, specifically a retrobulbar hemorrhage. The orbit is essentially a continuous cone-shaped fascial envelope with rigid bony walls on all sides (except anteriorly where the orbital septum forms an inflexible boundary), in which there is little room to accommodate an increase in volume.[21] A retrobulbar hemorrhage from a ruptured infraorbital or ethmoidal artery occurring with intact orbit walls will increase intraorbital pressure and can cause an orbital compartment syndrome, resulting in ischemia to the optic nerve and retina. The triad of proptosis, ophthalmoplegia, and vision suggests this process. Blunt trauma can also cause a direct optic nerve injury, causing decreased visual acuity, if not vision loss. A rare complication of an orbital fracture or retrobulbar injury is an oculocardiac reflex from periorbital soft tissues that—through an afferent signal via the trigeminal nerve and efferent signal via the vagus nerve—can trigger bradycardia, junctional rhythm or even asystole, with nausea and vomiting, and is potentially fatal if unresolved.

Diagnostic Testing

A thorough examination paying particular attention to extraocular eye movements, assessing for an afferent pupillary defect (APD; Fig. 61.11) and checking facial sensation is imperative in the diagnostic evaluation of a possible retrobulbar or orbital injury. An orbital wall fracture is most readily diagnosed with an orbital CT scan with axial and coronal fine (minimum 1.5 mm) cuts (Fig. 61.12). Less sensitive diagnostic screening tools include plain x-ray, in which suggestive signs are a bulge extending from the orbit into the maxillary sinus ("teardrop" sign) and an air-fluid level in the maxillary sinus, and bedside ultrasound (sensitivity 56% to 92%) in which a suggestive sign is bright acoustic shadowing from subcutaneous or orbital air.

Clinical signs strongly suggesting a retrobulbar hematoma include the presence of three or more of the following: pain, decreased vision, proptosis with resistance to retropulsion, chemosis, limited extraocular motility, diplopia, diffuse subconjunctival hemorrhage, increased IOP, and an APD.[21] Funduscopy may reveal edema of the optic disc or retina or retinal venous congestion. A CT scan of the orbit can provide additional evidence if clinical findings are inconclusive. However, if there is significant suspicion, treatment (see the following Management and Disposition section) should not be delayed because vision loss can be permanent.

An optic nerve injury may or may not be coincidental with a retrobulbar hematoma, and clinical findings may not necessarily differentiate whether there is compression or transection. An optic nerve injury may manifest as a decrease in visual acuity, visual field deficit, a relative APD (Marcus-Gunn pupil), or a change in optic nerve appearance. The optic disc can appear normal or swollen. CT can help determine the nature and degree of optic nerve injury.

Management and Disposition

Orbital Wall Fractures. Prophylactic antibiotics are frequently recommended by consultants for orbital wall fractures, but there is no literature evidence to support their use outside of intra-operative administration.[22,23] Antibiotic prophylaxis may be warranted, however, in patients with coincident sinusitis seen on CT scan, because it increases the risk of developing orbital cellulitis.[23] The patient should be instructed to avoid exerting Valsalva maneuvers, and nose blowing and should be prescribed nasal decongestants, such as pseudoephedrine 30 mg every 6 hours, so as to decrease the risk of orbital emphysema. Lastly, to improve healing and decrease swelling, ice packs to the orbit for at least 48 hours is recommended. Some ophthalmologists use steroids to reduce swelling, however, this is a case by case decision. Clinical findings that warrant urgent surgical exploration (ie, within 24 or 48 hours) include early enophthalmos greater than 2 mm; large (>2 cm^2) defects of the orbital floor/medial wall; pediatric trapdoor fractures; and when CT evidence of entrapment is associated with symptomatic diplopia, gaze restriction, or a non-resolving oculocardiac reflex.[24] Outside of these indications, persistent diplopia and cosmetic concerns (such as, enophthalmos) are generally not addressed until swelling subsides after 7 to 10 days. Patients can be discharged for reevaluation by an ophthalmologist in 1 to 2 weeks. Children with orbital wall fractures are a special consideration, because they are more predisposed to "green-stick" fractures of the orbital wall and develop fibrosis and shortening of the affected muscle within a couple of days, affecting ocular function; thus, children with orbital wall fractures should be seen by an ophthalmologist in 1 to 2 days.[23]

Retrobulbar Hemorrhage. The loss of vision associated with a retrobulbar hematoma is irreversible within 60 to 100

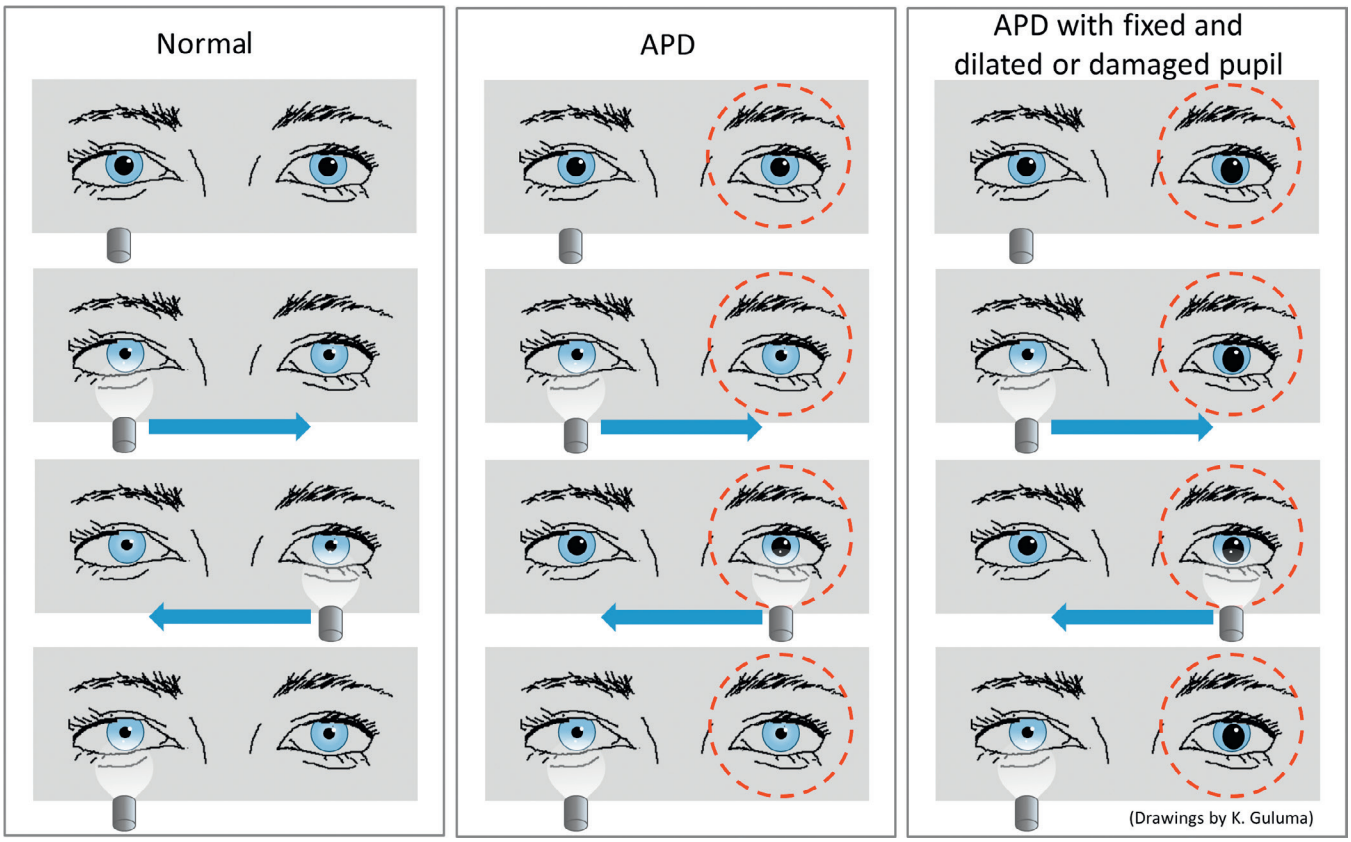


Fig. 61.11. The "swinging flashlight test" for an afferent pupillary defect *(APD),* which is otherwise known as a *Marcus Gunn pupil*. Normally *(panel on left),* both pupils constrict regardless of which eye is illuminated, due to intact afferent stimulus into the direct and consensual pupillary light reflexes. With an APD *(panel in the middle),* the pupils dilate upon "swinging" the flashlight to the pathological eye with dysfunction in the retina or optic nerve (dashed circle), because of a sudden loss of afferent stimulus into light reflexes. With a fixed and dilated or damaged pupil *(panel on the right),* the same will hold true, except that the damaged pupil may not react due to an intrinsic problem, regardless of the presence of an APD. In each condition, whether normal or with an APD, the pupillary findings will reverse on swinging the flashlight back across to the other eye. The flashlight should be held over each eye for at least 3 seconds to ensure time for a response.

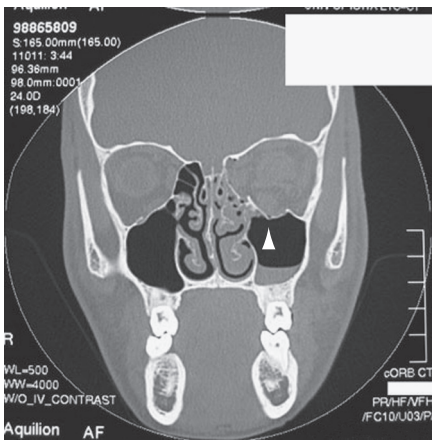


Fig. 61.12. Facial computed tomography (CT) scan showing left inferior orbital fracture with blood in maxillary sinus. (Courtesy University of Iowa Department of Ophthalmology, http://webeye.ophth.uiowa.edu/eyeforum/Images/floorfx_08232004.jpg.)

minutes after the onset of ischemia. Emergent ophthalmologic consultation for decompression is therefore indicated, keeping in mind that the clock does not start with the injury but at the time at which the intraorbital compartment pressure from the hematoma reached a pressure critical enough to start to cause vision loss. In the meantime, IOP-lowering agents (such as, intravenous [IV] carbonic anhydrase inhibitors, topical beta-blocker, alpha agonists, and in some cases 1 to 2 g of IV mannitol per kilogram) can be used. However, once ischemia and vision loss sets in, time is of the essence, and—depending upon the availability of an ophthalmologist in this time frame—a lateral canthotomy may need to be performed by the emergency clinician as a temporizing, vision-saving measure before definitive decompression (Fig. 61.13).[25]

Optic Nerve Injury. Once the determination of the type and degree of optic neuropathy is determined, treatment options can be considered. Surgical decompression of orbital canal fractures that impinge the nerve is not clearly beneficial, and steroids for traumatic optic neuropathy in general do not provide any additional benefit over observation.[26] In both cases, an ophthalmologist should be consulted in the ED for potential therapy options.

Chemical Exposures and Glues

Clinical Features

In addition to blunt or penetrating trauma, the eye can also be injured by chemical exposures. Chemical burns can lead to devastating vision loss. Acids burns precipitate and do not penetrate as deeply into tissue (due to coagulative necrosis, in which the

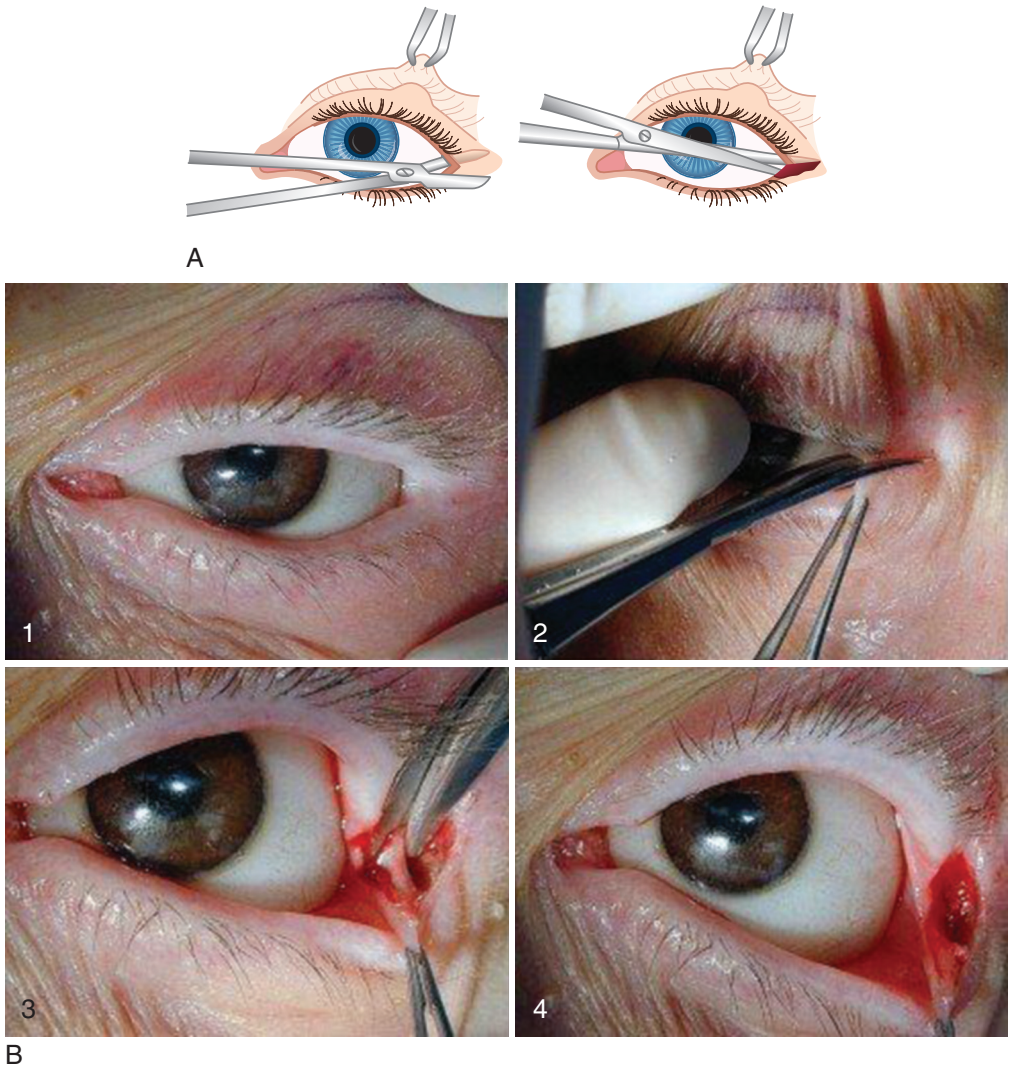


Fig. 61.13. A, Lateral canthotomy. **B,** *1,* Preoperative view of orbit. *2,* Incision for lateral canthotomy. *3,* Identification and incision of inferior canthal tendon, completing cantholysis. *4,* View after lateral canthotomy and inferior cantholysis, creating maximal immediate decompression by allowing eyeball and orbital contents to move anteriorly. (**B,** From Ramakrishnan VR, Palmer JN: Prevention and management of orbital hematoma. Otolaryngol Clin North Am 43:789–800, 2010.)

precipitation of tissue proteins limits the depth of the injury). The one exception to this is hydrofluoric acid, which may rapidly pass through cell membranes and enter the anterior chamber.[27] Alkaline burns are more severe because they produce a liquefactive necrosis (because damaged tissues then secrete proteolytic enzymes as part of an inflammatory response), leading to cataract formation, damage to the ciliary body and trabecular meshwork, and irreversible intraocular damage in as little as 5 to 15 minutes.[27] Another chemical exposure that may present in the ED is superglue to the eye. Cyanoacrylate is often used in ophthalmological surgical procedures and is relatively nontoxic to the eye. The main issues arising from superglue exposure are adhesion of eyelashes, which is difficult to reverse, and concurrent conjunctival and corneal abrasion.[28]

Differential Diagnosis

It is important to treat all unknown chemical exposures as an acidic or alkali exposure until proven otherwise. Certain substances, such as detergents and solvents, can lead to epithelial injury and anterior chamber inflammation, which then should be treated based on their particular findings (abrasion/iritis). Signs of a potent chemical exposure include periorbital edema and erythema, de-epithelialized skin, and loss of eyelashes and eyebrows, corneal and conjunctival epithelial defects, chemosis, corneal cloudiness, sterile ulceration, edema, and perforation. Elevated IOP may result from damage and/or inflammation of the trabecular meshwork. Although a determination of the pH of the solution involved is the most important consideration, other factors in the exposure (such as, temperature, amount, impact force, concentration, osmolarity, and redox potential) can greatly influence the pathophysiology of chemical tissue damage.[28a] Accessing the material safety data sheet (MSDS) of the agent involved or consulting with a Poison Center can greatly facilitate identification of the offending agent and guide the appropriate treatment. If the exposure occurred as a result of explosion, penetrating globe injury may also be present.

Diagnostic Testing

Treatment of a chemical exposure should begin as soon as possible with copious irrigation, even prior to arrival to the ED. The initial basic ophthalmic examination should pay attention to an inspection of the fornices, to ensure that there is no remaining chemical

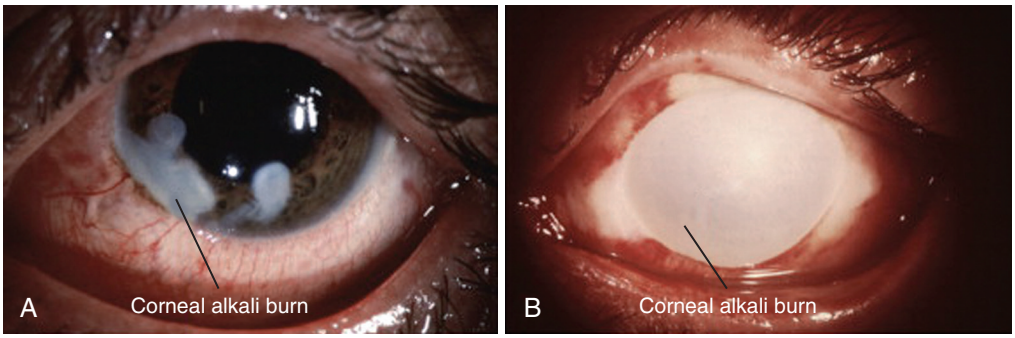


Fig. 61.14. A, Alkali burn demonstrating corneal burns and conjunctival injection on the day of the accident. **B,** Complete corneal tissue destruction 7 days after alkali burn. (From Kaiser PK, Friedman NJ, Pineda R II: The Massachusetts Eye and Ear Infirmary illustrated manual of ophthalmology, ed 2, Philadelphia, 2004, WB Saunders.)

gel or solid material (such as, alkaline lime or plaster), as well as screening for ocular trauma, facilitated with the use of a topical anesthetic for patient comfort.[27] With known chemical exposures, irrigation should be continued for a minimum of 10 minutes until a quick evaluation can be performed. This should include a pH measurement (Nitrazine paper dipped in lower lid fornix) to evaluate for acidity or alkalinity. If the pH is not in the neutral 7 to 7.5 range, irrigation should be continued. Superglue exposure represents a special circumstance, and there are two main principles in the evaluation: (1) to separate the lids so that a detailed eye examination can be performed and to remove visible superglue, and (2) to identify any corneal abrasion with fluorescein staining.[28]

Management and Disposition

For acid or alkaline burns, irrigation of the eye should be performed immediately. The longer irrigation is delayed, more irrigation volume will likely be required because the chemical can deposit within the tissue.[27] It may take up 20 L or more to change the pH to a physiologic level (a goal pH of 7 to 7.5). Based on animal studies, traditional isotonic saline irrigation solutions may be relatively ineffective at neutralizing a significant exposure to an alkaline agent (such as, sodium hydroxide) within the 20 minute time frame required to reduce injury and that buffered irrigation products specially designed for the task are significantly more effective.[29,30] This being said, initiation of irrigation with whatever solution is most readily available should not be delayed while such a solution is being obtained. Surprisingly, tap water is more effective than saline at normalizing pH, specifically for alkali burns.[29] It is also better tolerated than saline and is therefore recommended in situations in which a buffered product is not available. Use of topical anesthesia (see Table 61.1) and assistive devices, such as a Morgan lens and an eyelid retractor, can aid in delivering the irrigation more effectively. Emergent ophthalmological consultation is warranted in significant acid burns, and all alkaline burns, especially those in which irrigation to a pH of 7 required copious irrigation.[29] In chemical exposures deemed to have a low risk of significant injury (an assessment of facilitated by contact with a local poison center) with no signs of immediate ocular injury (such as, corneal burns), the patient can be treated and referred for follow-up with an ophthalmologist in 24 to 48 hours.

For more significant chemical injuries, cycloplegics, antibiotics (ointments are usually preferred because they also provide comfort), and occasionally steroid drops are indicated (see Table 61.1 for agents and dosing). After the acute treatment has been completed, obtaining additional history (such as, the nature of the substance) can be useful in determining prognosis; substances with pH ranging from 2 to 12 with limited contact time tend to have a better prognosis. However, at the time of presentation, the severity and complications of the injury may not be completely assessed because the full extent of the injury has not yet occurred. These complications can include permanent corneal injury (Fig. 61.14), glaucoma, palpebral and conjunctival adhesions, cataracts, and retinal injury.

In the case of superglue, cyanoacrylate does not bond well to wet surfaces, and an exposure into the eye typically results in a forceful blink and extrusion of the glue onto the dry surfaces of the lid margins.[28] Gentle traction will often separate glued eyelashes; if not, trimming with Westcott scissors can help. Examination by slit lamp can help determine which lashes can be more readily separated. Time will help loosen the adhesions and allow for removal of the glue. If there is eyelid malposition, cutting the lashes can often allow for normalization of the eyelid position. Attempts to dissolve the glue with other substances (especially acetone) should be avoided, because they may cause ocular damage. Ophthalmology consultation in the ED is recommended for cases in which the above measures fail to separate the lids to enable an examination, if there is residual eyelid mal-positioning, or if there is a suspected corneal abrasion from the hardened glue. If separating eyelids reveals no evidence of subsequent lid malpositioning and no sign of conjunctival involvement or injury, the patient can be referred to an ophthalmologist for follow-up as an outpatient in the next day.

INFLAMMATORY CONDITIONS

Principles

Inflammatory conditions of the eye tend to present as a "red eye," which is a general term that encompasses a variety possible etiologies in the conjunctiva, cornea, globe and surrounding orbit. The clinical approach to the red eye in general (which includes not just inflammatory processes but also infectious processes) is described in detail in Chapter 19.

The Conjunctiva and Cornea: Keratitis, Pterygium and Pinguecula

Clinical Features and Differential Diagnosis

Conjunctival and corneal inflammatory conditions present in a somewhat stereotyped fashion and include allergic conjunctivitis, superficial punctate keratitis, ultraviolet (UV) keratitis (radiation keratitis), and pterygium and pinguecula. Allergic conjunctivitis, although technically an inflammatory process, is similar enough in presentation to infectious conjunctivitides that it is considered together with the infectious processes outlined later this chapter.

Superficial punctate keratitis presents with pain or foreign body sensation, photophobia, and redness due to poor lubrication of the corneal surface from any one of several etiologies, including dry eyes, drug toxicity, and contact lens overuse. UV keratitis is a specific form of keratitis that presents when prolonged exposure to UV light (from a source such as a tanning booth, reflection from snow or water, or a welder's arc) causes a direct injury to the corneal epithelium, at times severe enough to cause ulceration. There is a latency of 6 to 10 hours before symptoms arise, at which point patients have a significant degree of pain and discomfort, photophobia, and mild conjunctival injection.

Another set of conjunctival inflammatory conditions, somewhat similar in appearance to one another, are pterygium and pinguecula. A *pterygium* is a chronic fibrovascular growth of conjunctiva triggered by chronic exposure to UV light that grows temporally from the nasal side of the eye (or vice versa), eventually covering the cornea. A pterygium can get acutely inflamed, whereupon patients experience foreign body sensation, dry eyes, and redness, but they should not have loss of vision unless the process has started to infringe upon the visual axis (a very gradual and chronic process). A *pinguecula* is of similar pathology and pathophysiology to a pterygium, resulting in similar symptoms, except that it stops at the limbus and does not enter the cornea or visual axis.

Diagnostic Evaluation

Examination with a slit lamp is an integral part of the diagnostic evaluation of conjunctival and corneal inflammatory conditions. With superficial punctate keratitis and UV keratitis, multiple punctate epithelial erosions are seen upon fluorescein staining. A patient with a pterygium or pinguecula will have a visible, opaque conjunctival overgrowth on the conjunctiva of one or both eyes, typically triangular or pie-shaped, with the apex of the triangle pointing towards the pupil.

Management and Disposition

Superficial Punctate Keratitis and Radiation Keratitis. Determination of etiology of the keratitis is important for definitive treatment. In general, however, care is supportive. The treatment considerations for superficial punctate keratitis and UV keratitis are the same as with corneal abrasion (because both entail an injury to the corneal epithelium and superficial cornea, see Corneal Abrasions) and include limited use of topical anesthetics and topical antibiotics administered for 3 to 5 days only if infection is a concern (see Table 61.1). UV keratitis will typically resolve in about 24 hours or so, and given the nature of the injury, patients should be instructed to avoid damaging UV rays.[31] Ophthalmologic follow-up in 24 hours is recommended if symptoms have not resolved.

Pterygium and Pinguecula. Treatment of pterygium and pinguecula are similar, and it includes UV protection, lubrication, and treatment of acute inflammation with topical NSAIDs (see Table 61.1). The inflammation of a pterygium or pinguecula is usually self-limited, and encroachment into the visual axis from a pterygium is typically very gradual; non-emergent referral to an ophthalmologist is recommended for surgical treatment of severe cases, and for evaluation of the rare coexistence of an ocular surface squamous neoplasia.

The Globe: Uveitis, Scleritis, and Episcleritis

Clinical Features and Differential Diagnosis

The globe itself can on rare occasion be afflicted by a variety of autoimmune conditions, typically involving the uvea as an autoimmune uveitis, or the sclera, as a scleritis. Three noninfectious, inflammatory considerations causing a painful red globe are uveitis, scleritis, and episcleritis.

Uveitis is an autoimmune inflammation of the uvea, the part of the middle layer of eye that includes the highly vascularized and pigmented iris, ciliary body, and choroid.[32] The iris and ciliary body are most commonly involved, a condition called *iritis* or *anterior uveitis,* but uveitis may rarely involve the intermediate and posterior chambers as a rare panuveitis. No cause is identified in 60% to 80% of people, although uveitis is one of the most frequent extra-articular features in seronegative arthritides (including ankylosing spondylitis, psoriatic arthropathy, arthritis from inflammatory bowel disease [ie, Crohn's], and reactive arthritis [ie, Reiter's syndrome]). The typical patient with an acute anterior uveitis will present with a very painful red eye, often with photophobia, and occasionally with decreased visual acuity.[33]

Scleritis is a similar autoimmune inflammatory process, but involving the sclera (the tough connective tissue layer that begins at the limbus and surrounds the eye) instead of the uvea.[34] It is divided into anterior scleritis and the less frequent posterior scleritis (inflammation of the sclera posterior to the insertion of the rectus muscles). Scleritis can also be infectious, treated much in the same way an endophthalmitis would be (see The Globe: Endophthalmitis).

Episcleritis, which can be confused with scleritis, is caused by inflammation in the episcleral layer of the eye rather than the deeper scleral layer. Episcleritis, unlike scleritis, is not vision-threatening and is not associated with as much discomfort.

Diagnostic Evaluation

On slit-lamp examination, uveitis will typically reveal conjunctival injection, ciliary flush in the peri-limbal area, and cells and flare in the anterior chamber. Episcleritis can be distinguished from scleritis in that it is associated with more peri-limbal injection and has a redness that described as salmon pink as opposed to the deeper purple hue seen in scleritis; instillation of 10% phenylephrine drops will constrict and blanch injected superficial episcleral vessels in episcleritis but will not do so to the injected deeper vessels involved in scleritis.[34] Scleritis is often more severe than episcleritis and has a much higher association with systemic diseases, such as Wegener granulomatosis, rheumatoid arthritis, and connective tissue disease (an evaluation that can be deferred to outpatient follow-up).

Management and Disposition

Treatment of both uveitis and scleritis typically involves topical corticosteroid drops (and cycloplegics for symptoms of iridospasm; see Table 61.1), with a transition to systemic corticosteroids and immunosuppressants if these treatments fail. NSAIDs are helpful for scleritis, although systemic steroids may be more useful in severe cases.[35,36] Decisions about treatment are typically made in concert with an ophthalmologist, and patients should be referred to an ophthalmologist for close follow-up in the next day or so; scleritis has a higher association with ocular complications, including keratitis, increased IOP, and vision loss.[35]

The Orbit: Orbital Pseudotumor, Orbital Apex Syndrome, and Thyroid Orbitopathy

Clinical Features, Differential Diagnosis, and Diagnostic Evaluation

The orbit may be affected by typically idiopathic, noninfectious inflammatory processes that lead to diffuse eye pain, redness,

swelling, and potentially disordered vision. Considerations include orbital inflammatory pseudotumor and orbital apex syndrome (which are unilateral), as well as thyroid myopathy (which is usually bilateral).

Orbital inflammatory pseudotumor (also known as *idiopathic orbital inflammation syndrome, orbital pseudotumor,* or *orbital inflammatory syndrome*) presents as an acute to subacute tumor-like inflammation consisting of a pleomorphic cellular response and a fibrovascular tissue reaction, and it is associated with various rheumatologic disorders, including Wegener's granulomatosis, giant cell arteritis, systemic lupus erythematosus, dermatomyositis, and rheumatoid arthritis.[37] In orbital apex syndrome, the apex of the orbit (through which the cavernous sinus drains the eye and orbit, and cranial nerves [CNs] III, IV, and VI travel) may be selectively affected by a cavernous sinus mass or vasculitis. Etiologies include infection, carotid-cavernous fistula, inflammatory vasculitides (such as, giant cell arteritis), Tolosa-Hunt syndrome (a rare idiopathic vasculitis), or tumor or infiltration (eg, sarcoidosis). Both orbital inflammatory pseudotumor and orbital apex syndrome may result in proptosis, chemosis, and/or conjunctival injection; and with orbital apex syndrome, there may be palsies of CNs III, IV, and VI (see Chapter 18).

Inflammatory thyroid orbitopathy from Grave's disease is the most common cause of ocular myopathy in older adults, and it presents with oculomotor muscle swelling and restriction that may be bilateral in 85% of cases. It classically affects the inferior and medial recti muscles first, leading to restriction of elevation and abduction of the eye with orbital muscle dysfunction and misalignment of the visual axes.[38] The examination may reveal stigmata of the underlying disease process, such as lid lag or periorbital swelling or proptosis, as well as diffuse conjunctival edema, and vascular injection near the insertions of the rectus muscles.

The diagnostic evaluation of a suspected orbital inflammatory process primarily involves imaging of the orbit. Options include a magnetic resonance imaging (MRI) scan of the orbits with gadolinium, which can allow an assessment for enlargement or enhancement in extraocular muscles and orbital structures, or—as a likely more readily available second-line option—a contrast-enhanced orbital CT (with fine cuts through the orbit).[39]

Management and Disposition

The mainstay of therapy (assuming infection is excluded) for orbital pseudotumor and orbital apex syndrome is systemic corticosteroid therapy, although there is increasing use of antimetabolites, cytotoxic agents, and other immunosuppressive agents.[37] Treatment choices will typically be made in concert with an ophthalmologist. For thyroid orbitopathy, the treatment of the underlying Graves' disease will address the ophthalmological issues but may involve immunosuppressive medications, radiation, or surgery.

INFECTIOUS CONDITIONS

Principles

A critical clinical distinction that comes into play in a patient with a red, irritated, or painful eye is whether or not there is an infectious process in play. This is based on clinical features, keeping in mind that the globe of eye and the encompassing tissues of the orbit represent a pristinely organized and functional arrangement of tissue planes and glandular structures, and that any disruption to these structures, whether from minor trauma, prior surgery, or inflammation, can predispose to an infectious process.

The Conjunctiva: Allergic, Viral and Bacterial Conjunctivitis, and Ophthalmia Neonatorum

Clinical Features, Differential Diagnosis, and Diagnostic Testing

Symptoms of conjunctivitis—which may be allergic, toxic, or infectious—include redness, discharge, foreign body sensation, photophobia, and blurry vision.

The most common form of conjunctivitis is thought to be allergic conjunctivitis. This is not infectious per se, but it is considered in the differential diagnosis here because it is sometimes a challenge to distinguish from a viral conjunctivitis. Allergic conjunctivitis is a type 1 histaminergic hypersensitivity reaction with red itchy eyes, clear discharge, and is classically bilateral, associated with pollen and dust. In more severe cases, moderate to severe injection with glassy chemosis is observed. A toxic conjunctivitis (from topical ocular medications) may appear similar to allergic conjunctivitis; a contact dermatitis (from a trigger like eye makeup) should be suspected if there is an associated lichenified, eczematous periorbital dermatitis and edema.

Of the infectious etiologies, viral causes are most common. Viral conjunctivitis is classically preceded by a viral infection with upper respiratory symptoms, with sequential involvement of both eyes, but many viral conjunctivitis episodes have no preceding upper respiratory infection (URI) syndrome. It is most commonly by adenovirus, easily spread by contact with fomites. The conjunctival discharge with viral infections tends to be more watery and less purulent than that in bacterial conjunctivitis, with signs such as preauricular lymphadenopathy and follicular changes of the conjunctiva (Fig. 61.15). Viral conjunctivitis, and keratoconjunctivitis, however, can present with impressive purulence, including having the eyelids stuck shut when awakening from sleep. Such findings do not distinguish bacterial from viral causes. Viral infections typically last 1 to 3 weeks. Epidemic keratoconjunctivitis is a highly contagious and more virulent viral conjunctivitis often presenting in epidemics, with which the patient may also complain of foreign body sensation and have a mild keratitis.

Bacterial conjunctivitis is significantly less common than viral. The organisms involved include Staphylococcus organisms, as well as *Moraxella catarrhalis, Streptococcus pneumoniae, Haemophilus influenzae* and rarely *Neisseria gonorrhoeae,* with an increased prevalence of methicillin-resistant *Staphylococcus aureus* (MRSA) conjunctivitis over the last decade.[40] Conjunctivitis from gonorrhea classically presents with copious purulent discharge (Fig. 61.16) and carries a high risk for corneal involvement and

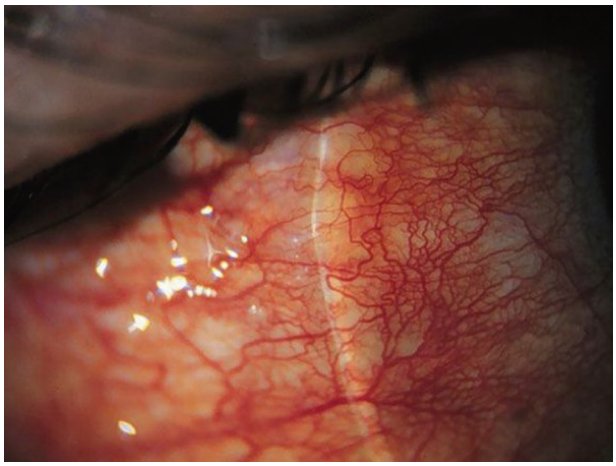


Fig. 61.15. Conjunctival injection resulting from viral conjunctivitis. (Courtesy www.tedmontgomery.com.)

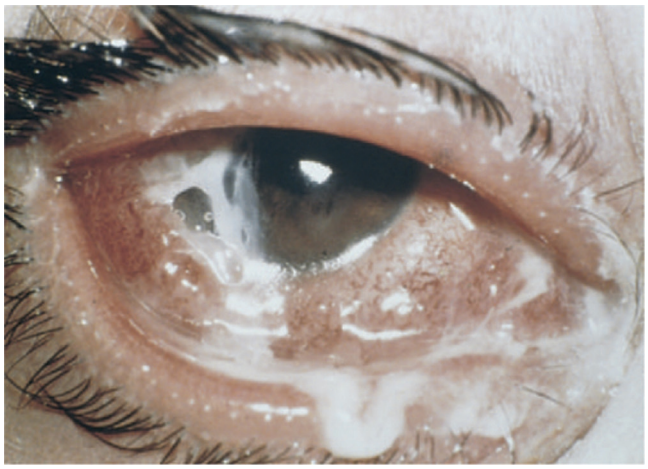


Fig. 61.16. Purulent discharge and conjunctival hyperemia suggest bacterial conjunctivitis. (From Goldman L, Schaefer AI, editors: Goldman's Cecil medicine, ed 24, Philadelphia, 2012, Saunders.)

subsequent corneal perforation.[40] A gram stain and culture (or a polymerase chain reaction [PCR] test as done for genital samples) can aid in the diagnosis.[41]

Distinguishing a viral from a bacterial conjunctivitis can sometimes be a challenge in the ED. A systematic review found that redness of the conjunctival membrane that is intense enough to obscure the tarsal vessels (likelihood ratio [LR], 4.6; 95% confidence interval [CI], 1.2 to 17.1), physician-observed purulent discharge (LR, 3.9; 95% CI, 1.7 to 9.1), and matting of both eyes in the morning (LR, 3.6; 95% CI, 1.9 to 6.5) increase the probability of a bacterial cause, whereas inability to discern that the patient's eye is red from 20 feet away (LR, 0.2; 95% CI, 0 to 0.8) and absence of morning matting of either eye (LR, 0.3; 95% CI, 0.1 to 0.8) decrease the probability of a bacterial cause.[42]

What appears to be an infection of the conjunctiva may actually represent an infection of the cornea, and therefore a slit-lamp examination is important; if signs of corneal involvement are present, a keratitis is in play (see Diagnostic Evaluation in The Conjunctiva and Cornea: Keratitis). Epidemic keratoconjunctivitis may have some mild punctate keratitis on fluorescein staining.

A consideration specific to neonates is ophthalmia neonatorum, which is a neonatal conjunctivitis developing the first 30 days of life. It can be from allergic or chemical causes but most concerning are bacterial and viral causes, presenting with tearing and discharge followed by scarring and blindness. The evaluation involves gram stain and cultures geared to infections such as *N. gonorrhoeae*, *Chlamydia,* and herpes simplex virus (HSV) that are transmitted from mother to infant through the birth canal. HSV may have associated corneal microdendrites and lid edema. Infection with *N. gonorrhoeae* manifests within 2 to 4 days after birth (but may take up to 20 days). The infant should be carefully examined for any evidence of a systemic gonococcal infection.

Management and Disposition

Allergic and Viral Conjunctivitis. Allergic conjunctivitis and viral conjunctivitis are usually self-limited, and can be treated with supportive measures, such as cool compresses (nature's anti-inflammatory). Topical antibiotics should be avoided unless there is concern for a bacterial superinfection. For allergic conjunctivitis, the patient should be counseled to avoid the offending agent and can be prescribed topical and systemic anti-allergy medications (see Table 61.1 for choices and dosing of topical options). Medications with preservatives in them should be avoided, because they may exacerbate symptoms. Presumed viral conjunctivitis rarely requires a culture. If symptoms and findings of either allergic or viral conjunctivitis worsen after 2 to 3 days, other etiologies should be considered. If inflammation is severe (with pseudomembranes, bleeding), then an ophthalmology evaluation in the ED for steroid treatment is recommended, otherwise patients can be discharged with a referral to an ophthalmologist if they worsen or if they do not improve by 7 to 10 days. Children with viral conjunctivitis should usually be kept out of school until symptoms have resolved, which will be 3 to 5 days, keeping in mind that communicability is estimated to last up to 10 to 14 days.

Bacterial Conjunctivitis. Although bacterial conjunctivitis is typically self-limited, most resolving in 1 to 2 weeks without treatment, topical antibiotics shorten the time to resolution.[43] Ointment is preferred given the smoothing effect on the eye and ease of instillation (patients know if ointment was applied or not and have to do it less frequently). The prescribed antibiotics (see Table 61.1 for options) should cover the organisms mentioned previously and be taken for at least 1 week; those with the highest level of evidence for the treatment of bacterial conjunctivitis are tobramycin, ciprofloxacin, moxifloxacin, ofloxacin, azithromycin, and trimethoprim/polymixin B.[40] Gentamicin and neomycin should be avoided due to toxicity. Contact lens wearers should have coverage for *Pseudomonas* (see Table 61.1). Treatment of a bacterial conjunctivitis suspected to involve *N. gonorrhoeae* consists of ceftriaxone 1 g intramuscularly once, and saline irrigation of the affected eye(s), with concomitant empirical treatment for *Chlamydia trachomatis* infection (either 1 mg of azithromycin orally once, or doxycycline 100 mg orally bid for 7 days).

Ophthalmia Neonatorum. Hospitalization of neonates with blood and cerebrospinal fluid examination may be indicated for ophthalmia neonatorum. *N. gonorrhoeae* conjunctivitis in a neonate is typically treated with single dose of ceftriaxone 25 to 50 mg/kg up to a total dose of 125 mg intramuscularly, topical erythromycin or polymyxin B–bacitracin ointment, and saline washes of the affected eye. Potential ocular chlamydial infection is often simultaneously treated with topical erythromycin ointment and oral erythromycin syrup 50 mg/kg/day divided into four doses per day for 14 days. HSV should be treated with acyclovir IV 45 mg/kg/day plus vidarabine 3% ointment five times per day for 14 to 21 days. Evaluation for systemic involvement is indicated and ophthalmology consultation in the ED is warranted.

The Cornea: Corneal Ulcers, Herpes Simplex Keratitis, and Herpes Zoster Keratitis

Clinical Features and Differential Diagnosis

What appears to be conjunctivitis may actually represent an infection of the cornea. A *corneal ulcer* (Fig. 61.17) is an infectious and/or inflammatory erosion, "ulcerative keratitis," of both the outer epithelial cell layer and the underlying stromal layer (which is the bulk of the cornea). Corneal ulcers present with pain and redness of the eye, tearing, sensitivity to light, and blurred, hazy, or otherwise decreased vision. There can also be discharge or a foreign body sensation. A corneal abrasion can become an ulcer if secondarily infected, which in turn lead to corneal perforation if severe and untreated. Although corneal ulcers are due to infection, most of the resulting corneal injury is due to the secondary inflammation. The most common bacterial pathogens are *Staphylococcus, Streptococcus, Mycobacterium,* and *Pseudomonas*, which is associated with contact lens wear. Fungal pathogens are typically seen in users of corticosteroid drops, and in agricultural workers and others who may have contamination of the eye with vegetable matter or soil.

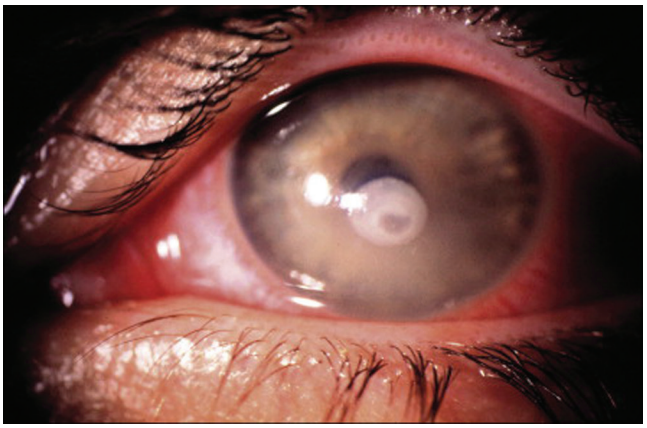


Fig. 61.17. This corneal ulcer caused by *Pseudomonas aeruginosa* occurred in a young man who wore decorative contact lenses without professional supervision. (From Yanoff M, Duker JS, editors: Ophthalmology, ed 3, Philadelphia, 2008, Mosby.)

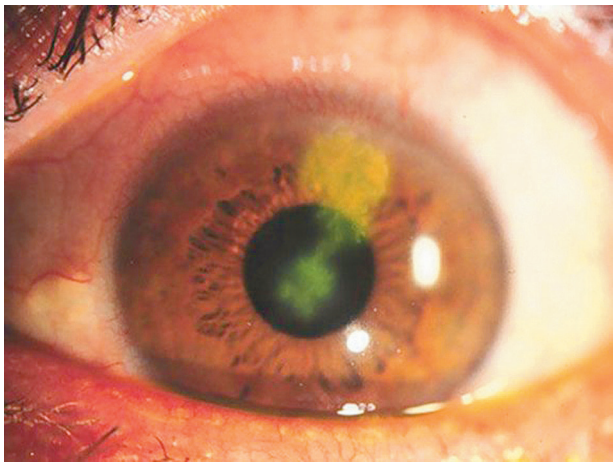


Fig. 61.18. Herpes simplex keratitis infection. Note typical dendritic pattern on cornea. (Courtesy www.tedmontgomery.com.)

The cornea can also be infected by viruses. Herpes simplex keratitis, one of the most common causes of viral keratitis, can produce recurrent corneal ulcers similar to recurrent herpes labialis or herpes genitalis. Herpes simplex may be either primary or reactivation of preexisting disease. Symptoms are similar to corneal ulcers. Herpes zoster keratitis can occur in the setting of herpes zoster ophthalmicus. Herpes zoster is re-activated along the ophthalmic division of the trigeminal nerve, and eye involvement is possible. Patients will typically present with a dermatomal rash over the forehead and upper eyelid and sometimes along the nose (branch of the nasociliary nerve—called *Hutchinson's sign*), or even have a local Horner's syndrome.

Diagnostic Testing

The foundation of the diagnostic evaluation of corneal lesions is a careful examination and biomicroscopy with the slit lamp and fluorescein staining to evaluate corneal epithelial surface disruptions. On slit-lamp examination, a corneal ulcer may appear to have more "heaped up" edges (seen with tangential lighting) than those seen with a corneal abrasion; this finding, combined with stromal edema or infiltration (whitening of the underlying or surrounding cornea), helps red-flag the process as an ulcer instead of an uncomplicated abrasion.

A corneal ulcer from herpes simplex keratitis may present with classic "dendritic" lesions on slit-lamp examination (Fig. 61.18), or with an amoeba-shaped ulceration, or have nonspecific findings such as punctate epithelial erosions, stromal whitening, and thinning of the cornea, possibly with classic herpetic vesicles located on the lids or conjunctiva. Herpes zoster keratitis may appear somewhat similar but will have signs of a dermatomal vesicular rash, and it is frequently associated with iritis, uveitis, and choroiditis.[44] Viral cultures of tissue can help direct therapy.

Management and Disposition

Corneal Ulcers and Infiltrates. Topical anti-microbial therapy for corneal ulcers and infiltrates is appropriate, although in some severe cases, systemic antibiotics may be warranted. The fluoroquinolones (see Table 61.1) have particularly good ocular penetration; doxycycline and other tetracyclines have good anti-collagenase properties that help preserve corneal integrity. Steroids may be used to decrease inflammation but must be used with caution, because they may exacerbate the clinical situation (and if a herpetic process is suspected, steroids may have to be avoided, or antivirals concurrently used). Ophthalmology consultation in the ED is important for management of corneal ulcers, because they can rapidly progress.

Herpes Simplex Keratitis. Herpes simplex keratitis is the most common cause for corneal transplants in the United States. Emergent ophthalmologic consultation is advised, because the severity of disease will dictate treatment. Herpes simplex keratitis is treated with topical antiviral agents, such as topical acyclovir trifluridine 1% nine times a day for 14 days. Topical prophylactic antibiotics, such erythromycin ointment, and a cycloplegic agent if there are symptoms of iritis can be considered (see Table 61.1). Topical steroids should be avoided because they worsen infection.[40] Systemic therapy should be considered (acyclovir 400 mg five times daily or valacyclovir 500 mg three times daily for 7 to 10 days) if topical treatment is not available or if the process is severe; admission will typically not be needed, but close follow-up with an ophthalmologist within 1 to 3 days is important.

Herpes Zoster Keratitis. Herpes zoster ophthalmicus accounts for approximately 10% to 20% of all zoster cases and necessitates emergent ophthalmologic consultation. If not treated and recognized immediately, herpes zoster ophthalmicus may result in permanent vision loss. Systemic therapy is the standard of care (unlike HSV, topical antivirals have little effect). If retinal involvement occurs or the patient is immunocompromised, inpatient treatment is recommended. Higher dose antiviral agents (acyclovir 800 mg five times daily, valacyclovir 1000 mg three times daily, or famciclovir 500 mg three times daily, all for 7 to 10 days[40]) are used, and occasionally topical steroid agents and systemic antibiotics may be added. Topical antibiotics are used to prevent bacterial superinfection of skin and lid lesions. Early treatment with antiviral therapy within 72 hours of the onset of the rash has been shown to reduce acute pain and ocular complications. Additional consideration for therapy includes pain management and aggressive lubrication to maintain a healthy ocular surface.

The Eyelids and Periorbital Area: Hordeolum, Chalazion, Dacryocystitis, Blepharitis, and Cellulitis

Clinical Features and Differential Diagnosis

The tissues of the eyelids and periorbital area are susceptible to any of a number of types of infections, which include those related to glandular or ductal structures, such as a hordeolum, chalazion,

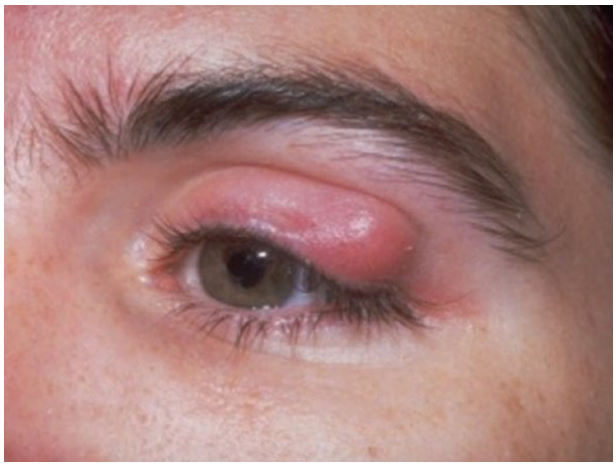


Fig. 61.19. Chalazion of the upper eyelid. (Courtesy www.tedmontgomery.com.)

or dacryocystitis, or more diffuse involvement of tissue, such as blepharitis or periorbital cellulitis. Hordeola and chalazia, also known as *styes of the eyelid,* are inflamed oil glands of the eye. A *hordeolum* is caused by acute inflammation of a gland of Zeis or hair follicle. It is typically painfully tender, erythematous, associated with swelling, and can be infected. On the other hand, a *chalazion* is a chronic sterile, granulomatous inflammation of a meibomian gland (and may evolve from a hordeolum), which results in localized swelling that is usually not acutely painful (Fig. 61.19).

Dacryocystitis is an infection of the lacrimal sac, usually resulting from a nasolacrimal duct obstruction. It is more common in females. Symptoms and signs include pain, tenderness, swelling, and erythema over the lacrimal sac medial to the eye (Fig. 61.20). Pressure over the sac may express purulent material from the puncta. The lacrimal gland itself can also become infected, appearing as a focal area of periorbital erythema, swelling and tenderness lateral to and above the upper eyelid.

Patients with blepharitis typically describe itching and burning of the eyelids with associated tearing and crusting of the eyelids. The eyelids become diffusely inflamed and thickened, with erythematous margins, and telangiectasias surrounding the eyelid margin. Blepharitis can be distinguished from a pre-septal cellulitis in that it is isolated to just the eyelid margin. Blepharitis has an association with atopic dermatitis, rosacea, and eczema.

Any one of the aforementioned focal infections, but especially dacryocystitis and blepharitis, may be complicated by a more diffuse, associated cellulitis. Cellulitis frequently presents, however, as an individual entity, and it has to be carefully distinguished as either pre-septal (also called *periorbital*) or post-septal (also called *orbital*). *Pre-septal* and *post-septal* are the most useful terms because (1) they incorporate the most impactful clinical distinction in the ED and (2) remove any chance of confusion as to what is being referred to in communications with consultants. Pre-septal cellulitis is limited to the tissue anterior to the orbital septum, whereas a post-septal cellulitis implies spread of the infection beyond the septum, which is concerning because it can lead to involvement of valuable orbital structures. Pre-septal cellulitis will present with lid erythema, warmth, tenderness, swelling, and even a low-grade fever. Post-septal cellulitis will present with the same but may also have more alarming symptoms including proptosis, ophthalmoplegia, pain with eye movement, chemosis, and systemic signs of infection. In very severe cases, visual loss can occur. In children, pre-septal cellulitis is often more difficult to differentiate from a post-septal cellulitis because of an incomplete orbital septum.

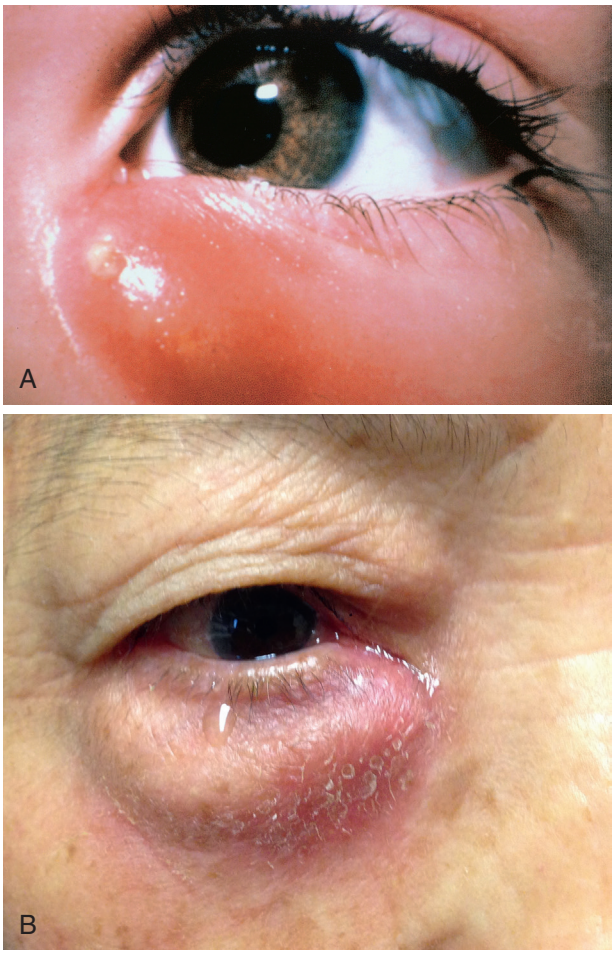


Fig. 61.20. **A** and **B,** Dacryocystitis. (Courtesy Jeffrey Lee, MD, University of California San Diego.)

Diagnostic Testing

For hordeolum, chalazion, dacryocystitis, blepharitis, and a cellulitis that is clearly pre-septal, the diagnosis is established on the clinical examination alone, and no additional diagnostics are needed. CT imaging is, however, indicated in cases concerning for an orbital abscess or in which localization of an infection (pre-septal or post-septal) is difficult. In such cases, a complete blood count (CBC) may also be helpful. The primary diagnostic decision for the ED patient with a cellulitis around the eye is deciding who needs further evaluation with a CT scan. Symptoms and signs of proptosis, ophthalmoplegia, pain with eye movement, and chemosis easily suggest the possibility that a cellulitis is post-septal, but upward of 50% of confirmed cases may not have these symptoms.[45,47] In these "no orbital symptom" cases, a peripheral absolute neutrophil count (ANC) of >10 000 cells/μL, moderate-to-severe periorbital edema (extending beyond the eyelid margins), absence of conjunctivitis as the presenting symptom, older than 3 years old, and recent antibiotic use have been shown to be predictors of an orbital abscess—specifically in the pediatric population.[45] In addition, a sudden onset is more typical of a post-septal orbital cellulitis.[47] The absence or presence of a fever has little discriminatory utility. Cultures obtained from swabs of the eyes are discouraged due to the risk of misleading results from inoculation with commensal organisms, and blood cultures have little diagnostic utility.[46]

Management and Disposition

Hordeolum and Chalazion. Often, hordeola and chalazia are self-limited and can resolve on their own when the glands become unobstructed. Conservative treatment to normalize flow of the obstructed oil glands is the primary goal. This includes warm compresses for 10 to 15 minutes, 3 to 5 times a day. Treatment of an underlying blepharitis may be indicated. Referral to an ophthalmologist is recommended for incision and drainage or additional management and evaluation in nonresponsive cases. Progression to an infected oil gland may indicate need for antibiotics, depending whether the process takes the form of a blepharitis or a cellulitis (see treatment of each in the following sections).

Dacryocystitis. The most common causative organisms in dacryocystitis are *S. aureus, S. pneumoniae*, *H. influenzae*, *Serratia marcescens*, and *Pseudomonas aeruginosa*, with an emerging prevalence of MRSA.[47] Treatment consists of massage, warm compresses, and systemic antibiotics selected so as to include coverage of MRSA. An attempt should be made to obtain a culture by applying gentle pressure to the nasal lacrimal duct and expressing fluid. In infants, acute dacryocystitis represents a medical emergency, because it can lead to complications including post-septal orbital cellulitis. Admission is warranted for severe cases. Occasionally, drainage of the sac is required; however, this can lead to fistula formation. Dacryocystorhinostomy is the definitive treatment, but the optimal time for surgery is when the infection is controlled. Discharged patients should follow-up with an ophthalmologist in 24 to 48 hours.

Blepharitis. The initial treatment of blepharitis is conservative, designed to remove residual oils and scurf, and entails warm massage with a moist washcloth about for 10 to 15 minutes, three to five times a day, and cleaning the lid margins twice a day with a cotton swab soaked in mild baby shampoo. Because blepharitis arises as a result of an inflammatory process, there is potential for bacterial overgrowth and superinfection (*Staphylococcus epidermidis* primarily, but also *Propionibacterium acnes,* and corynebacteria), and—if there is a concern for infection—topical azithromycin, erythromycin, or levofloxacin (see Table 61.1) can be considered.[47,47a] Uncomplicated cases can be discharged to follow-up with an ophthalmologist within a week or so, or within 1 to 3 days if there is concern for infection.

Periorbital Cellulitis. If pre-septal cellulitis with no other underlying medical conditions is diagnosed with certainty, the patient can be discharged on an oral antibiotics directed toward the most common organisms, *Streptococcus* and *Staphylococcus,* keeping in mind that orbital cellulitis tends to be polymicrobial.[47] Although many practitioners empirically cover for MRSA, this organism is actually very rare when it comes to orbital cellulitis (at least in published series involving primarily children).[48] An option is a beta-lactam antibiotic, such as oral amoxicillin-clavulanate, 875 mg two times daily for 10 to 14 days for adults (or 20 to 40 mg/kg divided into three doses for 10 to 14 days for children). Close follow-up, with a re-examination within a day at a primary care provider's office or with an ophthalmologist is important to assure response to treatment.

In more severe cases of pre-septal cellulitis, or with any concern of post-septal cellulitis, hospitalization with IV antibiotics is indicated to avoid complications, such as subperiosteal abscess, orbital abscess, meningitis, osteomyelitis, and cavernous sinus thrombosis. In children, the difficulty in localizing the spread of the infection dictates more aggressive management of any periorbital infection. An IV second- or third-generation cephalosporin, such as cefuroxime or ceftriaxone, is recommended. Other IV antibiotic options include ampicillin/sulbactam (Unasyn), or a combination of a first-generation cephalosporin with metronidazole.[47]

The Globe: Endophthalmitis

Clinical Features, Differential Diagnosis, and Diagnostic Testing

Endophthalmitis is an infection involving the globe itself. Pain and decreased vision are the primary symptoms. Examination findings include decreased visual acuity, chemosis, and hyperemia of the conjunctiva, intraocular inflammation (evidenced by hypopyon) (Fig. 61.21). The most common etiology of endophthalmitis is recent intraocular surgery. Other etiologies include previous perforated globe and endogenous infection. Early diagnosis and management is imperative for improved prognosis. The diagnosis is primarily clinical, and will typically have to be done in consultation with an ophthalmologist, because endophthalmitis can be difficult to distinguish from a uveitis, and the two have vastly different treatments and acuity.[49] An ultrasound of the eye (done in much the same way as to evaluate for retinal detachment) can be used to augment the evaluation; and with endophthalmitis, it may reveal numerous stands and membranes in a vitreous that would otherwise be uniformly hypoechoic.[49]

Management and Disposition

Endophthalmitis is a medical emergency that must be promptly treated. Systemic antibiotics are not effective, and therefore—although IV antibiotics can be considered (their effect is unknown)—intravitreal antibiotics must always be given.[50] The evaluation and treatment should be done in consultation with an ophthalmologist who can administer the intravitreal antibiotics at the bedside and perform a vitrectomy (removal of infected vitreous akin to draining an abscess) in the operating room if

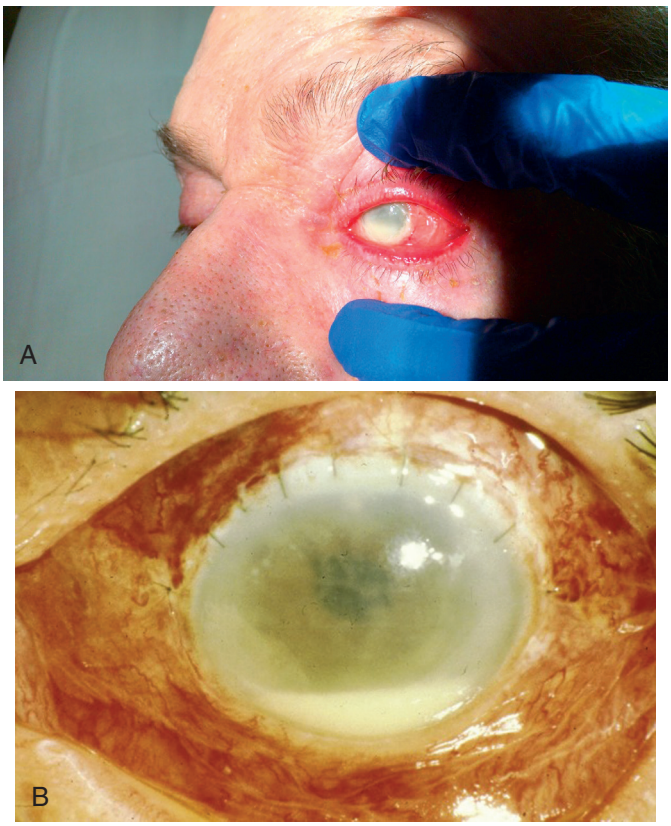


Fig. 61.21. Eye with endophthalmitis, illustrating a hypopyon (pus in the anterior chamber). (Courtesy Kama Guluma, MD, University of California San Diego.)

needed. The typical bacterial pathogen varies with the likely cause; coagulase-negative staphylococci are most common in post-cataract endophthalmitis, *Bacillus cereus* is a major cause of post-traumatic endophthalmitis, and *S. aureus* and streptococci are important causes of endogenous endophthalmitis associated with endocarditis.[50]

ACUTE ANGLE-CLOSURE GLAUCOMA

Principles

Aqueous humor provides structural support to the eye and delivers oxygen and nutrients to the avascular lens and cornea. It is produced by the ciliary processes, passes from the posterior chamber to the anterior chamber through the pupillary aperture, and then is transported into the trabecular meshwork located at the anterior chamber angle formed by the junction of the root of the iris and the peripheral cornea. This trabecular meshwork serves as a one-way valve and filter for the aqueous humor into the canal of Schlemm, which in turn drains into episcleral veins. IOP is determined by the rate of aqueous humor production relative to its outflow and removal, and it is normally between 10 to 20 mm Hg.

Clinical Features, Differential Diagnosis, and Diagnostic Testing

Glaucoma is an acquired chronic optic neuropathy. It is characterized by an enlarged ratio of the diameter of the optic cup to the diameter of the optic disc (termed *cupping*) and visual field loss. Glaucoma usually but not always is associated with elevated IOP. The two most common and important forms of glaucoma are primary open-angle glaucoma and acute angle-closure glaucoma.

Primary open-angle glaucoma is a chronic condition characterized by asymptomatic elevated IOP (but IOP may not always be elevated), and an enlarged ratio of the diameter of the optic cup to the diameter of the optic disc (termed *cupping*) and peripheral visual field loss. Patients may be on chronic topical ophthalmic medications designed to improve aqueous outflow. It is not typically a cause for an urgent visit to the ED (and therefore not discussed further), although it can lead to complete blindness over time.

Acute angle-closure glaucoma is the entity that typically precipitates an acute ED visit, at times in a patient with no prior knowledge or history of chronic glaucoma. A variety of rare conditions (such as, tumors, neovascular processes) can predispose a patient to this, but the more common predisposed patient has an anatomically shallow anterior chamber that further narrows with aging as the lens enlarges. Acute symptoms are often precipitated by pupillary dilation from being in a low-light environment (eg, movie theater) or taking an anticholinergic or sympathomimetic medication. This transient contraction of the iris crowds the angle ("pupillary block"), and continued formation of aqueous leads to an increased IOP, causing the iris to bulge forward, further inhibiting outflow, and eventually compromising arterial flow into the eye. The patient with acute angle-closure glaucoma typically presents with severe unilateral eye pain, redness, and blurred vision with "halos," as well as nausea and vomiting. On examination, the pupil may be moderately dilated and unreactive to light, the anterior chamber shallow when illuminated from the side with a penlight, the conjunctiva injected, and the cornea cloudy (steamy) (Fig. 61.22). The IOP is significantly elevated, and ischemia to intraocular structures, particularly the optic nerve, retinal nerve fiber layer, and the avascular anterior portion of the lens (which is sustained by aqueous humor) may occur. Sustained elevation in IOP can cause permanent corneal and optic nerve damage, and cause the peripheral iris to adhere to the trabecular meshwork, forming anterior synechiae and an irreversible occlusion that only can be corrected by surgery.

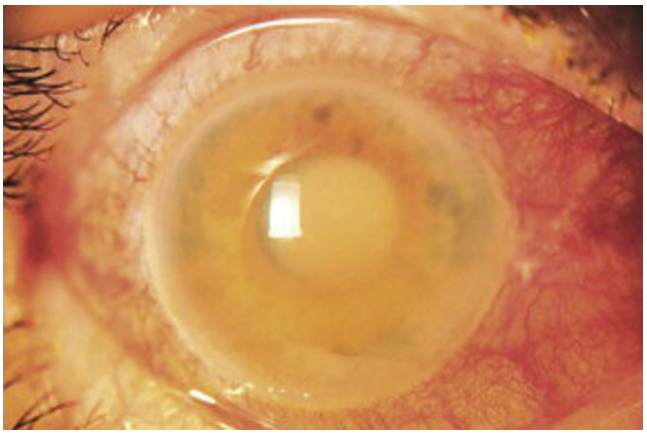


Fig. 61.22. Acute angle-closure glaucoma. (From Yanoff M, Duker JS, editors: Ophthalmology, ed 3, Philadelphia, 2008, Mosby.)

Management and Disposition

Treatment of acute angle-closure glaucoma begins with medications used to lower the IOP and then proceeds to definitive treatment of the anatomical abnormality that led to the elevated pressure in the first place.[51] Emergent ophthalmology consultation is necessary, and the treatment paradigm in the ED is as follows:

- Drugs that may be used to reduce production of aqueous humor:
  - Prostoglandins (latanoprost 0.005%—1 gtt)
  - Topical beta-blocker (timolol 0.5%—1 to 2 gtt)
  - Carbonic anhydrase inhibitor (acetazolamide 500 mg IV or orally)
  - Systemic osmotic agent (mannitol 1 to 2 g/kg IV over 45 minutes, to minimize cerebral effects, and typically reserved if topical medications and acetazolamide do not work within 1 hour)
- Drugs that may be used to increase outflow:
  - Topical alpha-agonist (phenylephrine 1 gtt)
  - Miotics (pilocarpine 1% to 2%)
  - Topical steroid (prednisolone acetate 1%—1 gtt every 15 to 30 minutes four times, then every hour)
- Definitive treatment—laser peripheral iridotomy within 24 to 48 hours

PRIMARY DISORDERS OF VISION

Principles

The process of visual perception is an orchestration of light refraction by the cornea and lens, signal transduction by the retina to generate electrical impulses, and transmission of those impulses through the optic nerve to be processed in each occipital cortex, being split and crossed at the chiasm along the way (Fig. 61.23). Primary disorders of vision can be caused by a derangement in any component in this process and may present as blurred vision, a focal disturbance somewhere in the visual field (in the form of dark objects or floaters, flashing lights (photopsia), or a visual field cut), or frank vision loss. Double vision has a very distinct presentation and is comprehensively addressed in Chapter 18.

The history enables a tailored approach to the evaluation of a patient with an atraumatic visual disturbance but may be fraught with challenges. One is the potential for a patient to use the term *blurred vision* to actually describe double vision, and vice versa. Another is that the patient with a cortical visual field cut may not

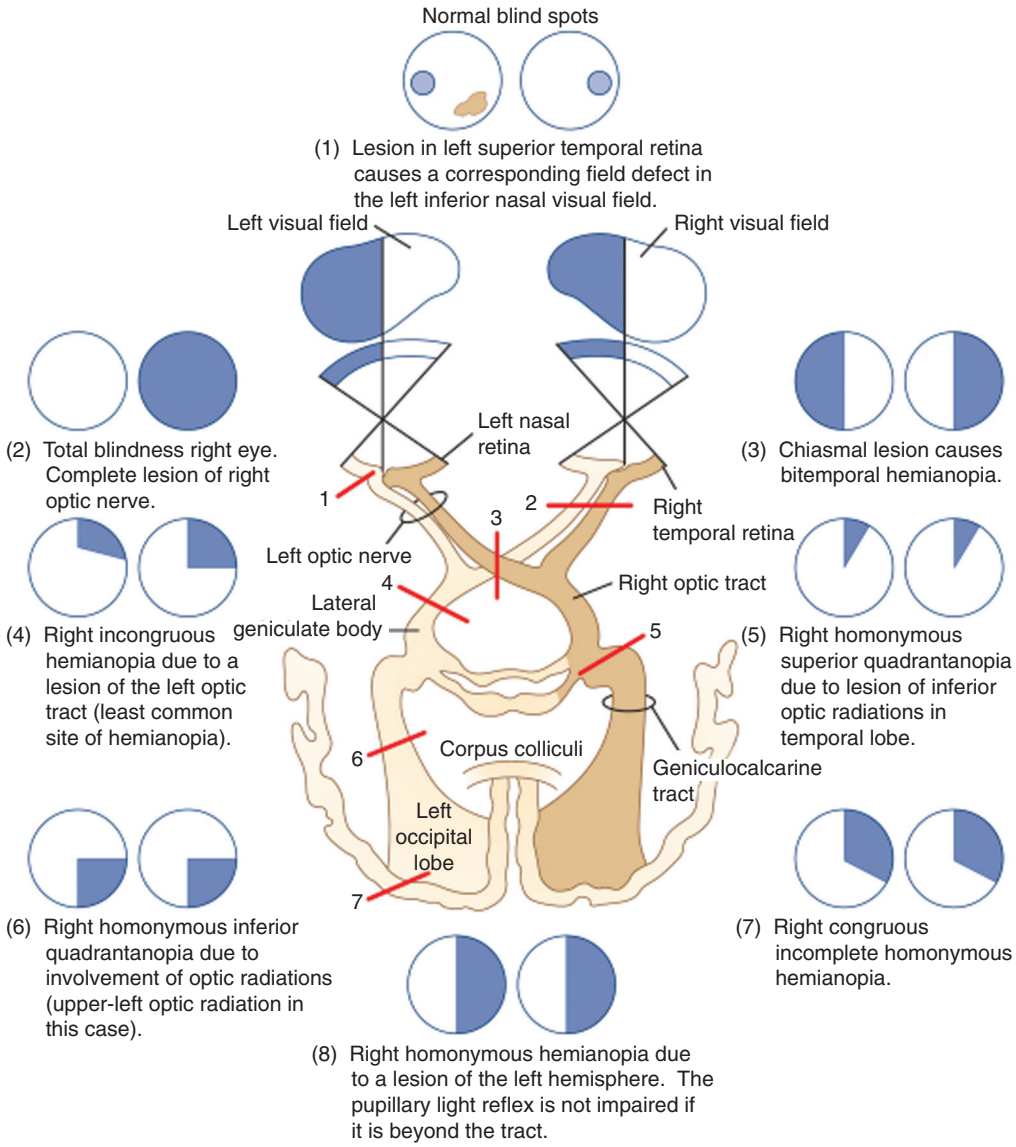


Fig. 61.23. Topographic diagnosis of visual field defects. (From Bradley WG, Duroff RB, Fenichel GM, et al: Neurology in clinical practice, ed 5, Oxford, 2007, Butterworth-Heinemann.)

even be aware there is a visual field deficit, being "blind to the blind spot." In addition, a patient may not notice a chronic problem in one eye until a problem finally develops in the remaining functioning eye. Finally, a patient may report what was actually a binocular defect of both visual fields on one side as a monocular issue involving a single eye on that side (a discrimination of which will be lost if the visual disturbance was transient and the patient did not check if the problem persisted with one eye or the other closed).

The elements of a screening ED ophthalmological examination are described in detail in Chapter 19 and should carefully incorporate a visual field and a neurological examination, in addition to the elements of visual acuity, pupillary examination, extraocular muscle movement, and examinations of the anterior and posterior segment (with a slit-lamp examination and a funduscopic examination). It should be kept in mind that a patient with a visual field defect or a retinal detachment may have a normal visual acuity, due to sparing of the macula.

An etiology of the visual loss occurring beyond the retina can be considered *neuro-ophthalmologic* and can be further divided into prechiasmal, chiasmal, and post-chiasmal (see Fig. 61.23). Patients with prechiasmal visual loss have monocular decreased visual acuity or visual field loss, typically from dysfunction of the optic nerve on that side, with an APD on the side involved on swinging flashlight test (see Fig. 61.11), and a visual field defect that does not respect the vertical midline and is often localized to the center of the visual field. Patients with chiasmal and post-chiasmal visual loss will typically have preserved visual acuity, and a visual field loss in both eyes that respects the midline (see Fig. 61.23).

Blurred Vision: Optic Neuritis, Toxic and Metabolic Disturbances, and Papilledema

Clinical Features and Differential Diagnosis

Any disturbance in the refraction of light may cause the symptom of blurred vision. Considerations include corneal infiltrates (from infections), significant pupillary dilation (which results in an increase in the scattered of light rays reaching the lens), and changes in the refractive properties of the lens or vitreous (due to edema from rapid osmotic changes). Blurred vision may also result from transductive dysfunction from retinal or optic nerve inflammation or edema. Considerations in the differential diagnosis of blurred vision include optic neuritis (which is usually

monocular) and toxic and metabolic visual disturbances (usually binocular), and papilledema from raised intracranial pressure (also usually binocular).

Optic neuritis is a primary, autoimmune inflammatory process of the optic nerve, affecting mostly young patients (range, 15 to 45 years old), has an association with multiple sclerosis, and is the presenting symptom of multiple sclerosis in 25% of cases.[52] The patient with optic neuritis typically presents with monocular blurring or fogginess of vision evolving over hours or days, mild pain with movement of the involved eye if the lesion is within the orbit, and at times bright, fleeting flashes of light with eye movement, as well as worsening of vision with small increases in body temperature (from exercise, hot baths, or hot weather).[52] The natural history of optic neuritis is for visual acuity to reach its poorest within 1 week and then slowly improve over the next several weeks. Approximately 30% of patients with acute optic neuritis develop multiple sclerosis within 5 years. Approximately 30% of patients with optic neuritis have a recurrence within 10 years of initial presentation.

With regards to toxic visual disturbances, perhaps the most characterized toxidrome presenting with acute visual change is methanol toxicity.[53] Orally ingested methanol (the toxicity of which is described in entries dedicated to it elsewhere in this text) is metabolized to formic acid, which accumulates in the optic nerve and leads to edema and compromised axoplasmic flow; in addition, it leads to widespread electrophysiological dysfunction that also affects photoreceptors in the retina, leading to visual loss. Other potential causes of a toxic visual disturbance are barbiturates, chloramphenicol, emetine, ethambutol, ethylene glycol, isoniazid, and heavy metals.

In terms of metabolic visual disturbances, any rapid osmolar shift in the cornea, lens, or even retina has the potential to cause visual changes. A representative scenario is acute hyperglycemia. A rapid elevation in blood glucose (or a rapid correction of severe hyperglycemia), as seen in poorly controlled diabetics, may cause an acute hyperopia (far-sightedness), presumably due to changes in refraction in the lens.[54] It may alternatively cause acute myopia when the rise in intracellular glucose levels in the lens overwhelms the normal glucose metabolic pathway such that it is converted to less absorbable sorbitol and fructose, generating an acute hyperosmolar state and stromal swelling. This may be followed by acute bilateral cataract formation within a matter of hours to days. Metabolic visual disturbances can also be from a nutrition-related optic neuropathy from causes such as thiamine deficiency and pernicious anemia.

Papilledema may be seen on examination and refers to the changes in the optic disc from increased intracranial pressure. The subarachnoid space of the brain is continuous with the optic nerve sheath. Any increase in the cerebrospinal fluid pressure (such as, from pseudotumor cerebri syndrome [otherwise known as *idiopathic intracranial hypertension*], cryptococcal meningitis in HIV/AIDS patients, or hydrocephalus or intracranial mass) can be transmitted to the optic nerve, resulting in swelling of the optic nerve head. Although visual symptoms may be isolated on rare occasion, patients with these entities will typically present with headache, which will provoke their consideration. That being said, a small percentage of patients with pseudotumor cerebri present with isolated subjective visual loss, blurred vision, or enlargement of the physiologic blind spot as the initial presenting symptom of the disease, and rapid deterioration may occur over days in severe cases.[55] Swelling of the optic disc and blurring of the disc margins, hyperemia, and loss of physiologic cupping are present (Fig. 61.24A). There may be obliteration of spontaneous venous pulsations. Flame-shaped hemorrhages and yellow exudates may appear near the disc margins as the edema progresses (see Fig. 61.24B). Visual acuity may be affected as the swelling becomes severe. Papilledema is typically bilateral but may be asymmetrical. There are conditions with optic nerve swelling (such as, ischemic optic neuropathy [ION], optic disk vasculitis, and diabetic papillitis) that may mimic papilledema.

Diagnostic Testing, Management, and Disposition

In the diagnostic evaluation of blurred vision, a standard ophthalmological assessment including visual acuity and a slit-lamp examination is important, but funduscopy (so as to screen for retinal or optic nerve edema or pathology) and an assessment of visual fields (to screen for associated sectoral abnormalities in retinal or optic nerve transduction) are critical.

Optic Neuritis. With optic neuritis, visual acuity will usually be abnormal (with the primary complaint of blurred vision), and the patient may have variable visual field defects (central, altitudinal, arcuate, hemianopic), with central defects being more common than peripheral ones.[52] An APD is usually present, and direct ophthalmoscopic examination reveals a normal or swollen disk (Fig. 61.25). An orbital MRI of the optic nerves with gadolinium is the mainstay of diagnosis, revealing optic nerve lesions in 95% of cases. A lumbar puncture can be done, which may show CSF pleocytosis and a raised protein concentration.[52] For treatment, steroids for optic neuritis has a long track record of investigation but with no demonstrated long-term effect on visual outcome.[55a] Some still recommend high-dose methylprednisolone (either 500 mg per day orally for 5 days or 1 g per day IV for 3 days), due to mild short-term benefits; plasmapheresis and IV immunoglobulins are also options.[52]

Toxic and Metabolic Visual Disturbances. A key component in the diagnostic approach to blurred vision from toxic and metabolic disturbances is recognizing the existence of a cause. These processes are bilateral, progressive, and symmetrical and may manifest with a drop in visual acuity, evident haziness in the lenses, or retinal edema on funduscopy. Visual loss can be severe and visual field testing reveals central defects. In each case, the treatment is aimed at the underlying toxin, metabolite, or deficiency involved (described in entries dedicated to them elsewhere in this text). Blurred vision due to hyperglycemia typically reverses when hyperglycemia is treated, although cataracts may sometimes be permanent.

Papilledema. The diagnostic evaluation and management of specific disease processes that result in bilateral papilledema can be found in entries specifically dedicated to them elsewhere in this text. An important part of the assessment is a funduscopic eye examination, with an assessment of the optic disc. Early or mild papilledema may be difficult to detect with the direct ophthalmoscope, and if the suspicion of such a process is high, consultation with ophthalmologist in the ED for stereoscopic viewing of the optic discs with indirect ophthalmoscopy is recommended, and patients should undergo neuroimaging (either with MRI or contrast-enhanced CT).[55]

Visual Field Disturbances: Floaters, Flashes, and Field Deficits

Clinical Features and Differential Diagnosis

Visual field disturbances may take the form of floaters (seeing objects in the field of vision, caused by material obstructing the light path), photopsia (flashing lights, caused by aberrant stimulation of the retina), or field deficits (focal areas of visual loss, caused by dysfunction in the transport or processing of impulses sent by the retina). Photopsia may be unilateral or bilateral, depending on the cause. The most common causes of unilateral

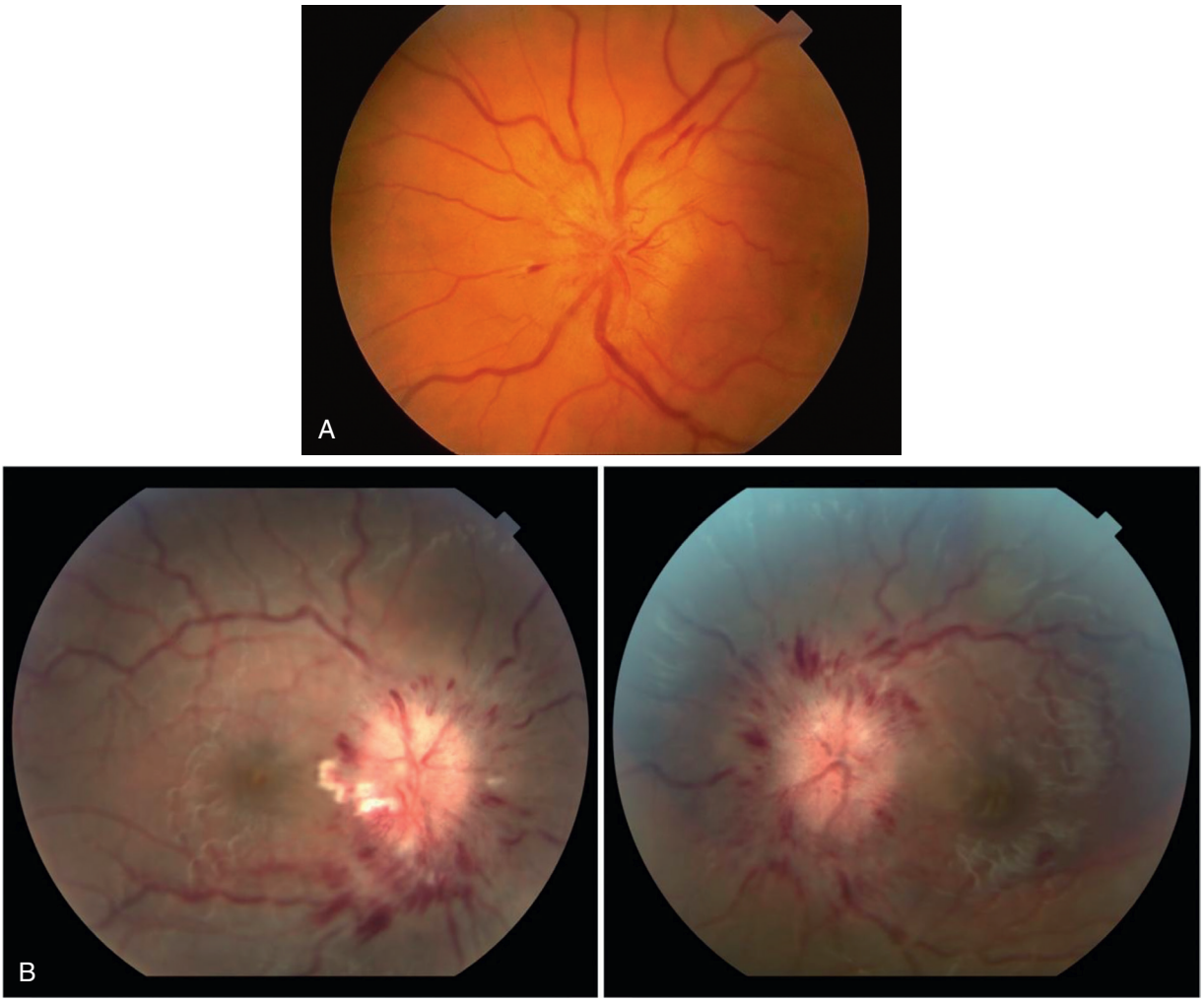


Fig. 61.24. A, Papilledema. Note the blurred disk margins. **B,** Papilledema. Note the blurred disk margins, exudates, and hemorrhages.

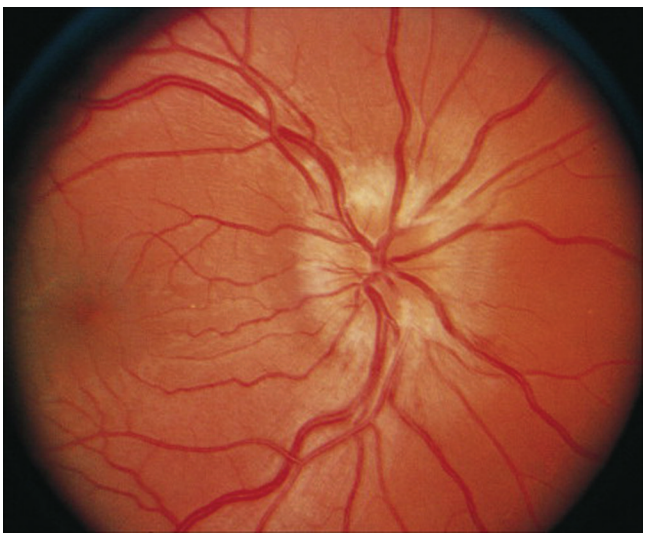


Fig. 61.25. Optic disk swelling (papillitis) associated with acute optic neuritis. (From Yanoff M, Duker JS, editors: Ophthalmology, ed 3, Philadelphia, 2008, Mosby.)

photopsia are vitreous or retinal detachment (see later), with which abnormal mechanical stimulation of the retinal photoreceptors leads to a cascade of action potentials that the visual system interprets as flashes of light. A less common cause of unilateral photopsia is uveitis involving the choroid. The most common cause of bilateral (and homonymous) photopsia is migraines, although scintillating scotomata is much more frequent. Less common causes of bilateral homonymous photopsia include lesions of the visual cortex with release hallucinations or epileptic seizures.

Considerations in the differential diagnosis of these visual field disturbances include intraocular (monocular) entities such as vitreous hemorrhage, vitreous and retinal detachment, and extraocular (binocular) entities at the optic chiasm and beyond.

Vitreous hemorrhage results from bleeding into the pre-retinal space or into the vitreous cavity. The most common causes are diabetic retinopathy and retinal tears. Additional causes include neovascularization associated with branch vein occlusion, sickle cell disease, retinal detachment, posterior vitreous detachment, trauma, age-related macular degeneration, retinal artery microaneurysms, trauma, and intraocular tumor. Symptoms begin with dark floaters or "cobwebs" in the vision and may progress over a few hours to painless visual loss. Floaters, described by the patient as dark or black dots or strands moving in the visual field in the direction of the preceding eye movement, are caused by vitreous blood.

Vitreous detachment is a common occurrence in patients older than 60 years old. With aging, the vitreous gel desiccates, shrinks, and pulls away from the retina, leading to symptoms similar to those of vitreous hemorrhage and retinal detachment.

Retinal detachment can occur by three mechanisms: (1) rhegmatogenous, (2) exudative, and (3) tractional. The retina has two layers—the inner neuronal retina layer and the outer retinal pigment epithelial layer—that can be separated by fluid accumulation. A retinal tear in the retinal membranes may or may not lead to a retinal detachment. A rhegmatogenous retinal detachment occurs as a result of a tear in the neuronal layer, allowing fluid from the vitreous cavity to leak between and separate the two retinal layers. It occurs in patients older than 45 years old, is more common in men, and is associated with degenerative myopia. Trauma can cause this type of detachment at any age, with patients with severe myopia being at greater risk. An exudative retinal detachment occurs as a result of fluid or blood leakage from vessels within the retina and is associated with hypertension, pre-eclampsia, central retinal venous occlusion, glomerulonephritis, papilledema, vasculitis, and choroidal tumor. Finally, a tractional retinal detachment is a consequence of contraction of a fibrous band that has formed in the vitreous. With retinal detachment, patients typically note flashes of light related to the traction on the retina, floaters related to vitreal blood or pigmented debris, and visual loss. The visual loss is commonly described as filmy, cloudy, or curtain-like in appearance, and is painless.

If a visual field disturbance is binocular, then a chiasmal or cortical disorder should be considered. Chiasmal disease is most commonly caused by chiasmal compression from pituitary tumors, craniopharyngiomas, or meningiomas. Visual loss is gradual and progressive. Beyond the optic chiasm, the most common causes of visual disturbances are infarctions, tumors, arteriovenous malformations, and migraine disorders. Patients report difficulty in performing a certain task, such as reading. Lesions can be located from the immediate post-chiasmal optic tract to the occipital cortex.

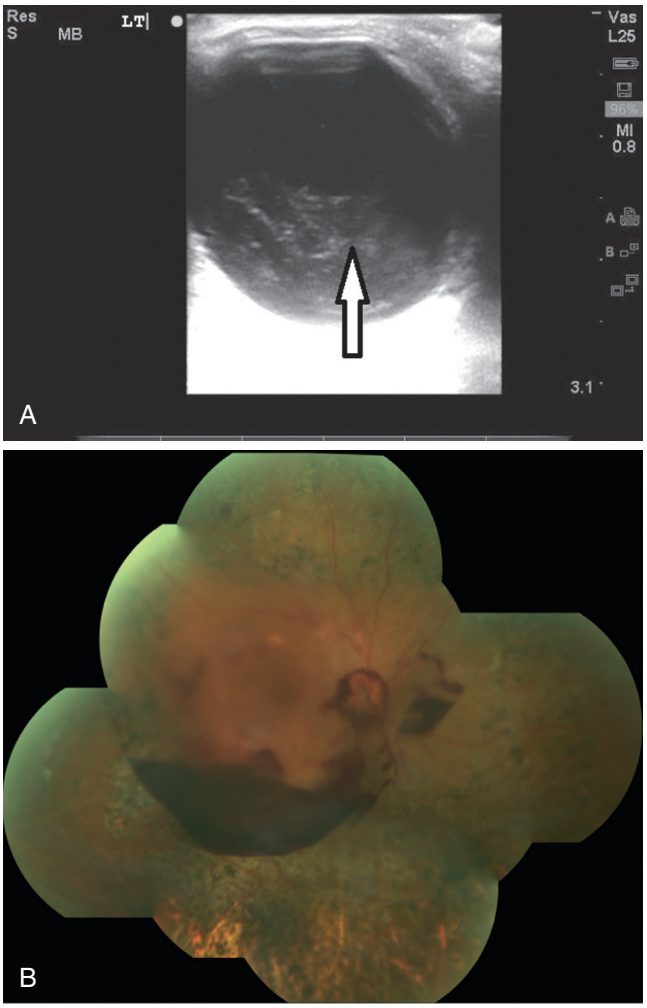


Fig. 61.26. **A,** Ocular ultrasound showing vitreous hemorrhage *(white arrow).* **B,** Boat shaped pre-retinal vitreous hemorrhage (**A,** Courtesy Douglas Brunette, MD. **B,** Courtesy Jeffrey Lee, MD.)

Diagnostic Testing, Management, and Disposition

In the diagnostic evaluation of visual field disturbances, the history should be specific enough to ascertain if the problem is an issue of an absence of vision (ie, a "blind spot," or visual field deficit, as seen in chiasmic or cortical etiologies), or of an obstruction of vision (ie, "floater," as seen in vitreous detachment or hemorrhage, or retinal detachment). In addition, a visual field examination should be detailed enough to determine if the disturbance is monocular or binocular and whether it respects the midline. Funduscopy is especially important to enable an assessment of the vitreous and retina, and ocular ultrasound is a helpful adjunct. With this approach, the considerations outlined earlier can be differentiated and addressed.

Vitreous Hemorrhage and Detachment. With a vitreous hemorrhage, direct ophthalmoscopy reveals a reddish haze in mild cases and a black reflex in severe cases. Details of the fundus are usually difficult to visualize. There is a diminished red reflex and an inability to visualize the fundus clearly with the direct ophthalmoscope. Ocular ultrasound, which will reveal echogenic debris in the vitreous, can be an effective diagnostic screening tool (Fig. 61.26A). A vitreous hemorrhage or detachment usually does not cause an APD by itself, and if an APD is present, an occult retinal detachment may be present. A hemorrhage may be evenly distributed throughout the vitreous, or—if trapped in the subhyaloid space as a pre-retinal hemorrhage—may be focal, with a boat shape (see Fig. 61.26B).

Ophthalmologic consultation in the ED, or a same-day evaluation by an ophthalmologist, will typically be needed to characterize the extent and complications of any suspected vitreous hemorrhage or detachment and manage vision-threatening complications. The management of a vitreous hemorrhage is otherwise largely expectant, with limitation of activity, avoidance of anticoagulants, and sleeping with the head of bed elevated to allow blood to settle and optimize visualization of the retina on subsequent examinations. Surgery is typically required if there is an associated retinal detachment. The same consideration applies for a posterior vitreous detachment, for which no specific emergent treatment is indicated unless accompanied by a retinal tear, vitreous hemorrhage, or retinal detachment.

Retinal Detachment. With a retinal detachment, visual acuity can range from minimally changed to severely decreased. Visual field deficits relate to the location of the retinal detachment, and an APD occurs if the detachment is large enough. When the detachment is visible on ophthalmoscopy, the retina appears out of focus at the site of the detachment. In large retinal detachments with large fluid accumulation, a bullous detachment with retinal folds can be seen (Fig. 61.27A). Retinal detachment cannot be ruled out by direct funduscopy. Indirect ophthalmoscopy is needed to visualize the more anterior portions of the retina. Bedside ED ultrasonography can be a useful tool in screening for a retinal detachment (see Fig. 61.27B).[56] It will reveal a billowing hyperechoic line that may undulate with side-to-side movements of the eye.

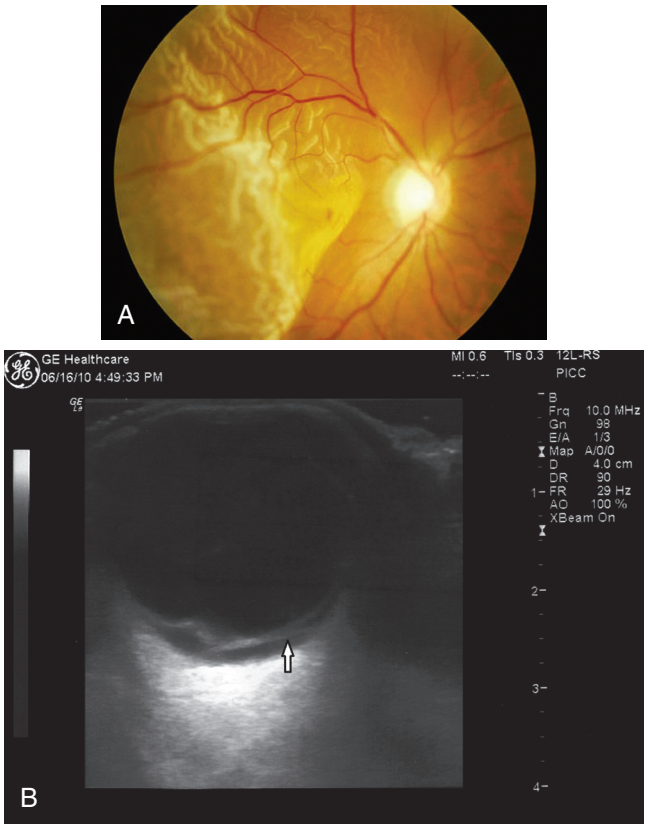


Fig. 61.27. A, Retinal detachment. Note large portion of retina billowing forward. **B,** Bedside emergency department (ED) ultrasound showing retinal detachment *(white arrow)*. (**A,** Courtesy www.tedmontgomery.com. **B,** Courtesy Nicholas Connors, MD, and Sophia Lin, MD, New York Presbyterian-Weill Cornell Medical Center.)

Any patient suspected of having a retinal tear or detachment requires immediate ophthalmologic consultation, because treatment with tamponade or retinopexy can prevent a retinal detachment that does not involve the macula (a "macula-on" retinal detachment) from progressing to involve the macula ("macula-off") and significantly degrade visual acuity. The duration of macular detachment, measured from the reported time of the loss of central visual acuity, is inversely related to final visual acuity. Even though the literature suggests that there is almost a day's-worth of leeway in the timing of repair of a "macula-on" detachment, and a fair amount of visual acuity is recoverable if a "macula-off" detachment is repaired early enough, a "macula-on" detachment that is close to the macula is at risk of converting to "macula-off" with even a few hours delay.[56-59]

Chiasmal and Cortical Disturbances. Although formal visual field testing may be necessary to stage the condition, the diagnosis of a chiasmic or cortical etiology to a visual field disturbance can usually be made by confrontation visual field testing. The classic defect for a lesion in or compression of the optic chiasm (chiasmal) is a bitemporal hemianopsia; however, tumors often compress the chiasm and optic nerves asymmetrically, resulting in combined central and temporal defects. When a visual field defect respects the vertical midline, the lesion is out of the globe and likely either chiasmal or post-chiasmal (see Fig. 61.23).

The classic visual field defect in post-chiasmal (cerebral or cortical) disease is a homonymous hemianopsia, a visual field loss on the same side of both eyes (see Fig. 61.23). Patients with such lesions have a focal neurologic deficit and need to be evaluated and treated based on the primary neurological diagnostic considerations, which include occipital infarction, neoplasm, an inflammatory process, or an infectious process (such as, encephalitis).

Sudden Vision Loss: Retinal Artery and Vein Occlusion, and Ischemic Optic Neuropathy

Clinical Features and Differential Diagnosis

Sudden onset of atraumatic, vision loss is usually due to a vascular process, such as infarction (although nonvascular processes, such as from retinal detachments and hemorrhages affecting the macula, are possible). Binocular processes include a sudden homonymous hemianopia from an infarction of the visual pathways in the temporal, parietal, or occipital lobes; and sudden total blindness in both eyes due to a basilar artery territory infarction of both occipital lobes. Central nervous system (CNS) processes (such as, ischemic stroke) that underlie these binocular events are discussed in entries specific to them elsewhere in this text. This section is, therefore, dedicated to sudden onset of painless monocular vision loss, which is ophthalmological and is usually due to a vascular process, such as infarction in either the retina or the optic nerve; the differential diagnosis primarily includes central retinal artery occlusion (CRAO), central retinal vein occlusion (CRVO), and ischemic optic neuropathy (ION). These typically present with sudden vision loss that is painless, severe, and develops over seconds, and may be permanent, or transient (amaurosis fugax).

In a CRAO, acute retinal ischemia develops from a sudden embolic, thrombotic, vasculitic or vasospastic occlusion of a branch of the retinal artery (a branch retinal artery occlusion [BRAO]) or the central retinal artery itself (a CRAO). A CRAO may be (1) non-arteritic and permanent (over two-thirds of all CRAO cases, due to platelet fibrin thrombi and emboli from atherosclerotic disease), (2) non-arteritic and transient (with transient monocular blindness, a transient ischemic attack [TIA] of the retina, related to transient vasospasm due to serotonin release from platelets on atherosclerotic plaques), or (3) arteritic (due to temporal arteritis and rare).[60] It generally has a poor visual prognosis with spontaneous resolution occurring in 1% to 8% of cases. Most commonly occurring in patients 50 to 70 years old, CRAO risk factors include hypertension, carotid artery disease, cardiac disease, diabetes, collagen vascular disease, vasculitis, cardiac valvular abnormality, and sickle cell disease. Patients with increased orbital pressure from acute glaucoma, retrobulbar hemorrhage, and endocrine exophthalmos are also at risk.

A CRVO leads to congestion of venous blood and fluid in the intraretinal space that may lead to secondary retinal ischemia. It is typically characterized as either non-ischemic or ischemic; a non-ischemic CRVO is associated with dilatation of retinal vessels and edema only, whereas an ischemic CRVO presents with the sudden onset of painless vision loss in one eye. Predisposing factors include hypertension, hyperlipidemia, diabetes mellitus, vasculitides, hyperviscosity, and smoking.

ION falls into two primary types, anterior ischemic optic neuropathy (AION; involving the optic nerve head) and posterior ischemic optic neuropathy (PION; involving the rest of the optic nerve). AION can further be divided into arteritic anterior ischemic optic neuropathy (A-AION; due to temporal arteritis) and—more commonly—non-arteritic anterior ischemic optic neuropathy (NA-AION; due to noninflammatory causes).[61]

Patients with A-AION may have concurrent symptoms of temporal arteritis (giant cell arteritis), such as weight loss, malaise, jaw pain, headache, scalp tenderness, polymyalgia rheumatica, and low-grade fever; in up to 25%, however, the acute vision loss is the only symptom.[60] Vision loss can be preceded by episodes of amaurosis fugax. Untreated it may progress to involve both eyes. Temporal arteritis is extremely rare in people younger than 50

years old, and the incidence rises with each subsequent decade. Vision loss has been shown to be unilateral in 46%, sequential in 37%, and simultaneously bilateral in 17%.

Patients with the much more common NA-AION lack the classic symptoms of temporal arteritis and tend to be younger with systemic vascular disease, diabetes, or hypertension. This is an acute ischemic event affecting the anterior optic nerve that typically occurs in patients over the age of 50 (typically 60 to 70 years old), at times associated with precipitant anemia, hypovolemia, dehydration, systemic hypotension, or fluctuations in blood pressure (especially that associated dialysis).[61]

A sudden complete loss of vision due to a vascular cause can be transient, whereupon it is called *amaurosis fugax,* and can be a manifestation of any of the aforementioned processes. It has been found in 2% of CRAO, 14% of BRAO, 5% of CRVO, just over 3% in NA-AION, and in 32% of patients with temporal arteritis who have ocular involvement.[62] Amaurosis fugax may also implicate proximal cerebrovascular disease and be a form of transient ischemic attack.

Diagnostic Testing, Management, and Disposition

Central Retinal Artery Occlusion. With CRAO, the examination reveals a markedly reduced visual acuity with a prominent APD, and an edematous with a pale gray-white retina with a cherry-red spot representing the fovea seen on funduscopy (Fig. 61.28). Patients younger than 50 years old should have a hypercoagulability evaluation, whereas older patients at risk for temporal arteritis should have an evaluation appropriate for that consideration.[60]

A number of interventions geared toward dislodgement of the embolus (via direct digital pressure through closed eyelids for 10 to 15 seconds and followed by a sudden release), dilation of the artery to promote forward blood flow (by increasing intra-arterial carbon dioxide level [pCO_2] with an inhaled mixture of 95% oxygen/5% carbon dioxide [carbogen]), and reduction of IOP (such as, with glaucoma, even using anterior chamber paracentesis) to increase in perfusion gradient have been recommended, but there is little evidence to support the benefit of any of these treatments.[62a] Other options in include hyperbaric oxygen. Overall, the efficacy of the above therapies varies between 6% and 49%, with a mean visual improvement rate of 15% to 21%.[60]

A CRAO may be amenable to the use of thrombolytic agents, with the caveat that it is usually an atheromatous embolic event, and thrombolysis is designed to lyse the fibrinoplatelet occlusion in a non-arteritic CRAO.[59] Studies are heterogenous, using different agents, dosing regimens, and time-windows in largely retrospective case series with different findings, but it appears that intra-arterial thrombolytic therapy might be effective if given less than 6 hours from onset.[60,63] IV thrombolysis might be effective if given less than 4.5 hours from onset, with a post-thrombolysis major hemorrhage rate significantly lower than that seen with ischemic stroke (none documented with tissue-plasminogen activator or urokinase).[64] Until a large randomized controlled trial of the safety and efficacy of thrombolysis for CRAO is performed, management should be tailored to individual patient circumstances in consultation with an ophthalmologist.

Central Retinal Vein Occlusion. A CRVO is differentiated from CRAO based on findings on funduscopic examination. Appearance can vary but classically includes dilated and tortuous veins, retinal hemorrhages, and disk edema (Fig. 61.29). Branch retinal vein occlusion is an incomplete CRVO and carries about a better prognosis. Neovascular glaucoma and macular edema are the major complications of ischemic CRVO.

Over 80% of patients with a non-ischemic CRVO will have an ultimate visual acuity that is better than 20/200, whereas less than 10% of patients with ischemic CRVO will have an ultimate visual acuity better than 20/200. Treatment of CRVO includes treating the underlying etiology and monitoring for potential sequelae. Ophthalmology should be consulted in the ED to secure timely initiation of therapy, which largely centers around treating the macular edema associated with the occlusion. Treatment involves anti-vascular endothelial growth factor (anti-VEGF) pharmacotherapies, intravitreal corticosteroid injection with a dexamethasone intravitreal implant or triamcinolone, as well as retinal photocoagulation, normalization of IOP, and cyclocryotherapy.[65] The use of antithrombotic therapy, in particular the use of low–molecular-weight heparin, has also shown recent promise.[65a] Underlying medical disease should be managed;

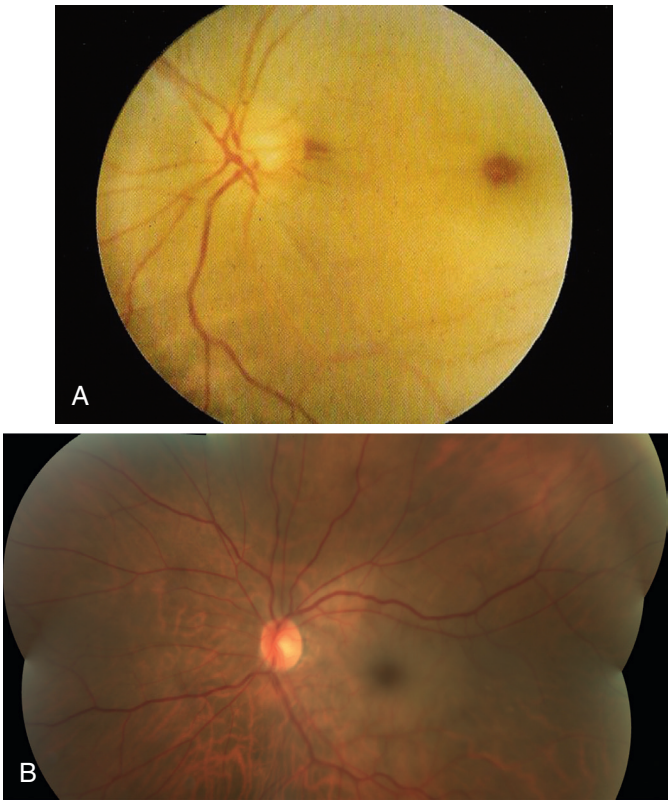


Fig. 61.28. Central retinal artery occlusion (CRAO). **A,** Note the cherry-red spot representing the fovea. **B,** Note whitening of the retina, with a less prominent cherry red spot. (**B,** Courtesy Jeffrey Lee, MD, University of California San Diego.)

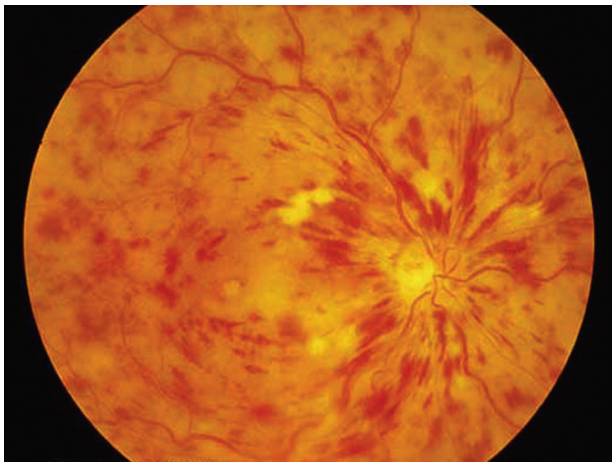


Fig. 61.29. Central retinal vein occlusion (CRVO). Note the "blood and thunder" appearance. (Courtesy www.tedmontgomery.com.)

the prognosis depends on the degree of obstruction and resultant complications.

Ischemic Optic Neuropathy. Examination findings are similar in A-AION and NA-AION, and include a large APD, visual loss, and a visual field defect that may respect the horizontal (as opposed to vertical) midline, with a pale and swollen optic disc on funduscopy.

The diagnosis of a temporal arteritis underlying an A-AION is outlined in entries specific to it elsewhere in this text, and it may include an erythrocyte sedimentation rate (ESR). Patients with NA-AION, on the other hand, do not have an elevated ESR, and an MRI may reveal abnormalities to the optic nerve head.

Temporal arteritis with evolving vision loss or amaurosis fugax from A-AION—as opposed to just headache alone—represents a distinct clinical emergency. Untreated, vision loss becomes bilateral in days to weeks in at least 50% of cases.[61] Patients should therefore be admitted for high-dose IV methylprednisolone (typically 500 mg to 1 g daily for 3 days) before transition to oral medications.[66] Patients treated with high-dose IV methylprednisolone are more likely to have visual improvement (a 34% chance of improvement) and are less likely to develop fellow eye involvement than those receiving oral prednisone.

The visual loss in NA-AION is less severe than with temporal arteritis, and improvement occurs in one-third of patients. There is no known treatment (intravitreal and systemic steroids have been tried without success, as have anti-VEGFs). Emergent ophthalmological consultation in the ED is warranted for any apparent ION to aid with differentiation of the type and extent of the process and management.

Functional Vision Loss

Clinical Features, Differential Diagnosis, Diagnostic Testing, Management, and Disposition

Functional (or factitious) vision loss may be a hysterical conversion reaction (a non-deliberate, imagined visual loss in a patient with a relatively flat affect) or malingering (a vision loss for secondary gain in a patient who somewhat dramatically demonstrates blindness). Although the evaluation may require collaborative consultation with an ophthalmologist, neurologist, and a psychiatrist, some tests can be performed in the ED that will suggest a functional overlay, given that the most common presentation of functional vision loss is a decreased visual acuity.[67,68] They include (1) rotating an optokinetic drum or rocking a mirror slowly back and forth in front of the patient (which will induce nystagmus or eye movements in the functional patient, but not in the truly blind patient), (2) rapidly moving the examiners hand toward the eye in question (which will induce a blink to a visual threat in the functional patient, but not in the truly blind patient), (3) checking for an APD as in Figure 61.11 (which will be absent in the functional patient but not in the truly blind patient with an optic nerve problem), (4) having the patient raise his or her arms and touch both index fingers together (which a functional patient will feign inability to do, but a truly blind patient will be able to do, given that the test is actually one of proprioception and not vision). The other presentation of functional visual loss is a defect in the visual field, typically with a central scotoma.[67,68] This can be identified as functional by having the patient sit in front of a picture (or grid) and describe the extent of a visual field defect vis-à-vis what is missing, and then moving him or her further away and asking for another description of what is missing. The functional patient may describe same missing elements in the picture (in an effort to convince the examiner that the defect is stable), whereas the patient will a real visual field deficit will notice that more elements in the picture (or grid) are missing.

DIPLOPIA

Chapter 18 provides a comprehensive overview of the approach to diplopia in the ED expounding on a methodological consideration of whether a binocular diplopia is due to an due to (1) a simple restrictive, mechanical orbitopathy from inflammatory or infectious mass-effect directly restricting of the movement of the eye, (2) a palsy of one or more of the oculomotor CNs, (3) a more proximal neuro-axial process involving the brainstem and related CNs, or (4) a systemic neuromuscular process.

ANISOCORIA

Principles

Dilation (mydriasis) of the pupil is controlled by the dilator muscle, innervated by sympathetics that exit spinal cord at the level of C8, T1, and T2, and then come back up under the subclavian artery and over apices of the lungs, enter the superior cervical ganglion, then the internal carotid plexus, and finally the ophthalmic division of CN V (the trigeminal nerve), whereupon they reach the eye through the superior orbital fissure. This sympathetic innervation serves a largely inhibitory role, facilitating pupillary dilation in darkness.

Constriction (or miosis) of the pupil is controlled by the pupillary sphincter muscle, innervated by parasympathetics that originate in the nuclei of CN III. This parasympathetic innervation is the primary means of regulating pupillary size in response to different intensities of light. Afferent input from the retina of each eye bifurcates to innervate the Edinger Westphal nuclei of each CN III, and each nucleus in turn provides efferent output to its pupillary constrictor muscle, underlying the direct and consensual pupillary light reflexes. Anisocoria, or a difference in pupillary size, can result from a process affecting the nuclei or the innervation pathways or from pharmacological interference at the neural endplates in the pupillary muscles.

Clinical Features and Differential Diagnosis

The differential diagnosis of anisocoria include an Adie's or Argyll Robertson pupil, pharmacologic mydriasis and miosis, a third-nerve palsy, Horner's syndrome, and a physiologic or headache-associated anisocoria.

Adie's and Argyll Robertson Pupils

An Adie's tonic pupil results from dysfunction or lesion of the ciliary ganglion or short ciliary nerves, and may be idiopathic (seen more frequently in women than men), for from local ocular or orbital damage from surgery, trauma, procedures, infection, inflammation, or ischemia. An Adie's tonic pupil may also be part of a condition causing systemic autonomic dysfunction, such as diabetes, dysautonomia, neurosyphilis, amyloidosis, or sarcoidosis. These patients present with a large pupil, sensitivity to light in that eye, and blurred vision when looking at things near them (but may be asymptomatic, with the pupil noticed incidentally). The Argyll Robertson pupil is typically smaller than an Adie's and similarly constricts poorly to direct light, but it briskly constricts when a target within reading distance is viewed. It is attributable to a dorsal midbrain lesion (such as from neurosyphilis) that interrupts the pupillary light reflex pathway but spares the more ventral pupillary near reflex pathway.

Pharmacologic Mydriasis and Miosis

Anisocoria can be caused by a variety of accidental medication and plant exposures. Parasympathomimetic miosis may be

induced by exposures to organophosphate esters, pilocarpine drops, or dust containing cholinesterase inhibitor from a dog's flea collar. Parasympatholytic mydriasis may be seen with anticholinergic medications (such as, transdermal scopolamine), aerosolized ipratropium administered through ventilator masks, cycloplegics (such as, homatropine, cyclopentolate, or tropicamide), and plants containing anticholinergic agents, such as Jimsonweed *(Datura stramonium)* and Angel's trumpet *(Datura suaveolens)*.[69-70] *Sympathomimetic mydriasis* may occur from sprays containing phenylephrine (Neo-Synephrine) and from apraclonidine (a glaucoma medication).

Third-Nerve Palsy

CN III innervates the medial, inferior, and superior recti muscles, the inferior oblique muscle, and the levator palpebrae superioris muscle, which lifts the upper eyelid. It also provides parasympathetic innervation to two intrinsic ocular muscles, the ciliary and constrictor pupillae muscles, which constrict the pupil. A CN III palsy, therefore, results an eye that appears deviated "down and out" with a dilated pupil and ptosis. The parasympathetic fibers that affect pupillomotor constriction are located peripherally and on the superomedial surface of CN III, where compression from an aneurysm or other source may cause pupillary involvement before other oculomotor signs, such as ptosis or diplopia, develop.

Horner's Syndrome

Horner's syndrome presents with ptosis, miosis, and facial anhidrosis resulting from a disruption of sympathetic innervation anywhere along the chain of sympathetic innervation.[71] The presence of associated symptoms may help localize etiology, as outlined in Table 61.2.[72] In children, the most common cause of acquired Horner's syndrome is a neuroblastoma of the paravertebral sympathetic chain, although it may be from a mediastinal tumor. Horner's syndrome can also be congenital, suggested by heterochromia or hypopigmentation of the ipsilateral iris.[71]

Physiologic and Headache-Associated Anisocoria

In physiological anisocoria, the difference in pupil size will typically be 1 mm or less. A more prominent transient mydriasis (benign episodic unilateral mydriasis) may occasionally accompany a migraine headache, either from sympathetic hyperactivity, or—with an ophthalmoplegic migraine—parasympathetic hypoactivity from CN III dysfunction.[73,74] A non-migrainous benign episodic unilateral mydriasis can occur without headache, ptosis, or ocular motility disorder, in episodes lasting minutes, hours, or even days and is also thought to be caused by over-activity of sympathetic innervation to the pupil. Patients are typically female, relatively young, and episodes last a median duration of 12 hours.

Patients can also present with a "tadpole pupil," in which the pupil becomes distorted and pulled in one direction like the tail of a tadpole, possibly occurring several times a day for several days and then resolving. This is likely the result of a sectoral spasm of the dilator muscle, thought to be benign, and has been associated with strenuous exercise. If, on the other hand, the patient has a baseline anisocoria and the tadpole pupil manifests in the smaller of the pupils, testing for Horner's syndrome is recommended.

Diagnostic Testing, Management, and Disposition

Determination of the potential etiology of an anisocoria can be facilitated by the approach outlined in the explanatory algorithm in Figure 61.30. Assuming no damage to the iris (implying a purely structural problem) is evident on slit-lamp examination, the strategy is to differentiate a benign cause of anisocoria (eg, physiological or pharmacological) from one that requires additional neuro-ophthalmological consultation (eg, Horner's syndrome) or emergent neuro imaging (eg, CN III compression potentially due to an aneurysm). The first step is to determine which pupil—the larger or the smaller—is the pathological one, keeping in mind that that parasympathetic innervation constricts a pupil in bright light, whereas sympathetic stimulation helps dilate a pupil in the dark. The subsequent steps incorporate the principles that an abnormally large pupil may be due to either a decrease in parasympathetic stimulation or an augmentation of sympathetic stimulation, and an abnormally small pupil may be due to either a decrease in sympathetic stimulation or an augmentation of parasympathetic stimulation.

The type of response to a topical application of cocaine (which specifically blocks norepinephrine uptake) can be diagnostic of Horner's syndrome, in that with no norepinephrine available to block the re-uptake, the Horner's pupil will typically not dilate. Other medications, such as hydroxyamphetamine (an

TABLE 61.2

Potential Locations of Lesion Causing a Horner's Syndrome, Based on Symptoms and Signs

SYMPTOMS AND SIGNS	POTENTIAL LESION LOCATION	POTENTIAL LESION TYPE
Brainstem symptoms (vertigo, ataxia, diplopia, and focal sensory and motor deficits)	Pontine or midbrain	Infarction or neoplasm
Myelopathic symptoms (paraparesis, sensory deficit, bowel or bladder symptoms, or hyperreflexia)	High spinal cord	Neoplastic or demyelinating process
Arm pain, weakness or numbness, neck lymphadenopathy (especially with hoarseness from recurrent laryngeal nerve compression)	Brachial plexus or cupula of the lung	Neoplastic process, such as a Pancoast tumor
Ipsilateral ear or neck pain (especially with symptoms of phrenic or vagus nerve involvement)	Carotid sheath	Carotid dissection; inadvertent injection of an anesthetic into the sheath during dental or line-placement procedures
Hearing loss and ear pain; trigeminal nerve dysautonomia (ipsilateral facial pain, rhinorrhea, conjunctival injection, and tearing)	Skull base	Neoplasm; inflammatory or infectious mass effect

Flaherty PM, Flynn JM: Horner syndrome due to carotid dissection. J Emerg Med 41(1):43-46, 2011. Davagnanam I, Fraser CL, Miszkiel K, et al: Adult Horner's syndrome: a combined clinical, pharmacological, and imaging algorithm. Eye (Lond) 27(3):291-298, 2013.

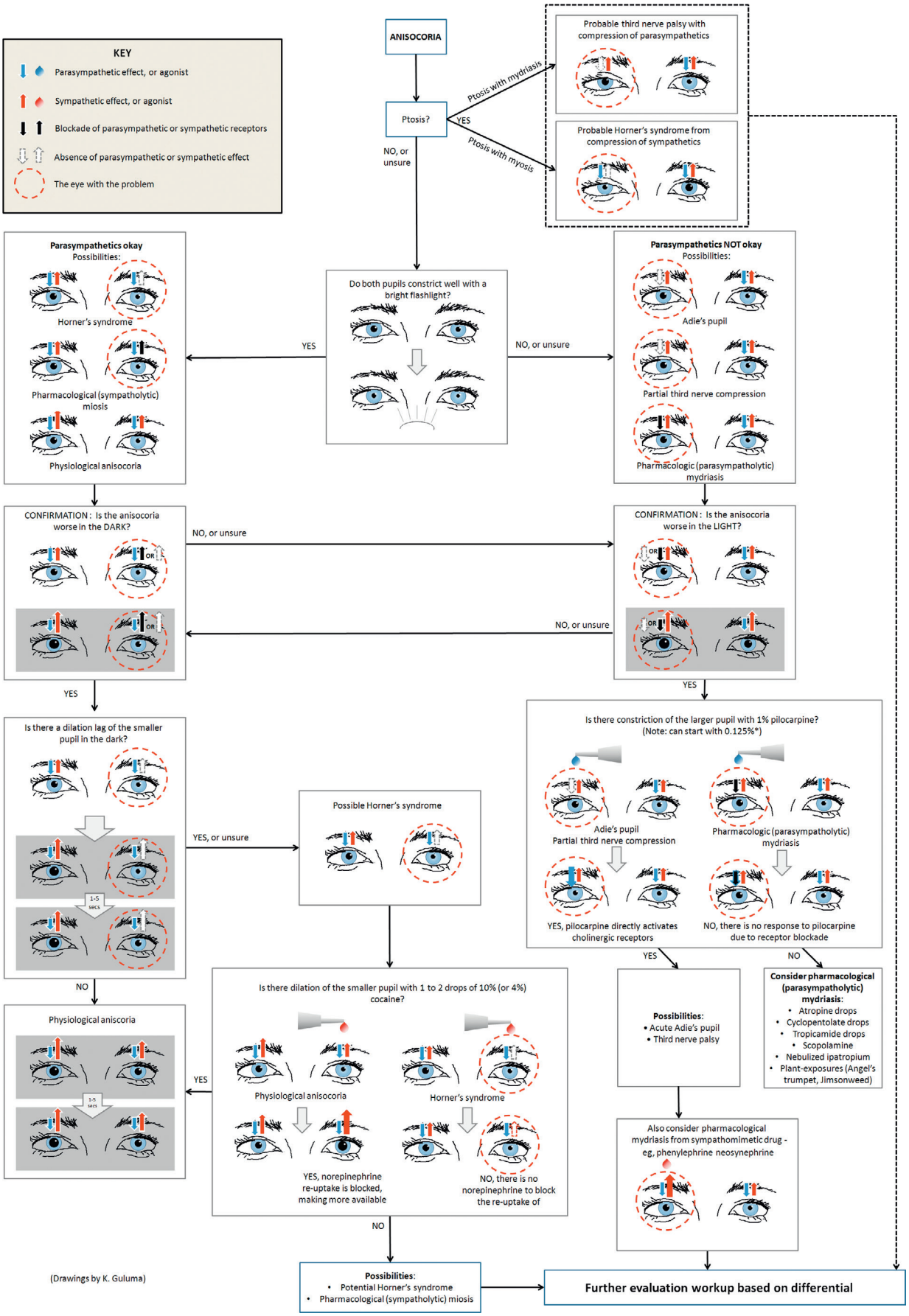


Fig. 61.30. The approach to anisocoria in the emergency department (ED), an explanatory algorithm. *Some authors advocate that a marked response to low concentration (0.1% or 0.125%) pilocarpine is more consistent with an Adie's pupil and can be used to differentiate it from an acute third nerve palsy (which may require the more concentrated 1% to elicit a reaction). This approach may be impractical, however, as a sole means to rule out a third nerve palsy from something like an aneurysm.

indirect-acting adrenergic mydriatic that causes endogenous norepinephrine to be released from sympathetic nerve endings without directly stimulating the effector cells[75]), as well as direct adrenergic agonists, such as a phenylephrine or 1% apraclonidine, can be used with ophthalmological consultation to perform a secondary evaluation of a Horner's pupil. Pilocarpine is a direct is a cholinergic receptor agonist and is used to differentiate hypo-parasympathetic conditions (see Fig. 61.30).

Once one of the typical presentations of anisocoria is identified, the evaluation progresses based on the clinical indications. Examination of an Adie's pupil typically reveals poor reaction to light with sectoral palsy of the iris sphincter, and a lack of (or slow) constriction with near accommodation (at least in the acute phase; later on, with re-innervation, the pupil will constrict strongly, and will thus be a "tonic" pupil).[76] Slit-lamp examination may reveal sectoral palsies of the iris, and a weak cholinergic agent (pilocarpine 0.1%) causes an intense pupillary constriction (compared to the patient's normal pupil) as a result of the cholinergic supersensitivity in the affected pupil. These patients should be referred non-emergently to an ophthalmologist for further evaluation. The Argyll Robertson pupil, like the Adie's pupil, will demonstrate segmental, slow, or little iris sphincter constriction with light, but normal constriction with near accommodation ("light-near dissociation," which distinguishes it from an acute Adie's pupil). A patient with bilateral Argyll Robertson pupils should be screened for neurosyphilis as per standard (please refer to entries dedicated to syphilis elsewhere in this text). The patient with a new-onset Horner's syndrome should undergo an evaluation to determine the cause and will typically require targeting imaging based on the diagnostic considerations outlined in Table 61.2, with MRI for brain, skull-base, and spinal cord lesions, and computed tomography angiography (CTA) for chest and neck/carotid pathology.[72] The cadence of the evaluation (and which components are done in the ED) will be dictated by the acuity of the primary considerations in the differential diagnosis, with aneurysm, dissection, brainstem stroke, and a rapidly progressive myelopathic process evaluated emergently in the ED and a more subacute or chronic process (such as, a tumor) being worked up urgently as an outpatient. The diagnostic evaluation and management of a third nerve palsy is covered in Chapter 18 and Chapter 95. With regards to pharmacologic mydriasis and miosis, most of the exposures and their effects will be self-limited and transient, and the specific management will dictated by the toxicological sequelae expected. The first-time clinical presentation of physiological and headache-associated anisocoria (mydriasis) may provoke a neuro-imaging evaluation for the presence of aneurysmal or mass compression of CN III; although this is being excluded, treatment can be rendered along lines that are standard for migraine headache. Physiological and headache-associated anisocoria is otherwise self-limited and will not typically require urgent ophthalmology referral unless persistent.

NYSTAGMUS

Principles

Three specific mechanisms keep an object of visual interest on the fovea: (1) fixation, wherein the visual system detects retinal drifts and programs corrective eye movements; (2) the vestibulo-ocular reflex (VOR), which keeps the eyes on target despite head movements; and (3) eccentric gaze-holding, which requires ongoing signals from the brainstem and cerebellum to overcome the natural elastic pull of orbital tissues when the eyes are deviated away from the mid-position to fixate on a target.[76] Dysfunction in any of these three mechanisms removes the visual target from the fovea and may result in nystagmus and oscillopsia (a subjective sense of movement of the visual field).

Nystagmus is a repetitive horizontal, vertical, or torsional back and forth movement of the eyes that may appear as an equal "to and fro" motion (pendular nystagmus), or demonstrate an alternating, slow phase followed by a corrective fast phase (jerk nystagmus). In jerk nystagmus, although the slow phase is the abnormal one, the directionality of the nystagmus is described as that of the fast phase. Gaze-evoked nystagmus (GEN) is an inability to hold the eyes in a fixed position at the eccentric extremes of gaze.

Nystagmus can be physiologic or pathologic and congenital or acquired. Patients may have an incidental nonspecific physiological nystagmus with a very small amplitude and a very fast velocity, non-sustained (less than three beats), only elicited in extreme eccentric gaze, only horizontal and symmetric, and without other signs or symptoms of cerebellar system dysfunction.[77] A patient may also have congenital nystagmus, typically identified as chronic or present since birth, which requires no acute intervention in the ED. The focus in the ED is therefore on acquired pathological nystagmus, of which the etiologies can be classified as either (1) peripheral (such as, seen with benign peripheral vertigo or vestibular neuronitis), (2) central (such as, seen with ischemic stroke or CNS mass lesions), or (3) toxic and metabolic (such as, that induced by medications, alcohol or illicit drugs). The clinical priority is to distinguish a peripheral (which is relatively benign and can be treated as an outpatient) from central (which may imply focal CNS pathology and require targeted neuro-imaging) from toxic or metabolic etiologies (which may imply toxic levels of a medication, or an underlying illicit drug intoxication).

Clinical Features, Differential Diagnosis, Diagnostic Evaluation, Management, and Disposition

Peripheral Nystagmus and Central Nystagmus

Because peripheral and central nystagmus from lesional processes (eg, from otoconia, vestibular neuronitis, posterior circulation stroke, brain tumor, and so on) present with prominent vertigo, a detailed discussion of these entities is deferred to the entries on vertigo and dizziness in Chapter 16. Table 61.3 highlights the specific features of the nystagmus associated with these conditions. The key clinical goal in the ED with regards to nystagmus caused by a lesion somewhere is differentiating more subtle presentations of a central cause from benign peripheral one. This can achieved along the lines of (1) the direction of the nystagmus, (2) how its intensity changes with extremes of gaze, and (3) how it is affected by visual fixation, as outlined in Table 61.3.

Toxic and Metabolic Nystagmus

Nystagmus from drug or medication toxicity may be suggested by a concurrent toxidrome and, depending on the agent and the degree of toxicity, a lack of prominent vertigo or ataxia (keeping in mind that the specificity of nystagmus findings as an indicator of toxicity is unknown). Drug-induced GEN, although symmetric, is different from physiological nystagmus in that it has a larger amplitude and slower velocity and beats in the direction of the gaze (ie, upbeat nystagmus with the patient looking up, rightward nystagmus with the patient looking to the right, and so on). GEN from a focal cerebellar or brainstem lesion may look similar to that which is drug-induced, but it is characterized by a sustained asymmetric and rebound nystagmus in which, although the slow phase is directed toward primary position where the eyes are deviated, a few slow phases may be directed toward the prior gaze direction after the eyes return to the primary position.[77,78] The management is targeted toward the overall toxicological profile of the specific offending agent.

TABLE 61.3

Forms and Causes of Nystagmus

TYPE OF NYSTAGMUS	PRESUMED AREA OF DYSFUNCTION	CHARACTER/ PRIMARY DIRECTION	TRIGGERED BY HEAD MOVEMENTS?	SUPPRESSES ON VISUAL FIXATION ON AN OBJECT?	CHANGES DIRECTION WITH GAZE?	SUSTAINED?
PERIPHERAL NYSTAGMUS						
Labyrinthitis or vestibular neuronitis	Labyrinthine dysfunction or viral infection of the superior portion of the vestibular nerve trunk	Horizonto-rotatory, one direction only, slow phase towards dysfunctional nerve	Yes	Yes	No, just gets more pronounced the further the patient looks away from dysfunctional nerve	Yes
Benign paroxysmal positional vertigo (BPPV)	Otolithic, posterior canal (most common)	Torsional combined with vertical, one direction only	Yes, raising the head from horizontal to vertical	Yes	No	No
Benign paroxysmal positional vertigo (BPPV)	Otolithic, other canals	Horizonto-rotatory, one direction only, slow phase toward dysfunctional canal	Yes, turning head side-to-side	Yes	No, just gets more pronounced the further the patient looks away from dysfunctional canal	No
CENTRAL NYSTAGMUS						
Downbeat nystagmus	Vestibulocerebellum Drugs: Lithium, phenytoin, carbamazepine, alcohol, toluene, felbamate, lamotrigine, phencyclidine (PCP), ketamine Nutritional deficiencies: magnesium, vitamin B_{12} or thiamine	Pure vertical, with fast component downward	No	No	No, just more pronounced on looking down	Yes
Upbeat nystagmus	Pontomesencephalic or pontomedullary junction, or the superior vestibular nucleus and tracts Nutritional deficiencies: Thiamine (Wernicke's)	Pure vertical, with fast component upward	No	No	No, just more pronounced on looking up	Yes
Torsional	Cerebellum or brainstem Drugs: PCP, ketamine	Pure rotary, with bidirectional fast component	No	No	Yes	Yes
Horizontal	Cerebellum or brainstem Drugs: PCP, ketamine	Bi-directional	No	No	Yes, fast component beats in direction of gaze, and gets worse with more extreme deviation	Yes
Gaze-evoked nystagmus (GEN)	Cerebellum or brainstem Drugs: Phenytoin, alchohol	Multi-directional, but asymmetric intensity	No	No; in fact worsens on eccentric fixation	Yes, fast component beats in direction of gaze, and gets worse with more extreme deviation	Yes, specifically if vision is eccentrically fixated on an object

TABLE 61.3

Forms and Causes of Nystagmus—cont'd

TYPE OF NYSTAGMUS	PRESUMED AREA OF DYSFUNCTION	CHARACTER/ PRIMARY DIRECTION	TRIGGERED BY HEAD MOVEMENTS?	SUPPRESSES ON VISUAL FIXATION ON AN OBJECT?	CHANGES DIRECTION WITH GAZE?	SUSTAINED?
OTHER MISCELLANEOUS CENTRAL NYSTAGMUS PRESENTATIONS						
Acquired pendular nystagmus	Paramedian pontine tract (seen in multiple sclerosis) Drugs: Phenytoin	Oblique or elliptical movements, can even be monocular	No	No	No	Yes
Periodic alternating nystagmus	Nodulus and ventral uvula of the vestibulocerebellum Drugs: Phenytoin	Horizontal nystagmus with a slow phase that changes direction every 1 to 2 min	No	No	No	Yes
Superior oblique myokymia	Possible cranial nerve (CN) disorder	Torsional oscillopsia, in one eye	No	No	No	Yes
See-saw nystagmus	Parasellar mass, or stroke to mesodiencephalic regions	Elevation with intorsion of one eye, with simultaneous depression and extorsion of the other eye	No	No	No	Yes
Oculopalatal myoclonus	Dentate, red, and inferior olivary nuclei in brainstem	Vertical-torsional or pure vertical (with one eye being more prominent), associated with palatal myoclonus	No	No	No	Yes

From Baier B, Dieterich M: Incidence and anatomy of gaze-evoked nystagmus in patients with cerebellar lesions. Neurology 76:361-365, 2011; Ehrhardt D, Eggenberger E: Medical treatment of acquired nystagmus. Curr Opin Ophthalmol 23(6):510–516, 2012; Shaikh AG: Fosphenytoin induced transient pendular nystagmus. J Neurol Sci 330 (1-2):121-122, 2013.

KEY CONCEPTS

- Routine prophylactic topical antibiotics are not indicated for the treatment of corneal abrasions, and eye patches are not recommended because they can mask a worsening infection.
- Eyelid lacerations that may require referral to a plastic or ophthalmic surgeon include those with lid margin lacerations, a canalicular laceration, or levator or canthal tendon injuries.
- Alkaline burns to the cornea and conjunctiva need to be copiously irrigated until a neutral pH is attained, because they produce a liquefactive necrosis that penetrates and dissolves tissue.
- Admission should be considered for traumatic hyphema patients with sickle cell trait, uncontrolled elevations in intraocular pressures (IOPs), hyphema of greater than 50%, and concern for re-bleeding.
- Any manipulation, palpation, or tonometry on a suspected globe rupture should be avoided, pending ophthalmological consultation and further examination.
- Scleritis, an autoimmune inflammatory process involving the sclera, can be confused with episcleritis, caused by inflammation in the more superficial episcleral layer of the eye. Episcleritis, unlike scleritis, is associated with much less discomfort, a pinker and more pronounced peri-limbal injection, and has injected superficial episcleral vessels that—unlike the deeper injected scleral vessels in scleritis—will vasoconstrict and blanch with 10% phenylephrine. Treatment of both involves topical corticosteroid drops.
- Endophthalmitis is an infection of the eye itself, and the most common etiology is recent intraocular surgery. Intravitreal antibiotics are indicated for endophthalmitis.
- Herpes zoster keratoconjunctivitis can complicate herpes zoster ophthalmicus, and necessitates emergent ophthalmologic consultation and treatment with systemic antiviral agents.
- The acute treatment of acute angle-closure glaucoma uses a two-armed approach: (1) reducing the production of aqueous humor with a topical beta-blocker (timolol 0.5%—1 to 2 gtt), a carbonic anhydrase inhibitor (acetazolamide 500 mg IV or PO), and a systemic osmotic agent (mannitol 1 to 2 g/kg IV); and (2) increasing the outflow of aqueous humor with a topical alpha-agonist (phenylephrine 1 gtt), miotic drops (pilocarpine 1% to 2%), and topical steroids (prednisolone acetate 1%, 1 gtt every 15 to 30 minutes four times, then every hour).
- With anisocoria, the following considerations help in the determination of which pupil—the larger or the smaller—is the pathological one: (1) parasympathetic innervation constricts a pupil in bright light, whereas sympathetic stimulation helps dilate a pupil in the dark; (2) an abnormally small pupil may therefore be due to a either a decrease in sympathetic stimulation or an augmentation of parasympathetic stimulation—but likely the former (eg, Horner's syndrome); (3) an abnormally large pupil may therefore be due to a either a decrease in parasympathetic stimulation or an augmentation of sympathetic stimulation—but likely the former (eg, partial third-nerve palsy from compression, Adie's pupil, pharmacological mydriasis); or (4) the abnormally small pupil will usually look worse in the dark, whereas the abnormally large pupil will usually look worse in the light.

The references for this chapter can be found online by accessing the accompanying Expert Consult website.

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CHAPTER 61: QUESTIONS & ANSWERS

61.1. A 23-year-old male presents with left periorbital pain after being struck with a fist. On examination, there are no globe injuries but marked periorbital swelling is noted. Computed tomography (CT) of the face reveals an orbital floor fracture. Which of the following would be the most likely physical findings?

A. Cheek anesthesia, enophthalmos, and limitation of upward gaze
B. Cheek anesthesia, ptosis, and limitation of inferior gaze
C. Forehead anesthesia and afferent papillary defect
D. Forehead anesthesia, diplopia, and limitation of lateral gaze
E. Ptosis, miosis, and ipsilateral anhydrosis

Answer: A. An orbital floor fracture may entrap the inferior rectus and inferior oblique muscles, resulting in diminished upward gaze. Other findings may include ptosis, enophthalmos, ipsilateral cheek/lip anesthesia, and orbital emphysema. Ten percent to 25% of such patients have associated globe injuries. Option E describes Horner's syndrome, which is not a typical finding.

61.2. A 20-year-old male presents with periorbital pain and swelling after a blow to the eye by a softball. Physical examination reveals proptosis with blurred vision and limitation of ocular motion in all planes. Tonometry reveals an intraocular pressure (IOP) of 35 mm Hg. Which of the following should be the first indicated maneuver?

A. Acetazolamide 500 mg IV, mannitol 20 g IV, and topical timolol
B. Computed tomography (CT) scan of the head and face
C. Endotracheal intubation and hyperventilation
D. Immediate lateral canthotomy and cantholysis
E. Ophthalmologic consultation

Answer: D. These findings should make one suspect retrobulbar hemorrhage. All of these interventions are likely indicated. Intraocular hypertension may compromise central retinal artery flow. Although immediate ophthalmologic consultation and pressure-lowering maneuvers are indicated, lateral canthotomy and cantholysis will provide the most rapid temporizing measure to preserve vision.

61.3. A 43-year-old male presents with acute ocular pain after a splash injury from drain cleaner. What should be the sequence of interventions?

A. Copious irrigation for 10 minutes, pH testing, cyclopentolate cycloplegia, topical antibiotics/intraocular pressure (IOP) measurement
B. Intravenous (IV) analgesia, cyclopentolate cycloplegia, IOP measurement, isotonic irrigation
C. IOP measurement, analgesia, head-up position, cycloplegia
D. Phenylephrine cycloplegia, isotonic irrigation for 10 minutes, pH testing, slit-lamp examination for foreign bodies
E. Phenylephrine cycloplegia, slit-lamp examination for foreign bodies, isotonic irrigation for 10 minutes, pH testing

Answer: A. Copious irrigation, ideally beginning at the scene, is the cornerstone of management. Nitrazine pH testing after 10 minutes should guide the need for continued irrigation. Cycloplegia, IOP measurement, and topical antibiotics come after pH normalization. Phenylephrine is contraindicated for cycloplegia in these cases because of its vasoconstrictive properties.

61.4. A 17-year-old girl who wears contact lenses presents with a 24-hour history of right eye pain. Physical examination reveals a right corneal abrasion at the six-o'clock position of the limbus. Appropriate treatment consists of which of the following?

A. Cessation of contact lens wear, eye irrigation (qid) with isotonic saline solution, followed by instillation of undiluted topical tetracaine for 5 days
B. Emergent ophthalmology consultation
C. Tetanus prophylaxis, eye patching for 48 hours, antibiotic ointment, and a 24-hour recheck
D. Tetanus prophylaxis, topical nonsteroidal anti-inflammatory drugs (NSAIDs), cessation of contact lens wear, and a 24-hour recheck
E. Topical nonsteroidal medications, topical antipseudomonal antibiotic, and a 24-hour recheck

Answer: E. Tetanus prophylaxis is not indicated for corneal abrasion unless there is corneal perforation or contamination with organic material. Topical NSAIDs reduce corneal abrasion pain. Antipseudomonas coverage with cessation of contact lens wear is appropriate. Eye patching is not indicated. Administration of undiluted topical anesthetics for more than 24 hours is untested and may be dangerous. Oral analgesics may be needed.

61.5. How do patients with subconjunctival hemorrhage most commonly present?

A. Asymptomatic blood in the eye, noticed in the mirror or by a friend
B. Decreased visual acuity
C. Foreign body sensation
D. Modest pain
E. Photophobia

Answer: A. Any significant symptoms, such as pain, decreased vision, foreign body sensation, or photophobia, should spark the search for more serious pathology. Bilateral hemorrhage in the absence of a clear cause (eg, severe vomiting) should raise suspicion for coagulation issues.

61.6. A 38-year-old man presents with unilateral left-sided visual loss after a motor vehicle collision (MVC). The only clinical finding is a left-sided hyphema rising to 50% of the height of the anterior chamber. Intraocular pressure (IOP) is 17 mm Hg in the unaffected eye and 29 mm Hg in the affected eye. Appropriate management should include which of the following?

A. Cycloplegia, intravenous (IV) mannitol, ophthalmology consultation
B. IV analgesia and antibiotic, immediate ophthalmologic consultation for decompression resulting from intraocular hypertension
C. Oral acetazolamide, patch and shield, antiemetics, 24-hour recheck
D. Topical beta-blocker, patch and shield, modest analgesia, admission
E. Topical beta-blocker, topical nonsteroidal anti-inflammatory drugs (NSAIDs) for pain, patch and shield, 24-hour recheck

Answer: D. Significant hyphema is an indication for admission. The presence of elevated IOP requires urgent treatment (which might also include topical alpha-agonists or IV acetazolamide,

and so on), patch and shield, elevation of the head, and cautious use of systemic analgesics. Any form of platelet inhibition would be contraindicated (ie, NSAIDs).

61.7. What is the major complication of hyphema?
A. Detached retina
B. Glaucoma
C. Horner's syndrome
D. Rebleeding
E. Vitreous hemorrhage

Answer: D. Rebleeding typically occurs 2 to 5 days later as the clot retracts. It is most common in patients with elevated intraocular pressures (IOPs), hyphema greater than 30% of the anterior chamber, and with delayed presentation. Rebleeding may lead to glaucoma and synechia formation.

61.8. A 48-year-old woman presents with right eye pain, photophobia, and decreased vision after a motor vehicle collision (MVC). Physical examination reveals an irregularly shaped pupil and a small hyphema. Photophobia, decreased acuity, minimal pupil reactivity, and bloody chemosis are seen on examination. What is the most likely diagnosis?
A. Acute angle–closure glaucoma
B. Blunt ciliary injury
C. Iridodialysis
D. Scleral rupture
E. Traumatic miosis

Answer: D. Scleral rupture occurs either at the insertion of the extraocular muscles or at the limbus, where the sclera is the thinnest. A "teardrop" pupil is often seen and may be accompanied by bloody chemosis or severe subconjunctival hemorrhage. Brownish black pigment prolapse may also be seen. Intraocular pressure (IOP) may be low, but tonometry is generally contraindicated in cases of suspected globe injury.

61.9. A 26-year-old man presents with a 3-day history of right eye pain, decreased vision, and photophobia. He reports a history of left eye trauma 6 weeks prior, with hyphema, traumatic iritis, and persistent decreased vision. He is otherwise healthy. Physical examination reveals photophobia in the right eye with bilateral decreased vision. Before the past 3 days, the vision in the right eye had been perfect. What is the most likely explanation for his right eye symptom?
A. Collagen vascular disease
B. Post-traumatic conjunctivitis
C. Post-traumatic retinal tear
D. Spontaneous vitreal hemorrhage
E. Sympathetic ophthalmia

Answer: E. Sympathetic ophthalmia is an autoimmune inflammatory response in the unaffected eye, days to months after uveal trauma in the opposite eye. Pain, photophobia, and decreased vision are common. This patient had no findings consistent with conjunctivitis or collagen vascular disease, and a retinal tear would not typically be painful.

61.10. Oral antibiotics are indicated for which of the following?
A. Blepharitis
B. Chalazion
C. Dacryocystitis
D. Endophthalmitis
E. Hordeolum

Answer: C. Dacryocystitis is an infection of the lacrimal sac from nasociliary duct obstruction. Warm compresses are also recommended and may be helpful, although evidence is lacking. Warm compresses and topical antibiotics are appropriate for the other conditions. Intravitreal antibiotics are indicated for endophthalmitis.

61.11. Emergency department (ED) bedside ocular ultrasonography can provide useful information for which of the following conditions?
A. Lens dislocation
B. Retinal detachment
C. Vitreous hemorrhage
D. All of the above

Answer: D. A displaced lens can be seen in the relatively hypoechoic vitreous. Vitreous hemorrhage and retinal detachment can both be diagnosed with ED bedside ultrasonography.