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OPHTHALMIC ASPECTS OF ORBITAL INJURY

A Comprehensive Diagnostic and Management Approach

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Trauma is the second leading cause of blindness in the United States, with 40,000 new cases of visual impairment occurring each year.[47] The incidence of ocular injury associated with facial fracture, in retrospective reviews, varies from 2.7%[36] to 67%.[39] In prospective studies, ocular injuries occurred in 29%[23] and 25%[46] of patients and complete blindness developed in 6%[23] and 14%[46] of patients, respectively.

The risk of ocular injury is greatest when the principal fracture locus involves bones of the orbit[19, 29, 41, 44] (Table 1). Fractures that decrease orbital bony volume, produce orbital soft-tissue hemorrhage, and extend to the optic canal have an added risk of secondary ocular or optic nerve injury.[2, 25, 27, 34, 35, 56] Eye injury may also accompany soft-tissue adnexal injuries, especially full-thickness eyelid and brow lacerations, and perforating eyelid injuries may be associated with occult or overt intraocular foreign body.[14, 52]

Ocular and extraocular injuries accompanying fracture-associated contusions may be minor and may not represent a serious threat to vision per se. On the other hand, serious ocular injuries often occur in association with those of less apparent significance.

In a prospective evaluation of 283 consecutive facial fracture patients seen at The University of Louisville between 1986 and 1987, 71 had ocular injuries. Eight had a single ocular injury and 63 had multiple injuries. Thirty-two of the patients with ocular injuries, or 12% of the facial fracture patients (32/283), had injuries classified as serious based on their potential for visual morbidity.[46] A summary of these injuries, their frequency of occurrence, and a brief summary of typical ocular symptoms and signs can be found in Table 1. Fracture types and percentage of associated serious ocular injuries are listed in Table 2.

The prognosis for visual recovery has improved significantly over the past 20 years owing largely to the advent of microsurgery, particularly vitreoretinal techniques. In 1945 Snell[58] reported final vision of 20/40 or better in 30% of patients following treatment of penetrating ocular injuries. By 1983 70% of patients with penetrating ocular injuries achieved at least 20/40 vision.[6] Meredith and Gordon[40] reported successful treatment of traumatic vitreous hemorrhage in 82% of treated eyes and a 52% success rate in eyes with both vitreous hemorrhage and retinal detachment.

Awareness of the possibility of injury and

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Table 1. VISION-THREATENING OCULAR INJURIES AND THEIR FREQUENCY ASSOCIATED WITH FACIAL FRACTURE

Injury	Percent of Ocular Injuries	Typical External Signs and Symptoms
Commotio retinae	21 (15/71)	Painless decreased vision due to derangement of the rods and cones of the retina if macular; if peripheral, usually asymptomatic.
Optic neuropathy	11.3 (8/71)	Painless loss of vision, decreased color perception. Afferent pupillary defect. Central scotoma, altitudinal field defect.
Ruptured globe	10 (7/71)	Vision normal or decreased depending on area of injury or associated injury. Normal to gross globe deformation. Deep or shallow anterior chamber, oblong pupil.
Hyphema	6 (4/71)	Anterior chamber turbidity to grossly visible blood in anterior chamber. Pain present if ocular pressure is increased. Normal to reduced vision depending on extent of hemorrhage.
Intraretinal hemorrhage	6 (3/71)	No external signs. Normal or decreased vision depending on location and extent of hemorrhage.
Vitreous hemorrhage	4 (3/71)	No external signs. Decreased vision. Absent red reflex on ophthalmoscopy or blurred view of optic nerve, retina, and blood vessels.
Choroidal rupture	2.8 (2/71)	No external signs. Decreased vision is common. Most breaks occur in macular or optic nerve region.
Corneal laceration	2.8 (2/71)	Usually grossly obvious to examination. Iris may be incarcerated with pupil deformity. Shallow anterior chamber and hyphema may be present. Vision usually decreased but may be normal if laceration is peripheral and anterior chamber not deformed.
Elevated intraocular pressure	2.8 (2/71)	May occur with orbital hematoma, subperiostal hematoma, "blow-in" fractures, and hyphema. Pain only if pressure significantly elevated (50–60 mm Hg). Vision normal if cornea clear and optic nerve uncompromised.
Retrobulbar/orbital hemorrhage	1.4 (1/71)	Proptosis, eyelid and conjunctival edema, pain and motility abnormality. Vision decreased if secondary optic neuropathy develops.

prompt recognition are critical to improved visual prognosis in the patient with an eye injury. Diagnosis is usually straightforward when the primary injury is overtly ocular or the primary symptoms are visual. However, significant injuries may be present in grossly normal eyes with good vision. Additionally, seriously injured patients may be obtunded or intoxicated, making communication difficult and unreliable. In this atmosphere, the diagnosis of serious eye injuries must not be delayed or overlooked. The initial evaluation of all orbital fracture patients, therefore, should include a brief ocular examination that must be carried out immediately after systemic stabilization and must precede fracture repair.

This article provides information essential to early diagnosis and insight into vision-threatening ocular injuries that commonly accompany facial fractures. It also suggests management of these injuries as it relates to facial fracture repair. The primary focus is upon pure ocular and optic nerve injuries. Minor attention will be given to ocular motility disturbances and adnexal injuries.

THE FIVE-POINT OCULAR ASSESSMENT

The objectives of the initial ocular evaluation in facial skeleton fracture(s) are to (1) develop a brief historical profile of pre-injury vision, current subjective visual status, current eye diseases, and previous intraocular surgery; (2) obtain an objective baseline visual acuity, examine the pupils, anterior segments, and posterior segment of the eye and eyelids, and ascertain ocular motility; and (3) determine if ophthalmic consultation is needed.

The following is a five-point guide suggested for initial ocular evaluation. It can be carried out by the nonophthalmologist in 2

Table 2. FRACTURE TYPE AND FREQUENCY OF SERIOUS OCULAR INJURY

Type	Percent
Tripod	37.5
Frontal/supraorbital	25
Orbital (isolated blow-out fracture of the orbital floor and/or medial wall)	12.5
LeFort II/III	9.5
Complex (panfacial fracture)	6.25
Nasal	6.25
Nasoethmoid	3.0

to 3 minutes with equipment normally found in emergency wards. Its elements in their recommended order of performance are subsequently discussed.

Visual Acuity

Accurate assessment of initial visual acuity is indispensable to the diagnosis and treatment of ocular and optic nerve injuries. Assessments that result in statements such as "vision grossly normal" are inadequate. Near or distant vision should be examined first, while completely occluding the eye not being tested. If the patient's glasses are available, they should be worn. Preprinted acuity charts are ideal but rarely available. In their place, print from package labels may be temporarily substituted; however, the test object used should remain with the patient until an appropriate test card can be obtained. If vision is so poor that printed material cannot be read, the patient's ability to count fingers, and the maximum distance at which this can be accomplished, should be noted. If fingers cannot be seen, the ability to perceive light in four quadrants should be tested. Inability to identify light location may indicate retinal injury. A patient should not be declared unable to perceive light unless the examination light is very bright (such as that of an indirect ophthalmoscope). It is imperative during testing of light perception that the fellow eye be totally occluded with an opaque object to ensure testing reliability.

Pupils

Abnormal pupillary reactions—in particular, the afferent pupillary defect—are of critical importance to the trauma surgeon. Pupil examination follows visual acuity testing because bright light may transiently reduce acuity. Size, shape, and symmetry should be noted in ambient room illumination. Reactions, however, should be tested in dim illumination. If the patient is conscious and cooperative, gaze is fixed on a distant rather than a near target.

The direct response should be elicited with a bright light. Once noted, the pupil is retested directly while the briskness and amplitude of the contralateral consensual response is observed. Each eye is thus examined in turn. Next, the alternating light test is performed as follows: The test light is passed briskly between the two eyes several times with the examiner's attention focused on the direct responses. Normal pupils will dilate slightly in the absence of stimulation as the light is moved between the two eyes. Therefore, the *first* movement of each pupil should be *constriction* to direct stimulation during the alternating light test. A lack of pupil reaction or dilation during direct testing is abnormal. A poor direct reaction may indicate traumatic mydriasis (poor direct *and* consensual responses), glaucoma, iris incarceration in a corneoscleral laceration, efferent third nerve lesion, massive internal derangement of the eye, or optic nerve injury.

An afferent pupillary defect, in the presence of a normal eye, indicates an optic nerve injury. It is diagnosed when the pupil reacts to direct stimulation but shows a noticeably more brisk consensual response when the contralateral pupil is directly tested (relative afferent pupillary defect or Marcus Gunn pupil). During the alternating light test, this direct-consensual reaction disparity will again be noted and, in addition, the *first* pupillary movement on direct testing will be *dilation* as the light is moved from the normal contralateral pupil. When an absolute afferent pupillary defect (amaurotic pupil) is present, there is no reaction to direct stimulation while a brisk consensual response is present. It too will dilate during the alternating light test when light is moved to it from the contralateral side (Fig. 1). Pupillary symmetry is not affected by the afferent pupil defect, nor does it indicate the location of the lesion along the course of the optic nerve.[9, 61] Pupil asymmetry implies an efferent lesion or an iris lesion or possibly both.

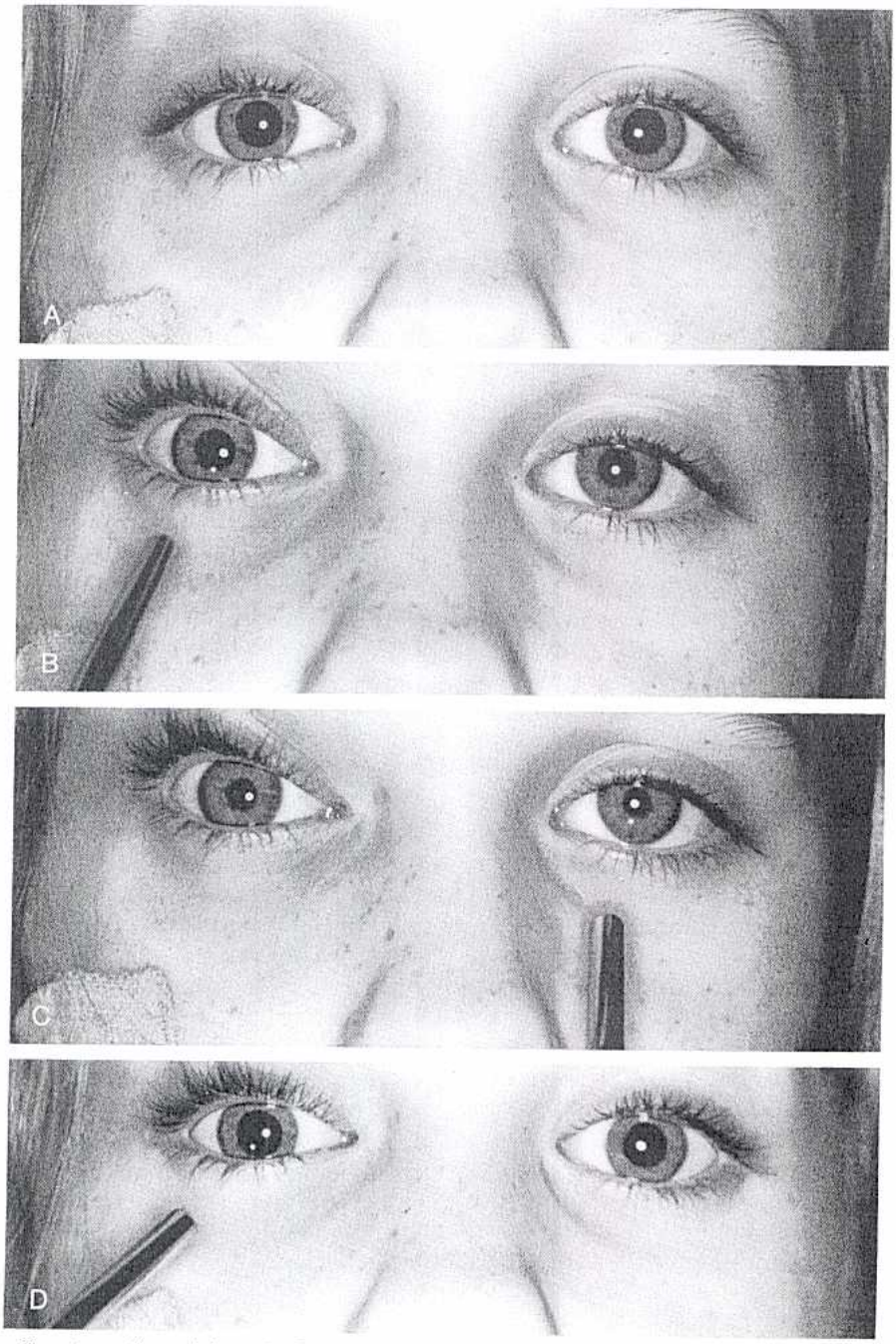

Figure 1. The afferent pupillary defect. *A*, Pupillary symmetry and size are observed in ambient room light. *B*, The direct response is first tested with a bright light, and direct and consensual responses are observed. *C*, The fellow eye is then tested. Comparing the reactions with those of Figure *B* shows greater constriction of the left pupil and a more complete consensual response of the right pupil *relative* to the direct response. This pattern indicates an afferent pupil defect. *D*, The alternating light test follows direct and consensual pupillary testing. The penlight is quickly moved between the eyes while the direct responses are observed. The first movement of the pupils should always be constriction; however, in this case the pupils dilate as the light is moved to the right eye. This is the typical pattern of an afferent pupillary defect as determined by the alternating light test. In the presence of a normal eye, this reaction indicates optic nerve injury.

Anterior Segment

The anterior segments and eyelids are examined following pupil evaluation. A full-thickness or marginal eyelid laceration should raise suspicion of globe rupture. To examine the anterior segment, a penlight is held temporally, slightly anterior and parallel to the plane of the iris and perpendicular to the sagittal plane of the eye (Fig. 2). Alterations of corneal clarity, clarity of the anterior chambers, and subconjunctival hemorrhage should be specifically noted. Asymmetry of chamber depths may imply globe rupture, lens subluxation, glaucoma, or vitreous hemorrhage.

Posterior Segment

The posterior segment of the eye encompasses the vitreous, retina, and optic nerve and can be adequately examined only with an indirect ophthalmoscope. For initial screening, however, a direct ophthalmoscope is used. An absent red reflex or inability to visualize the macula and optic nerve may suggest hyphema, lens rupture, or vitreous hemorrhage. Irregular folding of the retina indicates subretinal fluid and rhegmatogenous retinal detachment (Fig. 3). Inability to view the posterior pole of the eye in a patient with normal vision is usually the result of an improperly functioning ophthalmoscope. An indirect ophthalmoscopic examination should be carried out by an ophthalmologist as soon as neurologic status permits dilation of the pupil in a patient with decreased visual acuity, abnormal pupils, anterior segment abnormality, or suspected posterior segment injury.

Ocular Motility

The vertical and horizontal positions of gaze should be tested *after* the possibility of globe rupture has been eliminated. The objectives are to identify extraocular muscle paresis or entrapment.[31, 69]

With the exception of ocular motility abnormality, decreased vision or other abnormality disclosed during the five-point assessment is an indication for urgent ophthalmic consultation. Fracture repair should be deferred until this consultation has been completed.

OCULAR INJURIES

Selected ocular injuries from Table 1 discussed subsequently have special signifi-

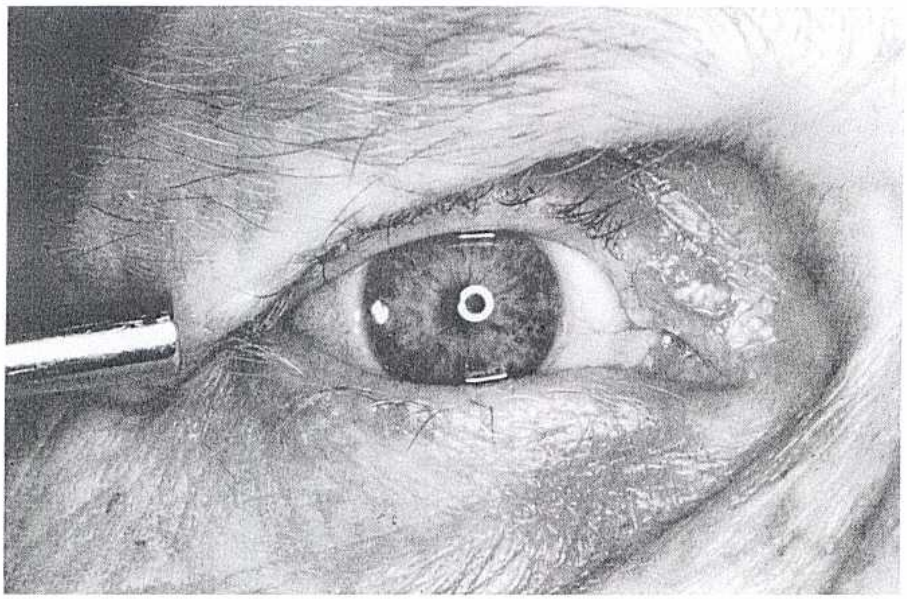

Figure 2. Estimation of the relative symmetry of anterior chamber depths is facilitated by holding a penlight temporally with its beam slightly anterior and parallel to the plane of the iris and perpendicular to the sagittal plane of the eye. The anterior chamber shown here is normal.

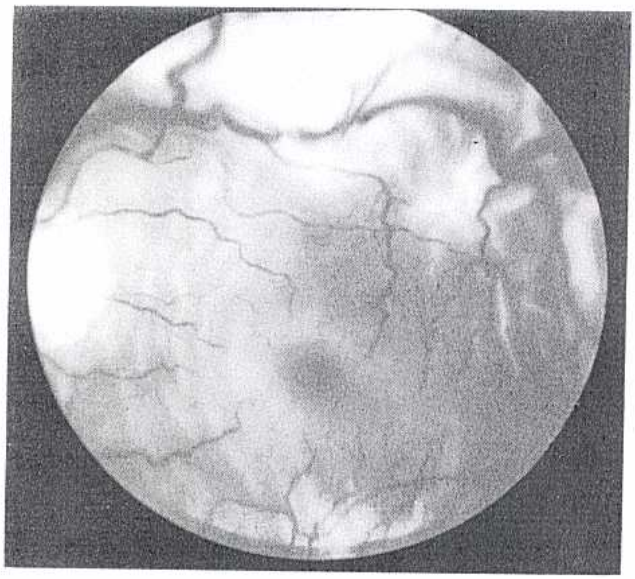

Figure 3. In comparison to Figure 4, the retina is irregular and folded, indicating the presence of subretinal fluid and a retinal detachment. Detachment in trauma usually results from horseshoe or giant tears located in the retinal periphery. They can be identified only by indirect ophthalmoscopy through a dilated pupil.

cance within the context of orbital fracture. They are injuries that not only pose a serious threat to vision but also can be exacerbated during fracture repair.

Commotio Retinae

Commotio retinae is seen as a grey to grey-white opacification of the retina as viewed during ophthalmoscopy (Fig. 4). It results from direct or indirect trauma to the affected area, and it is most frequent in the temporal quadrants of the retinae. It may also occur in the macular region and represents derangement of the outer retinal layers.[63] Embarrassment of the retinal microcirculation (e.g., by increasing orbital pressure) should be prevented, and long-term ophthalmologic follow-up is needed to detect possible late-developing retinal breaks in eyes with contusion retinopathy.[5]

Traumatic Optic Neuropathy

The diagnosis of traumatic optic neuropathy is established by the presence of acutely decreased vision, an afferent pupillary defect, and an otherwise normal eye. It may be

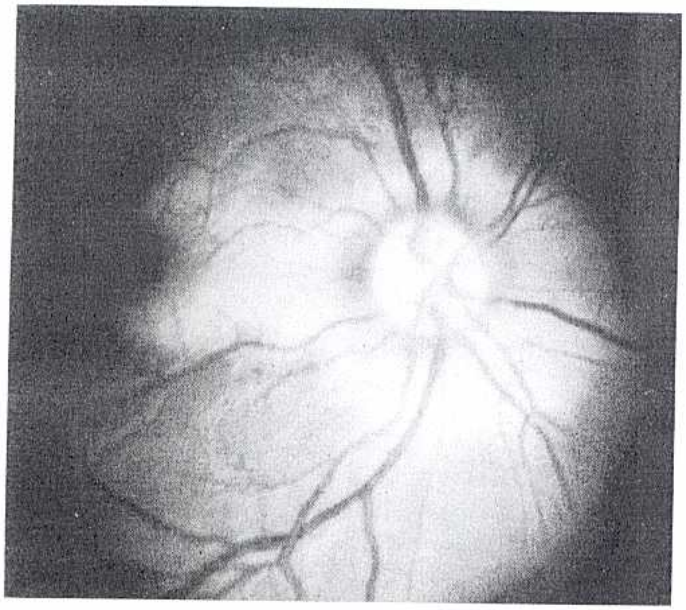

Figure 4. The dumbbell-shaped greying (white in the photograph) of the anterior retina surrounding the optic nerve and macula is the typical appearance of commotio retinae. It is common in fracture patients.

unilateral or, rarely, bilateral and is classified as either direct or indirect.

Direct traumatic optic neuropathy arises from compression along the course of the optic nerve by bone fragments, hematoma, foreign body, and fractures involving the optic canal that are demonstrated radiographically.

Indirect optic neuropathy occurs without fracture or radiographic evidence of other abnormality contiguous to the orbital, intracanalicular, or intracranial optic nerve. It may also accompany a variety of orbital fractures that spare the optic canal.[1, 7, 13, 54, 65] Postmortem examination of patients with indirect traumatic optic neuropathy has disclosed multiple abnormalities: neural hemorrhage, contusion, and severance of nerve fibers. Secondary edema and/or vasospasm are thought to produce nerve ischemia and infarction.[67] The etiology of this disorder, however, is largely speculative. Deceleration of the brain may compress the intracranial segment of the optic nerve against the falciform process, a thin dural fold that hoods the cranial opening of the optic canal[37] (Fig. 5). Deformation of the optic canal during frontal impact with resultant nerve contusion and edema has also been suggested as a cause.[50, 59] Support for this mechanism has been provided by a case report describing indirect optic neuropathy following the application of purely static force to the forehead.[55]

The optimal treatment of indirect traumatic optic neuropathy is an unsettled issue awaiting prospective study. It is known that some 25 to 30% of untreated patients improve spontaneously, and the degree of recovery correlates inversely with the severity of initial visual loss.[7, 8, 21, 71] There is a trend, however, toward early medical therapy of indirect neuropathy with high-dose corticosteroids.[1, 32, 50, 60] The rationale for their use is drawn from experience in the treatment of other optic neuropathies and experimental spinal cord trauma.[45, 62, 72] In a recent retrospective study of 36 patients, 15 received no treatment and 21 received high-dose intravenous dexamethasone (1 mg/kg/day). Sixty-two percent of the treated patients improved and 33% of those not treated improved. This difference was not statistically significant, although more rapid recovery of vision was noted with steroid treatment.[57] In the only prospective evaluation to date, conducted by the senior author, 16 consecutive patients were evaluated. Half were treated with high-dose corticosteroids and the other half received no treatment. Twenty-five percent of untreated patients improved and 25% worsened, whereas 87% of treated patients improved and none worsened. The difference was statistically significant.[16] These two studies, however, having small sample sizes, do not statistically support a definitive conclusion regarding steroid efficacy in this entity.

When traumatic optic neuropathy is diagnosed, CT evaluation of the full-length of the

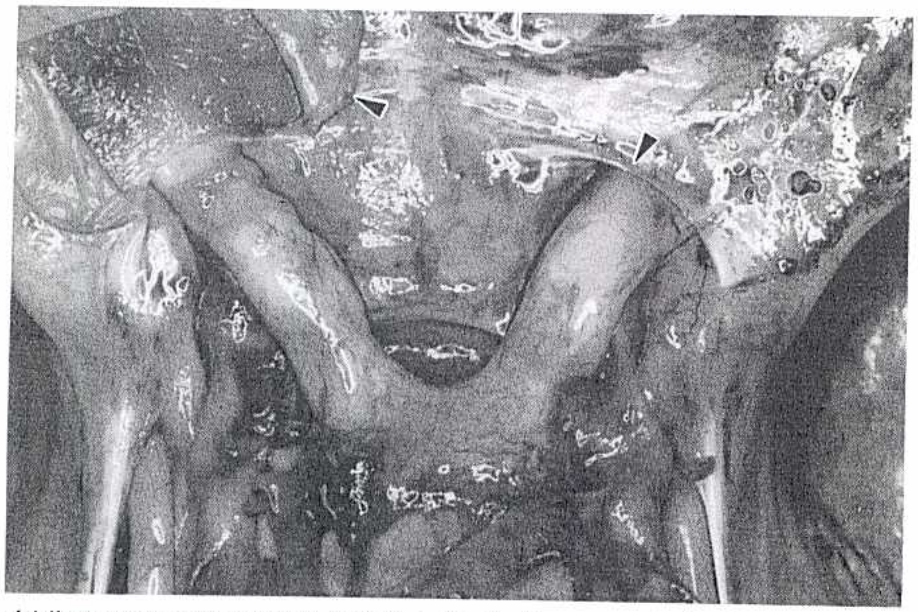

Figure 5. The falciform process (*right arrow*) may be a site of optic nerve contusion during rapid deceleration of the head. The dura is reflected (*left arrow*) revealing the intracranial aperture of the optic canal.

orbit, optic canal, and adjacent sinuses should be obtained immediately to differentiate between direct and indirect subsets.[18] Our current practice is to treat all cases of indirect and direct traumatic optic neuropathy at the time of diagnosis with high-dose corticosteroids (1.0 mg/kg dexamethasone loading, 0.5 mg/kg every 6 hours). If vision improves, dosage is maintained until a visual plateau is reached, after which rapid tapering is begun. Steroids are continued no longer than 7 days in patients showing no improvement, because none of our patients improved beyond this time[16] and none of Seiff's patients improved beyond 6 days.[57]

Extracranial optic nerve decompression for indirect neuropathy is also being used with greater frequency and increasing success compared with earlier transcranial decompression studies.[1, 7, 11, 12, 24, 27, 43, 50, 61] Fujitani et al,[11] comparing extracranial canal decompression and moderate-dose steroid treatment (prednisone, 60 mg per day), found a statistically significant difference between surgery (70 eyes with 47.7% improving) and moderate-dose steroids (43 eyes with 42.2% improving) only in patients with vision of no light perception immediately after injury and in those in whom the onset of treatment was delayed several weeks. In these two subsets, surgery was superior to steroids in restoring vision. More recently, Joseph et al[24] reported recovery of vision in 11 of 14 patients (78%) treated with extracranial transethmoidal optic nerve decompression. In these patients, decompression was carried out quickly in most cases (1 to 4 days) after diagnosis, and moderate-dose steroids were used briefly preoperatively.

Although initial treatment of indirect optic neuropathy with high-dose steroids is gaining acceptance, the indications and efficacy of surgical intervention have not been absolutely established. Walsh,[67] in 1966, proposed guidelines for the use of transcranial decompression: (1) decompression should not be performed on unconscious patients; (2) it may be contraindicated in patients who lose vision at the time of impact; (3) if vision was lost after the moment of impact, decompression may be helpful; (4) in patients in whom it cannot be determined if vision was present after impact, 4 to 6 days of observation may be indicated prior to decompression. The guidelines were suggestions and were not based on controlled clinical studies.

Twenty years later, Fujitani et al[11] suggested that patients with complete loss of vision at impact may be effectively treated with immediate surgical decompression. On the basis of the current literature and our experience with extracranial decompression, to the foregoing views we would add that transethmoidal canalicular decompression can be considered in (1) those patients not improving after 5 to 7 days of steroid therapy, (2) those worsening on steroids after initial improvement, (3) those with good vision after impact who then rapidly deteriorate despite steroid treatment, and (4) those having steroid contraindications. Orbital fracture repair should not be undertaken, in our opinion, until the traumatic optic neuropathy has conclusively stabilized. A prospective study comparing medical, surgical, and a combination of both treatments in terms of the final degree of visual recovery from the various subsets of traumatic optic neuropathy will refine the role of surgical and medical management in this disorder.

A variety of surgical methods have been effectively applied to direct optic neuropathies attributable to causes such as blow-in fracture of the orbit, orbital hemorrhage, optic canal fracture, subperiosteal hematoma, optic nerve hemorrhage, optic nerve sheath arachnoid cyst, and nonspecific optic nerve sheath enlargement.[17, 22, 25, 27, 28, 30, 35, 68] The surgical approaches, respectively, have included early fracture reduction, orbital decompression, extracranial optic canal decompression, hematoma drainage (subperiosteal or transneural), and nerve sheath fenestration. There is, in our opinion, no single best surgical approach to direct optic neuropathy. The technique must be tailored to the cause of the neuropathy.

Globe Rupture

Ruptured globe *must* be excluded before repair of eyelid lacerations or orbital fractures. Although the diagnosis is usually easily made by careful inspection, it may be missed. Subconjunctival hemorrhage should always suggest the possibility of ocular perforation and may obscure important signs of rupture such as uveal exposure and vitreous prolapse (Figs. 6 and 7). Hyphema should likewise raise suspicion of globe rupture.[52] Occult ruptures may develop beneath the rectus muscles at the time of impact. If a

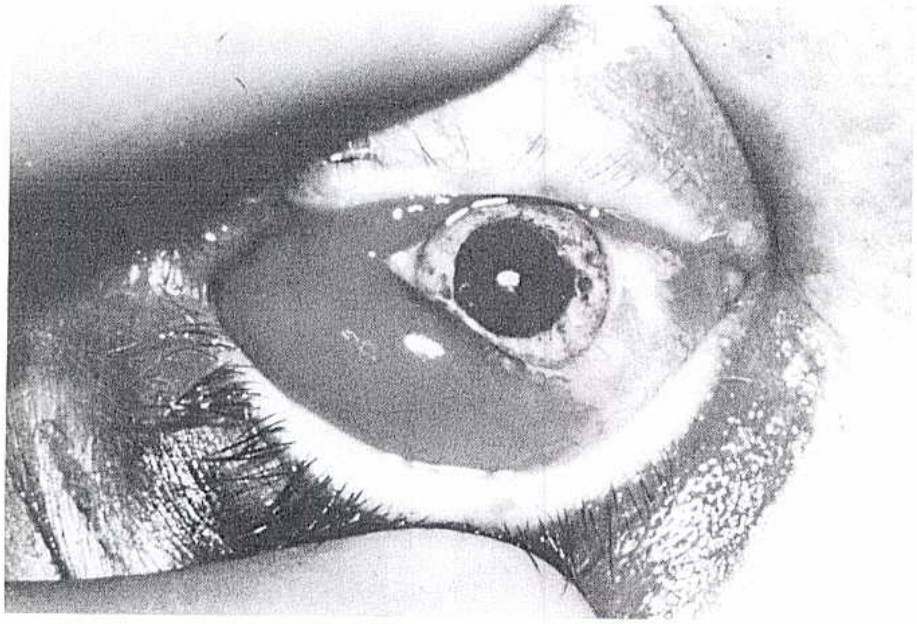

Figure 6. Subconjunctival hemorrhage is a minor ocular injury per se but may conceal signs of globe rupture such as uveal and vitreous prolapse.

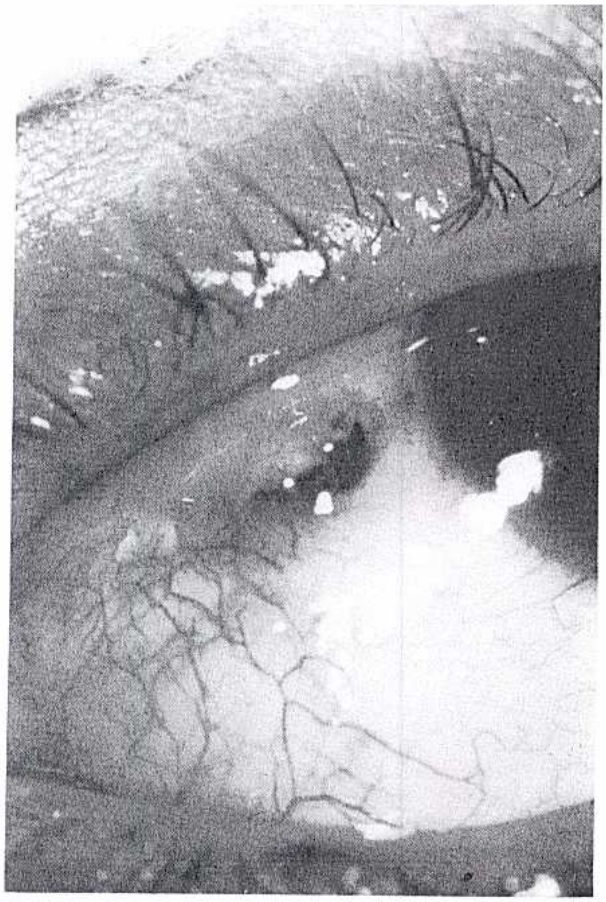

Figure 7. Uveal prolapse is identified as pigmented tissue that may protrude through the cornea or beneath the conjunctiva. (*Reprinted with permission from* Shingleton BJ: Eye trauma and emergencies. American Academy of Ophthalmology, 1985.)

scleral dehiscence is anterior in the region of the ciliary body, an oblong pupil may be seen that "points" at the site of injury (Fig. 8). Intraocular foreign body should be suspected and excluded in all cases of globe rupture.[53] If a rupture cannot be ruled out by clinical examination, the globe should be surgically explored prior to fracture repair in the orbital region. If an open globe is diagnosed or suspected, the eye must be protected against further injury by a metal shield until repair is undertaken.

Hyphema

Impact in the region of the cornea or ciliary body may precipitate bleeding into the anterior chamber.[70] Light bleeding produces biomicroscopically visible hemorrhage, whereas heavier bleeding produces an obvious fluid level of blood within the anterior chamber (Fig. 9). Vision may be reduced initially followed by gradual improvement as the blood settles into the inferior anterior chamber recess. Eyelid edema may hamper examination of the eye, and the complaint of ocular discomfort may be the only indication of a hyphema. Initial pain at impact that later gives way to a persistent dull ache with photophobia is typical.

Secondary hemorrhage may occur in 5 to 30% of cases and is usually more severe than the initial bleeding.[51] Risk is greatest within the first 5 days following injury. The goals of initial treatment, therefore, include the prevention of rebleeding and maintenance of normal ocular tension.[70] Secondary bleeding produces sudden, renewed pain in a heretofore comfortable eye. A clot may fill the anterior chamber and occlude the trabecular meshwork, which leads to protracted intraocular pressure elevation. Pain and vomiting ensue, and conjunctival engorgement and corneal clouding occur. The anterior segment becomes anoxic and the hyphema becomes very dark. These patients may also develop late-onset glaucoma secondary to damage to the anterior ciliary body.[26]

Because there is a risk of rebleeding during fracture repair, the latter should be delayed, if possible, at least until the period of greatest risk of rebleeding has passed, and the use of antifibrinolytic agents should be considered.

Corneal Laceration

Most of the injuries previously described result as a consequence of blunt impact upon the eye. Lacerations that damage adnexal structures may also produce laceration of the underlying cornea. Usually evident to penlight examination (they cast an iris shadow that moves *opposite* to the direction of the light), they often result in shallowing of the anterior chamber or prolapse of iris tissue (Fig. 10). At times, however, they may be

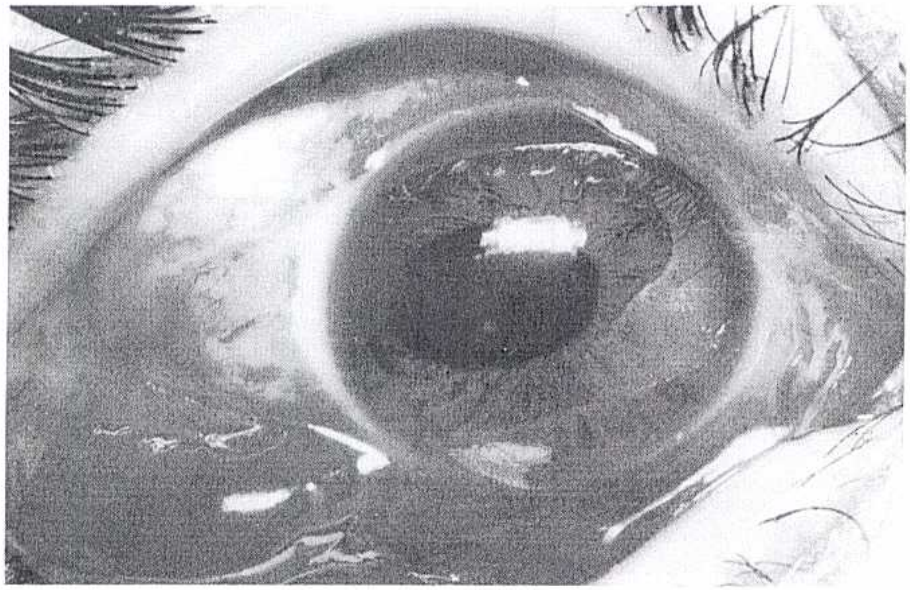

Figure 8. An oblong pupil may indicate globe rupture and "point" at site of injury. (*Reprinted with permission from* Shingleton BJ: Eye trauma and emergencies. American Academy of Ophthalmology, 1985.)

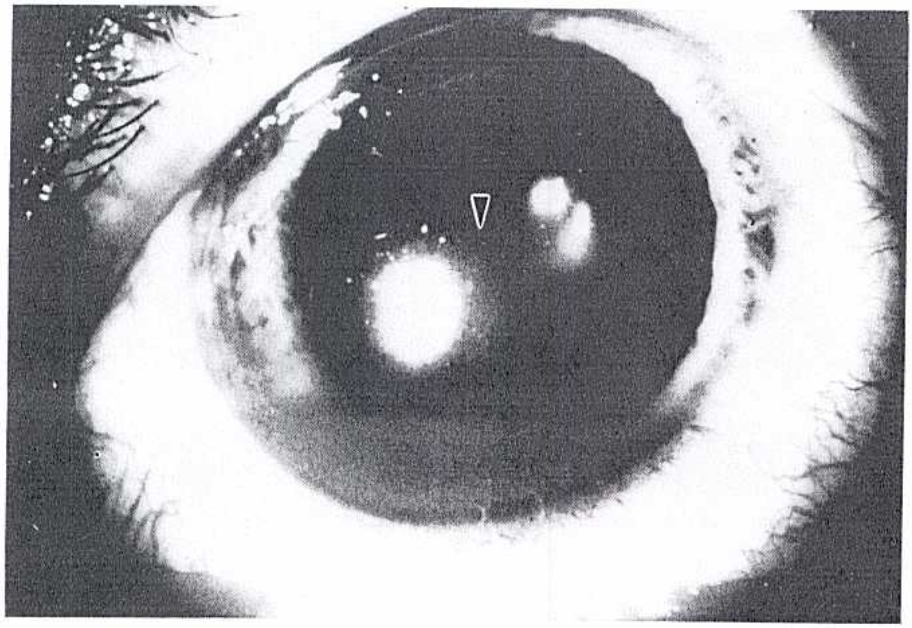

Figure 9. Gross hyphema may be difficult to recognize in patients in whom the globe is obscured by lid swelling. This hyphema was recognized after lid swelling had subsided but not before blood staining of the cornea (*inferior to arrow*) had occurred secondary to untreated intraocular pressure. Even in the face of massive eyelid edema, an ocular assessment must be performed.

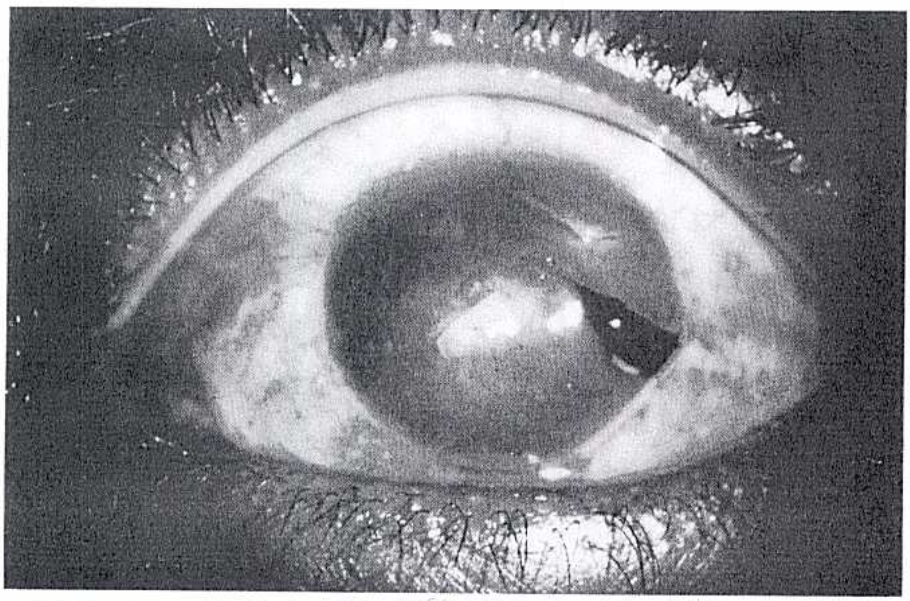

Figure 10. Laceration of the cornea often results in shallowing of the anterior chamber and prolapse of uveal tissue.

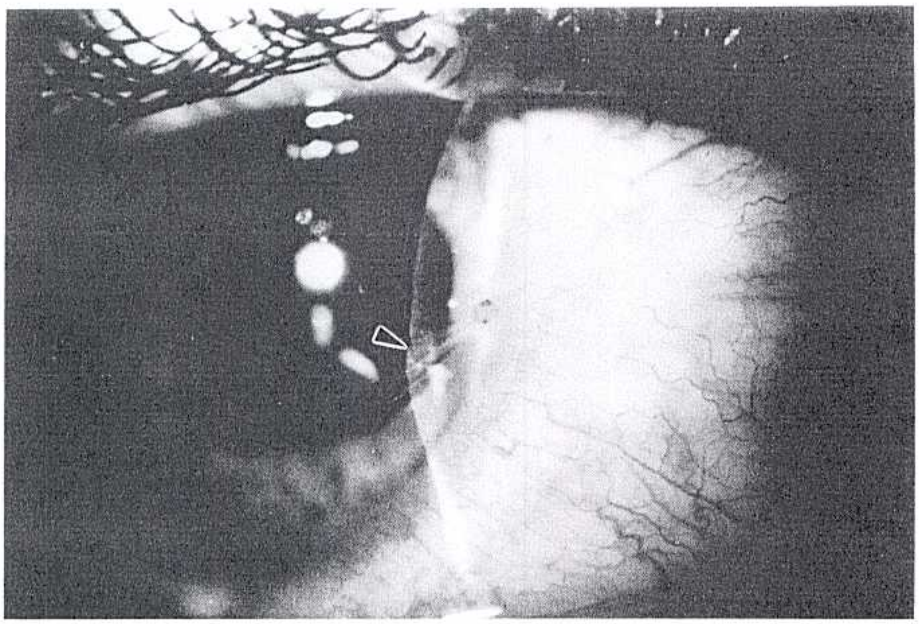

Figure 11. Corneal lacerations are usually associated with reduced vision unless peripherally located. In this case there is no iris prolapse although the laceration is full thickness (*arrow*). Subtle injuries of this type may be easily overlooked.

subtle and can be overlooked (Fig. 11). Although vision is often reduced, it may be normal if the injury is peripheral and self-sealing. An intraocular foreign body must be ruled out. Therefore, the historical background of the accident is important. If a foreign body is attached to the eye, protection must be provided against aggravation of the injury that may result from vomiting, from patient restlessness, or during radiographic evaluation of facial fracture (Fig. 12).

Postreduction Visual Loss

Loss of vision following orbital fracture reduction is rare and may be the result of optic nerve dysfunction secondary to increased orbital pressure from hemorrhage or edema, direct nerve compression, central retinal artery occlusion,[3, 4, 10, 15, 20, 33, 38, 48, 49, 66] or ocular injury. Its rarity notwithstanding, it is imperative that visual acuity be checked as soon as the patient can cooperate postoperatively. Measurement should be obtained daily during the first 48 to 72 hours after orbital surgery.

The five-point ocular assessment previously outlined should be performed at once if a patient experiences visual loss after surgery. Transient blurring of vision after surgery may be due to instillation of lubricating ointments. Simple irrigation of the ointment from the eye will improve vision. Lid and orbital swelling may distort the globe, inducing astigmatism or other refractive error that can be improved with a pinhole. Corneal abrasion may cause blurred vision and pain, with the latter being rapidly relieved by topical anesthetic. Ophthalmic consultation is urgently indicated if one of the previous diagnoses is not obvious.

High-dose corticosteroids should be instituted immediately if visual loss is due to optic neuropathy secondary to increased orbital pressure resulting from hemorrhage, edema or direct optic nerve compression. In addition, urgent surgical intervention may be required if vision does not improve, if the reduction is severe, or if CT demonstrates direct optic nerve compression owing to hemorrhage, bone fragments, bone grafts, or other orbital implant. The surgical objectives are to relieve direct optic nerve pressure or increase orbital volume in instances of neuropathy due to generalized orbital tissue swelling.

THE MONOCULAR PATIENT

The aesthetic or functional benefit of fracture repair must be carefully weighed (and discussed with the patient) against the surgeon's assessment of the potential risk to vision. Even though the five-point assessment may be normal, all monocular patients

Figure 12. The eye must be immediately protected against further damage in the presence of protruding intraocular foreign body. Vomiting, movement, and radiographic evaluation may exacerbate injuries of this type.

should receive ophthalmic consultation prior to fracture reduction in the orbital region.

SUMMARY

Improved prognosis for eye injuries sustained during facial trauma depends on early assessment and accurate diagnosis. The surgeon responsible for management of the orbital fracture(s) must perform an ocular assessment at the time of initial examination. Abnormalities found on the five-point assessment outlined herein should lead to urgent ophthalmic consultation. Fracture repair should not be undertaken until the results of consultation have been discussed with the ophthalmologist. Vision should be carefully monitored in the preoperative period because delayed optic neuropathy may occur.[42] Similarly, vision should be followed regularly in the postoperative period after reduction of fractures of the orbital bones. Decreased postoperative vision should lead to immediate ocular assessment and early ophthalmologic consultation.

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