CHAPTER 17

Posterior Segment Manifestations of Trauma

This chapter includes related videos. Go to www.aao.org/bcscvideo_section12 or scan the QR codes in the text to access this content.

Highlights

- In the initial evaluation of an ocular injury, the clinician should try to determine whether the globe is open or closed and whether an intraocular foreign body (IOFB) is present.
- In cases of ocular or orbital trauma, an IOFB should always be suspected and ruled out. If the foreign body material is unknown, a computed tomography scan is the preferred imaging modality over magnetic resonance imaging.
- Endophthalmitis occurs following 2%–7% of penetrating injuries; the incidence is higher in association with IOFBs and in rural settings.

Classification of Ocular Globe Trauma

Ocular trauma is an important cause of visual impairment worldwide. Ocular globe trauma can be classified as follows (terminology based on the Birmingham Eye Trauma Terminology System; Video 17-1):

- Closed-globe injuries
  - contusion (blunt trauma *without* break in eye wall)
  - lamellar laceration (partial-thickness wound of the eye wall)
  - superficial foreign bodies
- Open-globe injuries
  - rupture (blunt trauma *with* break in eye wall)
  - laceration (full-thickness wound of the eye wall, caused by a sharp object)
  - penetrating injury (entrance break; no exit break in eye wall)
  - perforating injury (both entrance and exit breaks in eye wall)
  - intraocular foreign bodies, penetrating or perforating

This chapter focuses on closed- and open-globe injuries primarily affecting the posterior segment of the eye.

VIDEO 17-1 Birmingham Eye Trauma Terminology System.
Animation developed by Shriji Patel, MD, MBA.

Microsurgical techniques have improved the ability to repair corneal and scleral lacerations, and vitrectomy techniques allow management of severe intraocular injuries (see Chapter 19). Ocular trauma is also discussed in BCSC Section 6, *Pediatric Ophthalmology and Strabismus;* Section 7, *Oculofacial Plastic and Orbital Surgery;* and Section 8, *External Disease and Cornea.*

Evaluation of the Patient After Ocular Trauma

In the initial evaluation of an ocular injury, the clinician should try to determine whether the globe is open or closed and whether an intraocular foreign body (IOFB) is present. The evaluation includes obtaining a complete history (Table 17-1) and performing a thorough examination. If possible, the visual acuity of each eye should be measured separately and the pupils evaluated for an afferent pupillary defect. To the extent possible, the clinician should perform external, slit-lamp, and fundus examinations and measure intraocular pressure (IOP) with care to avoid exacerbating the injury. Severe chemosis, ecchymosis, eyelid edema, low IOP, presence of an entrance wound, iris damage or incarceration, cataract, or other anterior segment pathology may suggest an ocular rupture or laceration. Normal IOP and/or absence of findings on examination does not exclude an occult penetration of the globe.

Table 17-1 Important Questions to Ask in Cases of Ocular Trauma

When exactly did the injury occur?
What was the exact mechanism of injury?
How forceful was the injury?
Was there any object (eg, wood stick, nail, knife) that may have penetrated the eye? If so, what was the object's material?
Is the presence of an intraocular foreign body a possibility? Could it be wood or organic material?
Was the patient hammering metal on metal or working near machinery that could have caused a projectile to enter the eye?
Was the patient wearing spectacles or was he or she close to shattered glass?
Was the patient wearing eye protection?
What was the health status of the eye before the injury?
Has the patient had previous ocular surgery, including laser in situ keratomileusis (LASIK), penetrating keratoplasty, and/or cataract surgery?
Are there concomitant systemic injuries?
What emergency measures were taken, if any (eg, tetanus shot given, antibiotics administered)?
When was the last tetanus toxoid administered?
When was the patient's last oral intake (in case surgery is required)?
Was the injury work related?

If an open-globe injury is suspected but cannot be confirmed on the basis of findings or because of lack of patient cooperation, a thorough examination with possible surgical exploration should be performed under general anesthesia in the operating room.

Multimodal imaging can help assess the status of the injured eye and facilitate detection of an IOFB, particularly in the presence of media opacities. For acute injury, the 2 most helpful imaging systems are computed tomography (CT) of the eye and orbits and ocular ultrasonography (B-scan). A CT scan without contrast should be considered for any trauma patient when an IOFB is suspected on the basis of the mechanism of injury. CT can also aid in identifying periocular trauma, including orbital fractures. When B-scan examination is performed, care must be taken to avoid expulsion of intraocular matter. It is advisable to perform ultrasonography through the patient's closed eyelids, aided by copious amounts of ultrasound gel. B-scan ultrasonography can be particularly helpful in detecting nonradiopaque IOFBs, such as wood and plastic. Signs of a scleral rupture that are visible on ultrasonography include the entrapment of vitreous strands into the rupture site. Intraocular air may cause image artifacts that can complicate image interpretation.

CT is very helpful in detecting radiopaque IOFBs; however, dense IOFBs may introduce image artifacts that cause them to appear larger than they really are, making exact localization difficult. Although magnetic resonance imaging (MRI) is not usually used with acute injuries, it can be helpful in visualizing detailed ocular anatomy and in identifying the presence and location of IOFBs, including those that are not radiopaque. However, MRI should be used only after the presence of ferromagnetic foreign bodies has been definitively ruled out, because of the possibility that the magnetic field will move such foreign bodies, causing additional damage. When the foreign body material is unknown, CT is preferred over MRI as the imaging modality.

Blunt Trauma Without a Break in the Eye Wall

In blunt trauma, the object does not penetrate the eye but may cause rupture of the eye wall. Serious sequelae from blunt trauma affecting the anterior segment include

- angle recession (see BCSC Section 10, *Glaucoma*)
- iridodialysis (see BCSC Section 8, *External Disease and Cornea*)
- iritis
- hemorrhage into the anterior chamber (hyphema) (see BCSC Section 8, *External Disease and Cornea*)
- subluxated or dislocated lens (see BCSC Section 11, *Lens and Cataract*)

Serious sequelae from blunt trauma affecting the posterior segment include

- commotio retinae
- choroidal rupture
- macular hole
- suprachoroidal hemorrhage
- retinal tears or detachment
- vitreous hemorrhage

- traumatic chorioretinal disruption (chorioretinitis sclopetaria)
- vitreous base avulsion
- optic nerve avulsion

See Chapter 16 for discussion of traumatic retinal breaks and retinal detachment and Chapter 19 for discussion of suprachoroidal hemorrhage. Sequelae of blunt trauma affecting the posterior segment are discussed in the following sections.

Commotio Retinae

The term *commotio retinae* refers to damage to the outer retinal layers caused by shock waves that traverse the eye from the site of impact following blunt trauma. Ophthalmoscopic examination reveals a sheenlike retinal whitening that appears some hours after the injury (Fig 17-1). This retinal whitening occurs most commonly in the posterior pole but may also be found peripherally. Spectral-domain optical coherence tomography (SD-OCT) findings suggest that the major site of disruption is in the photoreceptor and retinal pigment epithelial (RPE) layers, resulting in the observed retinal opacification (Fig 17-2). With foveal involvement, a cherry-red spot may appear because the cells involved in the whitening are not present in the foveola. Commotio retinae in the posterior pole may reduce visual acuity to as low as 20/200. Gradual visual recovery may occur if there is no associated macular pigment epitheliopathy, choroidal rupture, or macular hole formation. Disruption of the cone outer segment tips, ellipsoid zone, and external limiting membrane is associated with poorer visual and anatomical outcomes.

Choroidal Rupture

When the eye is compressed along its anteroposterior axis, tears may occur in Bruch membrane, which has little elasticity, as well as in the overlying RPE and fibrous tissue around the choriocapillaris. Adjacent subretinal hemorrhage is common. Choroidal ruptures may be single or multiple and occur typically in the periphery and concentric to the optic nerve

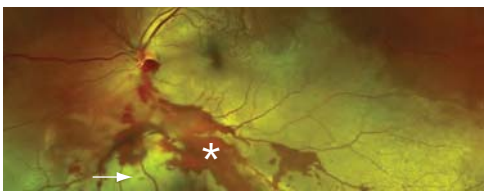

Figure 17-1 Extensive commotio retinae with sheenlike retinal whitening *(arrow)* and preretinal hemorrhage *(asterisk)* following blunt ocular trauma due to a rubber bullet. *(Reproduced from Barnes AC, Hudson LE, Jain N. Rubber bullet ocular trauma.* Ophthalmology. *2020;127(9):1190. Copyright 2020, with permission from Elsevier.)*

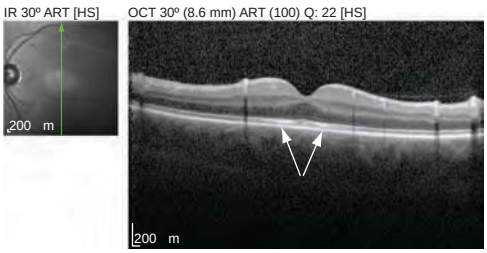

Figure 17-2 Spectral-domain optical coherence tomography (SD-OCT) image of the left eye of a patient who experienced a rock injury at work. Examination revealed macular retinal whitening with visual acuity (VA) decreased to 20/100. OCT image demonstrates increased reflectivity of the parafoveal ellipsoid zone *(arrows)*. *(Courtesy of Shriji Patel, MD, MBA.)*

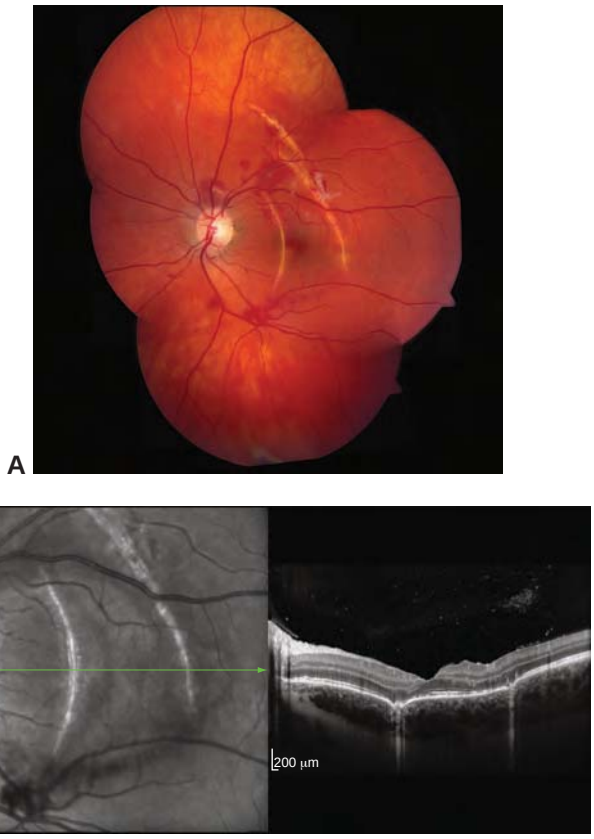

Figure 17-3 Images from a 24-year-old after a motor vehicle accident. **A,** Color fundus photograph montage reveals choroidal ruptures with subretinal hemorrhage along the superior arcade. **B,** SD-OCT shows breaks in the retinal pigment epithelium (RPE)–Bruch membrane complex with a highly reflective choroidal signal beneath the choroidal ruptures due to greater coherence of light penetration. *(Courtesy of Kenneth Taubenslag, MD, and Edward Chaum, MD, PhD.)*

head (Fig 17-3). Ruptures that extend through the fovea may cause permanent vision loss. There is no effective treatment.

Occasionally, choroidal neovascularization (CNV) develops as a late complication after damage to Bruch membrane (Fig 17-4). A patient with a choroidal rupture near the macula requires ongoing monitoring and should be alerted to the risk of CNV. Subfoveal CNV, if present, is generally treated with a vascular endothelial growth factor (VEGF) inhibitor. See Chapter 4 for information on the management of CNV.

Posttraumatic Macular Hole

Blunt trauma may cause a full-thickness macular hole by various mechanisms, including contusion necrosis and vitreous traction. Holes may be observed immediately after blunt trauma that causes severe commotio retinae, following a submacular hemorrhage caused by a choroidal rupture, or after a whiplash separation of the vitreous from the retina. In

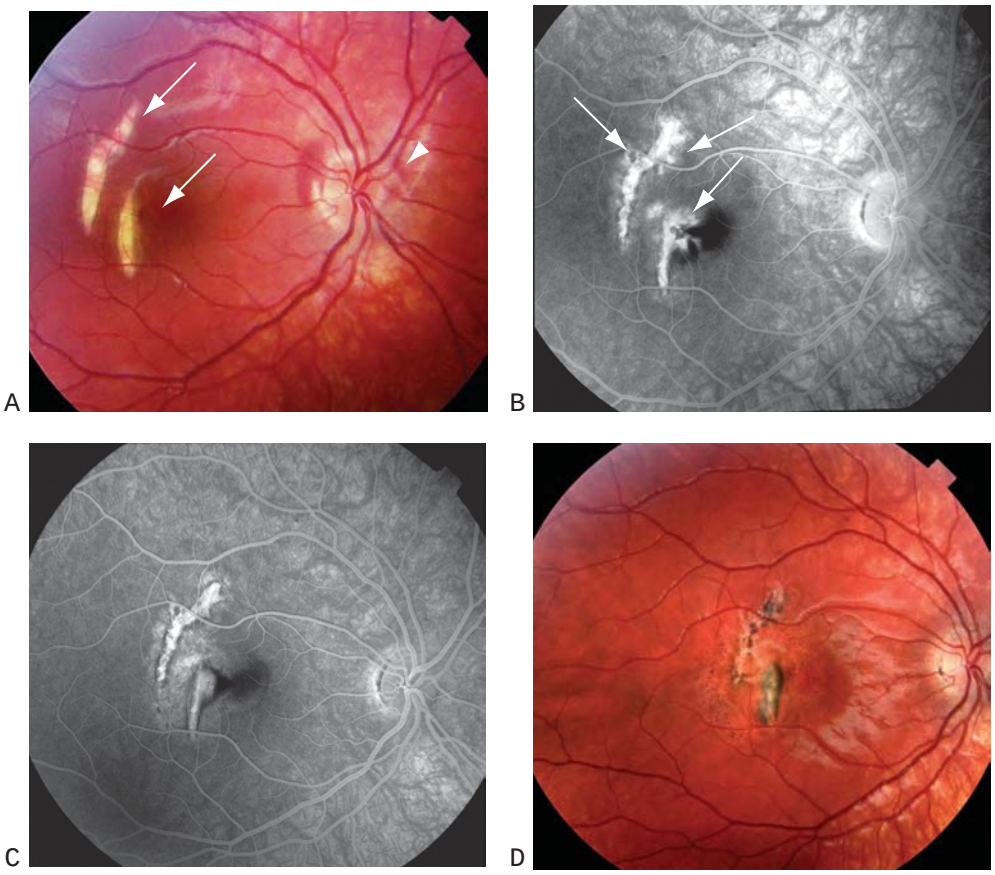

Figure 17-4 Images from a 10-year-old who was hit in the eye with a tennis ball. **A,** Color fundus photograph reveals choroidal ruptures *(arrows)*. A subretinal hemorrhage is present around the optic nerve head *(arrowhead)*. VA was 20/30. **B,** Six weeks later, VA decreased to 20/400. Late-phase fluorescein angiography image shows multiple fronds of choroidal neovascularization (CNV) arising from the choroidal ruptures *(arrows)*. **C,** Late-phase fluorescein angiography image taken 2 weeks after treatment with corticosteroids and photodynamic therapy shows that the CNV has regressed dramatically. **D,** Color fundus photograph taken 6 months after treatment. The scarring around the choroidal ruptures obscures their characteristic appearance. Some pigmentary changes have occurred in the macula as well, but VA is 20/25. *(Courtesy of Richard F. Spaide, MD.)*

addition, central depressions, or macular pits (similar to those observed in patients after sun gazing), have been described following blunt trauma to the eye and whiplash injuries. Lightning and electrical injury can also cause macular holes; patients with these injuries usually have signs of cataract and can have acute peripapillary retinal whitening. Posttraumatic macular holes may close spontaneously or may be successfully closed surgically.

Vitreous Hemorrhage

Vitreous hemorrhage is a common sequela of ocular trauma. Because a hemorrhage that is loculated at presentation can later become diffuse, a determination of the cause of the

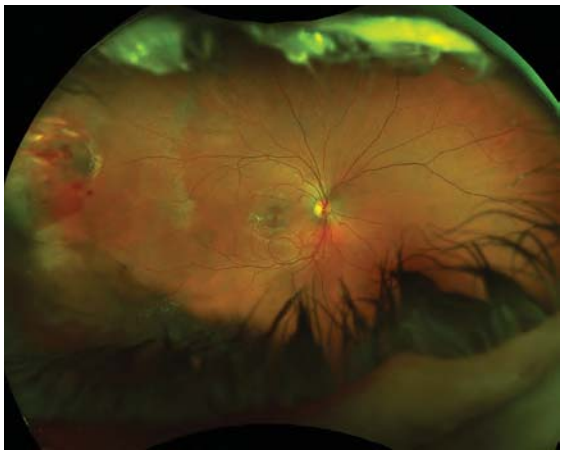

Figure 17-5 Wide-field color fundus photograph from a 32-year-old woman who presented after blunt trauma to her right eye. There is evidence of chorioretinitis sclopetaria, including temporal preretinal and subretinal hemorrhage and areas of whitening that are associated with bare sclera. *(Courtesy of Franco M. Recchia, MD.)*

hemorrhage should always be undertaken as soon as possible. Bed rest with elevation of the head may enable the hemorrhage to settle sufficiently to allow a more detailed ophthalmoscopic examination. If vitreous hemorrhage obscures the view of the posterior segment, B-scan or radiologic ocular imaging should be considered given the increased incidence of retinal tear and detachment in this setting. See also Chapter 19.

Traumatic Chorioretinal Disruption

Chorioretinitis sclopetaria is an unusual retinal pathology produced by high-speed projectile injuries to the orbit, leading to large areas of choroidal and retinal disruption combined with extensive subretinal, retinal, or vitreous hemorrhage (Fig 17-5). As the blood resorbs, the injured area is repaired by extensive scar formation and widespread pigmentary alteration. If the macula is involved, there is significant vision loss. Secondary retinal detachment rarely develops. The pattern of damage is ascribed to shock waves generated by the deceleration of the projectile passing close to the sclera. Blunt trauma due to injuries from paintballs or other projectiles may produce a similar fundus appearance.

Open-Globe Injuries

Open-globe injuries generally have a guarded prognosis regarding visual acuity outcomes (see the section Prognostication of Globe Injuries later in this chapter). The development of a retinal detachment is common; the detachment is usually caused by the primary injury or by traction resulting from proliferative vitreoretinopathy (PVR). In all cases of open-globe injuries, the presence of an IOFB should be ruled out.

Scleral Rupture

Severe blunt injuries can rupture the globe. Rupture injuries may be very severe; there is often expulsion of intraocular content (to varying degrees). The most common locations

for rupture are (1) the corneal limbus, especially through previous surgical wounds; and (2) through areas of physiologic scleral thinning parallel to and under the insertions of the rectus muscles. Important diagnostic signs of rupture include the following:

- marked decrease in ocular ductions
- very boggy conjunctival chemosis with hemorrhage
- deepened anterior chamber
- severe vitreous hemorrhage

The IOP is usually very low but may be normal or even elevated.

CLINICAL PEARL

The presence of 360° of hemorrhagic chemosis is highly suggestive of occult scleral rupture; there should be a low threshold for surgical exploration.

Lacerating and Penetrating Injuries

Lacerating injuries result from cutting or tearing of the eye wall by objects of varying sharpness. In a penetrating injury of the globe, there is an entrance break but no exit break in the eye wall. The prognosis is related to the location and extent of the wound, as well as the associated damage and degree of hemorrhage. An uncommon iatrogenic injury is scleral penetration with a needle during retrobulbar or peribulbar anesthesia for intraocular surgery.

Perforating Injuries

A globe-perforating injury has both entrance and exit wounds. Globe-perforating injuries may be caused by objects of varying sharpness such as needles, knives, high-velocity pellets, or small fragments of metal. Studies have shown that, after perforating injuries, fibrous proliferation occurs along the scaffold of damaged vitreous between the entrance and exit wounds. The wounds are often closed by fibrosis within 5–7 days after the injury, depending on wound size. Small-gauge injuries with only a small amount of hemorrhage and no significant collateral damage may heal without serious sequelae. Posterior exit wounds may be identified with gentle B-scan ultrasonography or CT scan.

Surgical Management

In most instances, primary repair of open-globe injuries consists of suturing of the corneal and scleral wounds. Although there are some theoretical reasons for performing an early vitrectomy, the priority at the time of the acute injury is to close the globe. Primary wound closure should not be delayed, particularly because closure will facilitate a later vitrectomy if it is needed. For more on vitrectomy, see Chapter 19. Open-globe trauma surgery is best performed with the patient under general anesthesia; use of peribulbar anesthesia is avoided because injection of local anesthetics into the orbit can cause compression of the globe and expulsion of intraocular contents.

Primary repair

The principles of primary repair of open-globe injuries include careful, gentle microsurgical corneoscleral wound repair, during which incarcerated uvea is reposited or excised. If a laceration crosses the limbus, or if there is any suspicion of a scleral laceration or rupture, a gentle and generous peritomy, usually 360°, should be considered for best possible exposure. Corneal lacerations may be closed with 10-0 nylon interrupted sutures, and scleral wounds may be closed with stronger 7-0, 8-0, or 9-0 nonabsorbable sutures. Vitreous should be excised from the wound and the anterior chamber re-formed. Any uvea or retina that protrudes should be amputated if devitalized or gently reposited into the eye. Chapter 3 of BCSC Section 4, *Ophthalmic Pathology and Intraocular Tumors,* discusses wound healing in detail.

Attempts should be made to safely explore any scleral laceration until its posterior extent has been located. If no laceration or rupture can be seen, and a posterior rupture is suspected, a meticulous scleral exploration, including underneath the rectus muscles, should be performed. This may necessitate disinserting 1 or more extraocular muscles to achieve adequate exposure. If the wound is very posterior, the site should be left to heal without suturing; attempts to suture very posterior wounds may result in expulsion of intraocular content. Some ophthalmologists advocate placing a prophylactic encircling scleral buckle at the time of primary repair to reduce the likelihood of a later retinal detachment.

Immediate vitrectomy

Immediate vitrectomy may be necessary or advisable in some circumstances—for example, when evaluation suggests the possibility of an IOFB, retinal detachment, or endophthalmitis.

Some surgeons favor immediate vitrectomy at the time of the primary repair, before cellular proliferation (PVR) begins. Inducing a posterior vitreous detachment and thorough dissection of the vitreous remove some of the scaffold on which contractile membranes grow. This may reduce the risk of late complications such as traction (also called *tractional*) retinal detachments, cyclitic membrane formation, and phthisis bulbi. Separating the posterior cortical vitreous from the retina may be difficult, especially in children and young adults and in eyes with retinal breaks or retinal detachment. If the injury is perforating, the posterior wound may present challenges because infusate may leak from it, making the maintenance of IOP during surgery difficult.

Delayed vitrectomy

Most practitioners in the United States prefer initially performing a primary repair of the wound(s) to restore the globe and IOP, followed by delayed vitrectomy, if necessary. The following are some advantages of delayed vitrectomy:

- reduces the risk of intraoperative hemorrhage in eyes that are acutely inflamed and congested
- allows the cornea to clear and improve intraoperative visualization
- permits spontaneous separation of the vitreous from the retina, which facilitates a safer and more complete vitrectomy
- allows posterior wounds in perforating injuries to heal, so there is ocular integrity during vitrectomy

The optimal timing of vitrectomy after primary repair remains controversial. Many advocate waiting at least 5 days if there are unsutured (posterior) wounds. Vitrectomy that is delayed more than 2 weeks following the injury may contribute to substantial worsening of PVR and associated worse anatomical and visual outcomes.

Typical indications for vitrectomy include the following:

- the presence of moderate to severe vitreous hemorrhage
- uncontrolled IOP despite medical management
- other tissue damage that requires repair
- phacoanaphylactic uveitis, which may occur if the lens is damaged
- signs of developing transvitreal traction
- retinal detachment
- posttraumatic endophthalmitis

Kuhn F. The timing of reconstruction in severe mechanical trauma. *Ophthalmic Res.* 2014;51(2):67–72.

Mieler WF, Mittra RA. The role and timing of pars plana vitrectomy in penetrating ocular trauma. *Arch Ophthalmol.* 1997;115(9):1191–1192.

Intraocular Foreign Bodies

In some cases, an IOFB is observed (Fig 17-6), or it is suggested by the presence of an entry site or the reported mechanism of injury. A detailed history helps the clinician assess the likelihood of the presence of an IOFB. As previously discussed, ocular imaging can be very

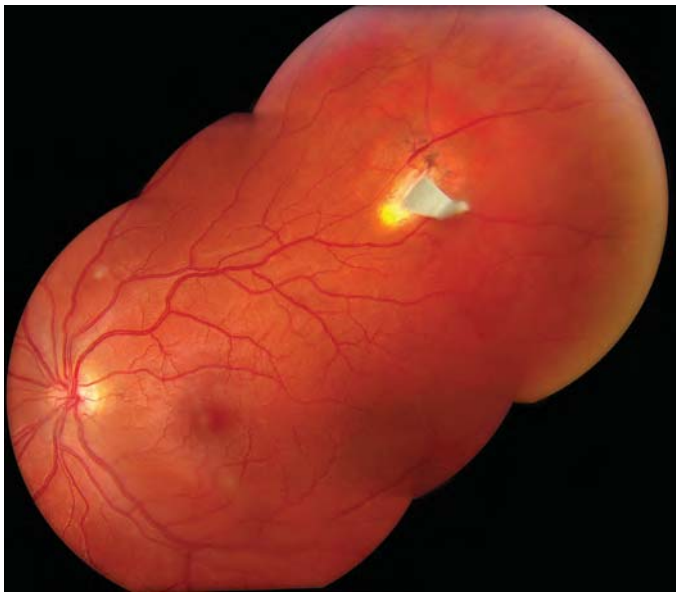

Figure 17-6 Color montage photograph of an aluminum intraocular foreign body (IOFB) with retinal penetration and visible sclera. Note the minimal reactive inflammation to aluminum, which the eye tolerates better than copper or iron. *(Courtesy of Edward Chaum, MD, PhD.)*

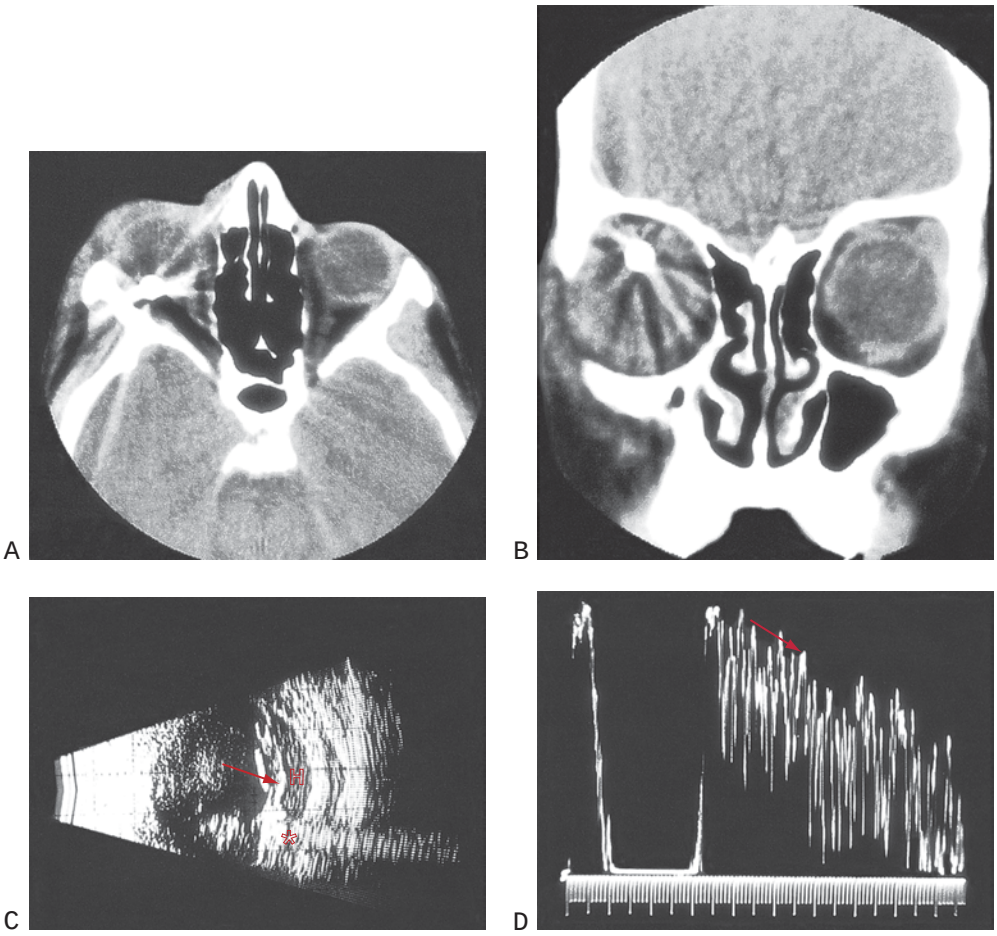

Figure 17-7 Images from a patient with an intraocular BB pellet. Axial **(A)** and coronal **(B)** computed tomography (CT) views show the pellet's position to be in the superior and posterior globe. B-scan **(C)** shows retinal detachment *(arrow)* and subretinal hemorrhage *(H)*. A characteristic reverberation of echoes between the front and back surfaces of the round pellet gives a "trail of echoes" artifact that extends posterior to the foreign body on B-scan *(asterisk)* and on A-scan **(D)** *(arrow)*.

helpful in the detection of IOFBs (Figs 17-7, 17-8). If surgical removal of an IOFB cannot be accomplished promptly, intravitreal injection and systemic administration of antimicrobial agents should be considered in order to minimize the risk of endophthalmitis developing.

Surgical techniques for removal of intraocular foreign bodies

Surgical planning should address the following issues:

- location of the foreign body in the eye
- surgeon's ability to visualize and localize the foreign body
- size and shape of the foreign body
- composition of the foreign body (ferromagnetic vs nonferromagnetic)
- encapsulation of the foreign body

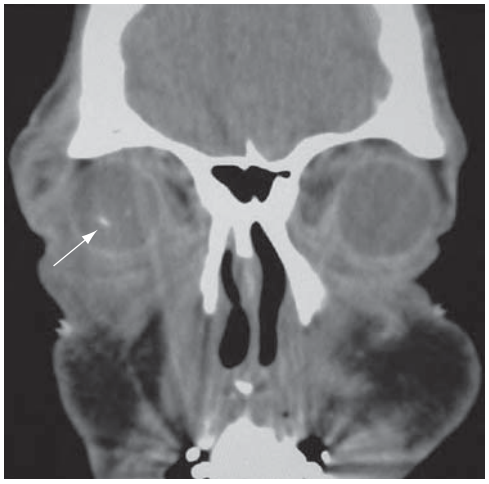

Figure 17-8 CT scan demonstrating a nonmetallic IOFB. *(Reproduced from Arey ML, Mootha VV, Whittemore AR, Chason DP, Blomquist PH. Computed tomography in the diagnosis of occult open-globe injuries.* Ophthalmology. *2007;114(8):1448–1452. Copyright 2007, with permission from Elsevier.)*

Pars plana vitrectomy allows removal of traumatized vitreous and facilitates controlled microsurgical extraction of IOFBs and media opacities such as cataract and hemorrhage (Video 17-2). Before forceps extraction is attempted, the IOFB should be freed of all attachments. A small rare-earth magnet or kidney stone basket may be used to engage and separate the foreign body from the retinal surface (Video 17-3). Although small foreign bodies can be removed through enlargement of the pars plana sclerotomy site, it may be safer to extract some large foreign bodies through the corneoscleral limbus or the initial wound to minimize collateral damage.

VIDEO 17-2 Intraocular foreign body with subhyaloid hypopyon.
Courtesy of Tomas A. Moreno, MD, and Shriji Patel, MD, MBA.

VIDEO 17-3 Removal of an intraocular foreign body using a tipless kidney stone basket.
Courtesy of Shriji Patel, MD, MBA.

Retained intraocular foreign bodies

The reaction of the eye to a retained foreign body varies widely and depends on the object's chemical composition, sterility, and location. Inert, sterile foreign bodies such as stone, sand, glass, porcelain, plastic, and cilia are generally well tolerated. If such material is found several days after the injury and does not appear to create an inflammatory reaction, it may be left in place, provided it is not obstructing vision.

Zinc, aluminum, copper, and iron are metals that are commonly reactive in the eye. Of these, zinc and aluminum tend to cause minimal inflammation (see Fig 17-6) and may become encapsulated. However, any very large foreign body may incite inflammation and thereby cause PVR. Epiretinal proliferations, traction retinal detachment, and phthisis bulbi may result.

Table 17-2 Symptoms and Signs of Siderosis Bulbi

Symptoms
Nyctalopia
Concentrically constricted visual field
Decreased vision
Signs
Rust-colored corneal stromal staining
Heterochromia iridis
Pupillary mydriasis and poor reactivity
Brown deposits on the anterior lens capsule
Cataract
Vitreous opacities
Peripheral retinal pigmentation (early)
Diffuse retinal pigmentation (late)
Narrowed retinal vessels
Optic nerve head discoloration and atrophy
Secondary open-angle glaucoma from iron accumulation in the trabecular meshwork

Chalcosis Pure copper (eg, copper wire) is especially toxic and causes *acute* chalcosis. Prompt removal is required to prevent severe inflammation that may lead to loss of the eye. Foreign bodies with a copper content of less than 85% (eg, brass, bronze) may cause *chronic chalcosis*. Typical findings in chronic chalcosis are deposits in Descemet membrane (a sign similar to the Kayser-Fleischer ring in Wilson disease and the result of copper's affinity for basement membranes), greenish aqueous particles, green discoloration of the iris, petal-shaped deposition of yellow or brown pigment in the lens capsule ("sunflower" cataract), brownish-red vitreous opacities and strand formation, and metallic flecks on retinal vessels and the internal limiting membrane in the macular region.

Siderosis bulbi In siderosis bulbi, iron from IOFBs is deposited primarily in neuroepithelial tissues such as the iris sphincter and dilator muscles, the nonpigmented ciliary epithelium, the lens epithelium (see BCSC Section 11, *Lens and Cataract,* Fig 5-14), the retina, and the RPE. Retinal photoreceptors and RPE cells are especially susceptible to damage from iron (Table 17-2). Electroretinography (ERG) changes in eyes with early siderosis include an increased a-wave and normal b-wave, a progressively diminishing b-wave amplitude over time, and eventually an undetectable signal during the final stage of iron toxicity of the retina (Fig 17-9).

Posttraumatic Endophthalmitis

Endophthalmitis occurs after 2%–7% of penetrating injuries; the incidence is higher in association with IOFBs and in rural settings. Posttraumatic endophthalmitis can progress rapidly. Its clinical signs include marked inflammation featuring hypopyon, fibrin, vitreous infiltration, and corneal opacification. The risk of endophthalmitis occurring after penetrating ocular injury may be reduced by prompt wound closure and early removal of IOFBs. Use of prophylactic subconjunctival, intravenous, or intravitreal antibiotics should be considered.

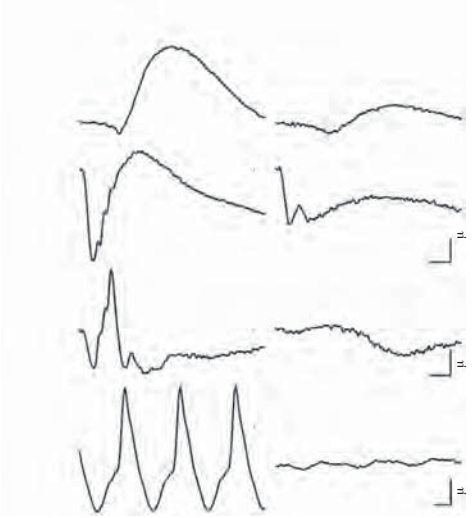

Figure 17-9 Standard full-field electroretinography (ERG) in the left eye of a patient with an iron IOFB removed several years after initial trauma. The ERG demonstrates moderate outer retinal dysfunction (a-wave reduction) and severe inner retinal dysfunction (b-wave loss) in the mixed cone- and rod-mediated responses, causing an electronegative configuration, as well as barely detectable cone responses. *(Reproduced from Sulewski ME Jr, Serrano LW, Han G, Aleman TS, Nichols CW. Structural and electrophysiologic outcomes in a patient with retinal metallosis.* Ophthalmol Retina. *2018;2(2):173–175. Copyright 2018 American Academy of Ophthalmology.)*

Intravitreal or periocular aminoglycoside antibiotics should be avoided because of their high risk of retinal toxicity. Anterior chamber and vitreous cultures should be obtained, and if endophthalmitis is suspected, antibiotics should be injected.

CLINICAL PEARL

- In penetrating or perforating ocular injuries, involvement of the crystalline lens raises the risk of endophthalmitis (even in the absence of an IOFB). A common mechanism is a dirty wire or stick that perforates the cornea, inoculates the anterior chamber or vitreous cavity, and then retracts from the eye. In such cases, there should be a low threshold for intravitreal injection of antibiotics.
- Fluoroquinolones have acceptable vitreous penetration after systemic (intravenous or oral) administration. Levofloxacin 500 mg daily or moxifloxacin 400 mg daily is a good option for treatment of posttraumatic endophthalmitis in addition to intravitreal antibiotics.

Bacillus cereus, which rarely causes endophthalmitis in other settings, accounts for almost 25% of cases of posttraumatic endophthalmitis. Endophthalmitis caused by infection with *B cereus* has a rapid and severe course and, once established, leads to profound vision loss and often loss of the eye. Most commonly, *B cereus* endophthalmitis is associated with injuries caused by soil-contaminated objects, especially in the setting of IOFB. Gram-negative organisms are also frequent pathogens in posttraumatic endophthalmitis. When there is contamination with vegetable matter, posttraumatic fungal infection, most commonly *Candida,* should be considered.

Bhagat N, Nagori S, Zarbin M. Post-traumatic infectious endophthalmitis. *Surv Ophthalmol.* 2011;56(3):214–251.

Jindal A, Pathengay A, Mithal K, et al. Endophthalmitis after open globe injuries: changes in microbiological spectrum and isolate susceptibility patterns over 14 years. *J Ophthalmic Inflamm Infect.* 2014;4(1):5.

Prognostication of Globe Injuries

The severity of the damage to the eye and its function at presentation have prognostic significance. Functional assessments include measuring visual acuity, determining whether an afferent pupillary defect is present, and noting the injury descriptors, specifically the type of trauma and the zone of injury. A higher zone of injury increases the risk for retinal detachment and portends a worse visual prognosis. The Ocular Trauma Score assigns a point value based on these assessments (Table 17-3). In this system, visual acuity is the most important predictor of injury severity; other characteristics are assigned a negative point value that is subtracted from the visual acuity score to produce the total raw score. The higher the raw score, the better the final visual acuity prognosis. This system is a useful general guide to roughly estimate visual acuity outcomes following globe trauma, but the clinician should use it cautiously when counseling patients.

Table 17-3 Calculating the Ocular Trauma Score

Step 1: Record any variables present and their associated raw points.

Variables Used		Raw Points
A. Visual acuity at presentation		
	NLP	60
	LP/HM	70
	1/200–19/200	80
	20/200–20/50	90
	≥20/40	100
B. Rupture		–23
C. Endophthalmitis		–17
D. Perforating injury		–14
E. Retinal detachment		–11
F. Afferent pupillary defect		–10

Step 2: Total the raw points of the applicable variables (A–F) to determine the raw score.

Step 3: Use the raw score to look up the estimate of the likelihood of various final visual acuity outcomes.

Raw Score	NLP	LP/HM	1/200–19/200	20/200–20/50	≥20/40
0–44	74%	15%	7%	3%	1%
45–65	27%	26%	18%	15%	15%
66–80	2%	11%	15%	31%	41%
81–91	1%	2%	3%	22%	73%
92–100	0%	1%	1%	5%	94%

HM = hand motions; LP = light perception; NLP = no light perception.

Adapted from Kuhn F, Maisiak R, Mann L, Mester V, Morris R, Witherspoon CD. The Ocular Trauma Score (OTS). *Ophthalmol Clin North Am.* 2002;15(2):163–165, vi. Copyright 2002, with permission from Elsevier.

Kuhn F, Maisiak R, Mann L, Mester V, Morris R, Witherspoon CD. The Ocular Trauma Score (OTS). *Ophthalmol Clin North Am.* 2002;15(2):163–165, vi.

Pieramici DJ, Au Eong KG, Sternberg P Jr, Marsh MJ. The prognostic significance of a system for classifying mechanical injuries of the eye (globe) in open-globe injuries. *J Trauma.* 2003; 54(4):750–754.

Sympathetic Ophthalmia

Sympathetic ophthalmia is a rare complication of penetrating ocular trauma in which the fellow, uninjured eye develops a severe autoimmune inflammatory reaction. Primary removal of the injured eye is not routinely necessary because the disorder is rare and treatable, and superior cosmetic and psychological outcomes are achieved with globe retention.

Avulsion of the Optic Nerve Head

A forceful backward dislocation of the optic nerve from the scleral canal can occur under several circumstances, including

- extreme rotation and forward displacement of the globe
- penetrating orbital injury, causing a backward pull on the optic nerve
- sudden increase in IOP, causing rupture of the lamina cribrosa

Total loss of vision characteristically occurs. Findings may vary from a pitlike depression of the optic nerve head to posterior hemorrhage and contusion necrosis (Fig 17-10). B-scan ultrasonography may reveal a hypoechoic defect in the area of the posterior sclera in the region of the optic nerve.

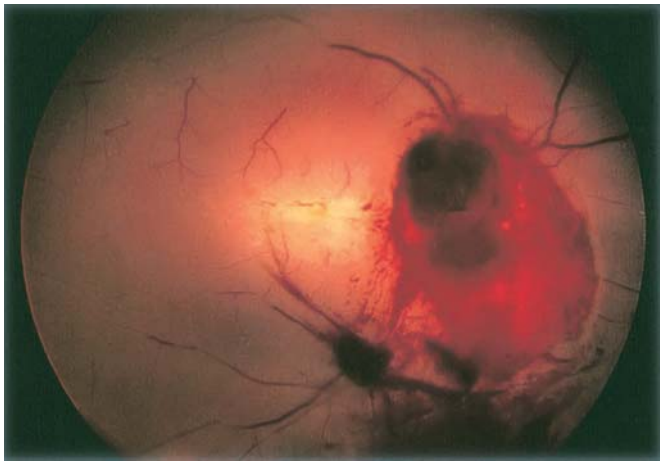

Figure 17-10 Avulsion of the optic nerve head. In this color fundus photograph, the nerve is obscured by hemorrhage, and a mixed vascular occlusion is present.

Abusive Head Trauma

Severe shaking of infants, a form of nonaccidental trauma, is the cause of abusive head trauma (formerly known as *shaken baby syndrome*). Patients with abusive head trauma are usually younger than 5 years and frequently younger than 12 months. The presenting sign of child abuse involves the eye in approximately 5% of cases. Any physician who suspects that child abuse may have occurred is required by law in every US state and Canadian province to report the incident to a designated government agency.

See BCSC Section 6, *Pediatric Ophthalmology and Strabismus,* for discussion of the multiple systemic symptoms associated with abusive head trauma. Ocular signs include

- retinal hemorrhages and cotton-wool spots (Fig 17-11)
- retinal folds
- hemorrhagic schisis cavities
- pigmentary maculopathy

The retinal hemorrhages associated with abusive head trauma often have a hemispheric contour. The retinopathy may resemble that observed in Terson syndrome or central retinal vein occlusion, neither of which is common in infants. Retinal hemorrhages may be present in cases of accidental trauma, blood dyscrasias, vaginal delivery, and sepsis.

Vitrectomy for vitreous hemorrhage should be considered when amblyopia is likely to occur but may be deferred when a bright-flash ERG response shows loss of the b-wave, which is indicative of extensive retinal damage.

Matthews GP, Das A. Dense vitreous hemorrhages predict poor visual and neurological prognosis in infants with shaken baby syndrome. *J Pediatr Ophthalmol Strabismus.* 1996;33(4):260–265.

Pierre-Kahn V, Roche O, Dureau P, et al. Ophthalmologic findings in suspected child abuse victims with subdural hematomas. *Ophthalmology.* 2003;110(9):1718–1723.

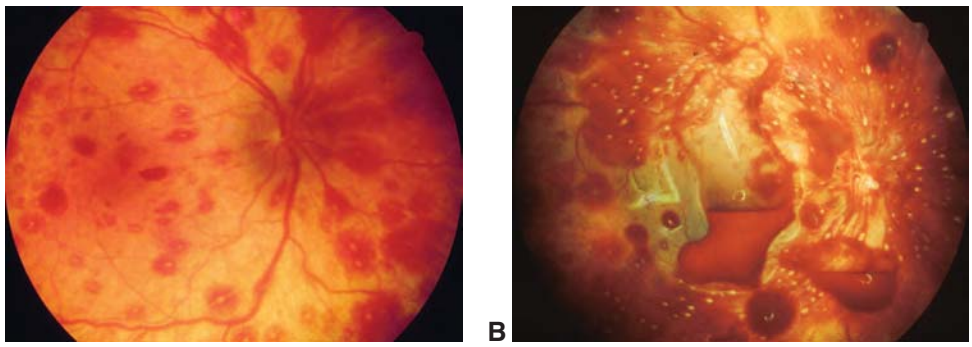

Figure 17-11 Color fundus photographs from a patient with abusive head trauma causing preretinal and retinal hemorrhages. **A,** Image taken several days after hospital admission, by which time many of the smaller hemorrhages had started to resorb. **B,** Numerous hemorrhages are located on and within the retina, and regions of hemorrhagic retinoschisis are observed centrally. Because the baby was upright, the red blood cells sank down into a dependent position within the larger regions of hemorrhagic retinoschisis. Some of the hemorrhages were white centered, whereas others had reflections of the flash from the fundus camera. *(Reproduced with permission from Spaide RF, Swengel RM, Scharre DW, Mein CE. Shaken baby syndrome.* Am Fam Physician. *1990;41(4):1145–1152.)*

Photic Damage

The eye has several mechanisms to protect itself against light damage, including pupil constriction, light absorption by melanin in the RPE, and the presence of antioxidants, such as lutein and zeaxanthin, in the macula. Light injures the retina by 3 basic mechanisms: mechanical, thermal, and photochemical.

Mechanical injury occurs when the power of the absorbed light is high enough to form gas or water vapor or to produce acoustic shock waves that mechanically disrupt retinal tissues. The absorbed energy may be enough to strip electrons from molecules in the target tissue, producing a collection of ions and electrons referred to as *plasma.* For example, a Q-switched Nd:YAG laser produces its therapeutic effect through mechanical light damage and uses this effect to disrupt a cloudy posterior capsule behind an intraocular lens.

Thermal injury occurs when excessive light absorption by the RPE and surrounding structures causes local elevation of tissue temperature, leading to coagulation, inflammation, and scarring of the RPE and the surrounding neurosensory retina and choroid. A therapeutic application of thermal light injury is the retinal burn caused by laser photocoagulation. See Chapter 18 for discussion of photocoagulation.

Photochemical injury results from biochemical reactions that cause retinal tissue destruction without temperature elevation. It is the result of the transfer of light energy to a molecule; the excess energy initiates reactions that cause tissue damage. Damaging reactions can include oxidation, photoisomerization, photochemical cleavage, and electrocyclic reactions. Such changes occur primarily at the level of the outer segments of the photoreceptors, which are more sensitive than the inner segments. Examples of photochemical injury are solar retinopathy and photic retinopathy that occurs after excessive exposure to illumination from an operating microscope.

Mainster MA, Turner PL. Photic retinal injuries: mechanisms, hazards, and prevention. In: Schachat AP, Wilkinson CP, Hinton DR, Sadda SR, Wiedemann P, eds. *Ryan's Retina.* Vol 2. 6th ed. Elsevier/Saunders; 2018:chap 93.

Solar Retinopathy

Solar retinopathy, also known as *foveomacular retinitis, eclipse retinopathy,* or *solar retinitis,* is a thermally enhanced photochemical retinal injury caused by direct or indirect gazing at the sun; it may also occur after viewing a solar eclipse without proper eye protection. The extent of the damage depends on the duration and intensity of the exposure. Younger, emmetropic patients with clearer lenses are at increased risk of solar retinopathy. Symptoms include decreased vision, central scotomata, dyschromatopsia, metamorphopsia, micropsia, and frontal or temporal headache within hours of exposure. Visual acuity is typically reduced to 20/25–20/100 but may be worse depending on the degree of exposure. Most patients recover within 3–6 months, with visual acuity returning to the level of 20/20–20/40, but there may be residual metamorphopsia and paracentral scotomata. Typical findings include a central opacified area of the fovea acutely and hypopigmentation after the acute changes resolve (Fig 17-12). No known beneficial treatment exists, and therefore prevention through education is critically important.

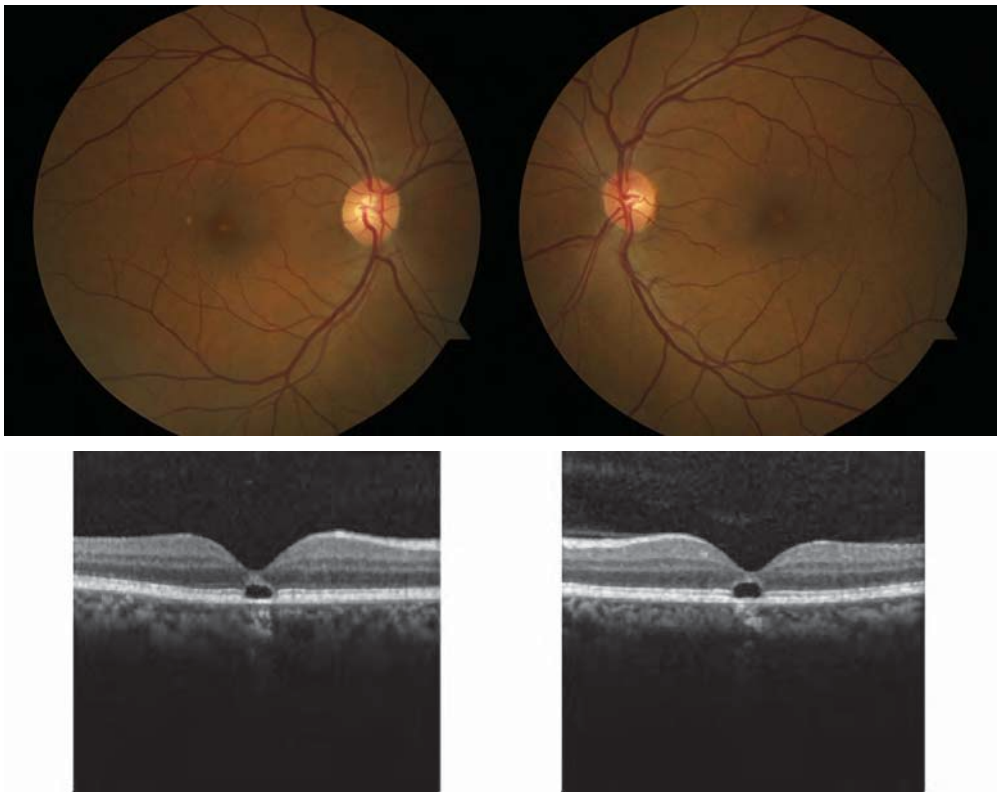

Figure 17-12 Solar retinopathy. The fundus photographs *(top)* show central foveal hypopigmentation. On OCT imaging *(bottom),* outer retinal cavitation in the fovea is typically seen. *(Courtesy of David Sarraf, MD.)*

Comander J, Gardiner M, Loewenstein J. High-resolution optical coherence tomography findings in solar maculopathy and the differential diagnosis of outer retinal holes. *Am J Ophthalmol.* 2011;152(3):413–419.e6.

Phototoxicity From Ophthalmic Instrumentation

There have been reports of injuries from operating microscopes and from fiber-optic endoilluminating probes used in vitrectomies. The incidence of photic retinopathy after contemporary cataract surgery is not known. However, cases continue to be reported after intraocular surgery. The incidence increases with prolonged operating times, but photic retinopathy can occur even with surgery times as short as 30 minutes. In retinal surgery, photic injury is more likely to occur with prolonged, focal exposure, especially when the light probe is held close to the retina, as it may be during macular hole and epiretinal membrane procedures.

Most affected patients are asymptomatic, but some will notice a paracentral scotoma on the first postoperative day. With acute injury, patients may have a deep, irregular yellow-white retinal lesion that is adjacent to the fovea and oval in shape, resembling the shape of

the light source. The lesion typically evolves to become a zone of mottled RPE that transmits background hyperfluorescence on fluorescein angiography.

Reports of photic macular lesions occurring after intraocular surgery underscore the need for prevention. During ocular surgery, the risk of photic retinopathy may be reduced by minimizing exposure time; avoiding the use of intense illumination; using oblique illumination when possible during parts of the surgery; filtering light wavelengths below 515 nm; and, when possible, using pupillary shields. See also BCSC Section 3, *Clinical Optics and Vision Rehabilitation*.

van den Biesen PR, Berenschot T, Verdaasdonk RM, van Weelden H, van Norren D. Endoillumination during vitrectomy and phototoxicity thresholds. *Br J Ophthalmol.* 2000;84(12):1372–1375.

Youssef PN, Sheibani N, Albert DM. Retinal light toxicity. *Eye (Lond).* 2011;25(1):1–14.

Occupational Light Toxicity

Occupational exposure to bright lights can lead to retinal damage. A common cause of this type of occupational injury is arc welding without the use of protective goggles. The damage from the visible blue light of the arc welder leads to photochemical damage similar to that observed in solar retinopathy. Occupational injury from stray laser exposure is also a serious concern.

Laser-Pointer Injury

The availability of high-power green and blue handheld laser devices has created a source of immediate accidental or purposeful sight-threatening macular injury (Fig 17-13). These

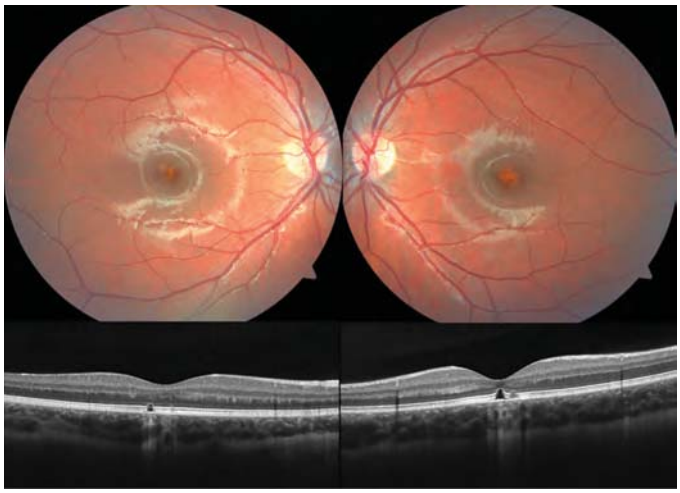

Figure 17-13 Fundus photographs and SD-OCT images from an 11-year-old boy with foveal atrophy and a focal, well-circumscribed area of photoreceptor loss bilaterally, which was due to prolonged laser-pointer exposure. *(Reproduced from Snyder L, Patel S. Laser pointer maculopathy.* Ophthalmol Retina. *2018;2(10):996. Copyright 2018, American Academy of Ophthalmology.)*

devices are easily obtainable via the internet and resemble the much safer, ubiquitous low-power red laser pointers. The devices also have the potential to cause secondary harm by visually incapacitating individuals while they are performing visually demanding functions, such as firefighting and piloting planes.

Alsulaiman SM, Alrushood AA, Almasaud J, et al; King Khaled Eye Specialist Hospital Collaborative Retina Study Group. High-power handheld blue laser–induced maculopathy: the results of the King Khaled Eye Specialist Hospital Collaborative Retina Study Group. *Ophthalmology.* 2014;121(2):566–572.e1.