Chapter 12

BLUNT INJURY OF THE POSTERIOR SEGMENT

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INTRODUCTION

Instruction in the management of ocular trauma usually emphasizes dramatic injuries such as penetration of the eye wall, retention of foreign bodies, and associated adnexal consequences. Nonpenetrating injuries, however, are far more common and can be disastrous to visual function. They have been consistently documented as a wartime injury, both from direct actions of combat and from the associated industrial hazards of the support environment.[1,2]

These blunt injuries may challenge the diagnostic acumen of the clinician and pose vexing therapeutic questions. In addition, the eye may display significant consequences in the fundus of injuries that are completely remote from the globe. The ophthalmologist treating combat casualties will benefit from a review of posterior segment manifestations of such injuries, including injury mechanisms, damage to the various tissue layers, and the sequelae of remote injury.

MECHANISM OF POSTERIOR SEGMENT INJURY

Direct blows to the eye transmit energy to the adjacent tissues as they produce compression, shearing, and tensile strains. The globe and orbital anatomy predispose the temporal fundus to such direct contact, although the variety of methods and locations of ocular injury seem unlimited. The terms *coup* and *contrecoup*[3] have been used to distinguish the effects of trauma on immediately adjacent tissues (coup) from the effects of forces transmitted to more distant portions of the eye (contrecoup). Clinical and experimental observations on such transmitted forces in the eye and in the brain confirm a consistent pattern of injury location at interfaces with significant differences in density (impedance mismatch). Common ocular sites are the lens–vitreous interface and the posterior vitreo–retinal–scleral interface.

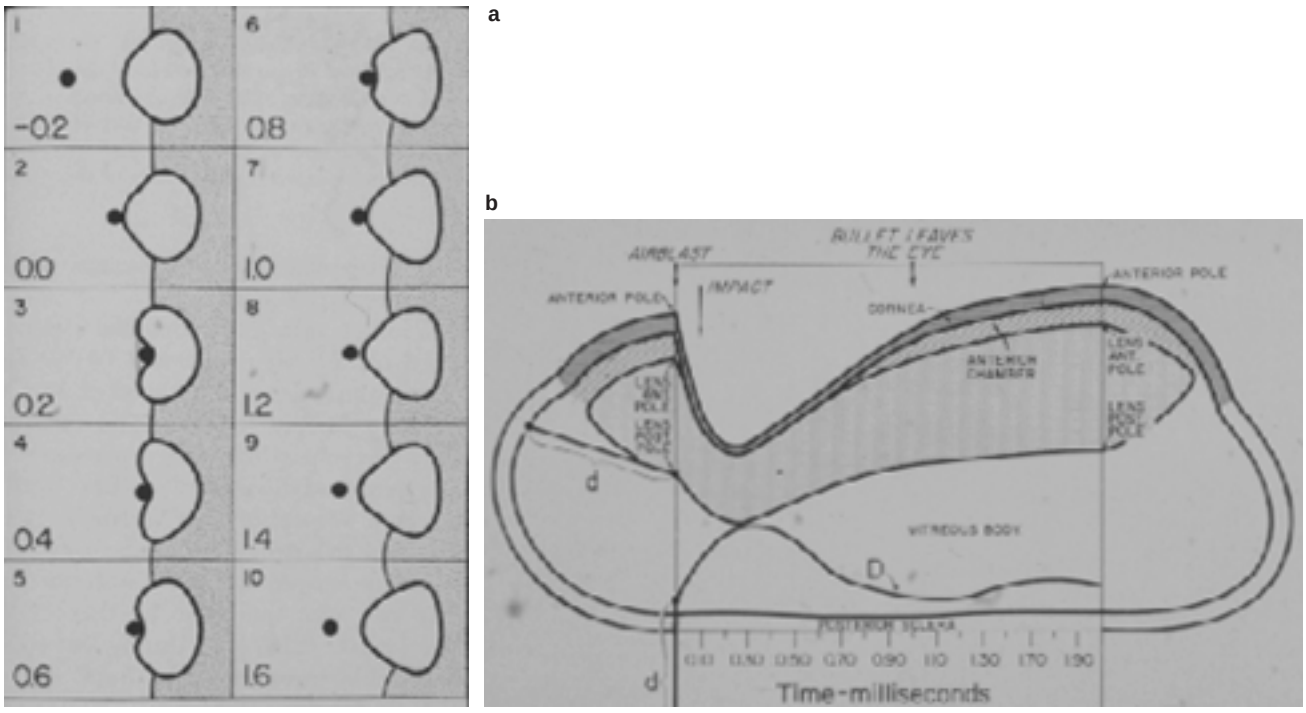


Fig. 12-1. (**a**) Schematic illustration of globe deformation sequence during high-speed impact and recoil (in milliseconds) demonstrates the occurrence of dramatic axial compression and transverse elongation, placing tension on the vitreous base. (**b**) The posterior indentation of anterior segment structures is accompanied by a simultaneous equatorial expansion. The vitreous base dimension *D* is maximally elongated at approximately 0.30 milliseconds, exerting forces on the peripheral retina that may produce retinal dialysis. Reproduced with permission from Delori F, Pomerantzeff O, Cox M. *Invest Ophthalmol.* 1969;8:294, 300.

Delori, Pomerantzeff, and Cox[4] studied globe deformation and injury under high-speed impact and were able to document the dramatic difference the speed of injury makes in ocular trauma. Ocular tissues are viscoelastic materials that exhibit the phenomenon of rate dependence. This means that "increased deformation rates result in increased levels of stress in the deformed tissues."[5(pp197–198)] For example, high-velocity projectiles caused retinal dialyses and other injuries, while low-velocity deformations—even with greater amounts of energy—caused no equivalent peripheral fundus injury.

High-speed impact creates impressive ocular indentation and other deformation (Figure 12-1). The impact site is displaced posteriorly while the globe's equatorial diameter dramatically expands. A 28% expansion of the vitreous base radius was documented 0.4 milliseconds after deformation begins.[4] The indentation process is followed, in nonpenetrating injury, by a recovery (recoil) that includes dampening oscillations of these structures. In addition, the anterior structures are displaced posteriorly during the first 0.3 milliseconds, followed by a more-gradual recovery phase with some overshoot displacement anteriorly. Measurements of the vitreous base (from the posterior pole of the lens to the insertion of the vitreous base) are maximal during the peak corneal indentation. The results include impressive inward tension exerted on the vitreous base, which is opposed by the high intraocular pressure that compresses the retina against the choroid. The resultant great shearing forces are usually strongest at the posterior border of the vitreous base, and a linear tear of the retina may result. If traction is strongest at the anterior border of the vitreous base, the nonpigmented ciliary epithelium is torn. Strong traction at both the anterior and posterior borders may produce vitreous base avulsion, which is pathognomic of ocular contusion.

Weidenthal and Schepens[6] used an experimental model that controlled these globe distortions by encasing the globe in rigid shells of variable geometry. When the equatorial expansion of the globe following impact was prevented, the damaging effects on the peripheral retina were dramatically reduced. Because these direct and indirect forces affect each tissue differently, the resulting injuries are discussed in this chapter based on the specific tissue layers involved.

RETINAL INJURY WITHOUT BREAKS

In 1873, Berlin[7] described retinal opacity (commotio retinae) following blunt ocular trauma that was assumed to represent extracellular retinal edema. However, Sipperley[8] documented in an owl monkey injury model that the outer retinal opacification, or whitening, corresponded with photoreceptor outer segment disruption (Figure 12-2). Histological evidence indicated that neither intracellular nor extracellular edema was present. This photoreceptor disruption was followed by phagocytosis of the fragmented outer segments by cells of the retinal pigment epithelium (RPE) and subsequent migration of these cells into the retina. Fluorescein angiography and vitreous fluorophotometry

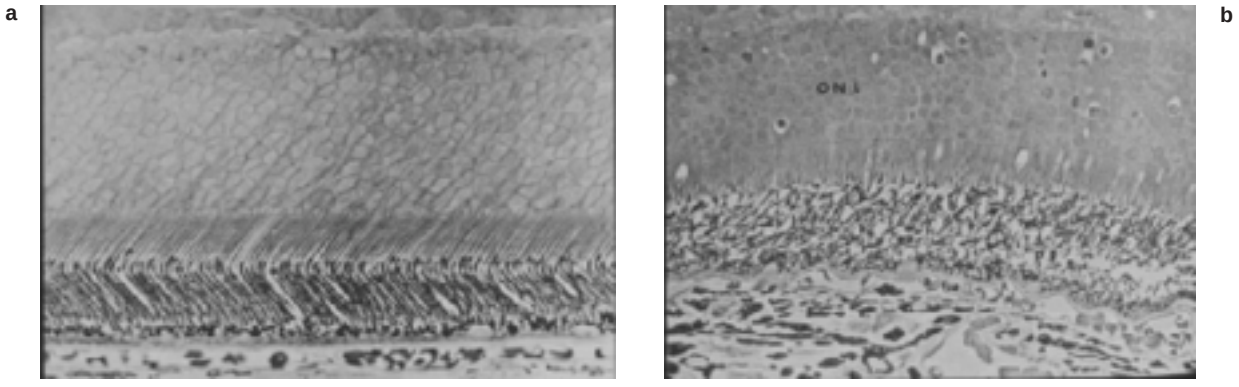
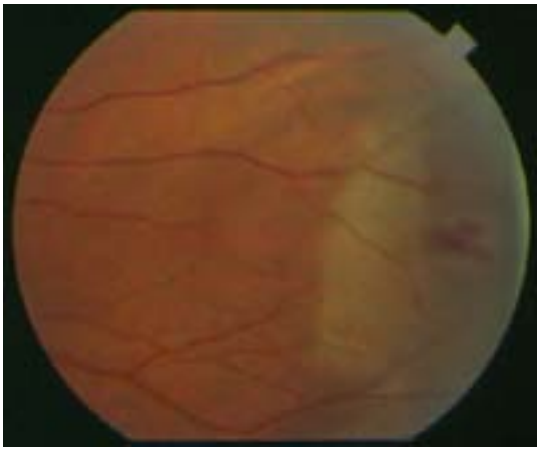


Fig. 12-2. Photomicrographs of (**a**) a normal owl monkey retina, contrasting with (**b**) the damage observed to the experimental animal 4 hours after blunt trauma. The photoreceptor outer segments are disrupted and disorganized, and the outer nuclear layer (ONL) demonstrates pyknotic nuclei. The pathological appearance corresponds with clinically observed retinal opacification. Reproduced with permission from Sipperley JO, Quigley HA, Gass JDM. *Arch Ophthalmol.* 1978;96:9226.

Fig. 12-3. Commotio retinae observed in the peripheral fundus of a patient seen 1 day following blunt injury. The zone of whitened retina is characteristically sharply demarcated, and associated retinal hemorrhage is seen. The outer retinal whitening obscures choroidal detail, but the view of the more superficial retinal vessels is unaffected.



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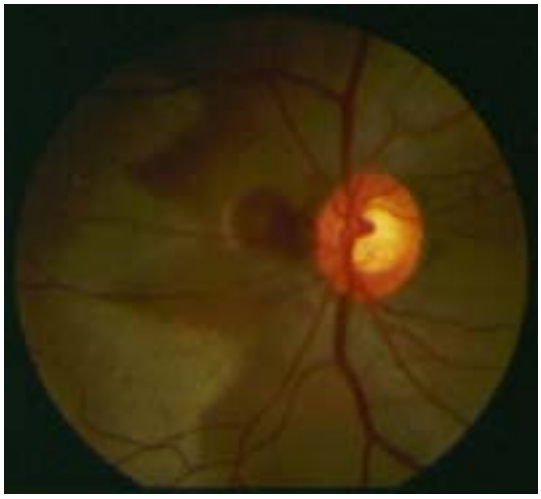
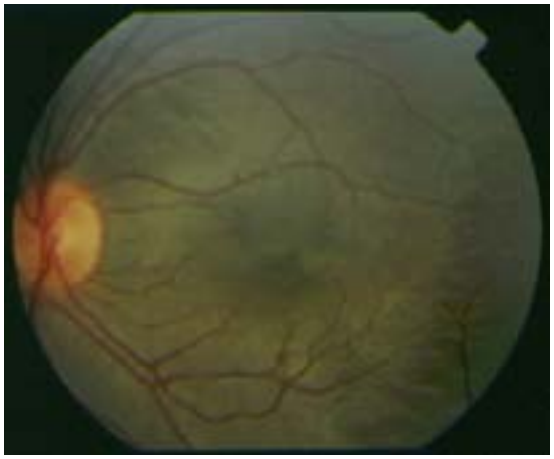




b

Fig. 12-4. (**a**) Retinal opacification in the posterior pole reveals a typical gray-white hue and sharply demarcated margins, which here have assumed a peculiar geometric pattern of scalloping. Peripapillary hemorrhage is also seen. (**b**) Fundus appearance 3 days after blunt injury reveals a large zone of outer retinal whitening affecting the macula, with sharp, geometric borders. The patient's vision was reduced to 20/400.

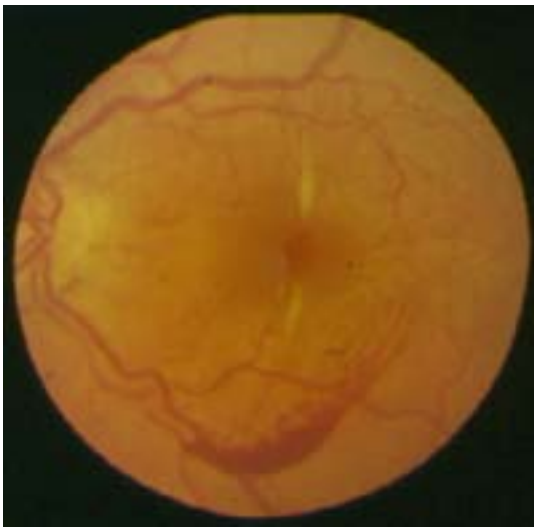


Fig. 12-5. This macular hole was noted 1 week after blunt injury. A macular hole may be seen as an immediate consequence of trauma or observed after a latent interval. Patients with an interval of better vision prior to macular hole development are more likely to benefit from macular hole surgery.

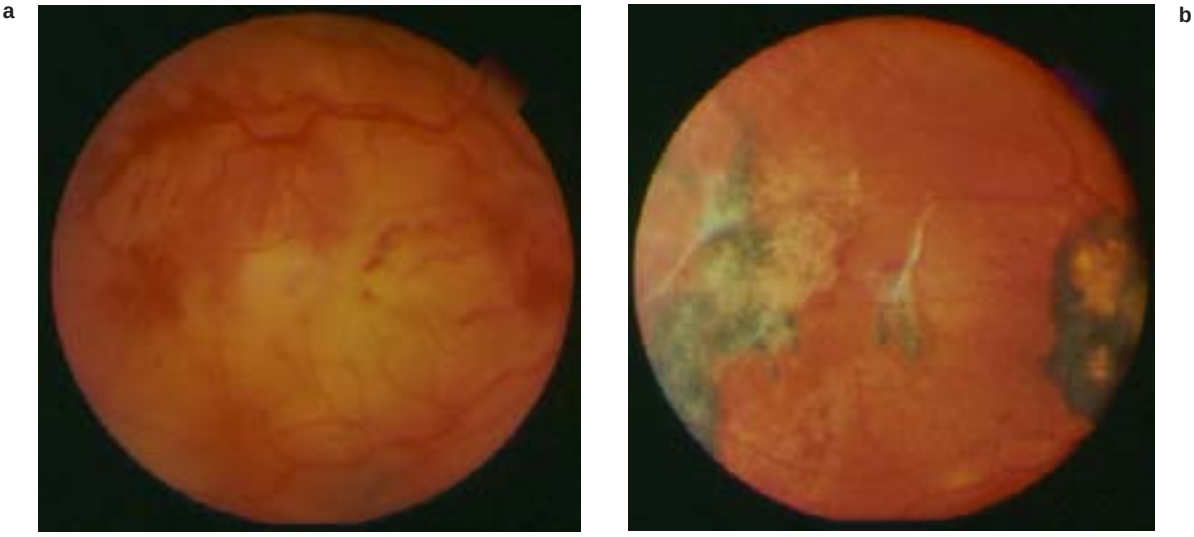


Fig. 12-6. (**a**) Retinal pigment epithelial contusion injury shows a cream-colored change at the level of the retinal pigment epithelium (RPE), associated with shallow overlying neurosensory retinal detachment and areas of retinal and vitreous hemorrhage. (**b**) By 4 months after the injury, the same patient had widespread retinal pigment epithelial hyperplasia and migration. Vision remained stable at 20/400.

confirmed the intact blood–retinal barrier.

Clinically, opacification may develop peripherally,[9] often at the site of impact (Figure 12-3) or in the posterior pole (Figure 12-4). The patient's visual loss, which depends on the location and the degree of the photoreceptor disruption, occurs promptly, and delayed visual loss should lead to investigation for other origins. The peripheral opacification may resemble either white without pressure or retinal detachment. Because foveal anatomy essentially includes only photoreceptors and their axons, extensive foveal photoreceptor loss results in significant risk of macular hole formation (Figure 12-5). Contusion of the RPE may present a confusing differential diagnosis. This cream-colored change in the RPE develops within 48 hours of injury (Figure 12-6), shows leakage and staining on fluorescein angiography, and may have serous elevation of the RPE or neurosensory retina. Resolution of edema and elevation is commonly accompanied by development of pigmentary migration and hyperplasia.

RETINAL BREAKS

Retinal breaks may be subdivided into two categories: (1) those caused by *vitreous traction*, which have such manifestations as dialyses, horseshoe tears, or operculated tears, and (2) those *without apparent vitreous traction*, with typical appearances including inferotemporal retinal defects of varied size.

Breaks Due to Vitreous Traction

The most common break produced by traumatic vitreous traction is the retinal dialysis. Tension on the vitreous base may result in tears at any location, but the inferotemporal and supranasal quadrants strongly predominate, representing approximately 60% of all traumatic retinal tears. Several clinical series[10,11] have demonstrated that the inferotemporal quadrant is most commonly affected; however, the presence of a clear history of antecedent trauma was less consistent than that noted in patients with supranasal dialyses. This distinction has suggested an anatomical predisposition of the inferotemporal quadrant to develop dialysis in milder injuries. Such a tendency—or even the possible spontaneous occurrence of such defects—may contribute to the bilaterality of inferotemporal dialyses, emphasizing the critical need for the examiner to study the peripheral fundus of both eyes in every patient with an eye injury.

Vitreous base avulsion is present in about 15% of dialysis patients but is pathognomic of trauma when present; multiple small dialyses or giant retinal tears may also be seen (Figure 12-7).

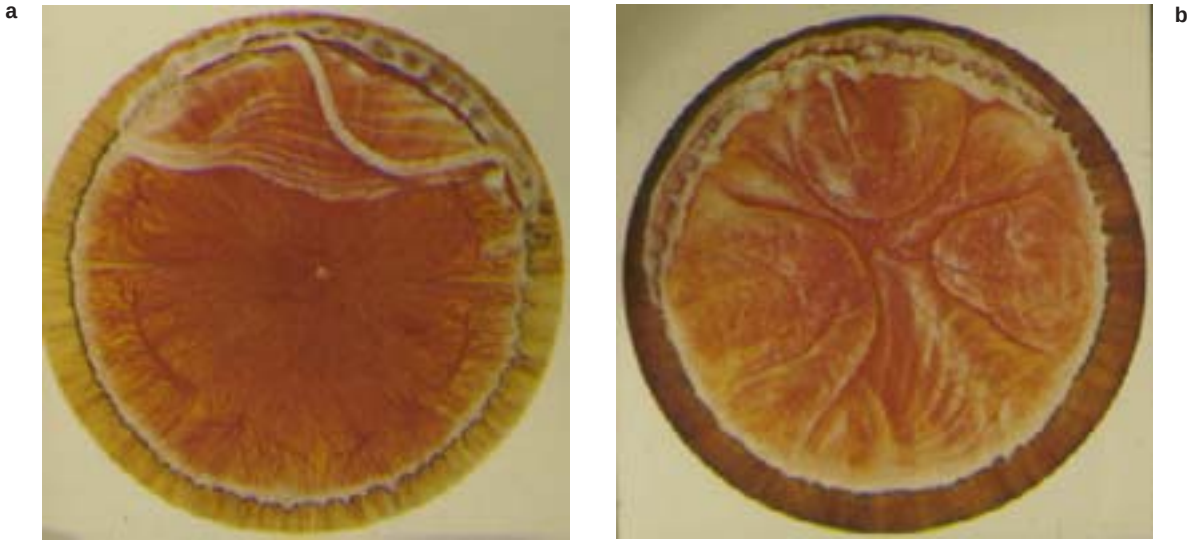


Fig. 12-7. Retinal breaks following contusion injury. (**a**) Supranasal retinal dialysis. The "bucket handle" avulsion of the vitreous base is pathognomic of trauma. Retinal tearing is seen both at the ora serrata and in the nonpigmented ciliary epithelium of the pars plana. (**b**) Retinal detachment. Careful peripheral fundus scrutiny with scleral depression is required to demonstrate the responsible linear tears located anterior to the ora serrata. Reproduced with permission from Weidenthal DT, Schepens CL. *Am J Ophthalmol.* 1966;62:465–477.

Detachment caused by dialysis occurs immediately following ocular contusion only in 12% of cases and typically presents after a latent interval: within 1 month (30% of cases), within 8 months (50% of cases), or within 2 years (80% of cases).[12] This delay in presentation affords an opportunity for effective prophylactic therapy using cryopexy or photocoagulation. When detachment does develop from dialysis, it has characteristics that differentiate it from nontraumatic rhegmatogenous detachments. These detachments are seldom bullous; they are usually smooth, thin, and transparent. Star folds or other stigmata of proliferative vitreoretinopathy (PVR) are rare. Features of chronicity are common (Figure 12-8), such as intraretinal cysts (20%) and demarcation lines (50%, often multiple). Detachment from dialysis is not related to refractive error. It seldom flattens with bed rest, so activity or position restrictions during transport of such patients are normally not valuable. The surgical prognosis is generally very good.

Tension on the vitreous base may also produce more-typical flap (horseshoe) or operculated breaks, usually at the posterior margin of the vitreous base or at the site of abnormal vitreoretinal attachments.

Fig. 12-8. Artist's depiction of typical findings of retinal detachment due to inferotemporal retinal dialysis. Parallel rows of demarcation lines ("high-water marks") are seen, along with retinal macrocyst formation. The absence of features of proliferative vitreoretinopathy is characteristic. Reproduced with permission from Hagler WS, North AW. *Arch Ophthalmol.* 1968;79:381.

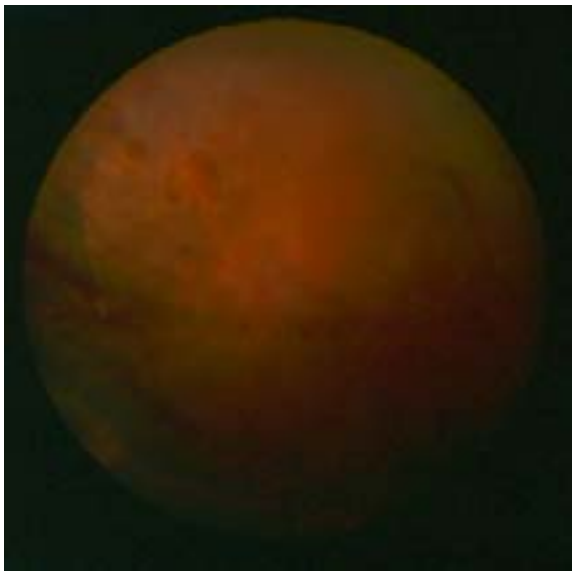


Fig. 12-9. A cluster of small, round retinal holes with localized retinal detachment is noted in the temporal periphery of an 11-year-old patient seen 1 week after injury from a soccer ball. Although the retinal holes are produced at the time of impact, the development of retinal detachment from traumatic holes may be delayed by a lengthy latent interval, or detachment may not occur at all.

Breaks Without Apparent Vitreous Traction

Traumatic retinal breaks may also occur in a sectoral or ovoid configuration, usually inferotemporal.[13] These defects are associated with extensive hemorrhage, ragged edges, and fragments of retina suspended in the overlying vitreous. Configurations vary from clustered, small, round holes (Figure 12-9) to a single, enormous defect (Figure 12-10). Choroidal ruptures may accompany the lesions, and the retina itself may be opaque. (Although the kinds of injuries seen in Figures 12-13 and 12-14 were initially [historically] suspected of being due to necrosis or a delayed effect of hemorrhage, they seem on reflection to be an immediate consequence of severe rapid deformation.) The retina and often the choroid are ruptured and both may retract leaving a zone of exposed bare sclera. (The choroid is not well anchored to the sclera in the anterior fundus.) The overlying hyaloid is usually not ruptured, explaining the usual absence of retinal detachment at presentation. Late detachment may occasionally develop, especially in association with PVR, but recent evidence[5] suggests that prophylactic treatment of these often very large breaks is ill-advised.

A macular hole may occur following contusion necrosis[14] or as a consequence of hemorrhage or vitreous traction.[15] Unlike spontaneous macular holes, the presence of associated photoreceptor disruption (suggested by severe vision loss immediately following the injury) may limit the value of any surgical intervention. In contrast, patients exhibiting visual decline due to delayed macular hole development may benefit from surgical efforts to close the macular hole.[16]

Traumatic retinal breaks generally occur at the time of injury. The production of these varied types of breaks seems to depend largely on the point of impact and the speed of deformation. The locations

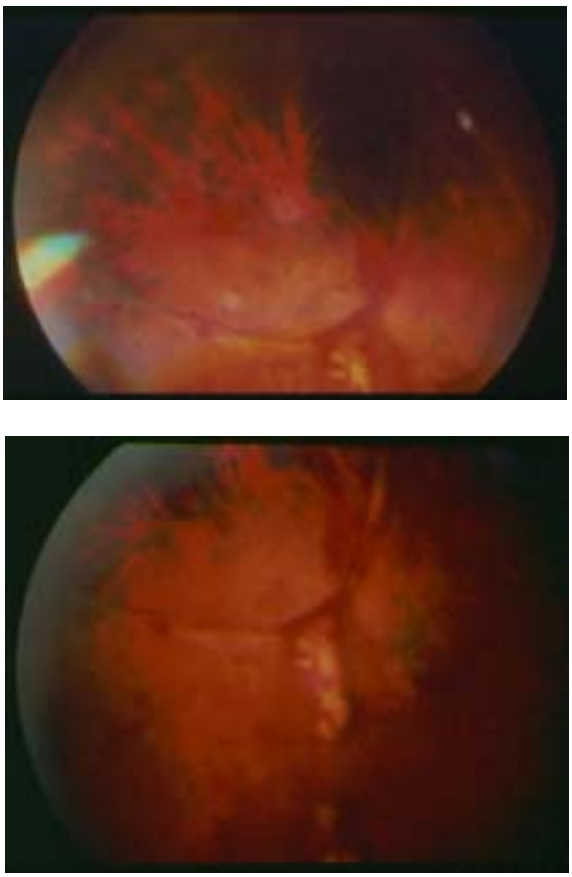


Fig. 12-10. Enormous retinal defect seen in the supratemporal periphery of a 22-year-old patient examined 1 day after being struck in the eye during a basketball game. (**a**) Note that the underlying choroid is intact in contrast to the defects seen in sclopetaria injuries. The retina remains attached. (**b**) Posterior extent of the same injury. Note the clumped material within the vitreous, which seems to descend from the retinal injury. This material may include blood, fibrin, and remnants of disrupted retina. The retina remained attached without treatment.

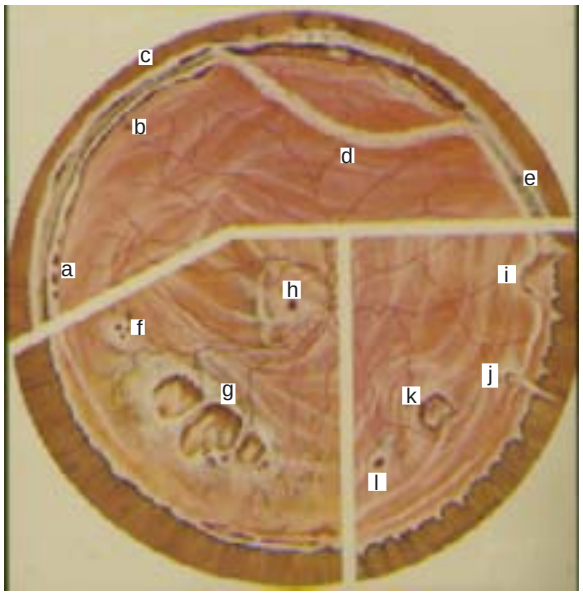


Fig. 12-11. This composite illustration depicts the variety of retinal breaks seen following contusion injury. The top portion depicts tears of the (**a**) anterior and/or (**b**) posterior margins of the vitreous base, including (**c**) complete dialysis, (**d**) small consecutive breaks, and (**e**) avulsion of the vitreous base. The lower left portion depicts breaks without apparent relationship to vitreous traction, including (**f**) small clustered round holes, (**g**) large ovoid breaks, and (**h**) a macular hole. The lower right portion **(i-l)** shows traumatic breaks associated with abnormal vitreoretinal traction, resulting in defects indistinguishable from most nontraumatic breaks. Reproduced with permission from Cox M, Schepens C, Freeman H. *Arch Ophthalmol.* 1966;76:678–685.

of retinal breaks in contusion injuries are elegantly summarized in Figure 12-11.

Head trauma without associated ocular trauma has been alleged to induce retinal breaks. Although such injury may be the precipitating event in some predisposed individuals, a clinical study of 247 cases of severe head trauma found that no retinal breaks were induced.[17]

CHOROIDAL RUPTURE

Choroidal rupture is a common manifestation of blunt injury, reflecting the mechanical vulnerability of Bruch's membrane. The retina is relatively elastic and the sclera mechanically strong, but Bruch's membrane does not share these characteristics. When ruptured, the choriocapillaris and the RPE also tear, and injury may extend through the full thickness of the retina.[18] Choroidal ruptures may be seen in the peripheral fundus (Figure 12-12), where they are caused by adjacent injury forces, or in the posterior pole (Figure 12-13), where indirect forces are transmitted to produce injury that is usually concentric to the optic nerve and temporally located.[19] The visual prognosis is variable and depends on the location, extent of rupture, associated hemorrhage, and subsequent fibrosis or vascularization. Ruptures may initially be obscured by extensive hemorrhage, which can also detach the choroid or retina or dissect into the vitreous (Figures 12-14 and 12-15). The proper therapy for these extensive hemorrhages remains controversial. Most such hemorrhages clear spontaneously, but poor vision following extensive hemorrhage under the fovea may be the result of the initial injury itself or due to photoreceptor loss or fibrosis caused by the hemorrhage.

Vitreous surgery to remove these extensive hemorrhages has significant associated risks and often is unsuccessful in fully evacuating the hemorrhage. The use of pneumatic displacement techniques, with or without intravitreous tissue plasminogen activator (tPA) injection to promote clot lysis, may successfully diminish foveal hemorrhage with less surgical risk.[20] A recently suggested approach[21] utilizes gas injection and positioning alone, followed in 24 to 48 hours by intravitreal tPA injection if hemorrhage is not displaced from the fovea. This approach is particularly appealing for the military ophthalmologist with limited equipment, although the impact of intraocular gas on subsequent aeromedical transport must be considered.

Neurosensory retinal detachments may be noted shortly after injury in some patients with extensive choroidal ruptures (Figure 12-16), but these normally resolve after several days. Late development of neurosensory detachment may herald subretinal neovascular membrane formation (Figure 12-17). Focal photocoagulation is usually appropriate for neovascular membranes not located under the foveal center.

Chorioretinitis sclopetaria is a term introduced into the German literature to describe a concussion injury to the globe that occurs adjacent to the path of an orbital missile (Figure 12-18). The term probably relates to

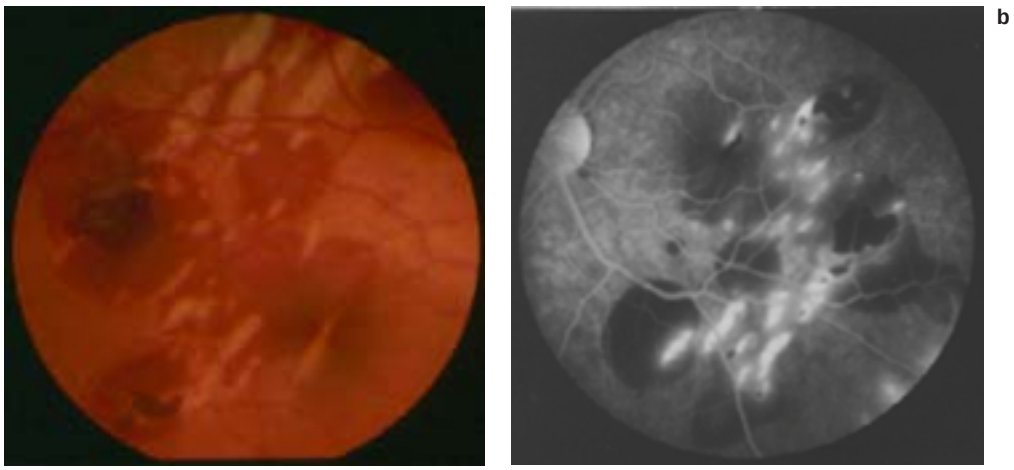


Fig. 12-12. Following contusion injury to the temporal globe, (**a**) multiple choroidal ruptures are seen and extend from the temporal macula to the equator. The linear ruptures are accompanied by subretinal and preretinal hemorrhage. (**b**) The fluorescein angiography of the fundus demonstrates intense fluorescence at sites of choroidal rupture that leaks into adjacent pools of hemorrhage and a shallow neurosensory retinal detachment. This fluorescein leakage typically ceases within several weeks, but transmission defects and blocking defects may remain indefinitely.

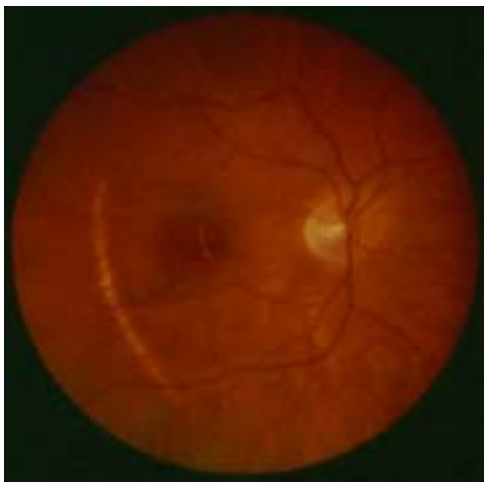


Fig. 12-13. Late appearance of two choroidal ruptures in the posterior pole. The curvilinear geometry is characteristic. Final visual outcome may depend primarily on the location of injury, such as the small foveal rupture seen here.

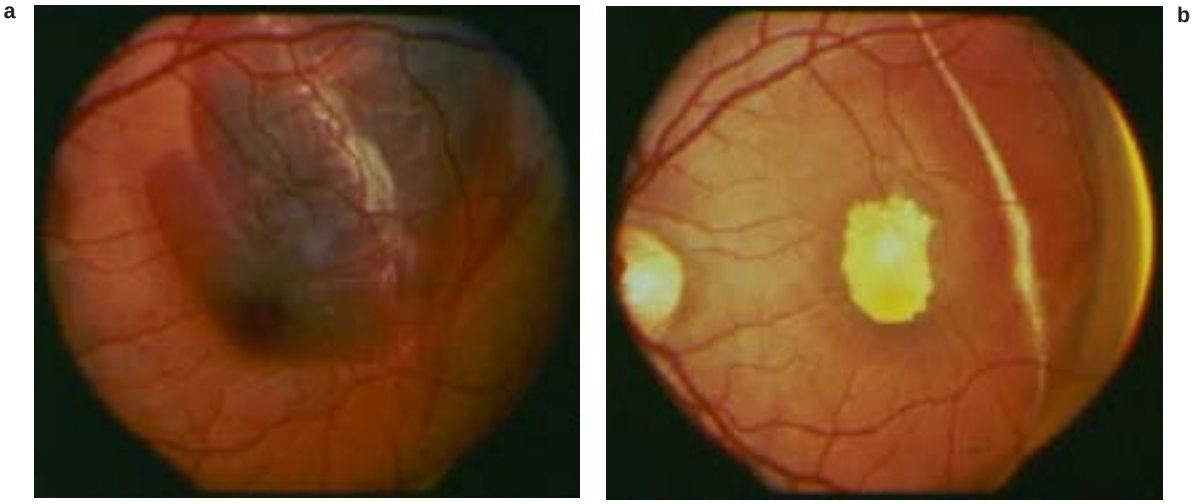


Fig. 12-14. (**a**) Large subretinal hemorrhage in the posterior pole following contusion injury extends to the foveal area. The underlying choroidal rupture responsible for the hemorrhage is largely obscured by the blood. (**b**) One month after the injury, the hemorrhage has cleared, exposing a large temporal choroidal rupture. A central macular scar and collection of absorbing hemorrhage elements is associated with persistently reduced vision of 20/400.

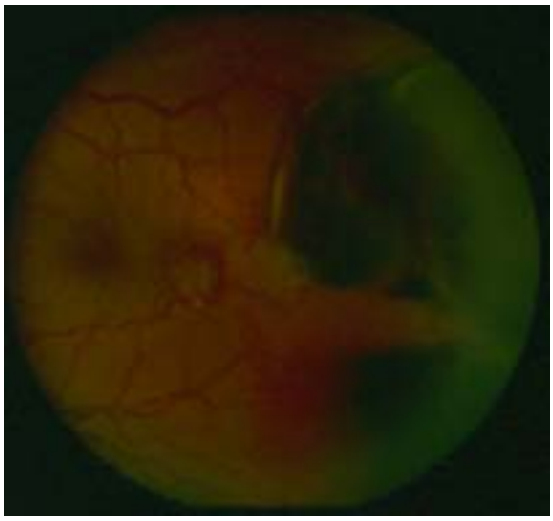


Fig. 12-15. A large, bullous subretinal hemorrhage is present in the temporal fundus obscuring the underlying choroidal ruptures. A meniscus separating blood and clear subretinal fluid is visible in the inferior balloon of hemorrhage. The fovea is spared from the hemorrhage, and visual acuity was 20/20.

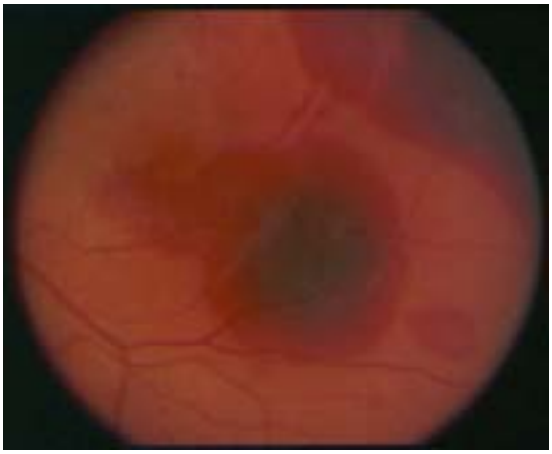
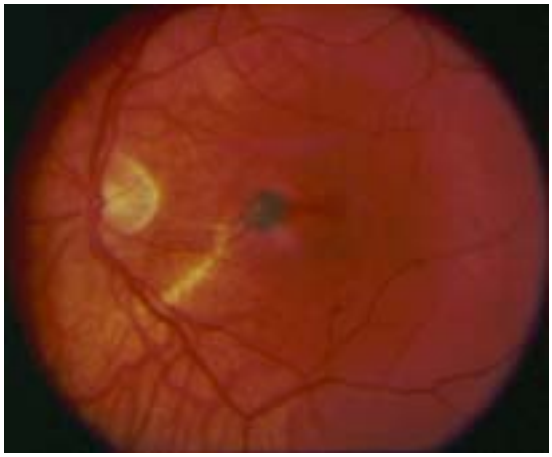


Fig. 12-16. Multiple, widespread bilateral choroidal ruptures are seen in a patient injured by a bomb blast. The partially obscured ruptures are accompanied by shallow neurosensory retinal detachments. Retinal striae have produced a fingerprint pattern to the shallow macular hemorrhage. The hemorrhage and detachments resolved over 1 month.

a



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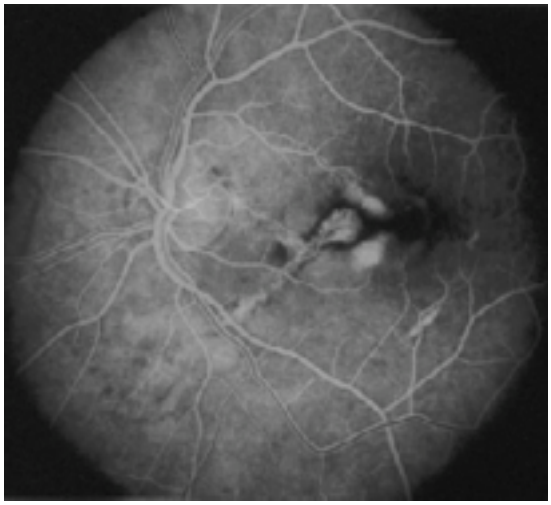


Fig. 12-17. A 23-year-old Marine noted reduced vision 11 months after being struck in the eye. (**a**) Neurosensory retinal detachment and subretinal hemorrhage were present. A previously unrecognized choroidal rupture is visible inferotemporal to the disc, with a choroidal neovascular membrane extending from the rupture to the edge of the fovea. (**b**) Fluorescein angiography of the fundus reveals a well-defined, classic, juxtafoveal neovascular membrane arising from the choroidal rupture site. The neovascularization was successfully treated with argon laser photocoagulation.

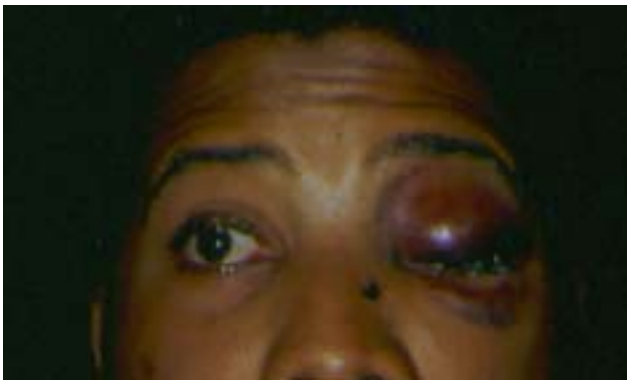


Fig. 12-18. A gunshot wound of the nasal orbit produced marked orbital edema and ecchymosis. The missile path avoided direct contact with the globe but resulted in retinitis sclopetaria injury to the peripheral fundus.

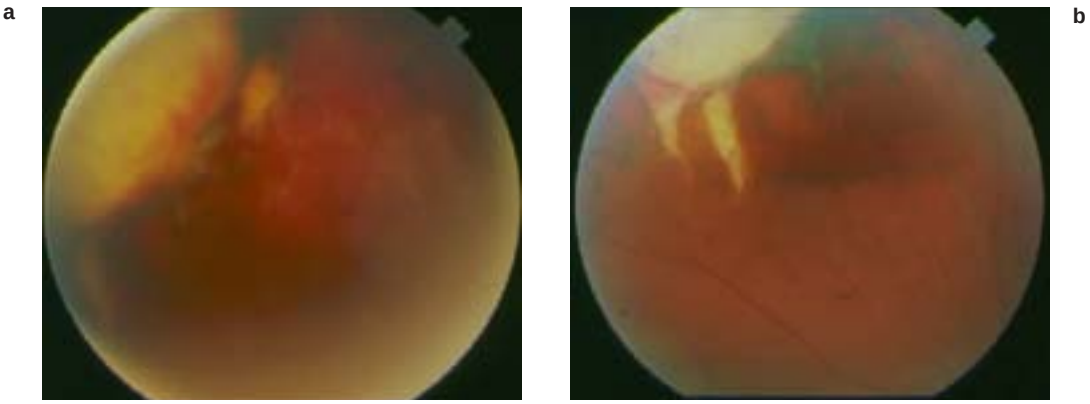


Fig. 12-19. A 20-year-old patient sustained an orbital missile wound that resulted in retinitis sclopetaria. (**a**) The large defect in the retina and choroid has a characteristic rolled and thickened posterior margin, representing retracted tissue. Despite the size of the defect, no treatment was applied. (**b**) The fundus is seen 6 weeks later, with maturation of the scarring process. The retina remained attached. Characteristic radial, claw-like scars are noted extending posteriorly from the large retinal and choroidal defect.

an old description of the claw-like breaks in Bruch's membrane and the RPE noted in many patients. Associated rupture and retraction of both choroid and retina may accompany severe injuries, but the posterior hyaloid commonly remains intact, substantially reducing the likelihood of retinal detachment.[5] Despite the impressive appearance of the retinal defects (Figure 12-19), treatment usually is not required.

SCLERAL RUPTURE

Scleral rupture must be strongly suspected in every significant contusion injury, and surgical exploration must be strongly considered even in the absence of hypotony.[22] Characteristic presenting features include very poor vision (hand motions [HM], light perception [LP]), marked hemorrhagic chemosis, severe hyphema, lacrimation, and loss of ocular motility (Figure 12-20).[23,24]

Optic nerve avulsion from the sclera (Figure 12-21) is an uncommon but disastrous event that has been well documented as a wartime injury.[1] The avulsion can be partial or complete with varying degrees of intraocular hemorrhage. The nerve head may be absent and the retinal circulation arrested. Imaging of the orbit reveals that the optic nerve sheath is usually intact.[25] No therapy is beneficial.

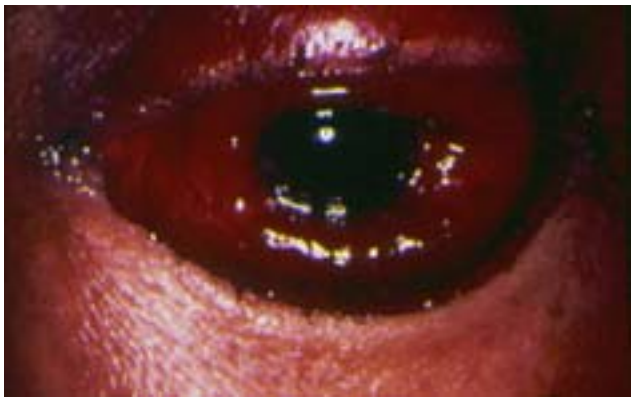


Fig. 12-20. A patient who sustained a blunt injury to the globe has massive hemorrhagic chemosis and a total "eight-ball" hyphema. Reduced ocular motility was noted. Surgical exploration disclosed a large posterior scleral rupture.

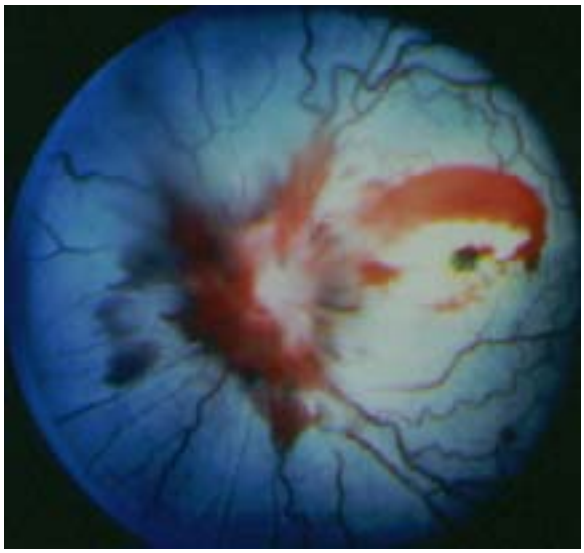


Fig. 12-21. Peripapillary and retinal hemorrhage, retinal edema, and arterial circulatory obstruction are seen in a patient with avulsion of the optic nerve. The dramatic variation in the inferotemporal diameter of the retinal arterioles confirms the interruption of the arterial circulation.

POSTERIOR SEGMENT EFFECTS OF REMOTE TRAUMA

Circulatory Influences

Remote effects may reflect venous, arterial, and mixed circulatory influences on the posterior segment. Valsalva retinopathy[26] follows a sudden rise in intraabdominal or intrathoracic pressure, including performance of Valsalva's maneuver. The rapid rise of venous pressure in the eye reflects the absence of competent venous valvular protection. Patients may have a history of coughing or straining, or they may not be able to relate the visual symptoms to any activity.

Rupture of retinal capillaries produces typical posterior pole preretinal hemorrhage (Figure 12-22), which may detach the internal limiting membrane. Intraretinal hemorrhage or vitreous hemorrhage may be seen. A fluid level (Figures 12-23 and 12-24) often develops over days before gradual, spontaneous clearing of the blood. Vision usually returns to normal. Investigation for predisposing circulatory defects may be appropriate, especially in the absence of a supportive history, but is commonly unrewarding. An extreme instance of venous pressure increase occurs in the compression cyanosis syndrome[27] (Figure 12-25), originally described as a consequence of bail-out from high-speed aircraft but now recognized in many traumatic settings where chest compression occurs, such as in trampling injuries.

Fat Embolism

Fat embolism occurs in approximately 5% of patients with fractures of the long bones and may be fatal in severe cases. About half of affected patients have retinal manifestations, including nerve fiber layer infarcts and blot hemorrhages (Figure 12-26). Recognition of the ophthalmic features can help establish the diagnosis and guide therapy of this condition.

Purtscher's Retinopathy

Purtscher (as cited in Marr and Marr[28]) described a retinopathy occurring after severe head trauma that appears to actually be more common after severe thoracic or abdominal injury. Within hours or days, the patient develops multiple patches of superficial whitening of the retina, which usually surround the nerve and may reflect the distribution of the radial peripapillary capillary plexus (Figure 12-27). These white patches are significantly larger than those seen in fat embolism. Retinal hemorrhages and disc edema may be seen. Intravenous fluorescein angiography shows capillary nonperfusion and leakage. The lesions slowly clear, often leaving optic atrophy, vessel attenuation, and variable visual defects.

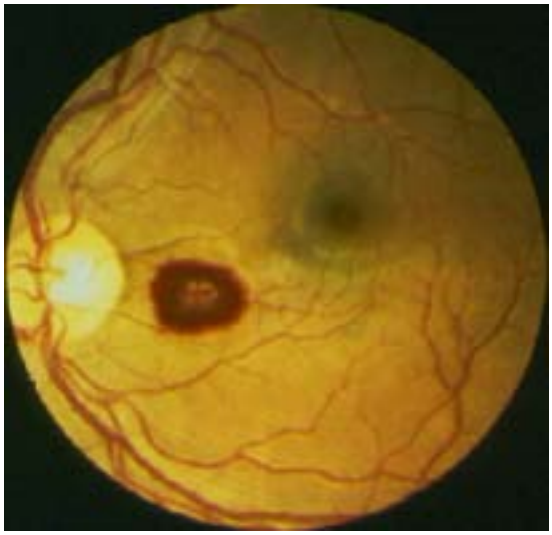


Fig. 12-22. Localized preretinal hemorrhage is seen in a 23-year-old Marine who noted a scotoma after completing a forced march with a 70-pound backpack. The hemorrhage cleared uneventfully, with vision remaining 20/20.

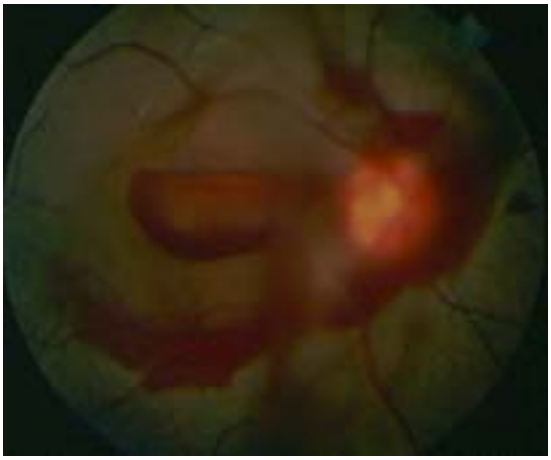


Fig. 12-24. Extensive retinal, subretinal, and vitreous hemorrhage is present in a 21-year-old patient with Valsalva retinopathy. Vision acutely was reduced to 20/400 but recovered to 20/20 over a 4-month period.

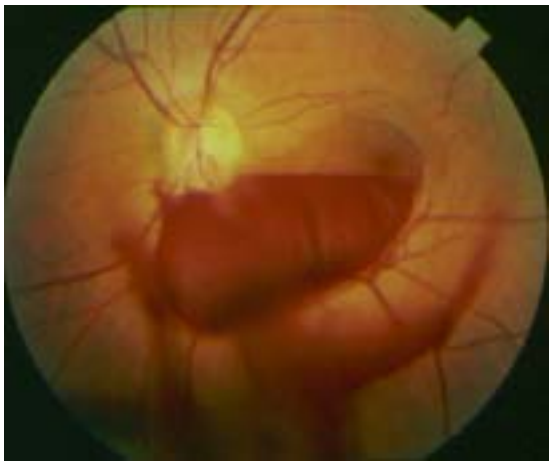


Fig. 12-23. Preretinal and vitreous hemorrhage due to Valsalva retinopathy. The hemorrhage cleared without treatment.

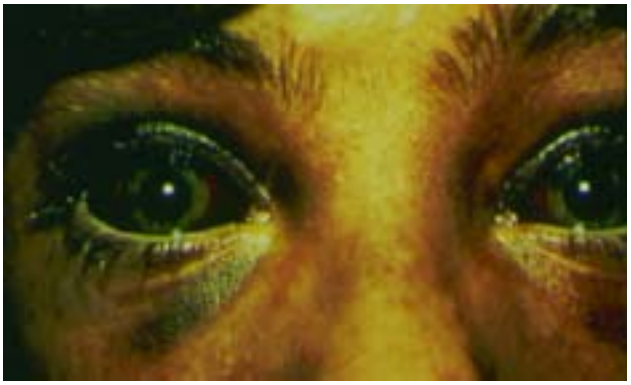


Fig. 12-25. Generalized ecchymosis of the head, neck, and thorax was seen in this patient with compression cyanosis syndrome following chest compression injury. Subconjunctival hemorrhage is present bilaterally.

Purtscher's retinopathy may be related to C5a complement activation and granulocyte aggregation resulting in embolic occlusion. This mechanism has reproduced the Purtscher's retinopathy clinical picture in animal models.[29] Similar lesions are also seen in patients with pancreatitis[30] and other illnesses with a presumably similar mechanism. Interestingly, a number of patients with unilateral Purtcher's retinopathy[31] have been observed. High-dose steroid therapy has been suggested for patients in whom the disorder is recognized early, although the efficacy of this approach has not been demonstrated.

Other Causes

Ocular effects of remote injury may also be seen in Terson's syndrome, in which subretinal, intraretinal, or vitreous hemorrhage develops in patients who have either spontaneous or posttraumatic sub-

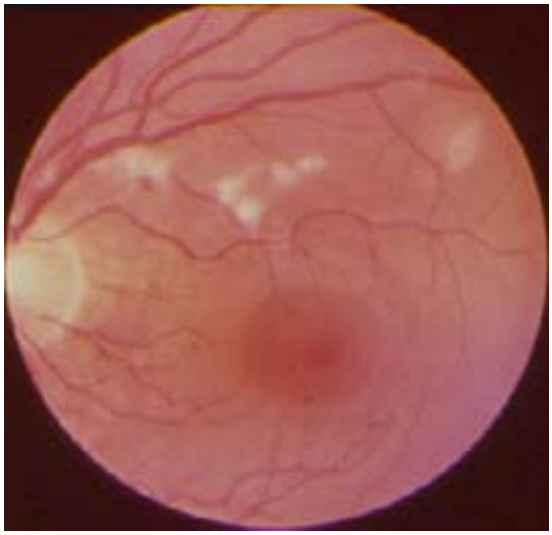
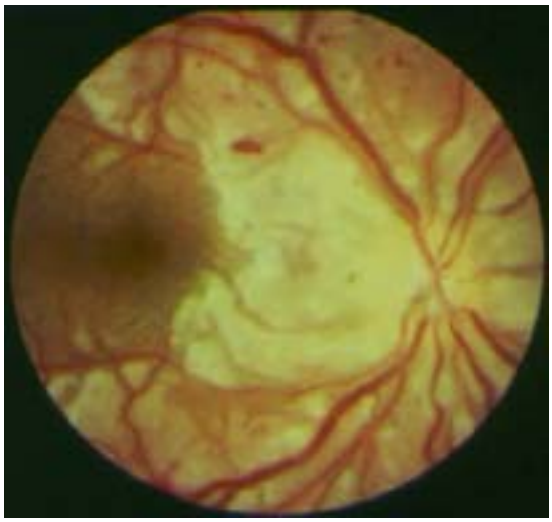
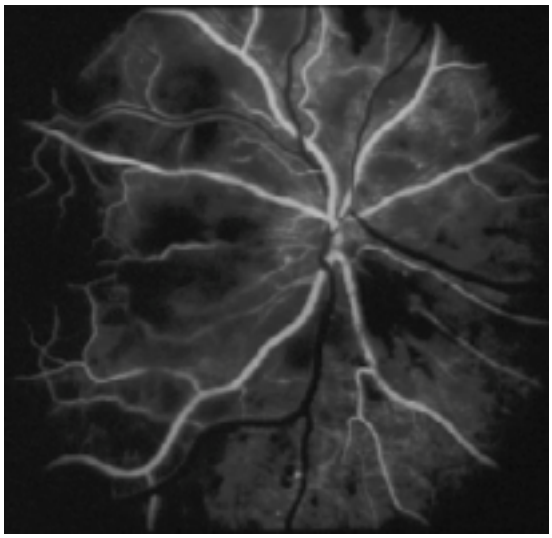


Fig. 12-26. Multiple white retinal lesions that are indistinguishable from nerve fiber layer infarcts are noted in this 22-year-old patient with long-bone fractures and fat embolism.





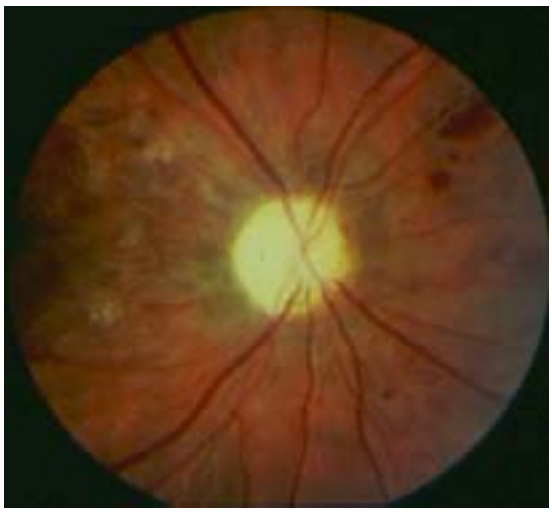


Fig. 12-27. Purtscher's retinopathy. (**a**) Striking peripapillary whitening is present bilaterally in this patient with Purtscher's retinopathy following severe chest injury sustained in a motor vehicle accident. The involvement of the radial peripapillary capillary plexus is a characteristic distribution in Purtscher's retinopathy, which may represent the vessel size most vulnerable to occlusions caused by granulocyte aggregation. (**b**) Fluorescein angiogram of the fundus reveals extensive zones of capillary nonperfusion in the peripapillary area. (**c**) Three months after the injury, the fundus reveals optic disk pallor and retinal vessel attenuation. The patient's vision remained reduced to 20/200 bilaterally.

arachnoid hemorrhage. Spontaneous clearing of the hemorrhage is common, and surgical intervention to evacuate the hemorrhage is only rarely indicated.[32]

Severe flexion–extension of the head and neck may generate macular changes in the form of whiplash maculopathy. This entity produces mild reduction in visual acuity (20/30) promptly after injury, with development of a subtle gray foveal change and a foveolar pit. The pathophysiology is not known, although speculation includes forceful avulsion of superficial foveal tissue from forces exerted on the hyaloid. No treatment is required.

MILITARY IMPLICATIONS

The variety and, sometimes, the subtlety of posterior segment manifestations of ocular trauma challenge the acumen of any clinician. For military ophthalmologists managing combat casualties, these difficulties may be aggravated by the challenges of simultaneously managing many patients with multisystem injuries, addressing complex disorders with a modest equipment inventory, and being isolated from consultation assistance. The following principles may be particularly helpful in managing casualties with posterior segment trauma:

- Consider posterior segment injury early in the patient management. Fundus examination opportunities at presentation may be lost to subsequent hemorrhage, lens opacification, and so forth.
- Careful peripheral depressed examination is imperative either primarily or, when appropriate, as a deferred follow-up examination.
- Do not overlook examination of the fellow eye.
- Clinical examination provides the overwhelming amount of useful information. Simple findings such as the presence of an afferent pupillary defect may be more valuable than imaging studies.
- Ultrasonography is a helpful and readily available mode with which to assess injured eyes with opaque media. Electrophysiology, in contrast, is unlikely to contribute useful findings.
- Despite their dramatic clinical appearance, many lesions of the posterior segment are best managed conservatively.

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