# Eye Care Following Disasters

**Michael G Weddle, MD, PhD, FACEP**

## Overview

The emergency phase of disaster response is that period immediately following a disaster when action is taken to save lives and reduce suffering. These actions include, but are not limited to, search-and-rescue and emergency medical assistance. For disasters like earthquakes and windstorms, the emergency phase is considered to be about 7-10 days (1). Surgical needs in casualty management, however, are increased only over the initial 72 hours (2). Following the emergency phase, medical care needs return to levels approximating long-term levels. The risk of infectious disease following loss of adequate sanitation requires ongoing surveillance and treatment capability. Likewise, injuries occurring during clean-up activities require treatment capability through the emergency and rehabilitation (recovery) phases.

The needs of ophthalmologic casualties mirror these general patterns of casualty care following disasters. The number of ophthalmologic casualties, however, may be large. In humans, the eyes account for only 0.1 % of the total body surface area, yet during an explosion as many as 10% of survivors may suffer eye trauma (3). Acute eye injuries during a disaster often require the care of an ophthalmologic surgeon and can result in considerable disability if not managed in an urgent fashion. It is vital for first responders in disaster response teams to be able to determine which victims require urgent evacuation and which can be managed in a more delayed fashion.

The eye exam also can reveal information about other head and neck injuries (eg., intracranial hematoma) and toxic exposures (eg., botulinum toxin and nerve agent). The lack of adequate sanitation and group living conditions in shelters may predispose individuals to eye infections. The loss of eye medication and ability to care for contact lenses may lead to special problems in these populations.

This chapter will cover specific patterns of injury in the following disaster types: volcanic eruptions, earthquakes, tropical cyclones, explosions, and bio-chem events. Specific ocular injuries and diseases will be described and their treatments discussed. It is assumed that the reader possesses a basic knowledge of eye anatomy and pathology. In addition, where procedures such as ophthalmoscopy and tonometry are discussed, it is assumed that any provider attempting to perform such work at a disaster sight will have basic knowledge and experience in the skill discussed.

A final section will discuss issues of eye care that providers may be faced with as they provide services for both victims and relief workers during the end of the emergency phase and ongoing rehabilitation.

## Objectives

By the conclusion of this session you should be able to:

1. Identify examination methods used to determine visual acuity.

2. Identify the anatomical structures and physical symptoms of eye injury.

3. Recognize the medical conditions and assessment techniques associated with eye injuries following disasters.

4. Identify the appropriate methods for treating eye injuries and illness to prevent unnecessary worsening of the patient's condition.

5. Identify the occult manifestations of toxidromes that provide clues to possible hazardous (bio-chem) exposures.

6. Recognize those eye injuries and illnesses requiring urgent care by an ophthalmologist, and those that can be referred in a delayed fashion or treated locally.

## Contents

## History

Following a disaster, much of the history may seem apparent, but important details need documented in the medical record or on the victim card to aid both yourself and future care providers in proving appropriate care. In the post-disaster setting, obtaining a thorough history, including letting the victim tell his or her 'story,' may be a luxury that you will not be able to afford. Some important details of the victim's eye history, however, should be obtained whenever possible.

- Does the victim wear glasses or contact lenses, and was either in place at the time of injury?
- Is there any history of lazy-eye (strabismus), unequal pupil size (Adie's pupil), glaucoma, or eye surgery?
- Does the eye hurt? Does it hurt to look at a light (photophobia)?
- Has the victim seen flashing lights, or a dark curtain coming across the visual field?
- Has there been any double vision? Does it persist if one eye is covered?
- Is the victim allergic to any medications?

## Examination

The eye examination may be as simple or complex as the skill of the examiner, available equipment and condition of the patient allows. Critical findings on simple observation of the traumatized eye can at times be sufficient to indicate urgent ophthalmologic referral, and every level of examiner responsible for evaluation of victims' eyes should be familiar with these. A simple drawing of the eye on the medical record or victim card is often more helpful than a scribbled note (4). Equipment available at the disaster site will vary. The patient's condition, and particularly his or her level of consciousness, will determine one's ability to completely evaluate the eyes. The most basic measurement of acuity will need to be deferred in the confused or poorly responsive victim.

- Acuity is the most basic measure of eye function, the 'vital sign' of the eye. It is measured for each eye independently, and corrected for eyeglass wearers with the victim's own prescription lenses whenever possible. Whenever possible, a Snellen eye chart at 20 ft. (6 meters) is used for acuity measurements. Acuity of 20/20 is considered normal. A reading of 20/40 indicates that a person can recognize a symbol at 20 feet that a 'normal' could recognize at 40 feet. If a persons misses 2 of the 5 letters on the '20/40' line, vision can be documented for that eye as '20/40 – 2.' If you need to move the chart closer for the person to see, the new distance becomes the numerator: '3/200' means that the eye measured can see the 'E' at 3 feet.

  If eyeglasses are lost, as will often be the case following a disaster, having the victim view the chart through a pinhole occluder can approximate corrected vision. If a Snellen chart is not available, a hand-held 'near vision card' can substitute, although

with this method pinhole testing may be needed for those over 45 years old (5). Other methods of documenting acuity include the ability to read newsprint or headlines.

If unable to see print, acuity can be documented as, in order of worsening ability, counting fingers at a distance ('CF at 5 ft.'), seeing hand motion at a distance ('HM at 5 ft.') or the ability to perceive light (LP). Intense pain caused by an eye injury can at times make acuity testing impossible until 1 or 2 drops of a topical anesthetic is used.

- Visual examination of the eyes can reveal much. The globe ('eyeball') and surrounding structures (adnexa) should be examined independently. Whether a globe is protruding (proptosis) or sunken (enophthalmos) can best be determined by looking down from over the top of the subject's head, comparing both sides.

  Looking at the globe can reveal penetrating or embedded foreign bodies. If penetration (open-globe) is suspected, either by a foreign body or laceration, the eye should be protected by a rigid eye shield and no further manipulation attempted. Subtle clues to penetration with no foreign body visible include a peaked, or teardrop-shaped pupil, bloody fluid causing swelling (bloody chemosis) beneath the conjunctival membrane that covers the white part (sclera) of the eye, and pigmented uveal tissue (including the iris – an 'iris prolapse') or jelly-like vitreous pooled on the surface of the eye.

  Visual examination will reveal if the pupils are round, if there is a fluid layer of blood (hyphema) in the chamber in front of the lens and iris (anterior chamber), and if there are any lacerations of, or hemorrhages beneath, the conjunctival membrane. An inflamed or infected eye will appear red, with conjunctival injection (engorged blood vessels) and either watery discharge or frank pus in the eye.

  Examination of the eyelids will reveal if a lid is drooping (ptosis), moving properly, swollen, bruised, or lacerated. If no globe penetrating injury is suspected, a cotton swab can be placed over the eyelid, and, with the eye lashes firmly grasped, the lid can be everted over the cotton tip to look for foreign bodies and examine the conjunctiva beneath the lid.

  The bony orbit should be examined both visually and with direct palpation. An area of point-tenderness, sometimes associated with a deformity or palpated 'step-off,' may indicate a fracture. Air palpated beneath the skin (subcutaneous emphysema), sometimes associated with a 'crackling' sensation (crepitus), should suggest an orbital or other facial fracture.

- The pupil exam requires little more than a pen-light and slightly darkened environment. The frequently documented 'PERRL' indicates that the pupils are equal, round and reactive to light. In the trauma patient, pupils may not be equal. Blunt trauma can cause either a small pupil (traumatic miosis) or an enlarged pupil (traumatic mydriasis). Chemical exposures can also cause these findings, with nerve

agent causing miosis, and atropine-related incapacitating agents (or atropine self-injectors used during a nerve agent threat) causing mydriasis.

APD stands for 'afferent pupillary defect,' the afferent referring to that which goes into and toward the eye and visual brain (light and nerve impulses). The normal eye will experience a reflexive pupil constriction when light is shone into the eye on the opposite side (consensual response). If the pupil constricts only in response to light in the opposite eye, and there is no response to direct light, and APD exists. This indicates that the light is being blocked between and lens and retina (a very dense vitreous hemorrhage) or that there is an injury to the retina or optic nerve.

- Extraocular muscles are tested by watching as the victim follows the examiner's finger through the 6 positions of gaze (left and right, left up and left down, right up and right down). Victims with deficits will sometimes complain of double vision with changes in gaze. These may represent direct muscle injury, fractures of the bony orbit with muscle 'entrapment,' swelling within the orbit, or non-traumatic nerve dysfunction ('palsies') including some exposures (eg, botulinum toxin).

- Begin the cornea exam with direct observation. The normal cornea is clear and, unless the anterior chamber is filled with blood or pus, the iris should be visualized without distortion. Foreign bodies seen on the cornea should be noted. A small, hazy white patch (infiltrate) indicates a corneal ulcer, a serious corneal infection. Conjunctival injection and pus in the anterior chamber (hypopyon) may also be present. A hazy, edematous cornea may indicate direct injury (burn) or underlying illness (acute glaucoma).

  Next, perform a fluorescein staining test. After a few drops of topical anesthetic are placed in the eye, a paper fluorescein strip is lightly swabbed along the sac formed by the bottom of the orbit and the lowest part of the visible conjunctiva. Blinking washes a thin film of fluorescein over the cornea. The eye is then examined with a wood's lamp or cobalt-blue filter (which may be attached to a pen-light) for staining defects, including abrasions and ulcerations. If penetration of the cornea is suspected, a moistened fluorescein strip can be used to 'paint' the suspicious site. If a perforation is present, a small stream of stained aqueous (lime-green under cobalt-blue light) will ooze from the perforation onto the outside of the cornea (positive Seidel test) (5).

- The retina can be examined using a hand-held direct ophthalmoscope, a procedure called ophthalmoscopy or funduscopy. Once the pupil exam has been documented, pupils may be dilated to facilitate the exam (1% tropicamide or 1% cyclopentolate drops). Certain patients, such as those with globe rupture, hyphema, or serious head injuries, should not be dilated. There is also a concern that pupil dilation may precipitate an acute glaucoma reaction in patients with a narrow anterior chamber. This condition is rare, generally bilateral, and mainly a concern for those over the age of 50 (6). Chamber narrowing occurs when the iris bulges forward and is dome-shaped. The normal iris is flat. Anterior chamber narrowing can be screened for by holding a pen-light to illuminate the eye from the side. A flat iris should not cast a

shadow when illuminated from the side. A shadow falling on the side of the iris away from the light suggests that the chamber may be narrow.

A normal visual acuity and red reflex in the trauma patient makes a direct retinal exam less critical, as a vitreous hemorrhage or large retinal detachment is unlikely. Even with a normal direct exam, if retinal detachment is suspected, a complete 360-degree retinal exam (indirect ophthalmoscopy) performed by an ophthalmologist is needed. If available, a new hand-held direct instrument with a five-fold increased field of view (Welch Allyn Panoptic®) may improve detection of small detachments by those not trained in indirect ophthalmoscopy.

- Intraocular pressure (IOP) can be measured with small, handheld devices which are relatively easy to use in the field. These include to Schiotz tonometer and the more recently introduced Tonopen®. If the Schiotz tonometer is used, remember to clean the device after each use. The normal range for IOP is between 10 and 20 mmHg. Both devices require the use of a topical anesthetic prior to pressure measurement. Pressing on the eye to obtain such a measurement is contraindicated in any victim with a suspected foreign body or laceration penetrating the globe. Injuries that can cause pressure elevation include hyphema and primarily increased intraorbital pressure, most commonly caused by hemorrhage behind the globe. A low IOP (less than 5 mmHg) should raise the suspicion of possible open-globe injury (7).

## Disaster Types

The following five sections will briefly describe disaster types to which a provider may be called to respond, and will address specific injury categories related to these events.

### Volcanoes

Erupting volcanoes affect the eyes mainly through the irritation and abrasions caused by ash-laden air. Close to the eruption, blocks and 'bombs' thrown from the volcano can crash through roofs and start house fires (8). After a 1968 eruption of the volcano Arenal in Costa Rica, houses were damaged by falling blocks up to 3 kilometers away. Later, heavy layers of ash, and especially heavy ash mixed with rainwater, caused the roofs of structures to collapse. Those exposed to thick, ash-laden air, including rescuers working outside, suffered irritation to both the eyes and the respiratory mucous membranes. Excessive inhalation of volcanic ash has resulted in death.

Following a volcanic eruption, residents and rescue workers exposed to ash fallout may present with eye complaints. Volcanic ash seems to possess ability to cause direct eye irritation. Following the eruption of Mount St. Helens in Washington State in 1980, emergency department visits increased for corneal abrasions, eye foreign bodies and conjunctival inflammation (conjunctivitis) (9). A group of ophthalmologists practicing within the area of ash fallout reported that about half their patients had

irritative conjunctivitis caused by ash, and fewer than 20% were found to have foreign bodies that required removal (10). Contact lens wearers were bothered more that non-lens wearers during ash exposure. The irritative conjunctivitis caused by ash presented as conjunctival swelling (chemosis) and engorged conjunctival blood vessels (hyperemia), and persisted for 24 to 48 hours after leaving the ash environment. Foreign bodies were divided nearly evenly between corneal and conjunctival.

No significant or lasting eye damage due to volcanic ash was reported following the eruption of Mount St. Helens. Similar studies in Japan have shown no major eye effects due to ash exposure, with most eye complaints resulting from simple mechanical irritation (11).

## Corneal Abrasion and Ocular Foreign Bodies

- **Corneal Abrasions**

Emergency departments treat more corneal abrasions than any other eye complaint (12). A corneal abrasion is simply an area of cornea where contact with an object has scraped off the outermost layer of cornea, the epithelium. If this area is in the center of the cornea (central corneal abrasion), visual acuity will be reduced. Victims present with a red, painful eye, tearing, and the sensation of a foreign body in the eye. The lid may be closed, and it may be difficult to coax the victim into opening the eye (blepharospasm) until topical anesthetic drops have been administered.

Any significant eye injury, including corneal abrasions, may result in traumatic iritis (iridocyclitis). This is an inflammatory response within the anterior chamber, and may develop over hours or even 1-2 days following injury (13). Ciliary muscle contraction (pupil constriction) becomes painful, and deep, aching eye pain (ciliary spasm) with photophobia is common. Light shone into the opposite eye will cause pain in the injured eye (consensual photophobia). Other findings include ciliary flush (swollen, injected blood vessels surrounding the limbus (corneal border), and, for those using a slit lamp, protein and inflammatory cells can be seen floating in the anterior chamber. Treatment for mild iritis includes pain medicine and sunglasses. More severe cases call for cycloplegic drops (cyclopentolate 1% every 8 hrs.) to reverse ciliary spasm.

The diagnosis of corneal abrasion is made after the fluorescein-stained corneal epithelial defect has been directly visualized under a cobalt-blue light. Be certain to exclude small foreign bodies that may remain beneath the lid or elsewhere in the eye. Treatment includes pain control and antibiotic drops to prevent bacterial superinfection. The choice of antibiotic is not critical, and this author prefers trimethoprim/polymyxin B (Polytrim) 1-2 drops 4 times per day. A special case would be contact lens wearers, who should be provided protection against *Pseudomonas* with either tobramycin (Tobrex) or ciprofloxacin (Ciloxan), also given 4 times per day. Contact lenses should not be placed

back into the affected eye until 2-3 days after healing. All victims diagnosed with abrasion should be rechecked in 24 hrs.

Other traumatic defects that stain with fluorescein include corneal lacerations and infected abrasions (corneal ulcerations). Corneal lacerations will be discussed more fully in the section on penetrating injuries. To reiterate, an abrasion is the removal of the thin, outermost layer of cornea, the epithelium. A laceration that cuts deeper into the underlying cornea (stroma) but does not penetrate it completely may be treated like an abrasion if not infected. The depth of an injury like this, however, is hard to judge without performing a slit-lamp exam. If the examiner cannot determine whether a laceration penetrates the cornea (open-globe injury), the eye should be treated as if it is perforated: rigid (Fox) eye shield, bed-rest (unless evacuation is necessary), broad-spectrum antibiotics (intravenous preferred, but an oral fluoroquinolone is a reasonable alternative), and emergent (as soon as possible) ophthalmologic referral.

If a careful examination reveals a small, hazy white patch (infiltrate) at the site of a fluorescein-staining epithelial defect, this indicates an infection (corneal ulceration), and this may spread quickly unless treated aggressively. Ulcerations are often associated with contact lens wear, but may begin as a simple abrasion in a non-contact lens wearer. One recommended treatment protocol is to use ciprofloxacin (Ciloxan) drops, 1 drop every 5 minutes for 3 doses, followed by 1 drop every 15 minutes for 6 hours and then 1 drop every 30 minutes around the clock (14). It is not unreasonable to admit these victims to a hospital if beds are available. Ulcerations are exceedingly painful, and cycloplegics and pain medication are required. Close ophthalmologic follow-up needs to be provided.

The finding of a corneal abrasion should prompt a search for a conjunctival foreign body. One particular pattern of linear abrasions over the superior cornea, called an 'ice-rink' sign, suggests a hidden foreign body which may be revealed with eversion of the upper lid.

- **Conjunctival Foreign Body**

A victim complaining of a red, irritated eye with a foreign body sensation warrants a thorough search, including eversion of the upper lid. If a foreign body is found, a cotton-tipped applicator moistened with a drop of topical anesthetic will often be all that is required to sweep the particle from the eye. In the absence of abrasion, relief will be immediate.

If no foreign body is found, and no corneal abrasion is found to explain the symptoms, it is reasonable to irrigate the eye thoroughly (at least 1 liter of fluid) to attempt to remove any small, poorly-visualized particles that may have been missed during the exam.

Note: the Morgan Lens® is a commercially available contact lens device through which the eye can be irrigated. It is designed for chemical eye exposures, and should not be used when the possibility of a foreign body exists.

- **Corneal Foreign Body**

Victims with a painful, red eye also may have a foreign body imbedded in the cornea. These are usually superficial, and often do not require magnification to see. Any attempt to remove a corneal foreign body requires adequate topical anesthesia.

If not using a slit-lamp, it is safer to attempt to remove the foreign body with irrigation or a moistened cotton-tipped applicator. These methods will often work for superficial foreign bodies that contacted the eye at low velocity.

More deeply penetrated foreign bodies need to be pulled from the cornea, a procedure that is much safer using the magnification of a slit lamp. The slit lamp also provides a rigid frame to keep the head from moving. The entire foreign object should be visualized before removal. Any foreign body penetrating the entire cornea constitutes an open globe injury, and should be left in place for the ophthalmologist to remove. Others may be removed mechanically at the time of exam, the easiest tool being a sterile 18-gauge hypodermic needle held on a 3 ml syringe. Obviously, this requires a cooperative patient. The needle tip is placed carefully beneath the object and lifted off of the cornea.

The removal of an iron-containing foreign body is not complete until the residual rust deposit ('rust-ring') is removed. The easiest method for those skilled in the technique is to grind out the rust-ring with a small, electric ophthalmic burr. The entire ring does not need to be removed at the time of foreign body removal. The resulting epithelial defect can be treated like an abrasion, and often after 24 hours the rust-ring will soften and elevate, and can be removed at that time like a small pellet.

## Earthquakes

Earthquakes injure people primarily through the collapse of structures. Structural collapse accounts for a full 90% of the deaths (8), the ratio of dead to injured being approximately 1 to 3 (2). Total numbers injured are a function of local construction methods, earthquake time of day and the population density in the affected area. Most injured survivors will not present with major trauma. After a 1968 earthquake in Iran, only 3.3% of those seeking emergency medical care required admission (368 of 11,254) (2). Injury mechanism is generally blunt trauma, and compressive eye injuries have also been reported in victims buried in debris, sometimes for extended periods (15). There will be a group of victims with multiple fractures and/or internal injuries requiring intensive treatment and an eye exam should not be overlooked in their initial evaluation.

**Blunt Eye Injuries**

- **Orbital Fracture**

The bones surrounding the eye may be fractured by a direct blow, or by force transmitted through the globe (eye-ball). When a blunt object strikes the globe and exceeds the globe's ability to absorb the force and 'bounce back,' the globe may rupture, resulting in an open-globe injury, or be pushed through the wall of the bony orbit.

The weakest surface of the bony orbit is the floor, with the medial wall being the next weakest (16). These 'blow-out' fractures occur typically in these two directions, usually through the orbit floor. The eye may be sunken (enophthalmos), and if the eye movement muscles (extraocular muscles) are 'entrapped' within the fractured bone, the victim may be unable to look up (paralysis of upward gaze) and complain of double vision. There may be decreased sensation (hypesthesia) below the traumatized eye, as a branch of the fifth cranial nerve passes through the bone in this area.

As long as the globe is not open and other injuries do not require in-hospital care, these victims may be observed with symptomatic treatment only for about 5 days (17), with delayed surgical repair if double vision or deformity does not resolve.

- **Subconjunctival Hemorrhage**

Hemorrhage beneath the conjunctiva may occur in the absence of trauma, as when blood pressure increases during sudden coughing, sneezing or vomiting. An eye contusion can cause bleeding, and victims may become alarmed as a blood-red patch spreads across the normally white conjunctiva. The cornea and spaces within the globe should be unaffected, and a subconjunctival hemorrhage alone will not affect vision. It helps to reassure victims that this is only a bruise, and its alarming appearance is only because, unlike the skin, the conjunctiva is clear. Resolution usually takes 2-3 weeks and is facilitated by warm soaks.

As always, a thorough history-taking and exam is important. The finding of a subconjunctival hemorrhage, a benign condition, does not exclude another more serious injury such as penetration of a high velocity foreign body.

- **Hyphema**

A blow to the eye may result in bleeding into the anterior chamber. If the chamber is clouded or blood-filled, the victim will complain of decreased vision. Smaller hyphemas are easily missed without a careful pen-light or slit-lamp exam. With the patient upright, a hyphema will be seen as a flat layering-out of blood at the bottom of the anterior chamber, between the iris and the cornea.

Hyphemas may resolve spontaneously. Conservative treatment has included hospitalization and emergent ophthalmology consultation. Some have suggested that bleeds that fill less than half of the anterior chamber and with normal intraocular pressure can be managed at home, but care must be taken in the disaster setting to assess whether a 'home' exists that can provide this care. Treatment includes bed rest, head elevation of 30-45 degrees, avoidance of aspirin and ibuprofen-like products, and eye rest (bilateral eye patches or atropine drops 1% 3 times per day to dilate the affected eye). The victim must be followed closely, as a delayed hemorrhage may occur after 2-5 days. This delayed hemorrhage can be much worse than the initial bleed.

- **Traumatic Iritis**

The discussion of traumatic iritis found in the section describing corneal abrasion will be repeated here. A corneal abrasion or other blunt eye injury may result in traumatic iritis (iridocyclitis). This is an inflammatory response within the anterior chamber, and may develop over hours or even 1-2 days following injury (13). Ciliary muscle contraction (pupil constriction) becomes painful, and deep, aching eye pain (ciliary spasm) with photophobia (pain with light exposure) is common. Light shone into the opposite eye will cause pain in the injured eye (consensual photophobia). Other findings include ciliary flush (swollen, injected blood vessels surrounding the limbus (corneal border), and, for those using a slit lamp, protein and inflammatory cells can be seen floating in the anterior chamber. Treatment for mild iritis includes pain medicine and sunglasses. More severe cases call for cycloplegic drops (cyclopentolate 1% every 8 hrs.) to reverse ciliary spasm.

- **Blunt Iris Injury**

In addition to traumatic iritis, the force of a blunt object may directly injure the iris. Blunt trauma may cause the pupil to be small (traumatic miosis) or enlarged (traumatic mydriasis). In traumatic mydriasis there will be no consensual response (light shone into the opposite eye will not result in pupil constriction) and no afferent pupillary defect. If the iris is torn away at the root, a clear space may be seen beneath the outer edge of the cornea. The victim may complain of double vision even with the opposite eye covered, as though seeing out of two pupils. No specific emergency treatment is required for iris injuries, although a tear at the root may result in a hyphema requiring care.

- **Blunt Lens Injury**

A blunt object striking the eye with enough force to push the pupillary ring of the iris onto the lens may leave a pigmented ring (Vossius' ring) on the Lens. No treatment is needed. A force strong enough to rupture the capsule covering the lens will result in lens clouding (traumatic cataract). These victims need rapid referral to an ophthalmologist as the injury can result in increased intraocular pressure (acute glaucoma) if the lens is not

removed (16). Conditions like traumatic cataract and total hyphema make it impossible to view the retina using ophthalmoscopy. The ability to refer these victims for ophthalmic ultrasound scanning is required to be able to evaluate for potential problems hidden behind the lens, including vitreous hemorrhage and retinal detachment.

Blunt trauma to the lens can also partially or completely tear the lens from the fibers holding it in place. If the lens is partially torn away (subluxed), the victim may complain of double vision even with the unaffected eye covered. The edge of the lens may be visualized with a crescent-shaped defect along the edge of a dilated pupil. The jelly-like vitreous may be bulging through the defect.

A lens completely torn away (dislocated) may move either forward or back. Without the support of the lens, the carefully observed iris may be seen to tremble following eye movements. If the lens moves forward, it may get stuck in the ring of the pupil and block the flow of fluid into the anterior chamber. This can cause an increase in intraocular pressure (glaucoma) that may not be immediately detected by a tonometer reading from the cornea. Subluxed and forward dislocated lens injuries require emergency ophthalmologic referral. Posterior dislocations do not require emergency surgery (16).

- **Vitreous Hemorrhage**

The spherical vitreous cavity is bordered by the lens in front and the retina in back. Bleeding into the cavity may be slight, causing the perception of 'floaters,' or severe, causing profound visual loss. The retina may not be able to be visualized with an ophthalmoscope and the red reflex may be absent when light is shone into the eye. The ability to see light is usually preserved. There is usually no afferent pupillary defect (AFD), although the response may be more brisk with light shone into the unaffected eye. An AFD suggests a hidden retinal detachment or optic nerve injury (16).

Trauma victims with vitreous hemorrhage are assumed to have a retinal injury until proven otherwise (13). This usually requires a dilated exam by an ophthalmologist or an ophthalmic ultrasound scan. Like hyphema, these hemorrhages are treated with bed rest, head elevation, monitoring of intraocular pressure, eye protection (eye shield), avoidance of aspirin and ibuprofen-like products, and ophthalmologic referral. Victims should be warned against heavy lifting and straining. It must be kept in mind that a retinal detachment may require surgery within 24 hours.

- **Globe Rupture**

A blunt object that is small enough to fit inside the orbit can strike the globe and cause a sudden increase in intraocular pressure. Sufficient pressure can rupture the globe. A globe rupture is usually a scleral rupture, and does not extend through the conjunctiva surrounding the sclera (16). This makes a globe rupture harder to find. Bloody chemosis (bloody swelling) of the conjunctiva is the most common finding (18), as bleeding from

beneath the sclera fills the conjunctival space. The eye may feel soft and 'squishy,' with an intraocular pressure of less than 5 mmHg, although if rupture is already suspected the eye should not be squeezed or pushed on with a tonometer. If the anterior chamber in the affected eye is flat, the globe is ruptured (5).

A globe rupture is treated the same as a perforation. The eye is protected with a rigid metal (Fox) eye shield or the equivalent, and the victim is referred to ophthalmology as quickly as possible. Antibiotics can be given to help protect against eye infection (endophthalmitis). If intravenous antibiotics are not available, ciprofloxacin (750 mg twice each day) is a reasonable temporary alternative. Tetanus immunization status should be checked and updated if needed. Concerns over barotrauma caused by air bubbles entering a ruptured globe should not delay air evacuation from a disaster site if that is the only route to the required level of care (19).

- **Retinal Detachment**

As the force of a blunt impact distorts the shape of the globe, the retina may become torn or detached and displaced. A small tear may also lead to a delayed detachment, sometimes even years later (16). Victims with a retinal tear will describe flashes of light, and may describe floaters if a small vitreous hemorrhage results. If detachment occurs, they may perceive a dark curtain being drawn across their visual field.

Large or central retinal tears and detachments may be seen using the hand-held direct ophthalmoscope. Others may require the ophthalmologist's examination. A retina obscured by hemorrhage or a lens cataract may be evaluated for detachment with an ophthalmic ultrasound scan. Depending on the injury, surgery within 24 hours may be indicated. Victims with suspected retinal tears or detachment awaiting the ophthalmologist should be kept on bed rest with both eyes patched.

- **Blunt Retinal Injury**

In addition to retinal tears and detachment, other injuries can result from blunt trauma. Hemorrhage can occur both within and behind the retina. These usually resolve over time without treatment and do not affect acuity unless they occur in the retina responsible for central vision (the macula). A blow to the front of the eye can cause a patchy whitening of the retina opposite the impact (Berlin's edema). In many cases the retinal exam and vision will return to normal over a week or two, but in severe cases or with macula involvement, a permanent vision loss may occur (13,16).

Another kind of blunt trauma occurs when compressive forces are applied to the eye for long periods of time. This may occur in disaster victims buried in rubble while search and rescue attempts are ongoing. They are at risk for all of the discussed retinal injuries, but may also suffer extreme eye compression with decrease retinal blood flow

and eventual thrombosis and occlusion of the retinal arteries (15). With complete occlusion the retina is pale and swollen, vessels are thread-like and the optic disc is milky white. Since blindness will occur quickly in the of absence retinal blood flow, prognosis is poor. Victims with some preserved flow (disc pale but not milky, etc.) have a better chance of sight recovery.

- **Optic Nerve Injury**

The optic nerve travels from its origin at the optic disc, visualized on retinal exam, through the orbit and through the bony optic canal on its way to the brain. Any sudden and transient force compressing the globe will also result in its 'bouncing back,' pulling on the nerve and stretching its support structures. If the blood vessels are torn, the nerve may loose its blood supply. Pulling on the nerve may also pull it away from the retina, an optic nerve avulsion. A fracture of the bone surrounding the optic canal may either swell and compress the nerve, or result in a sharp bone fragment cutting (transecting) the nerve.

An injury to the optic nerve as it travels through the bony canal may not be apparent in direct examination of the retina for several days. The victim will have severe vision loss in the affected eye with an afferent pupillary defect. Nerve compression due to a fracture is a surgical emergency. An avulsed nerve will appear as a hole where the optic disc should be on the retina exam. There is no treatment for this injury.

## Tropical Cyclones

Tropical cyclones are large, rotating circular storms with wind speeds greater than 120 km/hr (75 miles per hour). Wind velocity can reach 221 km/hr (136 miles per hour) or more in extreme cases. In the Atlantic and Caribbean these storms are called hurricanes. More than half of all deaths associated with these storms are actually due to flooding (1), as storm surges built up by the advancing storm are combined with heavy rainfall. When flooding is not a factor, destructive winds cause relatively few deaths (2), and the loss of life has continued to be limited where improved forecasting and evacuation has been utilized. Structures in high winds do not get 'blown over,' but rather 'explode,' as buildings are pulled apart by the low pressure created by moving air encircling a structure (1). Not surprisingly, injuries resemble those following explosions. Reports have been made of metal roofing material, nails and broken window glass flying at speeds of 100 miles per hour down streets and across fields. Penetrating injuries, including eye injuries, should be anticipated.

The discussion of penetrating eye injuries will be presented following the next section on blast injuries. It must be kept in mind that windstorm victims presenting with seemingly simple corneal abrasions and imbedded foreign bodies – conditions that are treated daily in most civilian emergency departments – may have been exposed to high projectile velocities. It is possible for small glass and metal splinters to enter the eye painlessly (18). Such exams should always be approached with the thought that a

penetrating eye injury (open globe) exists. At the time of examination, you may not have the equipment or special skills to perform a slit lamp exam and radiographic imaging, but you can still progress cautiously with an awareness of the signs and symptoms that may indicate that a foreign object has entered, or passed through, the globe.

## Explosions

Recent experiences, including the World Trade Center bombing in New York City in 1993, the Alfred P. Murrah Federal Building bombing in Oklahoma City in 1995, and the bomb attack at a U.S. military base in Dhahran, Saudi Arabia in 1996, emphasize the importance of understanding and being prepared to treat blast injuries.

Of the above three examples, the World Trade Center bombing was unique in that the explosion occurred underground. This somewhat contained the explosive force of the blast, and the majority of injuries following the attack were due to smoke inhalation (20). As an interesting aside, the explosive used in the attack was deliberately chosen because it contained sufficient cyanide to contaminate the inside of the Trade Center towers. The cyanide component was destroyed by the blast (21,22).

Explosions, and particularly above ground explosions, injure through three principal mechanisms (23). Primary blast injury results from the pressure wave directly impacting the body. Secondary blast injury results from objects being thrown against, into or through the body by the blast wave. Finally, tertiary blast injury occurs when the body itself is picked up and thrown against another object. Other injuries can result from thermal burns and smoke inhalation.

Primary blast injury has traditionally been described as affecting air-filled organs, most commonly the ear and the lung (24). It has also been shown that primary blast overpressure can cause both direct and indirect eye injury. Direct injury includes loss of eye movements, loss of pupillary reflexes, retinal hemorrhages, scotomas (blind spots) and blindness (25). These injuries were associated with degeneration of the optic nerves and other visual pathways in the brain. Retinal findings following primary blast exposure include hemorrhages, ischemia (areas of retinal blanching due to loss of blood flow), scotomas and retinal detachment. Primary injury can also result in conjunctival hemorrhage. Indirect eye injury includes air emboli (literally, bubbles) from damaged lungs, which can be seen ophthalmoscopically in retinal arterioles (24). These emboli can result in retinal ischemia and cell death.

Most of the described eye injuries following explosions are secondary injuries – fragments, particularly window glass, being propelled by the force of the explosion into the eye. It has been noted that whereas the surface of the eyes make up only 0.01% of the body surface area, up to 10% of blast survivors suffer eye injuries (3).

Of the 684 survivors treated following the bombing of the Alfred P. Murrah Federal Building in Oklahoma City, 55 (8%) sustained a total of 115 eye injuries (3). A

total of 67% of these injuries were penetrating eye injuries caused by projected glass fragments. Most of this was window glass, although light fixtures and computer screens also contributed. Most eye injuries (71%) occurred within 300 feet of the point of detonation, and eye-injured victims had higher overall injury severity.

Among the 115 eye injuries, there were 25 (21%) corneal abrasions, 23 (20%) eyelid and eyebrow lacerations, 12 (10%) globe penetrating (open-globe) injuries, 6 (5%) cases of hyphema, 6 (5%) fractures of the bony orbit and 5 (4%) cases of retinal detachment. Only 4% of eye injuries were caused by tertiary blast effect – being thrown by the explosion.

Approximately 20% of the injuries reported among rescue workers following the Oklahoma City explosion were eye injuries – 14.5% were ocular foreign bodies and 4.9% were corneal abrasions (26). These injuries were reported by workers with protective eyewear as well as those without.

Of the 26 victims requiring evacuation following the bombing of the U.S. compound in Dhahran, Saudi Arabia in 1996, 3 (11%) were evacuated for eye injuries (27). Glass fragments caused by the blast resulted in all of these injuries.

## Penetrating Eye Injuries

- **Eyelid Lacerations**

Small lacerations of the skin overlying the eyelid but not going through the entire lid, and not involving the edge of the eyelid (lid margin) can be repaired like any other facial laceration. If the proper equipment or expertise is not available to perform a suture repair, the laceration should be irrigated with the cleanest water available and treated with topical antibiotic ointment (19). Loose approximation with adhesive closures strips may be attempted. The status of tetanus immunization should be checked and updated if required.

The examiner must be very careful to perform lid eversion and look for any sign that the laceration penetrated all the layers of the lid. Any evidence of a ‘full thickness’ eyelid laceration must begin an examination to confirm or exclude a penetrating injury to the globe (see below). Any full thickness laceration (including those involving the lid margin) and any upper eyelid laceration resulting in a drooping lid (ptosis) should be referred to an ophthalmologist for repair.

- **Conjunctival Lacerations**

Lacerations of the conjunctiva are caused by the same type of injuries that result in corneal abrasions, and like corneal abrasions, they can usually be treated with topical

antibiotics and time. Lacerations longer than 1 cm should be referred to an ophthalmologist for repair (13). The most important thing is to be sure that the laceration stops at the conjunctive, and does not continue through the white sclera beneath. The thin, film-like conjunctiva wrinkles and folds when cut, and this often exposes the sclera for examination. When available, the slit lamp is the best instrument to use to perform this examination. A cotton-tipped swab moistened with topical anesthetic can be used to probe the sclera looking for an open area.

Scleral lacerations, once detected, should prompt placement of a rigid Fox-type eye shield and referral to an ophthalmologist. If a subconjunctival hemorrhage makes the scleral exam impossible, treat and refer as though a laceration exists until it can be proven otherwise.

- **Corneoscleral Lacerations**

All lacerations that penetrate the cornea or sclera are open globe injuries, and are treated with eye protection (a rigid metal Fox-type eye shield), antibiotics to prevent endophthalmitis, an update of tetanus immunization if required, and emergency evacuation and/or referral to an ophthalmologist. Intravenous antibiotic regimens include combination ceftazidime and vancomycin or ciprofloxacin and vancomycin (13), but if this level of care is not available, it is prudent to begin an alternative such as oral ciprofloxacin 750 mg in the field. Patients are kept at bed rest as much as possible. If the laceration is in the cornea, cycloplegic drops (1% cyclopentolate or 1% atropine every 8 hours) may be used. Care should be taken to apply no external force to the globe, including the force of tonometer readings, as this could result in the pushing-out (prolapse) of orbit contents.

It is sometimes difficult to tell the difference between a superficial corneal laceration and one that penetrates the cornea. Other times it will be obvious. If the anterior chamber is flat and drained of fluid, the globe has been opened. Any injury inside of the eye (an iris or lens injury, a peaked and teardrop shaped pupil, or hyphema) or orbital contents found outside the eye (jelly-like vitreous or the iris, or other dark, pigmented uveal tissue) suggests an opening. If these obvious signs are not found, a slit lamp can be used to follow the injury tract through the corneal stroma to see if penetration is complete. A superficial laceration that does not pass through the stroma can be treated as an abrasion. A Seidel test can be used to see if fluid (aqueous humor) is leaking from the tract. A positive Seidel test means that the globe is open. It should be kept in mind that small lacerations may seal themselves, and leakage of fluid through the cornea will not always be found on the examination of a deep corneal laceration.

As with lacerations of the cornea, scleral lacerations can be difficult to detect. Bloody swelling of the conjunctiva (bloody chemosis) may be diffuse or found just surrounding the site of a laceration. Decreased intraocular pressure (IOP less than 5 mmHg, a flat anterior chamber or a soft, ‘squishy’ globe) indicates that penetration has occurred. Again, if penetration is suspected, don’t push to see if the eye is ‘squishy’ or

not. A teardrop shaped pupil suggests penetrating laceration, with the peak of the 'drop' pointing toward the injury. Any pigmented uveal tissue (including iris) or jelly-like vitreous sitting on the surface of the globe indicates penetration. A foreign body in the eye indicates penetration. In the absence of other injuries, a vitreous hemorrhage, traumatic cataract (lens capsule injury) or retinal detachment suggest that the globe has been opened. An unexplained decrease in visual acuity also suggests penetration.

- **Orbital Lacerations**

Penetrating objects may miss the globe altogether, lacerating structures within the cup of the orbit, or may injure the orbit after passing through the globe. If the victim complains of double vision and cannot move the eye normally in each direction, one or more extraocular (eye movement) muscles may have been cut. If the victim is suddenly blind in the affected eye with an afferent pupillary defect, the optic nerve may have been cut.

Another injury that causes decreased vision, afferent pupillary defect and limited eye movement is a laceration or fracture with an enlarging collection of blood behind the eye (retrobulbar hemorrhage) with a dangerous increase in orbital pressure (orbital compartment syndrome). This condition will both directly transmit increased pressure to the intraocular space (glaucoma) and, through swelling, cause vascular congestion and obstruct venous return blood flow from the eye and, most importantly, the retina.

Orbital compartment syndrome is suggested by forward bulging of the eye (proptosis) following penetrating trauma. Visual acuity will be decreased with an afferent pupillary defect. The pupil may be dilated (mydriasis) and poorly reactive. Signs of congestion may be found on the conjunctiva (chemosis). This is similar to the prolonged eye compression seen in victims buried beneath fallen buildings after earthquakes. As with external compression, prolonged increases in intraorbital and intraocular pressure can result in a cut-off of the retina's blood supply (ischemia), and cause blindness if allowed to continue. If the measured intraocular pressure in a victim with suspected retrobulbar hemorrhage is greater that 50 mm Hg (measured with tonometry) (18), the space behind the eye should be surgically decompressed, if possible, within an hour of the injury. This requires an operator familiar with the minor surgical technique called lateral canthotomy.

- **Foreign Bodies inside the Eye**

A foreign body inside the globe is absolute evidence that penetration has occurred and treatment should proceed as with other open globe injuries (see corneoscleral lacerations, above) with prompt ophthalmologic referral. As with lacerations, some foreign bodies – especially glass shards, nails and other objects found protruding from the eye – will be quite obvious. These objects should be removed in the operating room, and some thought may need to be put into your method of shielding the eye while not moving

the foreign object with respect to the globe. In locations where x-ray capability exists, small metallic foreign bodies may be found using plain radiographs of the orbit.

Other foreign bodies in the orbit may be quite difficult to locate. One victim injured during the terrorist bombing of the U.S. barracks in Dhahran, Saudi Arabia, had 6 intraorbital glass foreign bodies found at the time of her initial CT scan and surgery. A seventh retained foreign body was found 6 months later when it was extruded from the upper lid (27). At the time of initial treatment and triage, however, that exact nature and position of a foreign body is an academic question. If an object is inside of the globe, a laceration exists. All of the signs and symptoms used to detect a corneoscleral laceration will also apply here.

## Toxic Exposures

The eye has long been a 'window' peered into by clinicians looking for signs of systemic illness. The combination of exposed mucous membrane, well-described pathways of nerve control, and the ability to directly visualize both the nervous system (optic disc) and the vascular system (retinal veins and arterioles) make the eye a unique diagnostic indicator. One quick look at a fixed and dilated pupil following head injury suggests a patient that will die quickly without rapid surgery to evacuate a cerebral hematoma – not an eye problem at all. The sequelae of systemic diseases such as hypertension and diabetes, as well as many systemic intoxications, can be directly assessed through examination of the eye.

In the immediate aftermath of a mass exposure to an injurious chemical or biological agent, there may be precious few indicators available to aid us in understanding what processes are attacking the victims' bodies, and in directing what therapeutic measures we should or should not deploy. The early indicators will be people, initial victims who may give their lives to provide clinical clues, which we must use to deduce the nature of the agent. The eye exam is a critical piece of the puzzle when trying to describe the effects of an agent or class of agents. This description of characteristic symptoms and signs associated with a particular agent or class of agents is sometimes called a 'toxidrome.'

The exposure may be due to an act of chemical or biological terrorism, as in the 1995 release of Sarin nerve agent in stations along the Tokyo subway by the Aum Shinrikyo sect. The attack left 11 dead and resulted in 5,500 casualties (28). The same sect had stockpiled a biological agent, botulinum toxin, throughout Tokyo. Botulinum toxin is 100,000 times more potent than Sarin. Anyone who lived through the World Trade Center bombing in 1993 and the Alfred P. Murrah Federal Building bombing in Oklahoma City in 1995 knows enough to never consider U.S. soil immune to terrorism. A toxic exposure may also result from an industrial or transportation error. Such was the case after the 1984 release of 40 metric tons of methyl isocyanate from a Union Carbide pesticide plant in Bhopal, India. Over 3000 persons near the plant died in the first 3 days following the release (29).

The discussions that follow will discuss the ophthalmologic symptoms of methyl isocyanate exposure, called 'Bhopal Eye' in the literature (30), as well as eye findings following nerve agent and botulinum toxin exposure. Findings after exposure to the important class of anticholinergic agents, which includes the nerve agent antidote atropine and weaponized incapacitating agents, will also be discussed.

## Bhopal Eye

Isocyanates are a large class of industrial chemicals, widely used in the manufacture of paints, plastics, pesticides, adhesives and polyurethane foam (29). They all react aggressively with water, increasing their industrial usefulness. This reactivity is the source of their toxicity. They react with the moist airways, and are the most common cause of occupational asthma (31). They also react with the moist eyes. Methyl isocyanate, the compound released in Bhopal, India, is one of the most reactive of all isocyanates. Deaths in Bhopal were due to pulmonary edema, but the first indication of the spill was eye irritation among those living next to the plant. This immediate reaction included persistent watering (described as 'streaming eyes' (30)), photophobia, profuse lid swelling and ulcerations of the cornea (30, 32).

All of the eye effects were reversible and no case of blindness was documented among the survivors (30). Animal studies have shown that the most severe eye effects were caused at intermediate levels of exposure, indicating that at high doses there was a protective response (33). This agrees with the findings in Bhopal, that high concentrations leading to death were associated with modest eye effects. This was thought to be due to the copious tearing and tight lid closure triggered at high concentrations (29).

Isocyanates should not be confused with cyanide, a substance that can be absorbed through the eyes, but has no direct eye effects. Cyanide prevents oxygen utilization in the cells, producing respiratory distress without cyanosis, seizures and, without treatment, rapid death.

## Nerve Agents

Nerve agents are potent weapons derived from the less-potent organophosphate insecticides. All of these compounds inhibit cholinesterase, the enzyme that degrades the neurotransmitter acetylcholine at cholinergic receptor sites. The effects of nerve agents, therefore, are due to excess acetylcholine accumulated at the receptor. A lethal dose can cause seizures followed by muscle flaccidity and respiratory arrest within minutes.

Eye findings are characteristic of nerve agent exposure. Contact with nerve agent vapor will cause miosis within seconds or minutes (34). Nerve agent induced miosis is accompanied by eye pain, blurred or dim vision, conjunctival injection, and occasionally vomiting. A dull frontal headache is common (34). Following Sarin exposure during the

Tokyo subway attack, miosis and miosis-related visual darkness was the most common sign and symptom, present in 105 of 106 hospitalized patients at one facility (28). Most victims had pupils of less than 1 mm in diameter. Eye pain, blurred vision, headache, nausea and vomiting were also experienced (28).

Treatment for nerve agent exposure is covered elsewhere. However, it is worth noting that the initial treatment agent atropine, an anticholinergic (cholinergic blocking) compound, will result in mydriasis. During nerve agent threats, U.S. soldiers are deployed with atropine self-injectors, and may receive atropine without nerve agent exposure. These people may be seen at health facilities for heat illness, as atropine will also inhibit sweating. Many drugs have anticholinergic properties. Weaponized incapacitating agents (for example, NATO code 'BZ') are atropine-like. A person prescribed skeletal muscle relaxants due to a disaster-related injury may overuse the medication, and present with anticholinergic toxicity. Certain drugs of abuse, for example, jimsonweed, will also result in anticholinergic toxicity. This anticholinergic 'toxidrome' can be remembered as:

- Blind as a bat (due to mydriasis)
- Dry as a bone
- Red as a beet
- Mad as a hatter (slightly higher doses than those causing the above effects will result in delirium)

**Botulinum Toxin**

Botulinum toxin is classified as a biological weapon because it is of natural origin (produced by a bacteria). The toxin itself is not a living organism, but rather a protein that binds irreversibly to a nerve terminal preventing the release of acetylcholine. As a neurotoxin, symptoms are the same following inhalation, as during an aerosolized attack, and botulism poisoning due to eating improperly canned foods.

Following inhalation, symptoms may begin within 24 hours but may also develop after several days (35). Bulbar palsies are among the earliest symptoms, including eye symptoms. These include double vision (diplopia), mydriasis, photophobia and drooping eye lids (ptosis). Other bulbar palsies will include difficulty swallowing and producing speech. It is important to make the diagnosis early if possible. Bulbar palsies are followed by a spreading (descending) paralysis with eventual respiratory failure. An antitoxin is available that can arrest the progression of paralysis, but as the toxin binds irreversibly, the antitoxin cannot restore strength already lost.

The only treatment after respiratory failure is endotracheal intubation and mechanical ventilation. As long as blood oxygen and CO2 are kept controlled by ventilation, the victim will remain awake and alert though totally paralyzed. If they do not succumb to pneumonia, infected bedsores and sepsis, the victims will be able to be removed from the ventilator after a recovery period of weeks or even months.

## Post-emergency Phase Eye Care

In the aftermath of a disaster, victims and rescue workers alike may present to a health post seeking treatment for eye problems. Presenting complaints may be as mundane as contact lens overwear (due to disruption of sleep cycles or loss of wetting solution) or as dramatic as an untreated injury that has progressed into a devastating globe infection. Loss of sanitation and clean water, as may occur with flooding, can predispose to certain infectious eye diseases in both individuals and populations. This unit will discuss some of these problems, which may present in the late emergency phase or the rehabilitation phase of disaster response.

### Anoxic Contact Lens Overwear Syndrome

In the aftermath of a disaster, daily routines and normal sleep-wake cycles may be disrupted for some time. Both victims and relief workers will be affected. Contact lens wearers may also have lost their spare eyeglasses and their sterile rinsing and soaking solutions.

Contact lens overwear syndrome is a consequence of a contact lens depriving the cornea of adequate oxygen. It can occur with both rigid and soft/extended wear lenses. Once cell damage has occurred, eye pain can begin even hours after the lens has been removed. Sufferers present with a red, painful eye and photophobia. Initial cornea swelling (edema) progresses to corneal trauma, inflammation (keratitis), and traumatic iritis. Physical exam will reveal conjunctival injection with ciliary flush, fluorescein-staining central corneal abrasion, and neovascularization, an encroachment of new blood vessels trying to grow over the cornea (a reaction to the low-oxygen environment).

Overwear syndrome is treated the same as corneal abrasion, with topical antibiotics, cycloplegics (for iritis) and adequate pain medication. The contact lens should not be put back in until 2-3 days after complete healing and resolution of pain.

### Untreated Open-Angle (chronic) Glaucoma

Open-angle glaucoma is a chronic illness normally affecting the elderly. Patients are generally not symptomatic, although they may experience mild headaches. Diagnosis is determined through routine screening. Management is predominantly medical, usually consisting of ophthalmic drops. Disaster victims may present having lost their regular home medications. The goal of treatment should be to re-establish regular medication regimens as soon as possible. Stopping medication should not precipitate an acute glaucomatous attack.

## Conjunctivitis

Either bacteria or viruses can cause infectious inflammation of the conjunctiva. These infections are extremely contagious, and may spread rapidly through populations living in close proximity and under conditions of poor hygiene. The distinction between bacterial and viral is largely academic, as many practitioners will treat both with topical antibiotics. It is difficult to exclude bacterial disease on the basis of physical exam alone, and the possibility of bacterial superinfection exists as patients rub their irritated eyes. Both forms of conjunctivitis produce red, irritated, itching and tearing eyes. The bacterial disease is more likely to produce actual pus, and the viral disease is more likely to affect both eyes – but these distinctions are not absolute. Neither conjunctivitis should affect vision unless a collection of mucous or pus is actually covering the cornea.

The actual topical antibiotic chosen to treat conjunctivitis is not critical, although it is recommended to treat the infection in contact lens wearers with a choice that would cover *Pseudomonas* – examples are tobramycin (Tobrex) and ciprofloxacin (Ciloxan), given 4 times per day.

## Other Infections

- **Periorbital (Preseptal) Cellulitis**

Previously traumatized eyes can develop infections in and around the orbits. Periorbital cellulitis is an infection of the soft tissue surrounding the eye. It is often secondary to a lid laceration or other trauma. The globe is not affected, and the visual acuity is normal. The victim will frequently have a fever in addition to swelling, redness, warmth and tenderness of the skin surrounding the eye. Recommended treatment is admission to hospital for intravenous antibiotics. Initial treatment with oral antibiotics (cephalexin 500 mg 4 times per day, levofloxacin 500 mg once each day or ciprofloxacin 750 mg twice each day) should begin if evacuation or other delay in hospital care is anticipated (19).

- **Endophthalmitis**

Endophthalmitis is an uncommon infection of the globe secondary to traumatic contamination of the intraocular space by bacteria. This is usually the result of an object like a wooden twig penetrating the eye. There is increasing pain and vision loss associated with swelling and injection of the conjunctiva and lid swelling. Sometimes pus is seen in the anterior chamber (hypopyon). Untreated, the infection can progress to blindness. Broad-spectrum antibiotics are used, and often need to be directly instilled into the globe by an ophthalmologist.

- **Acanthamoeba Keratitis**

Acanthamoeba keratitis is an extremely rare infection of the cornea caused by the free-living freshwater amebae, *Acanthamoeba*. It is discussed here because of the severity of the infections, and the association of the infection with eye trauma and water contaminated with sewage. It is generally an infection of contact lens wearers, as the lens itself provides both a mechanism of microtrauma and a vehicle for transferring non-sterile, contaminated water to the cornea (36). It has been reported in the non-contact lens wearer following eye trauma (37). It is suggested by pain that is disproportionately severe in the early stages compared to other corneal infections. Fluorescein-staining defects are found in 60% of cases, and in 29%, central ring-shaped infiltrates (ulcerations) are seen. Definitive diagnosis and treatment is performed by an ophthalmologist, and referral or evacuation should be prompt.

- **Leptospirosis**

Following the landing of Hurricane Hortense in Puerto Rico in 1966, a more than 4-fold increase in leptospirosis was found (38). Leptospirosis is a bacterial infection spread by animal 'renal carriers,' and contracted by humans in contact with infected animal urine. Flooding, as occurs following hurricanes, could facilitate *Leptospira* transmission by preventing urine drying and absorption. Sixteen of 17 cases in Puerto Rico presented with eye pain and watering. Other symptoms included fever, chills and muscle pain. Laboratory confirmation was needed to rule out the similarly presenting Dengue Fever, although eye pain was more typical in cases of Leptospirosis.

- **Trachoma**

Trachoma is a chronic conjunctivitis associated with conditions of crowding and poor hygiene. It may be found in refugee camps following long-term disasters such as drought and famine. It often begins in early childhood, and in the acute phase may be spread between family members, often by flies. If untreated, blindness follows after years of chronic infection, and trachoma remains the leading cause of preventable blindness in the world.

Acute infection has been described as leaving the conjunctival membrane beneath the everted eyelid 'red and velvety' (39), with numerous small blisters called follicles. This conjunctivitis heals with scarring, turning the edge of the upper lid inward (entropion), with the eyelashes then chronically traumatizing the cornea. Corneal ulceration leads to scar formation and blindness. A thick sheet of neovascularization (pannus) can also encroach upon the cornea from above.

The initial conjunctivitis is caused by the organism *Chlamydia trachomatis*, and treatment in the acute phase is effective (tetracycline ointment 3 times each day for 6 weeks). Victims may present with combined infections, and it may be easier to screen for

trachoma once a superimposed bacterial conjunctivitis has subsided. By the time scarring has affected lid position, surgery may be needed to prevent corneal trauma and blindness. Improving sanitation and hygiene will do much to dramatically reduce rates of transmission.

**Xerophthalmia**

Along with poor hygiene, poor nutrition also causes blindness in the refugee setting (40). Xerophthalmia is a slowly advancing eye disease caused by vitamin A deficiency. It is associated with childhood malnutrition in long-term disaster refugee settings. It may also result from chronic intestinal disease and poor nutrient absorption. If a population's diet is deficient in vitamin A, children will begin to manifest disease symptoms by their second or third year. The first symptom is night blindness, and anyone complaining of poor night vision in such a community should be promptly treated. Untreated, the disease results in eye drying that produces a dry, leather-like conjunctiva and a wrinkled, cloudy cornea. At this point, a rapidly progressing corneal ulcer can appear that spreads in hours. This end result has been called 'corneal melting' (39).

The recommended World Health Organization vitamin A dosing is 200,000 IU orally on days 1, 2 and 8 for all malnourished or symptomatic children (39). With malabsorption, the first dose can be changed to 100,000 IU given as an intramuscular injection. All children with measles should be treated. Doses are cut in half for infants (less than 1 year). Disease may be prevented in adults and older children with a dose of 200,000 IU every 4-6 months. All women should receive a 200,000 IU oral dose within 1 month of delivering a baby.

## References


1. Cuny FC. Aim and Scope of Disaster Management. Madison, WI. Disaster Management Center at the University of Wisconsin-Madison, 1986.

2. Emergency Health Management after Natural Disaster. Washington, D.C., Pan American Health Organization, 1981.

3. Mines M, Thach A, Mallonee S, et al. Ocular injuries sustained by survivors of the Oklahoma City bombing. Ophthalmology. 2000; 107(5): 837-43.

4. Mallek D. Care of eye injuries in disasters. Can J Ophthalmol Jul 1980; 15(3): 109-10.

5. Mitchell, JD. Ocular Emergencies. In : Tintinalli JE, Kelen GD, Stapczynski, JS (Eds). Emergency Medicine: A Comprehensive Study Guide. New York, McGraw-Hill, 2000: 1501-1518.

6. MacCumber MW. Management of ocular injuries and emergencies. Philadelphia, Lippincott-Raven, 1998.

7. Kylstra JA, Lamkin JC, Runyan DK. Clinical predictors of scleral rupture after blunt occular trauma. Am. J. Ophthalmol. 1993; 115: 530-535.

8. Perez E, Thompson, P. Natural Disasters: Causes and Effects. Madison, WI. Disaster Management Center at the University of Wisconson-Madison, 1986.

9. Mount St. Helens Volcano Health Report 18. Atlanta, GA. Centers for Disease Control. August 15, 1980.

10. Fraunfelder FT, Kalina RE, Buist AS, et al. Ocular effects following the volcanic eruptions of Mount St Helens. Arch Ophthalmol Mar 1983; 101(3): 376-8.

11. Yano E, Yokoyama Y, Higashi H, et al. Health effects of volcanic ash: a repeat study. Arch Environ Health. Nov-Dec 1990; 45(6): 367-73.

12. Edwards RS. Ophthalmic emergencies in a district general hospital casualty department. Br. J. Ophthalmol. 1987; 71: 938-942.

13. Mahadevan SV, Savitsky E. Emergency Management of Traumatic Eye Injuries. Trauma Reports. July/August 2001; 2(4): 1-15.

14. Rhee DJ, Pyfer MF, Rhee DM (Eds). The Wills Eye Manual: Office and Emergency Room Diagnosis and Treatment of Eye Disease. New York, Lippincott Williams & Wilkins, 1999.

15. Cai YS, Zhou GJ. Compressive eye injuries caused by earthquake. Chin Med J Oct 1983; 96(10): 731-6.

16. Joondeph BC. Blunt Ocular Trauma. In: Mathews J, Zun LS (Eds). Ophthalmologic Emergencies and Ocular Trauma. Emergency Medicine Clinics of North America. Feb 1988; 6(1): 147-167.

17. Clark RB. Common ophthalmologic problems. In: Rosen P, Baker FJ, Barkin RM et al. (Eds). Emergency Medicine: concepts and clinical practice. St. Louis, Mosby, 1988: 1033-1049.

18. Lubeck D. Penetrating Ocular Injuries. In: Mathews J, Zun LS (Eds). Ophthalmologic Emergencies and Ocular Trauma. Emergency Medicine Clinics of North America. Feb 1988; 6(1): 127-146.

19. Butler, FK. The Eye in the Wilderness. In: Auerbach PS (Ed).Wilderness Medicine. St. Louis, Mosby, 2001: 531-551.

20. Quenemoen LE, Davis YM, Malilay J, et al. The World Trade Center Bombing: injury prevention strategies for high-rise building fires. Disasters. 1996; 20: 125-132.

21. Jenkins BM. Understanding the link between motives and methods. In: Roberts B (Ed). Terrorism With Chemical and Biological Weapons: Calibrating Risks and Responses. Alexandria, VA, Chemical and Biological Arms Control Institute, 1997: 43-52.

22. Brennen RJ, Waeckerle, JF, Lillibridge, SR, et al. Chemical Warfare Agents: Emergency Medical and Emergency Public Health Issues. Ann Emerg Med. Aug 1999; 34(2): 191-204.

23. Wightman JM, Gladish SL. Explosions and Blast Injuries. Ann Emerg Med. June 2001; 37(6): 664-678.

24. Mayorga MA.The pathology of primary blast overpressure injury. Toxicology Jul 25, 1997; 121(1): 17-28.

25. Petras JM, Bauman RA, Elsayed NM. Visual system degeneration induced by blast overpressure. Toxicology. Jul 25, 1997; 121(1): 41-9.

26. Dellinger AM, Waxweiler RJ, Mallonee S. Injuries to rescue workers following the Oklahoma City bombing. Am J Ind Med Jun 1997; 31(6): 727-32.

27. Thach AB, Ward TP, Hollifield RD, et al.Eye injuries in a terrorist bombing: Dhahran, Saudi Arabia, June 25, 1996. Ophthalmology May 2000; 107(5): 844-7.

28. Ohbu S, Yamaguchi T, Murai T, et al. Sarin poisoning on Tokyo subway. South Med J Jun 1997; 90(6): 587-93.

29. Varma DR, Guest I.The Bhopal accident and methyl isocyanate toxicity. J Toxicol Environ Health Dec 1993; 40(4): 513-29.

30. Andersson N, Muir MK, Mehra V. Bhopal eye. Lancet Dec 1984; 2(8417-18): 1481.

31. Cartier A, Grammer L, Malo J, et al., Specific serum antibodies against isocyanates: Association with occupational asthma. J. Allergy Clin. Immunol. 1989; 84: 507-514.

32. Dwivedi PC, Raizada JK, Saini VK, et al. Ocular lesions following methyl isocyanate contamination: the Bhopal experience. Arch Ophthalmol Nov 1985; 103 (11): 1627.

33. Salmon AG, Kerr Muir M, Andersson N. Acute toxicity of methyl isocyanate: a preliminary study of the dose response for eye and other effects. Br J Ind Med Dec 1985; 42(12): 795-8.

34. Medical Management of Chemical Casualties Handbook. Aberdeen Proving Ground, MD. U.S. Army Medical Research Institute of Chemical Defense, 2000.

35. Medical Management of Biological Casualties. Fort Detrick, MD. U.S. Army Medical Research Institute of Infectious Diseases, 1998.

36. McCulley JP, Alizadeh H, Niederkorn JY. The diagnosis and management of Acanthamoeba keratitis. CLAO J (Contact Lens Assoc. of Ophthalmologists Journal) Jan 2000; 26(1): 47-51.

37. Rumelt S, Cohen I, Lefler E, et al. Corneal co-infection with Scedosporium apiospermum and Acanthamoeba after sewage-contaminated ocular injury. Cornea Jan 2001; 20(1): 112-6.

38. Sanders EJ, Rigau-Perez JG, Smits HL, et al. Increase in leptospirosis in dengue-negative patients after a hurricane in Puerto Rico in 1996. Am J Trop Med Hyg Nov 1999; 61(5): 861.

39. Ott E, Bock R, Hawks W. Ophthalmologic Diseases. In: Sandler RH, Jones TC, (Eds). Medical Care of Refugees. New York, NY, Oxford University Press, 1987: 191-202.

40. Heldt JP, Frye BA, Wessels IF. Refugee eye care: a frequently overlooked dimension of disaster assistance. Ophthalmic Surg Jun 1988; 19(6): 440-9.